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Attention-Deficit/Hyperactivity Disorder (AD/HD) has been viewed to be a potential precursor for further behavior problems, particularly Oppositional Defiant Disorder (ODD). Because not every child with AD/HD develops this condition, factors may be present that protect these individuals who are at risk. The role of protective factors in the AD/HD population has largely been overlooked by prior research. The current study explored risk and protective factors that influence an ODD outcome within an AD/HD population. An ethnically mixed clinical sample of 60 boys (mean age = 9.3 years) and their mothers served as participants. As expected, both risk and protective factors emerged as significant predictors of oppositional-defiant behavior. Consistent with prior research on parenting style, a higher level of corporal punishment was associated with higher levels oppositional-defiant behaviors, whereas higher levels of child adaptability, intrapersonal skills, and involvement in family/community were associated with low levels of these behaviors. Categorical analysis found that impulsive symptoms served as a risk factor and child adaptability and intrapersonal skills served as protective factors, predicting with 85% accuracy which children had a co-occurring diagnosis of ODD and which did not. This study provides evidence for the importance of examining both risk and protective factors in a clinical AD/HD population with respect to comorbid disruptive behavior disorders. Implications for the assessment and treatment of children with AD/HD were discussed.

THE LINK BETWEEN ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND
OPPOSITIONAL DEFIANT DISORDER: RISK AND PROTECTIVE FACTORS

by

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CHAPTER I

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (AD/HD; American Psychiatric Association, 1994) is a chronic, disruptive behavior disorder with origins in childhood stemming from predominantly neurobiological causes (Swanson, Castellanos, Murias, LaHoste, & Kennedy, 1998). It is characterized by inattentive, hyperactive and impulsive behavior including not listening, being easily distracted, being fidgety, and interrupting. AD/HD has been shown to be associated with an antisocial behavior trajectory that begins with oppositional defiant disorder (ODD; American Psychiatric Association, 1994) and is followed by conduct disorder (CD; American Psychiatric Association, 1994). AD/HD has been identified as one of the best predictors of the early onset of aggression and defiance and of persistent antisocial behavior (Loeber, Green, Lahey, Christ, & Frick, 1992, Moffitt, 1993; Patterson, Reid, & Dishion, 1992). Nearly half of childhood referrals to outpatient clinics are for antisocial behavior and adult individuals with persistent antisocial behavioral outcomes comprise the majority of criminal offenders (Kazdin, 1995). Although there are a number of factors and potential pathways to antisocial behavior, there is reason to have concern about the frequency with which individuals with AD/HD develop co-occurring antisocial conditions. However, current literature fails to fully account for the tendency of some children with AD/HD to continue along a trajectory toward antisocial behavior, though others do not.

In contrast to AD/HD, ODD has typically been considered a disorder that relies on environmental influences to occur. The antisocial literature links ODD to a multitude of factors, the bulk of which stem from the family environment, particularly negative parent-child interactions (Patterson, Reid, & Dishion, 1992). The family environment may exacerbate child temperamental factors (Moffitt & Caspi, 2001). Although the origins of AD/HD and ODD differ, their frequent co-occurrence (approximately 40%) suggests a common connection. Current research indicates that the direct effects of AD/HD symptomology and indirect effects (e.g. psychopathology, parenting style, and family stress), which often accompany children with AD/HD, all play a role in antisocial outcomes (Anderson, Hinshaw, & Simmel, 1994; Danforth, Barkley & Stokes, 1991; Frick et al, 1993; Moffitt, 1990). Finally, child temperament has been implicated in antisocial outcomes: aggression has been identified as a temperamental trait preceding the onset of antisocial behavior (Lahey & Loeber, 1997; Moffitt, 1993; Moffitt & Caspi, 2001). Also, impulsivity, a core feature of AD/HD, is predictive of ODD development (Burns & Walsh, 2002).

Because not every child with AD/HD follows a developmental course that includes antisocial behavior, factors may be present that protect these individuals who are at risk. Such factors may include child temperament (Rae-Grant et al., 1989) and positive, involved parenting (Masten et al., 1999). However, resilience and protective factors have been virtually overlooked related to children with AD/HD. Preliminary findings in a community sample indicated that child adaptability and interpersonal skills are related to an AD/HD only outcome (Rhoads, 2001). Because the presence of AD/HD has been shown to place a

child at significant risk for comorbid conditions, there is reason to consider why some children with this disorder never develop other problems such as ODD. Understanding the route to resilience may be a critical piece to understanding developmental outcomes of AD/HD. This understanding will help inform disruptive behavior disorder theory, enhance methods of treating AD/HD, and reduce the negative outcomes that burden the mental health and criminal justice systems. Considering protective factors in children with AD/HD is an important and much needed area of research.

Keeping these issues of risk and protection in mind, this study addressed the question: *Why do some children with AD/HD develop ODD features while others do not?* In particular, this study examined how risk and protective factors impact the development of comorbid antisocial behavior, specifically ODD, in children with AD/HD.

In order to explore the question of risk and protection in an AD/HD sample, it is first necessary to provide overviews of AD/HD, comorbidity, and antisocial behavior. Next, the concept of risk is defined and a review of the literature as it pertains to risk factors for AD/HD and ODD/antisocial behavior. Then the concepts of resilience and protective factors are defined along with a review of the resilience literature as it relates to AD/HD and ODD/antisocial behavior. Finally, the hypotheses of the study are specified in terms of risk and protective factors.

Overview of AD/HD

AD/HD Characteristics and Diagnostic Criteria

AD/HD (American Psychiatric Association, 1994) is characterized by two empirically-derived clusters of behavioral symptoms: inattention and

hyperactivity/impulsivity. Inattentive symptoms include having difficulty maintaining attention to a task, having trouble following directions, and being easily distracted.

Hyperactive-impulsive symptoms include showing constant activity by fidgeting, running around, climbing or talking excessively and having difficulty waiting for a turn to act or talk. In order to meet criteria for an AD/HD diagnosis, at least six of nine symptoms of inattention and/or six of nine symptoms of hyperactivity-impulsivity must be endorsed as developmentally deviant. These symptoms must have persisted for at least 6 months, must have been present prior to age seven, and be associated with clinically significant impairment in two or more settings (e.g., home, school, social settings). Finally, these symptoms cannot be attributable to another mental, physical, or behavioral condition.

Diagnosis can fall into one of three subtypes of AD/HD: Predominantly Inattentive Type (AD/HD-I) with the presence of 6 or more inattentive symptoms and less than 6 hyperactive-impulsive symptoms, Predominantly Hyperactive-Impulsive Type (AD/HD-II) with the presence of 6 or more hyperactive-impulsive symptoms and less than 6 inattentive symptoms, and Combined Type (AD/HD-C) with 6 or more of both types of symptoms (American Psychiatric Association, 1994). Subtyping has relevance not only on the symptom presentation of AD/HD, but also on impairment, comorbidity, and developmental course (Millich et al., 2001).

Epidemiology

AD/HD is estimated to be present in 3-7% of school-aged children reported in the general population (American Psychiatric Association, 2000). Of the few epidemiology studies using newer (DSM-IV) criteria, which separate symptoms into subtypes, the

overall rate for all types of AD/HD is higher at 7.4% (Barbarese et al., 2002) to 9.9% (Hudziak et al, 1998). Prevalence rates vary according to gender, with boys being diagnosed more frequently than girls (4:1 to 9:1) depending on the sample (APA, 2000). In clinical populations, boys are thought to out-number girls (from 6:1 to 9:1) due to a higher referral rate associated with disruptive behavior symptoms including hyperactivity/ impulsivity as well as aggression and oppositional behavior (Szatmari, 1992). Although diagnostic criteria may not adequately fit non-white samples, differences in prevalence were found across ethnic groups for boys: 3.06% in Hispanics, 4.33% in Caucasians, 5.65% in African Americans (Cuffe, Moore, McKeown, 2005). Finally, AD/HD occurs and appears similar across socioeconomic levels but has been found to be more frequent among lower SES (Szatmari, 1992).

Psychosocial Impact

Being inattentive, hyperactive and impulsive can make it difficult to perform everyday tasks and activities to a full capacity. Symptoms can impact school and social functioning including poor school performance (Barkley, 1996) and negative peer relations (Pelham & Bender, 1982). Difficulties also emerge at home including negative and coercive parenting strategies (Anderson, Hinshaw & Simmel, 1994; Danforth, Barkley, & Stokes, 1991; Mash & Johnston, 1982), increased familial conflict (Patterson, Reid, & Dishion, 1992), marital conflict (Forehand, et al., 1991; Hinshaw, 1999), and parenting stress (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992). These problems exacerbate an already chaotic situation (Patterson, Capaldi, & Bank, 1991) and coupled with parental psychopathology can increase difficulties for the child and his or her

environment (Faraone, Biederman, Jetton, & Tsuang, 1997; Frick, Lahey, Christ, Loeber, & Green, 1991). Parental psychopathology including adult AD/HD, depression, anxiety, and antisocial personality disorder (Cunningham et al., 1988; Frick et al., 1993) has been found to be more prevalent in home with a child with AD/HD than in the general population.

Developmental Course

Symptoms of AD/HD emerge at different points across development and show different phenotypic expression during an individual's life. The peak age of onset of AD/HD occurs around 3-4 years (Barkley et al., 1990). Hyperactive-impulsive features are often the first behaviors observed at this age. However, the appearance of these symptoms may be difficult to detect as abnormal, as preschool children are expected to be highly active. Inattentive symptoms are often the last ones to be detected as developmentally deviant, most often emerging between the ages of five and seven (Loeber et al., 1992). They are most evident as the child enters more structured settings such as school. Hyperactive-impulsive symptoms have been found to steadily decrease over time while inattentive symptoms remain relatively constant (Barkley, 1996). Symptoms of hyperactivity often shift from behavioral to verbal forms of hyperactivity. The disorder has been found to persist into adolescence and adulthood at a clinical level of difficulty in about 50 - 80% and 30 - 50% of the cases, respectively. (Barkley, 1998; DuPaul et al., 1997). Although the rates of diagnosable AD/HD decrease, adults continue to display non-clinical levels of symptoms.

Comorbidity

The shared presence of more than one independent disorders in an individual is known as comorbidity (Pliszka, Carlson, & Swanson, 1999). The multitude of difficulties imposed by AD/HD may also incite or be related to other clinically significant problems. As a result, AD/HD is associated with a multitude of additional difficulties including higher rates of oppositional defiant disorder (ODD, 40%; Biederman, Faraone, & Lapey, 1992), conduct disorder (CD, 25%; Biederman, Faraone, & Lapey, 1992), internalizing disorders such as depression and anxiety (20-25%; Jensen et al., 1997; Mennin et al, 2000), and learning disorders (30-50%; Semrud-Clikeman et al., 1992) among others in clinic referred populations. Despite being separate disorder with varying outcomes, ODD and CD are often discussed jointly or as a group because of their common membership to a category of psychopathology in children known as antisocial behavior.

The term 'comorbidity' has various implications within psychopathology literature. It has been a term utilized when diseases or disorders occur together to imply overlapping symptom patterns, etiological commonalties, implications for treatment, or increase risk of negative outcomes (Lilienfield, Waldman, & Israel, 1994). For the purpose of this paper, the term 'comorbidity' will be analogous to 'covariation,' which describes the presence of two diagnoses occurring more often than by chance (Kaplan & Feinstein, 1974; Lilienfield, Waldman, & Israel, 1994).

Antisocial Behavior

Oppositional Defiant Disorder & Conduct Disorder

Among the problems that are comorbid with AD/HD, Oppositional Defiant Disorder (ODD; American Psychiatric Association, 2000) occurs most frequently, emerging in approximately 40% or more of the cases (August et al, 1996; Biederman, Faraone, & Lapey, 1992). The high rate of this condition is concerning, particularly because ODD is thought to be the link between AD/HD and a pervasive pattern of adult antisocial behavior known as Antisocial Personality Disorder (APD) (Loeber, 1990; Moffitt, 1993).

ODD is associated with a pattern of aggression, defiance to authority, externalization, and generally disruptive behavior (Hinshaw & Anderson, 1996). As its name suggests, it is characterized by angry, irritable, resistant, and uncooperative behavior. A typical child with ODD will frequently talk back to adults, lose his or her temper over something small and purposely do what s/he is told not to do, among other oppositional and defiant behaviors (American Psychiatric Association, 1994). In order to meet criteria for an ODD diagnosis, four of eight symptoms of “negativistic, hostile and defiant behaviors” must be endorsed as present to a degree that is developmentally deviant. These symptoms must have persisted for at least 6 months and be associated with clinically significant impairment. Finally, these symptoms cannot be attributable to another mental or behavioral condition including Conduct Disorder.

Although it is not the primary focus of this study, it is important to briefly address Conduct Disorder (CD; American Psychiatric Association, 2000). Because nearly all of

those who meet criteria for CD first met criteria for ODD and continue to display ODD symptoms, ODD is considered a developmental precursor for CD (Lahey et al., 1992). In this diagnostic hierarchy, if met, only the CD diagnosis is given as it presumes an ODD diagnosis (APA, 2000).

Individuals with CD participate in delinquent, cruel, or destructive acts that fall into overt (blatant acts of aggression) or covert (sneaky acts of nonaggression) behaviors (Loeber, 1990). A youth with CD will violate societal norms and/or the rights of others by such acts as starting fights, hurting or threatening others, vandalism, stealing, or running away (American Psychiatric Association, 2000). In order to meet criteria for CD, at least 3 of 15 symptoms must occur repetitively within a year and one of these within the most recent 6 months. These symptoms cannot be attributable to Antisocial Personality Disorder (in adults) and must cause impaired functioning. There are three subtypes of CD: childhood-onset (at least one symptom before age 10), adolescent-onset (no symptoms prior to age 10), or unspecified (onset age is unknown). A younger age of onset is predictive of a developmental pattern characterized by more severe antisocial behavior and a poorer prognosis, whereas an older onset is indicative of greater peer influences and better prognosis (Moffit & Caspi, 2001).

Epidemiology

The prevalence of ODD is approximately 12% of the general population of children (Nottelman & Jensen, 1995) in comparison to approximately 40% in the AD/HD population (Biederman, Faraone, & Lapey, 1992). In the general population, CD occurs less frequently than ODD, emerging in 2% to 6% of the population (Hinshaw &

Anderson, 1996). By contrast it develops in 20-30% of children with AD/HD. Among those with CD, over 50% have AD/HD (Nottleman & Jensen, 1995). Although AD/HD is not present in every child with antisocial behavior, the fact that the disorders overlap so frequently is notable.

As a group, antisocial behavior occurs at much higher rates (3-4:1) in boys than in girls during childhood (Kennan, Loeber, & Green, 1999). Boys are more likely to have the early onset form of CD, which is also more associated with AD/HD. The adolescent onset is about equal in boys and girls. Boys tend to exhibit more overt, aggressive behaviors than girls. Both groups exhibit nondestructive and covert behaviors such as lying, truancy, and defiance. Girls with ADHD are half as likely to have ODD as boys (Biederman, et al 2002).

Both disorders are more prevalent in children with low socioeconomic status (Lahey et al, 1999). Parenting variables may account for the differences seen in lower SES groups (Capaldi & Patterson, 1994). In a large national sample, comparisons across racial/ethnic groups indicates similar rates of ODD in Hispanics, non Hispanic black, and non-Hispanic white groups (5.4%, 5.6%, and 5.7%, respectively) (Breslau et al, 2006). The rates are higher, but not significantly, for CD in Hispanics (6.9% vs. 4.9% and 5.0%, in blacks and whites, respectively).

Psychosocial Impact

Difficulties with aggression, oppositional behavior, and defiance impairs an individual across domains of functioning. (Lahey et al, 1997) At home, difficulties include increased parent and sibling conflict, family distress, and parenting distress

(Cunningham & Boyle, 2002; Greene et al., 2002). As might be anticipated, these children also have difficulties outside the home including reading problems (Maughan et al., 1996), school refusal (Haranda, Yamazaki, & Saitoh, 2002), and negative peer relations (Carlson et al, 1997; Greene et al., 2002). In terms of a broader social impact, nearly half of childhood referrals to outpatient clinics are for antisocial behavior and adult individuals with persistent antisocial behavioral outcomes comprise the majority of criminal offenders (Kazdin, 1995).

Developmental Course

The earliest signs of antisocial behavior may be temperamental attributes. A difficult temperament early in childhood may be the first indicator of antisocial behavior, although this is not specific to antisocial behavior (Loeber, 1990). Early symptoms of antisocial behavior, particularly aggressive tendencies, may begin to emerge around age 3-4. The average onset of ODD is around age 6.

Two principal developmental courses, which are thought to be the paths of the DSM diagnostic subtypes of “childhood-onset” and “adolescent onset,” have emerged in antisocial behavior (Moffitt, 1993). One is a continuous or life-course-persistent (LCP) path with symptoms that emerge early and persist into adulthood. The symptoms show heterotypic continuity, meaning that although behaviors are continuously antisocial in nature, they change in form over development. For example, a LCP boy may throw tantrums and act aggressively as a child then subsequently commit felonies as an adult. The LCP group consists of a smaller group of those with conduct disorder. Among those on a LCP path, core symptoms of AD/HD, hyperactivity, inattention and impulsivity are

often present early on. ODD typically emerges next along with aggression around the age of 4. An early onset of conduct disorder follows with an onset occurring before the age of 10. LCP youth engage in crime by the teen years and may continue this pattern into adulthood as antisocial personality disorder (APD; American Psychiatric Association, 1994). For most delinquent youth, the criminal behavior ends in adulthood. Nevertheless, the LCP group tends to have many problems as adults including other psychopathology, relationship difficulties, low productivity, illness, and poor parenting practices.

In comparison, a second course, the “adolescent-limited” path (AL) tends to emerge and desist during the teens (Moffitt, 1993). This pathway is more common but is also less severe. The AL path is no more associated with AD/HD than the general population (Lahey & Loeber, 1997) and is not relevant to this review.

When AD/HD and antisocial behavior are present in the same individual, AD/HD tends to appear first developmentally at about three years old and is followed by ODD at about six years old (Loeber et al., 1992). About 50% of these children will continue to have problems with ODD into their teens and 25% will no longer display ODD (Hinshaw, Lahey, & Hart, 1993). Another 25% will develop CD with an average age of onset being approximately nine years old (Lahey & Loeber, 1997; Loeber et al., 1992). A portion of these children (approximately 15-25%) will meet criteria for antisocial personality disorder (APD) as adults (Biederman et al., 1992). Figure 1 illustrates antisocial developmental trajectories for children with AD/HD.

Further work on the developmental pathways involved in AD/HD and comorbid antisocial behavior have been explored by the Developmental Trends Study (boys ages 7-

12) and the DSM-IV Field Trials for the Disruptive Behavior Disorders (males and females age 4-17) (Lahey & Loeber, 1997). Findings indicate that children who have AD/HD are significantly more at-risk for externalizing disorders such as ODD or CD than the general population. Furthermore, children who have AD/HD who develop these problems show more persistent problems over the lifespan. Children with AD/HD are not only prone to further difficulties, they comprise a bulk of those who have antisocial behavior. Because of the presumed progression of these disorders developmentally, it is important to consider the factors pertaining to their relationships with each other. Finally, the association between these disorders is specific to an early onset (childhood); adolescents with AD/HD are at no greater risk than the general population for an adolescent onset of CD. Another important finding from this work suggests that ODD is the link between the development of AD/HD and CD. That is, “children with AD/HD who do not meet criteria for ODD in childhood are not at elevated risk for CD in adolescence or antisocial behavior during adulthood.” These studies have looked primarily at children who are in elementary school or older, focus on males, use a variety of criteria to determine problem behaviors, and focus on CD as an outcome (not on ODD).

Risk

In order to understand the connection between AD/HD and ODD, it is important to examine current conceptualizations of the biological and environmental influences on these disorders. When risk is present in a population, there is a greater chance that associated problems will occur (Cowan, Cowan, & Schulz, 1996). Vulnerability to

negative outcome is thought to increase as the degree of risk increases (Werner, 1989). Risk factors are those that increase the likelihood of developing an emotional or behavior disorder beyond that of random chance and can exist in a range of forms from situational to inherent conditions (Garmezy, 1983). According to Rutter (1987), it is more accurate to describe risks as sets of processes which connect the risk condition or variable to an undesirable outcome. Examining the etiologies and associated risk factors at play may help determine why some children with AD/HD proceed down a developmental pathway that includes ODD and potentially other antisocial behavior while others do not.

Current perspectives of AD/HD hold that its origins are predominantly neurobiological in nature (Swanson et al., 1998). In particular, neurological and genetic factors are the most influential, whereas social factors, while potentially influential are no longer considered causal (Barkley, 2006).

By contrast, antisocial behavior stems from a combination of both biological and psychosocial variables, with psychosocial variables thought to play a key role (Burke, Loeber, & Birmaher, 2002). Indeed, ODD and CD are disorders with developmental paths characterized by equifinality or multiple early experiences but similar outcomes (Sameroff, 2000). AD/HD is considered one of these early factors, but other factors include parent-child interaction (Patterson, Reid, & Dishion, 1992), social-cognitive influences (Crick & Dodge, 1994) family and socioeconomic environment (Moffitt, 1993) as well as intrinsic influences such as genetic inheritance (Edelbrock et al., 1995) and child temperament (Burns & Walsh, 2002; Moffitt & Caspi, 2001).

A caveat of this review relates to the research available on ODD. Although CD is a less frequent and later occurring outcome (approximately 25% of the ODD cases), it is preceded by ODD over 90% of the time (Lahey et al., 1992). However, more research has been conducted on CD or antisocial behavior in general than ODD. As a result, very little theory or etiological research is available on ODD alone. Instead, it is often assumed or incorporated into work on CD. The lack of distinction between the disorders is a criticism in the literature as a frequently overlooked and possible missing link when attempting to elucidate antisocial behavior (Loeber et al., 2000). In this review, effort has been made to address ODD when possible or to discuss the earlier onset of CD, which involves preceding ODD and often AD/HD (Hinshaw et al., 1993, Moffitt, 1990).

Neurobiological Contributions

The neurotransmitters and related systems in the brain that are thought to be involved in AD/HD have been described by the “catecholamine hypothesis” (Pliszka, McCracken, & Maas, 1996). This view originally proposed that the core symptoms of AD/HD were caused by deficits or excesses of three neurotransmitters, dopamine, norepinephrine, and epinephrine (McCracken, 1991). Pliszka and colleagues’ (1996) proposed a multistage hypothesis in which norepinephrine and epinephrine are more associated with attention regulation and dopamine is associated with hyperactive/impulsive features. However, other findings point to dopamine being the most influential neurotransmitter involved in the presence of all AD/HD symptoms (Swanson et al., 1998).

Neurophysiology studies have found evidence for differences in brain activity between children with AD/HD and children without AD/HD. For instance, children with AD/HD may have decreased cerebral blood flow in prefrontal regions and to the pathways connecting these regions to the limbic system through the caudate nucleus (Lou et al., 1989; Sieg et al., 1995). Increased severity of AD/HD symptoms has been associated with diminished metabolic activity in the prefrontal, cingulate, and caudate regions in some positron-emission tomography (PET) scan studies (Ernst et al., 1994) but not in others (Zametkin et al., 1993).

Converging neuroanatomical evidence points to the frontal lobe (particularly the prefrontal cortex) and areas of the basal ganglia (globus pallidus and caudate nucleus) as the source of the deficits in AD/HD (Swanson et al., 1998). The difficulties in AD/HD: inattention, disinhibition, emotion/motivation regulation deficits, and disorganization were found to be comparable to the effects of damage to the prefrontal region (Benton, 1991). Impaired performance on neuropsychological (Barkley, 1990) and vigilance tests (Frank, Lazar, & Seiden, 1992) provide further confirmatory evidence. MRI studies found that the frontal lobes and basal ganglia are smaller in groups with AD/HD (Castellanos et al., 1996). Frontal cortex differences in prefrontal and caudate-striatal areas in very small samples of children and adults with AD/HD implicate deficient development of the neuroanatomical circuitry of executive control as associated with AD/HD symptoms (Swanson et al., 1998).

Compared to the brain mechanisms and structures associated with AD/HD, those associated with antisocial behavior appear to be varied and largely unspecified (Hinshaw

& Anderson, 1996). It is likely that environmental experiences thought to cause antisocial behavior are mediated by neurobiology (Lahey et al., 1993). Although not examined in ODD, two physiological differences found between early onset children with CD and clinical and normal controls were lower levels of cortical arousal (Magnusson, 1988) and autonomic reactivity (Pennington & Ozonoff, 1996). These physiological conditions may play a role in the poor decision making of those with antisocial behavior. At normal levels these systems help individuals learn to avoid situations that result in punishment or negative consequences. At low levels, negative consequences may have little impact on the learning of appropriate social conventions. Serotonin was implicated in a study that measured 5-HIAA and HVA during an experimentally induced aggression response through plasma monoamine metabolites in the blood (van Goozen et al., 2002). The metabolites were lower in children with ODD in comparison to normal children.

Family genetics. The neurobiological evidence supporting differences between those with and without AD/HD is largely correlational and indicates multiple biological pathways. Nevertheless, genetics research indicates that heredity may be the principal mechanism responsible for the neurobiological abnormalities in children with AD/HD. Family, adoption, twin and molecular biology studies indicate that the core deficits of AD/HD are heritable traits (Barkley, 1998; Hinshaw, 1999).

In family studies, higher rates of AD/HD have been found in family members of children with AD/HD than in the general population. Approximately 10-35% of the siblings of children with AD/HD also have it (Levy et al., 1997; Faraone, Biederman, Keenan, & Tsuang, 1991). Additionally, it is thought that the inheritance rate when one

parent has AD/HD is about 50% for the child (Biederman, et al., 1995). Findings indicate that AD/HD behaviors are more likely to be observed in adopted children whose biological parents rather than adoptive parents also have AD/HD behaviors (Todd, 2000).

Twin studies provide strong evidence for a genetic contribution to AD/HD. The Virginia Twin Study (Nader et al., 1998) compared the phenotypes of 900 pairs of male and female identical (MZ) twins and fraternal (DZ) twins. Using DSM-IV criteria to investigate the occurrence of AD/HD, it was found that genetics accounted for 61% of the variance. Likewise, by comparing MZ and DZ twins, Edelbrock and colleagues (1995) found an overall genetic effect that accounted for 66% of the variance in attention problems. Other twin studies indicate concordance rates that range from 50% to 90% (variable according to the use of stringent diagnostic determinations of AD/HD versus symptom counts or dimensional determinations) but average approximately 65% (Todd, 2000). In general, more severe levels of AD/HD are associated with higher rates of genetic effects.

Family, adoption, and twin studies indicate that children's genetic inheritance may also play a role in antisocial behavior (Edelbrock et al., 1995; Faraone et al., 1997; Waldman et al., 2001). However, this inheritance may be a predisposition to develop problematic behavior patterns rather than an inheritance of antisocial behavior traits, *per se* (Moffitt & Caspi, 2001). A large community based twin study from the Australian Twin Registry looked at DSM-III-R symptoms of AD/HD, ODD, and CD in MZ twins, DZ twins, and siblings (Waldman et al., 2001). Results suggested high proportions of genetic effects for both AD/HD and for ODD (.90 and .85, respectively), whereas the

heritability estimate was approximately .51 for CD. Other twin studies indicate that there may be a genetic predisposition that is more associated with aggressive than delinquent behavior (Edelbrock et al., 1995). According to parent responses on identical and fraternal twins ages 7-25, aggression is more heritable than delinquency. It was found that genetic effects accounted for an estimated 60% of the variance in aggression versus only 35% of the variance in delinquency. Impulsivity, a tendency in the combined type of AD/HD, has been linked to the subsequent development of antisocial behavior and is thought to have genetic underpinnings (Moffitt & Caspi, 2001).

Family studies of children indicate differential histories for AD/HD and antisocial behavior. One study investigated the biological relatives of a large sample of 7 to 12 year-old boys with DSM-III-R diagnosed with AD/HD and/or CD (Frick et al., 1991). The AD/HD only group was associated with a family history of AD/HD and the CD only group was associated with fathers with conduct and substance abuse problems. However, the group with both AD/HD and CD had high rates of AD/HD as well CD and substance use in the family history. This study demonstrates the independence of these disorders due to differential familial patterns and also suggests that more factors are associated with CD than the presence of AD/HD alone. Several methodological limitations to this study include maternal report as the source of data as well as the failure to clarify the familial link to these disorders. Additionally, the role and presence of ODD was not indicated in this study.

Family studies also suggest the possibility of an inherited subtype of AD/HD that includes antisocial behavior (Faraone et al., 1991; Faraone et al., 1997). Similar to Frick

and colleagues (1991), familial patterns were found in groups of children with AD/HD only, AD/HD + ODD, AD/HD + CD and control groups at age 15 (Faraone et al., 1997). It was found that relatives of all groups with AD/HD were at-risk for AD/HD and for ODD but that the more severe behavior problems of CD and APD (Antisocial Personality Disorder) were associated with relatives of the AD/HD + CD group. A major limitation of these studies is that the dual genetic and environmental roles that parents play as suggested by Frick and Jackson (1993) are not well explored. These studies do not clarify the possibility that parents dealing with similar psychopathology may also contribute significantly to a family context that is associated with antisocial behavior. One possibility, suggested by the Minnesota Twin Family Study (Burt et al., 2001) is that AD/HD and ODD are individually influenced by genetic factors; however, their covariance is “a function of a single shared environmental factor.”

Molecular genetics. A growing literature of molecular studies has looked at specific genes that are likely candidates for roles in the expression of AD/HD. Gene knockout studies of mice indicate that dopamine excesses related to the D2 and the D4 receptor genes and deficits at the dopamine transporter (DAT), D1 and D3 genes contribute to AD/HD (see review in Swanson et al., 1998). In humans, researchers have found that the allele frequency of the DAT 10-repeat VNTR on chromosome 5p15.3 is higher for participants with AD/HD than normal controls (Gill et al., 1997). DAT1 has been called a “high risk allele” because it appears to be preferentially transmitted to children with ADHD who are showing hyperactive/impulsive symptoms (Cook et al., 1995). Another gene that has received attention in the literature is the dopamine 4

receptor (DRD4). Swanson and colleagues (1998) found that higher rates of the D4 7-repeat VNTR on chromosome 11p15.5 were associated with the presence of AD/HD. Finally, serotonin was implicated in a family based genotyping study by Manor and colleagues (2001) that found decreases in short/short 5-HTTLPR genotype in children with AD/HD Combined Type (N=68). Genetic findings, although promising, do not specify the mechanism or necessity of these genes in the causation of AD/HD and have often not been well-defined.

Very few molecular biology studies have looked specifically at ODD. Snoek and colleagues (2002) found that the postsynaptic 5-HT(1B/1D) receptor is "functionally more sensitive in children with ODD." They suggested that serotonergic functioning is abnormal in "impulsive aggression." Aggression and impulsivity have been associated with a 5-HTTLPR - 44bp deletion in the promoter region, which results in reduced transcription and lower transporter protein levels (Manor et al., 2001).

Prenatal and other influences. In some cases, AD/HD may result from developmental conditions that affect the biology of the child. Low birth weight was strongly associated with AD/HD but not ODD or internalizing disorders at 6 year follow-up of a birth cohort (Breslau et al., 1996). Maternal use of alcohol (Streissguth et al., 1995) or nicotine (Milberger et al., 1996) during pregnancy has been associated with the development of AD/HD symptoms in their children even after controlling for parent AD/HD symptoms. Additional biological contributions to AD/HD may stem from environmental toxins present at pre-, peri-, or postnatal periods such as exposure to lead,

disease, neurological trauma, and malnutrition (See reviews in Anastopoulos & Shelton, 2001 and Barkley, 1998).

Early onset antisocial behavior is linked to prenatal influences in some cases (See review in Burke, Loeber, & Birmaher, 2002). Such influences include maternal smoking, substance abuse, and environmental toxins such as lead.

Psychosocial Influences

Parenting factors. In the past, researchers have hypothesized that poor parenting has led to AD/HD (Willis & Lovaas, 1977). However, data support only 5-15% of the variance in AD/HD stemming from environmental sources (Edelbrock Rende, Plomin, & Thompson, 1995). Psychosocial factors including parenting and other family/home, neighborhood or school contexts are thought to exacerbate, rather than cause, symptoms. It is suggested that the “goodness of fit” between parent and child characteristics is an important determinant in child outcomes (Chess & Thomas, 1991). However, it is unlikely that poor parenting is the primary factor leading to the development of AD/HD in children, although it may maintain their disruptive behaviors through a poor parent/child fit. Furthermore, negative family interactions are thought to be more related to the development of ODD and CD rather than to the core symptoms of AD/HD (Campbell et al., 1991; Hinshaw, 1999).

Patterson’s Coercion Model: Patterns of parenting behaviors and parent-child interaction have been the focus of work by Patterson and colleagues (Patterson, 1982; Patterson, Reid, & Dishion, 1992). Using in-home observations of parent-child interactions a model explaining how antisocial behavior develops as a result of coercive

interactions has been proposed (see Figure 2). The Coercion Model asserts that the child learns antisocial behavior through parent-child interactions (Patterson, Reid, & Dishion, 1992). This is done through increasingly coercive and negative interactions in which the child learns to use antisocial behavior (e.g. arguing, tantrums, etc) to delay or avoid unwanted demands (e.g. clean room, go to bed, etc.). Such transactions are negatively reinforcing for the child and punishing for the parent as the increasingly noxious behavior often allows a child to avoid compliance, if even for a short time. Indeed, a child is 50% more likely to show increasingly aversive responses if the parent's response was aversive instead of neutral or positive. It is thought that mothers of children with antisocial behavior are up to 8 times more likely to be inconsistent with their parenting style by not following through on their demands. These interactions are associated specifically with the early onset of antisocial behavior (Patterson et al., 1998). Furthermore, the likelihood that the child's aversive behavior will be negatively reinforced (by the parent withdrawing the request or response) is increased when families are stressed (Patterson, 1982).

Coercive interactions consistent with Patterson's theory have been found to be characteristic of the interactions between children with AD/HD and their parents (Anderson, Hinshaw & Simmel, 1994; Danforth, Barkley, & Stokes, 1991; Mash & Johnston, 1982). Observational studies indicate that children with hyperactivity tend to elicit their mother's attention, show more noncompliance, and ask more questions than other children (Danforth, Barkley, & Stokes, 1991; Mash & Johnston, 1982). Their impulsive and aversive behavior increases the rate of negative (critical, disapproving,

corrective, and punishing) and eventually avoidant responding by their mothers. These mothers showed these behaviors with their AD/HD children even when they were behaving appropriately or interacting positively (Anderson, Hinshaw & Simmel, 1994). More powerful evidence that the child's behavior began the coercive cycle stemmed from the fact that mothers were less coercive and aversive with non-AD/HD siblings. A study of interactions between mothers and teenage sons indicated that this coercive relationship remains stable over time (Danforth, Barkley, & Stokes, 1991). Mothers of the boys with AD/HD and ODD used more commands and 'putdowns' and the teens also demonstrated significantly more negative talk toward the mother. Meanwhile, mothers of the AD/HD only teens or the normal control teens were not differentiated.

Parenting skills were distinguishing factors for covarying AD/HD and ODD when family processes were investigated in 7-11 year old boys (Lindahl, 1998). Four groups were compared: boys with AD/HD only, ODD only, both AD/HD and ODD, and normal controls. Results indicated that looking at differential levels of family cohesiveness and sensitivity and consistency differentiated the three groups with behavior problems. The AD/HD+ODD group had the most lax, inconsistent, and coercive parenting. On these factors the AD/HD only and control groups did not differ. This is more evidence that parenting style is associated with different pathways for those with AD/HD.

The preponderance of studies on early onset antisocial behavior suggests that parenting is one of the most powerful predictors. In a meta-analysis of over 300 studies Loeber and Stouthamer-Loeber (1986) found that several specific variables were associated with an outcome of delinquency: a lack of involvement, poor supervision of

the child's activities, and inconsistent and negative discipline practices. While they reviewed research that focused on a more severe level of antisocial behavior at an older age (teen years), it has important implications on the evolving relationship between parent and child. On the other hand, such associations cannot determine whether parenting variables cause or are an effect of antisocial behavior.

An important finding that has major implications on a young, at-risk AD/HD population was found by Frick, Christian, and Wootton (1999). Looking at age-group differences in antisocial behavior, they found that among five factors of parenting (involvement, positive parenting, monitoring, inconsistent discipline, and corporal punishment), younger children (6-8) who had ODD symptoms were associated with less involved mothers and parents who used inconsistent discipline. The other factors were associated with older groups with these symptoms.

Inadequate parenting was also found to predict a life-course persistent path (LCP) of antisocial behavior (Moffitt, 1990; Moffitt & Caspi, 2001). The LCP path is thought to be more related to biological components (neurocognitive abnormalities and genetic inheritance). However, family environmental factors were also more severe for this group. These include harsh and inconsistent discipline as well as high levels of overall family conflict. In contrast, peer factors play the main role in the adolescent limited (AL) course. Moffitt and colleagues (Moffitt, 1993; Moffitt & Caspi, 2001) have done extensive work and shed much light on the pathways and characteristics of developing antisocial behavior. Their work suggests that AD/HD behaviors are clear risk factors for early-onset of these difficulties and that family and parenting factors are more adverse.

However, they have not specified how these behaviors and family factors might be associated.

The evidence linking AD/HD to parenting behaviors and antisocial behavior has been summarized theoretically. As a part of a larger model based on a literature review of antisocial behavior, Dishion, French, and Patterson (1995) have hypothesized that “marginal deviations” from normal socialization processes in development lead to a chain reaction of negative, antisocial, and eventually even criminal behavior. They state that this type of process occurs with children with negative temperamental characteristics such as the core deficits in AD/HD. The child with these characteristics incites “rejection and erratic, ineffective parenting practices, yielding coercive parent-child exchanges.” From these exchanges come increasing levels of negative child behavior, which leads to more marginal deviations that may eventually escalate to have societal ramifications.

Family adversity, stress & psychopathology. Parenting is not influenced by child behavior alone; other factors can influence a parent’s vulnerability toward poor parenting skills. Community and other contextual variables that impact families (e.g. transitions, poverty, adversity, and parental psychopathology) that play into early onset of difficulties are indirect and play into how well parents can be involved with their children and increase coercive interactions.

Moffitt (1990) reported that early-onset antisocial behavior is more likely in children with AD/HD who live in families with high levels of adversity. The Dunedin longitudinal study of boys from birth to age 15 in New Zealand traced the developmental

pathways toward antisocial behavior. Four groups were defined at age 13 according to DSM-III diagnosis of Attention Deficit Disorder (ADD) and self-reported delinquent behavior. They included a comorbid group (ADD and delinquent), ADD only, delinquent only, and a control group. Family adversity was defined by family characteristics related to social class, including low parental education and income and high maternal psychopathology. The comorbid group was associated with more family adversity, lower verbal intelligence and poorer reading skills than the other three groups. This group began to show antisocial behavior much earlier than the other groups (as early as 4 years old). The early behaviors began as aggression and defiance and progressed with age to more severe antisocial behavior such as truancy and theft. By contrast, the boys who had only ADD by age 13 had slightly lower levels of family adversity, above average verbal intelligence and reading skills, and showed mild, non-clinical levels of later emerging antisocial behavior (in middle childhood).

It appears that effects of low SES and other adversity disappear when parenting variables are factored in (Capaldi & Patterson, 1994). Adversity is thought to impact children mainly through the availability and effectiveness of a parent. The likelihood that a child's aversive behavior will be negatively reinforced by a parent (as seen in coercive parent-child interactions) is increased when families are stressed (Patterson, 1982). These interactions are more likely in stressed families and AD/HD families experience more parenting stress, marital stress, psychopathology and other forms of adversity (Anastopoulos & Shelton, 2001; Danforth, Barkley, & Stokes, 1991). Those who are

stressed are less likely to be able to respond immediately and consistently to their AD/HD child.

Parental inconsistencies are more likely in caregivers who are dealing with their own AD/HD or other psychopathology. This is more common in a home with a child with AD/HD than in the general population (Frick et al., 1993). If a parent has antisocial tendencies, this can also result in a dysfunctional environment for child rearing (in addition to passing on a genetic predisposition) (Frick & Jackson, 1993). Consequently, parental psychopathology also contributes to child functioning indirectly by impacting family functioning. Children who have certain traits are more likely to be parented by a parent who also has those traits (Plomin, Chipuer, & Loehlin, 1990). A child who is impulsive, hyperactive, and inattentive is more likely to have a parent who is impatient, irritable and inconsistent versus a warm, “easy” parent. Like parents who are stressed, parents dealing with psychopathology will not “fit” well with their child and will fail to respond in a way that enhances child development.

These findings are consistent with the findings of Rhoads (2001), which investigated risk and protective factors for antisocial behavior in an ethnically mixed community sample of 96 six-year-old children. Higher levels of parenting stress, parental psychopathology, and inattentive symptoms were associated with increased risk for antisocial behavioral difficulties. These risk factors explained 51% of the variance in antisocial behavior. Anastopoulos and colleagues (1992) indicated that parenting stress may be an effect of antisocial behavior rather than a cause. Therefore, it could be that

parenting stress might better account for poor parenting skills because of its high correlation with overall parenting ability.

Summary of Risk Factors

Current perspectives of AD/HD support that it is a disorder with core symptoms that stem from predominantly inherited neurobiological origins (Swanson et al., 1998). Antisocial behavior also has biological underpinnings. However, the role of neurobiology and genetics appears to be one of an inherited predisposition, which depends on the environment to develop into clinical level antisocial behavior (Moffitt & Caspi, 2001). Evidence indicates that any biological predisposition that a child has for difficulties such as ODD depends on the environment, particularly parenting (Danforth, Barkley, & Stokes, 1991), to elicit these problems. The literature suggests that AD/HD is a risk factor for ODD and CD even when initial levels of antisocial behavior are controlled (Hinshaw & Anderson, 1996; Loeber et al., 1992, Moffitt, 1990). Because the heritable predisposition of impulsivity, which may precede the development of ODD, is also a core symptom of AD/HD, it warrants closer examination.

In terms of psychosocial factors, research indicates that these influences “weigh in” differentially for AD/HD and ODD. Environmental factors may exacerbate rather than cause symptoms of AD/HD (Hinshaw, 1999). However, the environment appears to be a necessary component for eliciting and shaping the behaviors that become ODD and subsequent antisocial problems. A more biologically based disorder, AD/HD creates conditions of risk for the early onset of antisocial behavior to develop (Anderson, Hinshaw, & Simmel, 1994; Danforth, Barkley & Stokes, 1991). Research shows that

among the environmental influences on the early onset of these difficulties, a home environment can be the most influential (Frick et al., 1993; Moffitt, 1990; Patterson, Reid, & Dishion, 1992). In particular, a home in which parents are stressed (due to SES, parenting, marital discord, and psychopathology) and parenting becomes increasingly negative and coercive is the most predictive of antisocial outcomes. By eliciting negative and coercive parent-child interactions and creating/adding to the stress in a family, AD/HD behaviors can set the stage for the onset of ODD and subsequent antisocial difficulties.

A proposition of Bronfenbrenner's ecological model of human development (1994) states that the greatest impact on development stems from the "reciprocal interaction" between the child and the factors in its immediate environment, which are dubbed "proximal processes." This is consistent with research that suggests that the most potent influences on antisocial outcomes are related to the proximal factors of child temperament and parenting. The array of risk factors at play in antisocial behavior is captured in an ecological model developed by Dishion, French, and Patterson (1995) and depicted in Figure 3. As mentioned earlier, developmental pathways observed for AD/HD and antisocial behavior indicate that AD/HD emerges during preschool years, ODD follows around age 6 and in some cases, CD emerges around the age of 9 (Loeber & Keenan, 1994). Consequently, associated risk factors for this pattern of comorbidity are thought to begin early in development when the most proximal influences on the child are related to parenting and child characteristics. For this reason child factors and parent related factors were the focus of this study.

Because not all children with AD/HD continue on an antisocial trajectory, identifying risk factors is not sufficient to explain the paths that children with AD/HD take. Little work has been done that focuses on both resilience as it relates to children who have AD/HD and whether or not they have ODD. The following section addresses resilience.

Resilience

The literature overwhelmingly indicates that AD/HD is a condition that puts a child at-risk for developing further behavioral difficulties. Because AD/HD exerts a profound impact on the environment, it should come as no surprise that so many with AD/HD develop environmentally rooted conditions such as ODD. On the other hand, the indication that approximately 60% of children with AD/HD grow up without displaying ODD is puzzling because of the strong association between these externalizing conditions. When considering what differentiates children with AD/HD who develop ODD from children who do not, it is important to consider whether looking at risk is the only piece to the puzzle. Because not every child with AD/HD is associated with a developmental course that includes antisocial behavior, factors may be present that protect these individuals. Exploring possible factors that lead to positive outcomes in children with AD/HD is critical for understanding the development of problems as well as how they might be treated.

A person on the “positive pole of individual differences,” who avoids adverse outcomes when exposed to stress or when considered otherwise at-risk for problems is considered resilient (Rutter, 1990; 1987; Werner & Smith, 1992). Children with AD/HD

who do not develop ODD can be considered resilient because they experience distress, but are not “debilitated” by it (Zimmerman & Arunkumar, 1994), are considered vulnerable to specific problems (Garmezy & Masten, 1991), and are resilient in one domain (antisocial behavior) but have difficulties in others (e.g. academic problems) (Rutter, 1985).

A protective factor is defined as an element or process that buffers an individual predisposed to an undesirable outcome when risk is present (Rutter, 1987; Werner & Smith, 1982). It is not simply the opposite of a risk factor; instead it interacts with a risk factor to determine the outcome. Through interaction, a protective factor moderates the effect of a risk factor and increases the likelihood of a positive result (Garmezy et al., 1984; Zimmerman & Arunkumar, 1994).

Protective Factors Related to AD/HD

Although the literature on the risks imposed by AD/HD for comorbid ODD is growing, currently very little research has addressed protective factors in this area. The need for the concepts of resilience and protective factors stems from the fact that an “average expectable environment” is not enough (Baumrind, 1993), especially when an individual is at risk. This appears to be true for the AD/HD population in relation to comorbid antisocial behavior. The following studies help to shed light on protective factors associated with resilient outcomes for children with AD/HD.

As a part of the Minnesota Competence Enhancement Project (MCEP), August and his collaborators (1996) investigated how levels of disruptive behavior at the first assessment predicted teacher rated competence at the second assessment 3.5 years later.

Competence was determined from a composite of scores on the teacher reported Behavior Assessment System for Children (BASC) ratings of Adaptability, Leadership, Social Skills, and Study Skills. Using the average of teacher and parent reports on dimensional BASC ratings, initial levels of both hyperactivity/attention problems (HAP) and aggression/conduct problems (ACP) were compared. Results indicated that initial levels of behavior problems accounted for the greatest amount of variance in regression equations with lower initial levels predictive of higher levels of competence. For the HAP children, having a child-perceived positive relationship with their parents (according to the BASC self report) appears to predict school competence. For the ACP children, both a positive relationship and a positive self-concept predicted competence in school. The measure of outcome reflected overall competence only in the school versus specific areas of competence (social, academic, etc.).

Rhoads (2001) examined the possible role of protective factors in children with AD/HD who do not have ODD in a community sample. It was found that child adaptability and interpersonal strengths added a small but significant amount (5%) to the variance explaining antisocial outcome after AD/HD severity, parenting stress, and maternal psychopathology.

The coercive interactions that AD/HD behaviors incite may be reduced through parent training techniques and stimulant medication for the child (Danforth, Barkley & Stokes, 1991). This occurs because both can reduce the severity of aversive AD/HD symptoms. By reducing symptoms, the negative chain reaction paving the road toward comorbid antisocial behavior desists. Stimulant medication has consistently been shown

to reduce inattention, hyperactivity and impulsivity (Swanson et al., 1998). Observations have been made of mother-child interactions when children with AD/HD are dosed with a stimulant such as methylphenidate. Results from these studies indicate that children demonstrate less aversive behaviors; and mothers respond less aversively and with more frequent and positive feedback (Danforth, Barkley & Stokes, 1991). When thinking back to the situations that normalize the behaviors of children with AD/HD, it is ironic that the types of responses that these children need are the responses that they are less likely to receive. Yet when they are not showing these symptoms (e.g. while on medication), mothers are more inclined to give frequent, positive feedback and interact without coercion.

Training in the use of parent management techniques has been found to be effective in reducing coercive interactions between children with hyperactivity and their mothers (see reviews in Danforth, Barkley & Stokes, 1991 and Hinshaw, Klein & Abikoff, 1998). The use of positive reinforcement and rewards, contingency-management, and consistency with non-physical punishment are all attributes of cognitive-behavioral family treatment programs found to reduce problematic behavior in AD/HD children. Mothers were taught to take notice of and give positive feedback to their children's compliant behavior. The way in which they delivered requests was also modified. Overall decreases were shown in child noncompliance. Increases in the child's ability to sustain attention on tasks of compliance were reported. Hinshaw and colleagues (1997) found that parenting characterized by warmth and clear expectations protected boys from peer rejection, which is a corollary of antisocial behavior. Such

features are present in caregivers with an authoritative parenting style (Baumrind, 1993). Treatment outcome studies, however, should not be assumed to reflect protective processes. Children in treatment may already be on a path toward antisocial behavior. Treatment may serve as an intervention leading to remittance. Nevertheless, these studies indicate that positive and consistent parenting skills lead to improved or better outcomes in children with AD/HD.

Some studies that focus on risk factors also provide some helpful insight into possible protective factors. For instance, when investigating groups of boys with AD/HD only, ODD only, AD/HD + ODD, and no disorders, Lindahl (1998) found results that could be interpreted in protective terms. Her findings indicate that boys who had AD/HD but did not have ODD were like normal controls and had consistent, supportive parents and cohesive, harmonious families. Whereas the two groups with ODD had parents who were coercive/rejecting, inconsistent, and less cohesive. Interestingly, the AD/HD only group was associated with significantly more parental commands than any of the other groups. This unique difference may be evidence that when parents are highly involved and instructive with their AD/HD children while still being supportive and non-coercive, resilience is more likely.

In her previously discussed longitudinal work that delineated the types and pathways of antisocial behavior, Moffitt (1993) demonstrated important findings related to resilience. It was found that children diagnosed with DSM-III ADD who did not display antisocial difficulties actually had slightly *higher than average* family and home environments. These results are promising because they indicate that intervention at the

family level may encourage resilience. However, it also is a reminder that the average expectable environment may not be adequate to buffer the risks put in place by AD/HD.

Protective Factors for Antisocial Behavior

Several studies have identified protective factors that are specifically associated with ODD or to antisocial behavior in general. In an effort to limit the scope of this study to the influences most proximal to a child, only child and parenting factors will be explored here.

Child characteristics. As a part of Project Competence, Masten and colleagues (1999) reported on resilience for antisocial behavior. Results indicate that higher levels of intellectual functioning, in addition to quality of parenting, buffered the effects of high stress and protected children from antisocial behavioral outcomes. Intellectual functioning was determined by IQ estimates from block design and vocabulary subtests. Research results also support “gets along” and “good student” as features of the child which ameliorate adverse responses to risk (Rae-Grant, et al., 1989). These factors were defined by the authors on a five-point scale of how well the child harmonized with teachers, parents and peers and performed in school, respectively. An easier temperamental style was also related to resilience. Children who are likeable, social and are adaptable to change are less likely to display antisocial behavior (Masten & Coatsworth, 1998). To an extent, measures of child sociability are by nature a resilient outcome if the negative outcome is antisocial behavior. Caution should be used when considering this child characteristic as a protective factor.

Parenting factors. Environmental factors such as parenting and caregiver-child relationships have may be key moderators of children's behavioral outcomes under conditions of adversity. Twin studies indicate that in comparison to emotional and behavior problems, competency is largely the result of environmental influences such as parenting (Edelbrock, et al., 1995). Treatment programs implementing parenting skills have had good results in promoting competence and preventing antisocial behavior. When the parents of children generally 'at-risk' for antisocial behavior were trained in skills that decreased the amount of coercive transactions with their children, the risk for ODD and CD decreased significantly (Dishion, Patterson, & Kavanagh, 1992).

Masten and colleagues (1999) also found that the quality of parenting buffered highly stressed children. Quality of parenting was determined by a composite factor comprised of more structure, warmth and high expectations. The level of parenting during childhood was more predictive of later outcomes than parenting during adolescence. Parenting appears to be a critical protective factor for resilient behavioral outcomes since it was significant even when SES and IQ were controlled. Several issues relevant to this review were not addressed. First, dimensional measures rather than categorical diagnoses for psychopathology were utilized in this study. Secondly, other than "adversity," no other adverse conditions or risk factors were examined including pre-existing psychopathology. Finally, early childhood experiences and outcomes were not addressed.

A study of 5th and 6th grade Latino children looked at protective factors specific to antisocial behavior (Morrison et al., 2002). Three principal domains were investigated:

individual personality, perceived environment, and child behavior. Results indicate less engagement in antisocial behavior when students perceive that they are more engaged in school, less supervised by their parents, and have more social support. The finding that child-reported rates of less supervision predicted resilience is surprising and may be due to the fact that the child has earned the parent's trust or due to cultural differences.

Nevertheless, this finding contradicts much prior research on the relationship between parental supervision and child behavior. Although this is one of the few studies directed at protective factors for antisocial behavior, it poses several major limitations. The age-group was limited to middle childhood and level of functioning was limited to the school setting. Of greatest concern is that antisocial behavior was measured using only a student self-report questionnaire, which identified approximate frequency of current behaviors.

Attachment. Greenberg and Speltz (1988) suggested that the establishment of positive, secure attachments in families with disruptive, externalizing children is important for positive outcomes. Forehand et al. (1991) found that in families where there is stress and marital discord, children are protected from externalizing problems if a positive relationship is maintained between parent and child. However, research indicates that even when a close relationship between parent and child is absent, the child can glean benefits from other relationships. The support of a significant person other than parents protects stressed children from negative outcomes (Werner & Smith, 1982). A close relationship to grandparents has been shown to buffer children from disruptive behavior disorders and other psychopathology (Grizenko & Pawlick, 1994; Rae-Grant et al., 1989). As suggested earlier, a secure bond between a child and an adult may be the

product of other factors at work (e.g. easy temperament, responsive caregiving, etc.) rather than serve as a moderator itself.

Summary of Protective Factors

It is important to consider the protective influences that may be involved in buffering children with AD/HD from having ODD. Research in the field of resilience has begun to produce promising and positive findings on the ability of children to withstand stress and adversity of risky circumstances and demonstrate competence. Protective factors for antisocial behavior may include both child characteristics (e.g. intelligence, easy temperament) and parenting characteristics (e.g. warm, structured, consistent, and involved). However, these factors have not been addressed in the literature for specific types of antisocial behavior such as ODD. Few studies look at resilient outcomes of children at-risk due to the presence of AD/HD. Factors that have been implicated as beneficial for this population are child characteristics (e.g. low levels of symptoms, positive self-concept) and parenting characteristics (e.g. consistent, supportive, and responsive). Studies of AD/HD tend to focus on negative outcomes or treatment benefits. The study by Rhoads (2001) is the only research that has looked at AD/HD as an at-risk group and examined how protective factors add to the variance explaining an antisocial outcome. However, this study did not use a clinical population in which diagnostic status was well-defined.

The aforementioned ecological model by Dishion and colleagues (1995) conceptualizes the risk for antisocial behavior, but does not incorporate protective factors that lead to resilience. However, Bronfenbrenner's ecological model, which considers

the many layers of an individual's environment that are reciprocally interactive and contribute to developmental outcomes can easily incorporate both positive and negative influences (Bronfenbrenner, 1994). Addressing both risk and protective factors that are reciprocally interactive and contributing to further pathology or resilience would better complete an ecological model of antisocial behavior and would add to the current literature.

Summary and Hypotheses

It is generally thought that given a poor fitting environment, a child with AD/HD can proceed down a path of further difficulties that can lead to ODD. The question remains, which factors interacting with the primary AD/HD symptoms, determine which developmental trajectory a child with AD/HD follows. Although researchers recognize that children with AD/HD are a population at risk for comorbid externalizing disorders, most do not specify factors of risk or separate ODD from CD. Finally, the current literature has not identified protective factors for children with AD/HD. The goal of this study, therefore, was to investigate such factors.

As a part of a program of research investigating the relationship between AD/HD and ODD, this study sought to build upon the findings of Rhoads (2001) by continuing to view AD/HD as a group vulnerable to ODD and capable of being resilient. However, both depth and breadth were added by looking at both risk and protective factors using a clinical population. Therefore this study sought to answer the general question: *Why do some children with AD/HD develop ODD features while others do not?* In order to add to the current literature, it is important to consider *whether protective factors add to the*

explanation of a child's diagnostic status. Finally, it was important to look at these questions in *a well defined clinical sample.* With these questions in mind, the following hypotheses were made:

1. Consistent with prior clinical research, it was expected that children with more severe AD/HD, in particular higher levels of Hyperactive-Impulsive features, would be associated with oppositional-defiant outcome.
2. Consistent with prior research, environmental risk factors associated with parenting (i.e. poorer parenting practices, negative parent-child interactions, maternal psychopathology/distress) would account for a significant amount of variance in oppositional-defiant outcome.
3. Finally, protective factors would enhance the amount of variance predicting an AD/HD only outcome. Protective factors would include positive child characteristics, consistent with the community sample findings of Rhoads (2001), and a new finding of high levels of positive parenting characteristics such as involvement and consistency.

CHAPTER II

METHOD

Participants

The participants were 60 clinic-referred male children between 5 and 12 years of age ($M=9.3$ years, $SD = 2.4$) and their maternal caregiver. Only male children were recruited to reduce variability in the clinical presentation of AD/HD and ODD features. They were recruited through the AD/HD Clinic in the Department of Psychology at the University of North Carolina at Greensboro following clinical evaluations. All children underwent a standardized multi-method assessment, which incorporates parent, child, and teacher information and uses interviews, rating scales, and psychological testing procedures (Anastopoulos & Shelton, 2001).

Each child met full Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition criteria for a diagnosis of AD/HD (DSM-IV TR; American Psychiatric Association, 2000). These criteria were addressed in part via maternal responses to the C-DISC-IV (NIMH, 1997). The C-DISC-IV is a structured interview that is designed to assess the criteria for DSM-IV Axis I disorders that could be present in children. Test retest reliability in a clinic sample was .79 for AD/HD (Fisher, et al., 1997) with a validity of .72 (Schwab-Stone et al., 1996). Additionally, each child displayed gender specific developmental deviance evidenced by T-scores at or above the 93rd percentile on

the Hyperactivity or Attention problems subscales of the BASC-2 (Reynolds & Kamphaus, 2004). The BASC-2 is a broad band rating scale that uses maternal responses to 150-160 items (depending on child's age) on a 4-point scale. The measure yields 9 clinical scales, 5 adaptive scales, and 4 composite scores. The BASC-2 has good reliability with internal consistency coefficients averaging around .85 in general norm samples as well as AD/HD clinical samples.

Participants recruited were diagnosed with either Combined Type or Inattentive Type of AD/HD. In addition to other DSM-IV-TR criteria, Combined Type was defined as the presence of 6 or more of both Inattentive and Hyperactive/Impulsive symptoms. Inattentive Type was defined as the presence of 6 or more Inattentive symptoms and less than 6 Hyperactive/Impulsive symptoms. For the purpose of the proposed project, children were not grouped according to subtype. Instead, children were grouped according to the presence or absence of comorbid ODD. ODD was defined as the presence of 4 or more symptoms of ODD and T-scores at or above the 93rd percentile on the Aggression or Conduct problems subscales of the BASC-2 (Reynolds & Kamphaus, 2004) described above. Thus, one group of children had a diagnosis of AD/HD in the absence of ODD. The second group had a dual diagnosis of AD/HD and ODD. Children with the diagnosis of CD were excluded from this project.

The sample was representative of the ethnic composition of the community, with 68.3 % Caucasian (N=41) and 31.7 % Minority (N= 19; 18 African American children and 1 biracial child). In terms of treatment, at the time measures were administered, 53.3% were taking stimulant medication, 11.7% were taking non-stimulant medication,

and 35.0 % were not on medication. Full Scale IQ was in the average range ($M = 100.9$, $SD = 14.1$). Mean family income was in the \$40,000-\$50,000 range ($M = 4.3$, $SD = 2.3$). The sample had an average of 7.3 ($SD = 1.3$) inattentive symptoms, 6.6 ($SD = 2.3$) hyperactive-impulsive symptoms, and 3.7 ($SD = 2.8$) ODD symptoms. Average BASC-2 T-scores were 69.0 ($SD = 6.9$) on inattention, 70.9 ($SD = 13.1$) on hyperactivity, 62.37 ($SD = 14.7$) on aggression, and 62.2 ($SD = 14.1$) on conduct problems. A summary of these sample characteristics appears in Table 1.

The AD/HD group consisted of $N = 32$ children and the dual diagnosis group (AD/HD+ODD) consisted of $N = 28$ children. Consistent with current epidemiological literature, 46.7% of the total sample of children with AD/HD had co-occurring ODD. A summary of group characteristics appears in Table 2.

Outcome Variables

Antisocial behavior was assessed both categorically and dimensionally. The dimensional analysis was utilized to examine the relative degree to which ODD behavior was present, whereas the categorical analysis was utilized to examine the clinically defined presence of ODD, which is often overlooked in the literature. The categorical diagnosis of ODD was assessed by a positive diagnosis on the NIMH Diagnostic Interview Schedule for Children - Version IV and a T-score at or above the 90th percentile on the Aggression or Conduct Problems subscales of the BASC-2. Each child participant was placed into the 'AD/HD Only' group if the C-DISC-IV was positive for AD/HD but not for ODD. Children with both AD/HD and ODD were placed into the 'AD/HD+ODD' group.

The ODD Rating Scale (Anastopoulos, 1999) served as the dimensional measure. This measure was modeled after the AD/HD Rating Scale-IV (DuPaul, Power, Anastopoulos, & Reid, 1998) and utilizes the 8 ODD symptoms that are listed in DSM-IV-TR (American Psychiatric Association, 2000). It has good concurrent validity ($r=.61$) when compared with the BASC-2 aggression T-score and its test-retest reliability shows increasing stability with age (.54 - .60 in early childhood to .78 from age 5 to 7). Each symptom was rated on a 0 (not at all) to 3 (very often) frequency scale. Items are then summed to yield ODD scores ranging from 0-24, with higher scores indicative of greater symptom severity. In this study, the ODD severity score was used as the dimensional outcome.

Predictor Variables

Demographic information (gender, race, and chronological age) was derived from information sheets gathered at the clinic visit.

Severity of AD/HD was assessed with the ADHD Rating Scale-IV (DuPaul, Power, Anastopoulos, & Reid, 1998). The scale consists of the 9 inattentive and 9 hyperactive/impulsive symptoms in the DSM-IV (American Psychiatric Association, 1994). Each symptom is rated on a 0 (not at all) to 3 (very often) scale. The total score on the scale ranges from 0 to 54, with subtype severity scores for Inattention and Hyperactivity/Impulsivity ranging between 0 and 27. Internal consistency is excellent with an alpha of .92. Ratings have been demonstrated to discriminate between children with AD/HD and clinic-referred children who did not have AD/HD. The inattention,

hyperactivity, and impulsivity severity scores and symptom counts were used as predictors in this study.

Intelligence was measured using the Weschler Intelligence Scale for Children, Fourth Edition (WISC-IV; Weschler, 2003). The scale consists of four domains of intelligence: verbal comprehension, perceptual reasoning, working memory, and processing speed. The first two domains are comprised of 3 core subtests and the latter two are comprised of 2 core subtests. Using the four domain scores, a Full Scale IQ score is produced. This scale has well-established norms, with a mean Full Scale score of 100 and a standard deviation of 15. It has excellent reliability with average internal consistency reliability coefficients ranging from .96 to .97. An estimate of the Full Scale score can also be obtained using the scores from the Block Design and Vocabulary subtests. The Full Scale IQ score or its estimate was used as the measure of child intelligence in this study.

Child strengths were assessed using Behavioral and Emotional Rating Scale (BERS, Epstein & Sharma, 1998). The BERS was developed to assess positive features in children referred for specialized services. The BERS is a 52-item scale that measures 5 broad areas of behavioral and emotional strengths: interpersonal strengths, family involvement, intrapersonal strengths, school functioning, and affective strengths. The internal consistency is high for each factor of this measure with Cronbach's Alpha's ranging from .91 - .98 (Epstein, 1998). The broad area scores for intrapersonal strengths, school functioning, and affective strengths served as child predictors.

An additional child strength of adaptability was measured with the previously described Behavior Assessment System for Children (BASC-2; Reynolds & Kamphaus, 2004). The t-score from the Adaptability scale was used.

Parenting style/skills was measured with the Alabama Parenting Questionnaire (APQ; Shelton, Frick, & Wooton, 1996), which was filled out by the mother. The APQ queries the areas of parenting most related to disruptive behavior in children. It consists of 42 items that are rated according to frequency (from 1 = never to 5 = always). The scale produces scores on five constructs: involvement, positive parenting, poor monitoring/supervision, inconsistent discipline, and corporal punishment. All but one of the subscores (corporal punishment) has been found to have high internal consistency. Furthermore, it has been found to be useful for differentiating between the mothers of children with disruptive behavior disorders and those without these problems. All five subscores were utilized in this study.

Maternal AD/HD was assessed using a modified self-report version of a scale used with children, the Adult AD/HD Rating Scale-IV (Murphy & Barkley, 1996). Indices of overall AD/HD Severity, Inattention and Hyperactivity/Impulsivity are provided by the measure. An earlier version of the same measure (Murphy, 1992) was shown to have high internal consistency with a Cronbach's alpha of .94 (Arnold et al., 1997). The overall severity score during the past 6 months (ranging from 0-54) was used in the current study.

Additional maternal distress, including depression and anger-related difficulties, was assessed with the Symptom Checklist 90-Revised (SCL 90-R; Derogatis, 1992). The

SCL-90-R has excellent validity and reliability and has been used extensively in clinical research. The scale consists of 90 items, which cover a range of adult psychopathology and somatic complaints. Each item is rated on a 4-point scale of distress from 0 (not at all) to 4 (extremely). The scale yields T-scores for nine primary symptom dimensions and three global indices of distress. T-scores for two specific dimensions (depression and hostility) and one global index of distress (general severity index) were used in this study.

Parenting Stress related to difficult parent-child interactions was assessed by the Parenting Stress Index – Short Form (PSI-SF; Abidin, 1990). The PSI-SF is a widely used measure of 36 items that are rated on a 5 point scale. The index is comprised of three domains: parental distress, parent-child dysfunctional interaction, and difficult child. The PSI-SF yields scores from each of these three domains and a Total Stress Score. The PSI-SF is well validated with good factor, concurrent, discriminant, and construct validity as well as internal reliability. The parental distress and parent-child dysfunctional interaction factor scores were used in this study.

Socioeconomic Status (SES) was based on family income. A scale of annual family income ranging from 1 = less than \$20,000 to 8 = more than \$100,000 was used as a measure of SES.

Family/Community Involvement was measured with the Behavioral and Emotional Rating Scale (BERS, Epstein & Sharma, 1998), described previously. The broad area score for family involvement, which taps involvement in family and community activities, was utilized as a predictor.

Procedure

Standard procedure for an evaluation at the AD/HD Clinic at UNCG was followed. Measures looking at child psychopathology and functioning were given to the mother, father (if available), and teacher prior to the 1st visit to be completed and returned. Interview and rating scale data assessing child, family and parent functioning were collected during one to three clinic visits. A semi-structured background interview and the C-DISC-IV interview was administered to the mother by a graduate student examiner using a computer. Graduate students administering the C-DISC-IV were trained and supervised by a Ph.D. level psychologist certified by instructors from Columbia University. After the completion of the C-DISC-IV, additional measures of parent and family functioning were completed by the mother and father, when available. The administration of a child interview and testing procedures occurred in one to two subsequent visits. The graduate student examiner, under the direct supervision of a Ph.D. psychologist, determined diagnostic status. The family then returned to the clinic for feedback regarding the results of the evaluation.

Following the clinical evaluation, graduate student examiners sought consent for the family to be contacted for research purposes. Some families first participated in a research project on the genetic basis of AD/HD and were then invited to sign consent forms and respond to additional questionnaires for the current study. Other families recruited directly from the AD/HD Clinic were asked to return to UNCG to review and sign consent forms and respond to additional questionnaires. Verbal assent from the child participant was also obtained. Families were given a \$5 giftcard for their participation.

Design and Statistical Analyses

The current investigation utilized a case-control, observational research design to test its hypotheses. Both dimensional and categorical outcomes of antisocial behavior (i.e. ODD) were analyzed. The dimensional analysis was utilized to examine the relative degree to which ODD behavior was present, whereas the categorical analysis was utilized to examine the clinically defined the presence of ODD. Categories were represented by an 'AD/HD' group and an 'AD/HD+ODD' group. Dimensional outcome was represented by a total ODD severity score ranging from 0-24 from the ODD Rating Scale (Anastopoulos, 1999). Clinically relevant categorical outcome was based on positive diagnosis on the C-DISC-IV (NIMH, 1997) and developmentally deviant T-scores on the BASC-2 ($\geq 93^{\text{rd}}$ percentile on the Attention Problems or Hyperactivity subscales for AD/HD and $\geq 90^{\text{th}}$ percentile on the Aggression or Conduct Problems subscales for ODD).

Oppositional-defiant behavior was analyzed continuously with a hierarchical stepwise regression. Antisocial behavior was represented by the total score of ODD severity with a range from 0 to 24. Following demographic variables, other predictor variables were entered within a hierarchy of five steps according to their presumed etiological importance (clusters of child variables, parenting, maternal psychopathology/distress, and family/social variables). In each step, predictors that were significant at the $p < .05$ level were retained and locked into the regression at the next step. Nonsignificant predictor variables were dropped from further analyses. A summary of these predictor variables, presented in their conceptual categories, appears in Table 9.

Further analyses examined antisocial behavior categorically using logistic regression. The categorical outcomes (AD/HD or AD/HD+ODD) were used to evaluate the predictive value of a set of independent biological and environment child and parenting variables evaluate. By using steps, each predictor variable was examined in sequence to identify the one with the strongest relationship to the outcome. Variables were entered in an order consistent with the ecological models presented by Bronfenbrenner (1994) and Dishion, French, and Patterson (1995), with core child variables entering first (demographics, symptomology, and other child characteristics), followed by parenting variables, maternal distress variables, and family/cultural variables. The strength of each variable's association with the AD/HD+ODD outcome was evaluated using relative odds (RO). Relative odds uses an odds' ratio to assess the unique contribution of each independent variable to predicting the outcome. Values greater than 1.0 indicate a positive relationship to having both AD/HD and ODD, whereas values less than 1.0 indicate a negative relationship to the presence of both disorders. Risk factors had values greater than 1.0 (predictive of AD/HD+ODD) and protective factors had values less than 1.0 (predictive of AD/HD only outcome).

CHAPTER III

RESULTS

Preliminary analyses

Checks were used to determine whether the assumptions of the analyses were met. All variables were distributed normally with the exception of the child's AD/HD – Inattentive symptom count, the Alabama Parenting Questionnaire (APQ) - Poor Monitoring/Supervision index score, and the APQ – Corporal Punishment index score. To correct for skewness, the Inattentive symptom count was normalized by squaring the score. The skewed APQ indices were normalized using square root transformation.

Group Equivalence

A summary of characteristics for the AD/HD only and AD/HD+ODD groups appears in Tables 2 - 4. Independent sample T-tests were conducted to test for equivalency between groups. There were no differences in age ($t = 1.70, p > .05$), race ($t = -.36, p > .05$), medication status ($t = 1.40, p > .05$), SES ($t = .51, p > .05$), or Full Scale IQ ($t = .34, p > .05$). As expected, differences between groups were evident with respect to levels of aggression ($t = -4.38, p < .001$), conduct problems ($t = -4.56, p < .001$), ODD symptom counts ($t = -14.75, p < .001$), and ODD severity ($t = -11.87, p < .001$). Groups also differed on levels of hyperactive-impulsive symptom counts ($t = -2.40, p = .02$) and symptom severity ($t = -2.71, p = .009$), but not on levels of inattention. Differences were

evident on adaptability ($t = 4.83, p < .001$), intrapersonal strengths ($t = 3.94, p < .001$), affective strengths ($t = 2.24, p < .05$), parenting stress associated with dysfunctional parent-child interactions ($t = -2.78, p < .01$), and family involvement ($t = 5.04, p < .001$). No differences were found with respect to other child, parenting, or maternal distress variables. A Chi-square analysis of age and ODD diagnosis was conducted to further analyze whether the diagnostic groups differed according to age. Age groups were based on two-year age intervals (5-6 years, 7-8 years, 9-10 years, and 11-12 years). A Pearson Chi-Square coefficient of 3.06 ($p = .38$) indicated that there was not a significant difference between groups by age.

Correlations among Predictor and Outcome Variables

Summaries of bivariate correlations among the predictor and outcome variables in each conceptual category are presented in Tables 5-8. Consistent with prior research, higher levels of hyperactivity-impulsivity ($r = .29, p = .027$) were associated with higher levels of oppositional-defiant severity. However, when the hyperactivity-impulsivity factor was separated into hyperactive severity and impulsive severity, only impulsive severity was significantly correlated with oppositional-defiant behavior ($r = .31, p = .016$). In terms of IQ, Full Scale IQ and the vocabulary subtest were not associated with oppositional-defiance, but a poorer performance on the block design subtest was ($r = .30, p = .020$). Among the other child variables, higher levels of adaptability ($r = -.49, p < .001$), interpersonal strengths ($r = -.70, p < .001$), intrapersonal strengths ($r = -.53, p < .001$), school functioning ($r = -.34, p = .008$), and affective strengths ($r = -.34, p = .007$) were related to lower levels of oppositional-defiance. Because of the high correlation and

conceptual overlap between (lack of) interpersonal skills and oppositional-defiant behavior, it was decided to remove the former as a predictor in further analyses.

In terms of parenting style, more inconsistent discipline ($r = .30$, $p = .019$), poorer monitoring/ supervision ($r = .29$, $p = .026$), and increased use of corporal punishment ($r = .45$, $p < .001$) was associated with higher levels of oppositional-defiant behavior.

Positive parenting and maternal involvement did not correlate significantly. Higher levels of two maternal distress variables were significantly correlated with higher levels of oppositional-defiant behavior: maternal hostility ($r = .28$, $p = .033$) and parenting stress related to dysfunctional parent-child interactions ($r = .40$, $p = .002$). Maternal AD/HD severity, maternal depression, maternal general severity, and parenting stress related to the parent's own distress were not significantly correlated.

Among family/social predictor variables, family involvement ($r = -.64$, $p < .001$) was associated with lower levels of oppositional-defiant behavior but SES was not. No significant correlations were found between age, race, or medication status and oppositional-defiance.

Risk and Protective Factors Associated with Oppositional-Defiant Behavior severity

A stepwise hierarchical regression analysis was used to determine which variables accounted for variance in oppositional-defiant behavioral outcome. A summary of the variables entered into the regression is presented in Table 9. Using the Bronfenbrenner (1994) and Dishion et al. (1995) ecological models, the ordering of the predictor steps was conceptually driven in the following way: In the first step, Demographic Factors were entered, followed in the second step by Child Factors. The third step was Parenting

Factors, followed by the entry of the fourth step, Maternal Distress Factors, and finally, the fifth step was Family/Social Factors. An inclusion criteria probability of F to enter was $p < .05$. The outcome, oppositional-defiant behavior, was represented by the total score of ODD severity from the ODD Rating Scale (Anastopoulos, 1999).

In the first step, race and age were entered into the regression. None of the demographic factors emerged as a significant predictor of oppositional-defiant behavior and therefore none was retained in the model. In the second step, intrapersonal strengths (Adjusted $R^2 = .270$, $p < .001$) and adaptability (Cumulative Adjusted $R^2 = .372$, $p = .002$) emerged as significant child factors in predicting oppositional-defiant behavior and were retained in the model. AD/HD symptom severity (inattentive, hyperactive, or impulsive), Full Scale IQ, school functioning, or affective strengths were not found to be significant predictors in the model. In the third step, only corporal punishment emerged as a significant parenting predictor, Cumulative Adjusted $R^2 = .450$, $p = .020$, and was retained in the model. No support was found for maternal involvement, inconsistent discipline, positive parenting, or poor monitoring/supervision. In the fourth step, maternal AD/HD symptom severity, hostility, depression, or general symptom severity were entered, but none was retained as significant in the model. In the final step of the regression, family/social variables of family involvement and SES were entered. Family involvement emerged a significant predictor (Cumulative Adjusted $R^2 = .478$, $p = .009$) and was retained in the model.

As seen in Table 10, the final regression model consisted of four predictors - intrapersonal strengths, adaptability, corporal punishment, and family involvement -

which together explained 48% of the variance in an oppositional-defiant behavior outcome, $F(4, 55) = 14.52, p < .00$. An analysis of the beta weights indicates that a higher level of corporal punishment was associated with higher levels of oppositional-defiant behavior. By contrast, high levels of intrapersonal strengths, adaptability, and family involvement were associated with lower levels of these difficulties.

Risk and Protective Factors Associated with Diagnostic Classification

To examine the hypotheses using the diagnostic classification of ODD as the outcome of interest, the AD/HD group ($N = 32$) and the AD/HD+ODD group ($N = 28$) were used. The variables were entered into a forward stepwise logistic regression in the same order and five conceptual categories as the dimensional analysis to determine which variables differentiated between an AD/HD only outcome and an outcome with AD/HD and ODD. Demographic Factors were entered first, but none was significant in classifying children into diagnostic groups. Next, adaptability [$\text{Exp}(B) = .884, p = .012$] followed by intrapersonal strengths [$\text{Exp}(B) = .604, p = .02$] emerged as significant child factors in predicting diagnostic classification and were retained in the model. Finally, impulsive symptom count emerged as nearly significant predictor [$\text{Exp}(B) = 2.98, p = .052$] Due to the significance in the change if removed from the model ($p = .03$), the variable was retained as a predictor. No other child, parenting, maternal distress, or family/social variables were retained in the final model. The final logistic regression model, therefore, consisted of adaptability, intrapersonal strengths, and impulsive symptoms. A summary of the variables entered into the final logistic regression model appears in Table 11.

An analysis of the odds ratio, Exp(B), and the confidence intervals for the odds ratio indicated that higher levels of adaptability and intrapersonal skills served as protective factors for a comorbid ODD diagnosis. By contrast, the odds ratio for impulsivity indicates that a high level of impulsivity was a risk factor for comorbid ODD. However, because 1 fell just within the confidence interval [95% C.I. for Exp (B) = .989 to 8.949], impulsivity is nearing significance as a risk factor.

The model correctly classified 26 out of 32 children (81.3% accuracy) as being in the AD/HD only group and 25 out of 28 children (89.3% accuracy) as being in the AD/HD + ODD group. The overall correct classification rate was 85%, with 51 out of 60 cases predicted accurately. The false positive rate was 10.7% and the false negative rate was 18.7%. The predicted and actual outcomes were compared in a contingency table, a summary of which appears in Table 12. The final model deviance ($-2LL = 48.83$, $p < .001$) and likelihood ratio ($\chi^2 = 21.0$, $p < .001$) were significant, indicating that the model predicted ODD diagnosis at a significantly higher rate than would be expected by chance alone (i.e. 50%). Adaptability alone predicted 75% of the cases ($\chi^2 = 21.0$, $p < .001$), intrapersonal strengths improved the model by 5% ($\chi^2 = 7.36$, $p = .007$), and impulsivity improved the model by another 5% ($\chi^2 = 4.71$, $p < .03$).

CHAPTER IV

DISCUSSION

This study explored risk and protective factors that are associated with oppositional defiant behaviors within an AD/HD population. As predicted, both risk and protective factors emerged as significant predictors of an oppositional-defiant outcome. Specifically, a higher level of corporal punishment was associated higher levels of these behaviors, whereas higher levels of child adaptability, intrapersonal strengths, and involvement in family/community were associated with fewer behavior problems. Overall, these factors explained 48% of the variance in the levels of oppositional-defiant behavior. From a slightly different perspective, categorical analysis found that impulsive symptoms served as a risk factor and child adaptability and intrapersonal strengths served as protective factors, predicting with 85% accuracy which children had a co-occurring diagnosis of ODD and which did not.

The results of the correlational and categorical analysis are supportive of Hypothesis 1. Consistent with prior clinical research (Hinshaw & Anderson, 1996; Loeber, Green, Lahey, Christ, & Frick, 1992), children with a more severe level of AD/HD were more likely to have comorbid ODD. As expected, levels of inattention were not associated with an ODD outcome but hyperactive-impulsive symptoms were. In particular, the impulsive symptoms were retained in the logistic regression model and

boosted its prediction rate by an additional 5%. It was not surprising that children who are impulsive are more likely to respond to others in more socially unacceptable ways (e.g. with defiance, aggression, and oppositionality). Impulsivity is a temperamental trait that is part of the symptomology of AD/HD and has been linked to antisocial behavior (Moffitt, 1990). An impulsive child is more likely to be emotionally reactive and to show lack of judgment when faced with even neutral social cues (Gronau & Weiss, 1997). By extracting the impulsive features from the Hyperactive-Impulsive symptoms of AD/HD, this study demonstrated that it appears to be the tendency to act without thinking that is more associated with ODD, rather than the hyperactive symptoms.

Evidence was also found for risk factors over and above AD/HD, thereby supporting Hypothesis 2. Specifically, higher levels of maternal-reported corporal punishment were associated with increased oppositional-defiant behavior in children with AD/HD. This is consistent with prior research (Frick, Christian, & Wootton, 1999; White et al, 1994) and suggests that within an AD/HD population, a more physical and negative parenting style is less effective in reducing disruptive behavior and is instead associated with increased oppositional-defiant behavior. Although hyperactive-impulsive AD/HD behaviors tend to elicit negative and punishing responses from mothers, mothers tend to respond this way even when their sons behave appropriately (Anderson, Hinshaw & Simmel, 1994). Thus, mothers using corporal punishment may model angry and aggressive behaviors, while also providing very limited modeling or reinforcement of positive behavior.

Although this study did not examine the developmental unfolding of these disorders, taking these risk factors together, the ecological model hypothesized by Dishion and colleagues (1995) is supported. These findings indicate that a child with more impulsive AD/HD features may be more likely to engage in negative interactions with his maternal caregiver, which may lead to increasingly negative, even corporal, parenting strategies and more antisocial behavior.

However, in contrast to the risks presented in children with AD/HD, a third hypothesis of this study addressed a new dimension, the presence of protective factors in children with AD/HD without comorbid oppositional-defiance. It was found that the children characterized by higher levels of adaptability and intrapersonal strengths have a more favorable outcome. Adaptability is characterized as having a flexible personality and includes attributes such as adjusting well to changes in routine/plans, recovering “quickly after a setback,” and as being able to be “soothed when angry.” In addition, when a child has a pleasant temperament with high intrapersonal strengths (e.g. smiles, has a sense of humor, can identify his own strengths) despite his hyperactive, impulsive and inattentive nature, an outcome free of comorbid antisocial problems is more likely. Such children tend to elicit more positive feedback from the world, more support from others, and are able to have a sense of competence despite their deficits. Children who are better able to cope with stress and adapt to change easily may be buffered from the chaos and stress elicited by AD/HD. These children may be less likely to respond to stress in an oppositional or defiant manner. August et al (1996) showed that adaptability was also related to having a more positive self concept and a more positive relationship with

parents. It is possible that while adaptability may be characterological in some children, it may also be enhanced by children's interactions with others, particularly parents. This underscores the need for caregivers to not only help children with AD/HD by treating their deficits, but to nurture the child's sense of self and sense of competence. This finding of positive child characteristics as protective factors in a clinical sample is consistent with the community sample findings of Rhoads (2001). Although the symptoms of AD/HD alone can be a source of frustration for a child and a parent, when a child also has "attractive" temperamental qualities, the impact of the child's difficulties may be buffered. An element or process that buffers an individual predisposed to an undesirable outcome (Rutter, 1987; Werner & Smith, 1982) is the very definition of a protective factor.

A third protective factor, the involvement of children with AD/HD in family and community activities, was associated with low levels of ODD severity. This finding is consistent with resilience literature (Werner & Smith, 1982), but is a new finding for children with AD/HD. In this study, groups did not differ according to the level of maternal involvement with the child, but instead differed according to level of child involvement in family and community. Family and community involvement has been viewed as a protective factor for general populations. The notion that "it's hard to have time to get in trouble when you are busy" is not new. Like other groups of children, children with AD/HD appear to be protected from antisocial outcomes when they are involved with their family and the community, even if their involvement is not specific to their maternal caregiver.

It was somewhat surprising that maternal distress did not emerge as a risk for comorbid disruptive behavior disorders. Prior research (Faraone et al., 1997; Frick et al, 1991) points to the chaotic and stressed environment that a parent's psychopathology can impart on a child. This study indicated that the child's behavioral and temperamental characteristics, coupled with parenting style and involvement in family/community, were more predictive of oppositional defiance than levels of distress. These mothers of boys with AD/HD had high levels of distress regardless of ODD status. It is possible that mothers dealing with their own difficulties may respond in different ways. Those who respond with a more negative parenting style have children with increased levels of ODD. Those who may expose children to less of their own distress by getting them involved in activities have children with low levels of ODD. Further research is needed to explore this domain.

In sum, the findings from this study provide support for multiple domains of influence on the presence of comorbid disruptive behavior disorders in children with AD/HD, with factors from the child, parenting, and family/community domains emerging as significant. This study corroborates prior findings related to risk factors for ODD and suggests that the new dimension of protective factors may be important to consider in the ecology of children with AD/HD. Children with greater difficulties with impulsivity, whose mothers use a more negative, corporal approach to parenting, were more likely to have comorbid ODD. In terms of protective factors, it appears that children with AD/HD who do not go on to develop further problems with aggression and defiance are perceived

by their mothers as having more pleasant and adaptive characteristics and are more involved in the family/community.

Although promising, such results must be tempered by a consideration of the limitations inherent in this type of design. All measures, except for Full Scale IQ, are based on self- and other-report questionnaires. Although the initial diagnostic assessment was based on multiple informants, the questionnaires in the analysis were responded to by maternal caregivers. Information gathered across settings and caregivers would provide a more comprehensive, less biased assessment of child behavior and situational variables. In addition, examining the role of fathers and teachers in term of risk and resilience for comorbid ODD behavior in children with AD/HD would be crucial. Furthermore, because this study aimed to determine factors that predict developmental outcomes, a longitudinal study would provide more conclusive answers to the hypotheses addressed here. For example, it would be important to confirm with longitudinal research that involvement and other protective factors precede the differentiation of the groups into AD/HD with and without ODD. The design of the study placed limits on the ability to make causal inferences.

This study investigated only boys with AD/HD and ODD. Therefore, the results cannot be generalized to the population of girls. Current research indicates that both AD/HD and antisocial behavior may differ in boys and girls (Waschbusch & King, 2006; Moffitt & Caspi, 2001), which the scope of the current study could not adequately address. This is an important area of research that warrants further investigation. It

would be of great value to determine if similar or distinct risks and protections are involved in the presentation of these disorders in girls.

Little is known about the combined impact of risk and protective factors on developmental outcomes, particularly for children with AD/HD. This study supports prior research that identifies risk factors, but also provides support for the association of protective factors related to AD/HD and co-occurring ODD. Further research is needed to determine the direction of these relationships and their theoretical and clinical implications. This study highlights the importance of continued work in the area of risk factors and need for more comprehensive investigations in the area of protective factors.

The findings have implications for the assessment and treatment of children with AD/HD. This study highlights the potential utility of identifying both aspects in clinical assessment and incorporating them into case conceptualization. Just as children benefit from positive reinforcement, families may benefit from hearing about what assets they or their child brings, what they are “doing right,” and what they should continue to do, in addition to learning about deficits, diagnostics conclusions, and recommendations for change. The findings also underscore the need for a multi-layered treatment approach that includes training caregivers in proper parenting strategies and involving children in activities that allow for structured social interaction and increase the child’s sense of competence (e.g. sports, clubs, family time, and religious activities). In addition to reducing aversive behaviors by learning more positive, consistent, and effective parenting skills, parents might also benefit from learning ways to enhance children’s sense of competence and reinforce positive features. It is possible that caregivers may be trained

to improve their “goodness of fit” (Chess & Thomas, 1991) with their AD/HD child in order to avoid oppositional-defiant and other antisocial behavior. Averting the progression of children with AD/HD to ODD is key to preventing more severe conduct problems. More research is needed to determine the utility and effectiveness of incorporating protective factors into assessment and treatment approaches.

In conclusion, this study addressed the frequent co-occurrence of antisocial behavior within an AD/HD population and explored why some children with AD/HD have ODD features while others do not. Risk and protective factors in the child, parenting, and family/social domains emerged as significant predictors of oppositional-defiant behavior. Although these findings lend further support to prior research related to risk factors, they also establish a new perspective, the importance of examining protective factor among an AD/HD population. Those with AD/HD are vulnerable to more severe problems and this study provided compelling evidence that factors involved in resilient outcomes should not be overlooked. The findings shed new theoretical light on the ecological model of antisocial behavior and have practical implications on how children with AD/HD are assessed and treated.

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APPENDIX A

TABLES

Table 1. Sample Characteristics

Sample Size	60
Gender	
Male	100.0%
Race	
Caucasian	66.7 %
Minority	33.3 %
	<hr/>
	<u>M</u> (<u>SD</u>)
Age in years	9.3 (2.4)
Socioeconomic Status	4.3 (2.3)
Child IQ	
Verbal	10.1 (2.5)
Block Design	10.3 (3.4)
Full Scale	100.9 (14.1)
AD/HD Symptom Counts	
Inattentive symptoms	7.3 (1.3)
Hyperactive/Impulsive symptoms	6.6 (2.3)
ODD Symptom Counts	3.7 (2.8)
BASC-2 T-Scores	
Inattention	69.0 (6.9)
Hyperactivity	70.9 (13.1)
Aggression	62.4 (14.7)
Conduct problems	62.2 (14.1)

Note: IQ = Intelligence Quotient; AD/HD = Attention-Deficit Hyperactivity Disorder;

ODD = Oppositional Defiant Disorder; BASC-2 = Behavior Assessment System for Children

Table 2. Group Characteristics

	AD/HD only	AD/HD + ODD
	(N = 32)	(N = 28)
Race		
Caucasian	68.8%	64.3%
Minority	31.3%	35.7%
Medication Status		
Stimulant medication	62.5%	42.9%
Non-stimulant medication	12.5%	10.7%
Not on medication	25.0%	46.4%
	<u>M</u> (<u>SD</u>)	<u>M</u> (<u>SD</u>)
Age in years	9.6 (1.5)	8.9 (1.9)
Socioeconomic Status	4.4 (2.4)	4.1 (2.1)
Child IQ		
Vocabulary	9.9 (2.4)	10.4 (2.7)
Block Design	10.8 (3.3)	9.8 (3.0)
Full Scale	101.4 (14.9)	100.2 (13.2)
AD/HD Symptom Counts		
Inattentive symptoms	7.2 (1.3)	7.4 (1.3)
Hyperactive symptoms	3.8 (1.9)	4.6 (1.6)
Impulsive symptoms *	2.1 (1.0)	2.8 (0.4)

Note: AD/HD = Attention-Deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder;

IQ = Intelligence Quotient

* p < .01.

Table 3. Means and Standard Deviations of Child Variables by Group

	AD/HD only	AD/HD+ODD	t
	<u>M</u> (<u>SD</u>)	<u>M</u> (<u>SD</u>)	
ADHD RS: Inattentive severity	19.4 (4.0)	20.6 (4.5)	-0.28
ADHD RS: Hyperactive severity	10.4 (4.7)	13.3 (3.7)	-2.60*
ADHD RS: Impulsive severity	5.9 (2.4)	7.4 (1.6)	-2.79**
BASC-2 - Adaptability	40.1 (8.2)	35.5 (7.2)	4.83***
BERS - Intrapersonal strengths	11.2 (2.2)	9.2 (1.6)	3.94***
BERS - School Functioning	9.3 (2.5)	8.2 (2.0)	5.04***
BERS - Affective strengths	11.8 (2.1)	10.5 (2.7)	2.24*

Note: AD/HD = Attention-Deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder;

ADHD RS = ADHD Rating Scale IV; BASC-2 = Behavior Assessment System for Children;

BERS = Behavioral and Emotional Rating Scale

*p<.05, **p<.01, ***p<.001.

Table 4. Means and Standard Deviations of Parenting, Maternal Distress, and Family Involvement Variables by Group

	AD/HD only		AD/HD+ODD		t
	M	(SD)	M	(SD)	
APQ - Involvement	0.27	(0.92)	0.25	(1.04)	0.06
APQ - Positive parenting	0.19	(0.91)	0.12	(1.00)	0.27
APQ - Poor monitoring (sq rt)	0.71	(0.68)	0.84	(0.50)	-0.89
APQ - Inconsistent discipline	-0.81	(1.08)	-0.15	(1.12)	-2.31*
APQ - Corporal punishment (sq rt)	0.85	(0.60)	1.17	(0.42)	-2.42*
Adult ADHD RS: Maternal AD/HD severity	12.8	(12.7)	13.1	(8.6)	-0.10
SCL-90R - Maternal depression	57.0	(12.6)	59.9	(8.1)	-1.02
SCL-90R - Maternal hostility	54.6	(8.9)	58.1	(11.1)	-1.39
SCL-90R - Maternal general severity	55.7	(12.0)	58.8	(9.8)	-1.08
PSI-SF - Parental distress	29.2	(10.1)	30.3	(7.7)	-4.66***
PSI-SF - Parent-child dysfunctional interaction	23.6	(7.1)	28.8	(7.4)	-2.79**
BERS – Family Involvement	11.6	(2.1)	9.0	(1.8)	5.04***

Note: AD/HD = Attention-Deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder;

APQ = Alabama Parenting Questionnaire; Adult ADHD RS = Adult ADHD Rating Scale IV;

SCL-90R – Symptom Checklist – 90 – Revised; PSI-SF = Parenting Stress Index – Short Form;

BERS = Behavioral and Emotional Rating Scale

*p<.05, **p<.01, ***p<.001.

Table 5. Correlations among Outcome, Demographic, & Family/Social Predictors

	1	2	3	4	5
1. ODD RS: ODD Severity	-				
2. Age (months)	-.14	-			
3. Race	-.08	-.10	-		
4. Socioeconomic Status	-.17	-.00	-.29*	-	
5. BERS: Family Involvement	-.64**	.07	-.02	.28	-

Note: ODD RS = Oppositional Defiant Disorder Rating Scale IV; BERS = Behavioral and Emotional Rating Scale

*p<.05, **p<.001.

Table 6. Correlations among Outcome and Child Predictors

	1	2	3	4	5	6	7	8	9	10
1. ODD RS: ODD Severity	-									
2. ADHD RS: Inattentive severity	.13	-								
3. ADHD RS: Hyperactive severity	.19	.10	-							
4. ADHD RS: Impulsive severity	.31*	.28*	.50***	-						
5. BASC-2: Adaptability	-.49***	-.08	.50***	-.27*	-					
6. BERS: Interpersonal strengths	-.70***	-.15	-.26**	-.26*	.54***	-				
7. BERS: Intrapersonal strengths	-.53***	-.30*	-.08	-.23	.33*	.58***	-			
8. BERS: School Functioning	-.34**	-.39**	-.07	-.07	.25	.46***	.47***	-		
9. BERS: Affective strengths	-.34**	.08	-.04	-.16	.29*	.39**	.51***	.38**	-	
10. WISC: Full Scale IQ	-.10	-.31*	-.15	.20	-.08	-.01	.05	.29*	-.03	-

Note: ODD RS = Oppositional Defiant Disorder Rating Scale IV, ADHD RS = Attention-Deficit/Hyperactivity Disorder Rating Scale IV; BASC-2 = Behavior Assessment System for Children; BERS = Behavioral and Emotional Rating Scale; WISC = Wechsler Intelligence Scale for Children – Fourth Edition IQ = Intelligence Quotient

*p<.05, **p<.01, ***p<.001.

Table 7. Correlations among Outcome and Parenting Predictors

	1	2	3	4	5	6
1. ODD RS: ODD Severity	-					
2. APQ: Involvement	-.04	-				
3. APQ: Positive Parenting	-.04	.64**	-			
4. APQ: Poor Monitoring (sq rt)	.29*	-.18	-.16	-		
5. APQ: Inconsistent Discipline	.30*	-.01	.05	.07	-	
6. APQ: Corporal Punishment (sq rt)	.45**	-.04	.11	.32*	.11	-

Note: ODD RS = Oppositional Defiant Disorder Rating Scale IV; APQ = Alabama Parenting

Questionnaire

*p<.05, **p<.001

Table 8. Correlations among Outcome and Maternal Distress Predictors

	1	2	3	4	5	6	7
1. ODD RS: ODD Severity	-						
2. Adult ADHD RS: Maternal AD/HD severity	.08	-					
3. SCL-90R: Maternal Depression	.11	.29*	-				
4. SCL-90R: Maternal Hostility	.28*	.37**	.54***	-			
5. SCL-90R: Maternal General Severity	.17	.44***	.84***	.73***	-		
6. PSI-SF: Parental Distress	.09	.41**	.36**	.40**	.44**	-	
7. PSI-SF: Parent-Child Dysfunctional Interaction	.40**	.17	.22	.44***	.32*	.42**	-

Note: ODD RS = Oppositional Defiant Disorder Rating Scale IV; Adult ADHD RS = Adult ADHD Rating Scale IV; SCL-90R – Symptom Checklist – 90 – Revised; PSI-SF = Parenting Stress Index – Short Form

*p<.05, **p<.01, ***p<.001

Table 9. Summary of Variables Entered into Stepwise Hierarchical Regression

Step	Domain	Variables
1	Demographics	Chronological Age Race
2	Child Characteristics	ADHD RS: Inattention ADHD RS: Hyperactivity ADHD RS: Impulsivity BASC-2: Adaptability BERS: Intrapersonal strengths BERS: School Functioning BERS: Affective strengths WISC: Full Scale IQ
3	Parenting	APQ: Inconsistent Discipline APQ: Involvement APQ: Positive Parenting APQ: Monitoring APQ: Corporal Punishment
4	Maternal Distress	Adult ADHD RS: Maternal AD/HD severity SCL-90R: Maternal Depression SCL-90R: Maternal Hostility SCL-90R: Maternal General Severity PSI-SF: Stress from Mother's Distress PSI-SF: Stress from Parent-Child Interaction
5	Family/Social Variables	BERS: Family Involvement Socioeconomic Status

Note: ADHD RS = Attention-Deficit/Hyperactivity Disorder Rating Scale; BASC-2 = Behavioral Assessment System for Children; BERS = Behavioral and Emotional Rating Scale; WISC = Wechsler Intelligence Scale for Children – Fourth Edition; IQ = Intelligence Quotient; APQ = Alabama Parenting Questionnaire; Adult ADHD RS = Adult Attention-Deficit/Hyperactivity Disorder Rating Scale-IV; SCL-90R – Symptom Checklist – 90 – Revised; PSI-SF = Parenting Stress Index – Short Form

Table 10. Summary of Regression Steps Predicting Oppositional-Defiant Behavior

Severity

Step	Variable	Std. β	Cum. R^2	Cum. Adj. R^2	F	p
1	Intrapersonal Strengths	-.53	.28	.27	22.86	<.001
2	Intrapersonal Strengths	-.42	.28	.27	18.48	<.001
	Adaptability	-.35	.39	.37		
3	Intrapersonal Strengths	-.26	.28	.27	15.25	<.001
	Adaptability	-.39	.39	.37		
	Corporal Punishment	.28	.45	.42		
4	Intrapersonal Strengths	-.10	.28	.27	14.52	<.001
	Adaptability	-.27	.39	.37		
	Corporal Punishment	.23	.45	.42		
	Family Involvement	-.36	.51	.48		

Table 11. Summary of Logistic Regression Steps Predicting ODD Classification

Step	Variable	B	p of B	Exp(B)	95.0% C.I. for Exp(B)		Change -2 Log Likelihood	p of Change	%Predicted by Model
					Lower	Upper			
1	Adaptability	-.16	<.001	.85	.78	.93	22.00	<.001	75.0
2	Adaptability	-.14	.003	.87	.79	.95	14.65	<.001	80.0
	Intrapersonal Strengths	-.49	.016	.61	.41	.91	7.36	.007	
3	Adaptability	-.12	.012	.88	.80	.97	10.01	.002	85.0
	Intrapersonal Strengths	-.50	.020	.60	.40	.92	6.72	.010	
	Impulsive Symptom Count	1.09	.052	2.98	.99	8.95	4.71	.030	

Table 12. Predicted versus Actual Diagnostic Group Classification

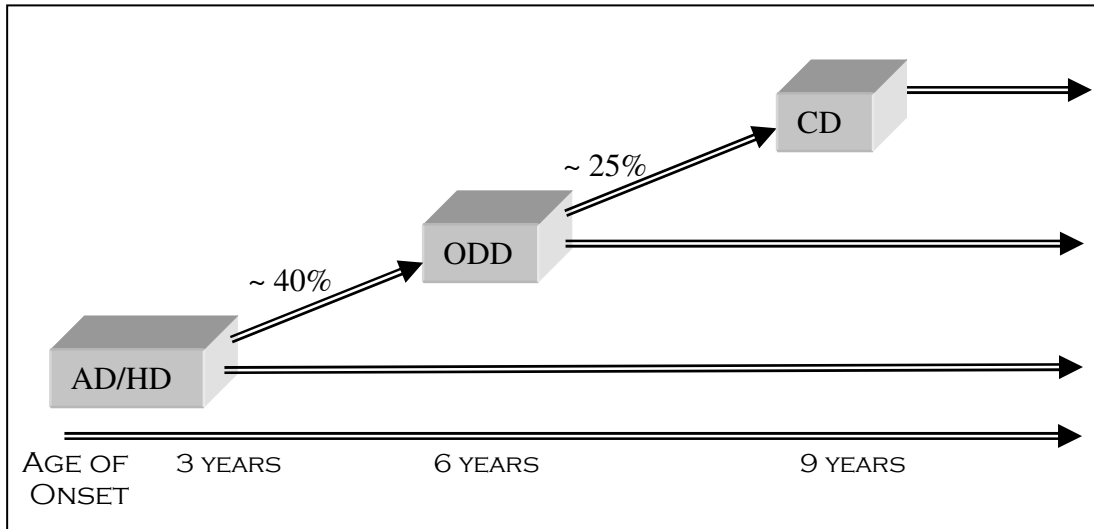
Cell Count % Correct (% Incorrect)		Correct Classification		Total
		AD/HD Only	AD/HD + ODD	
Predicted Classification	AD/HD Only	26 81.3	3 (10.7)	29
	AD/HD + ODD	6 (18.8)	25 89.3	31
Total		32	28	60

Note: AD/HD = Attention-Deficit/Hyperactivity Disorder, ODD = Oppositional Defiant Disorder

APPENDIX B

FIGURES

Figure 1. Developmental Course of Disruptive Behavior Disorders



Note: Percentages indicate proportion of children with AD/HD estimated to develop ODD or CD.

AD/HD = Attention-Deficit/Hyperactivity Disorder; ODD = Oppositional Defiant Disorder;

CD = Conduct Disorder

Figure 2. Example of Coercive Parent-Child Interaction (Patterson et al., 1992)

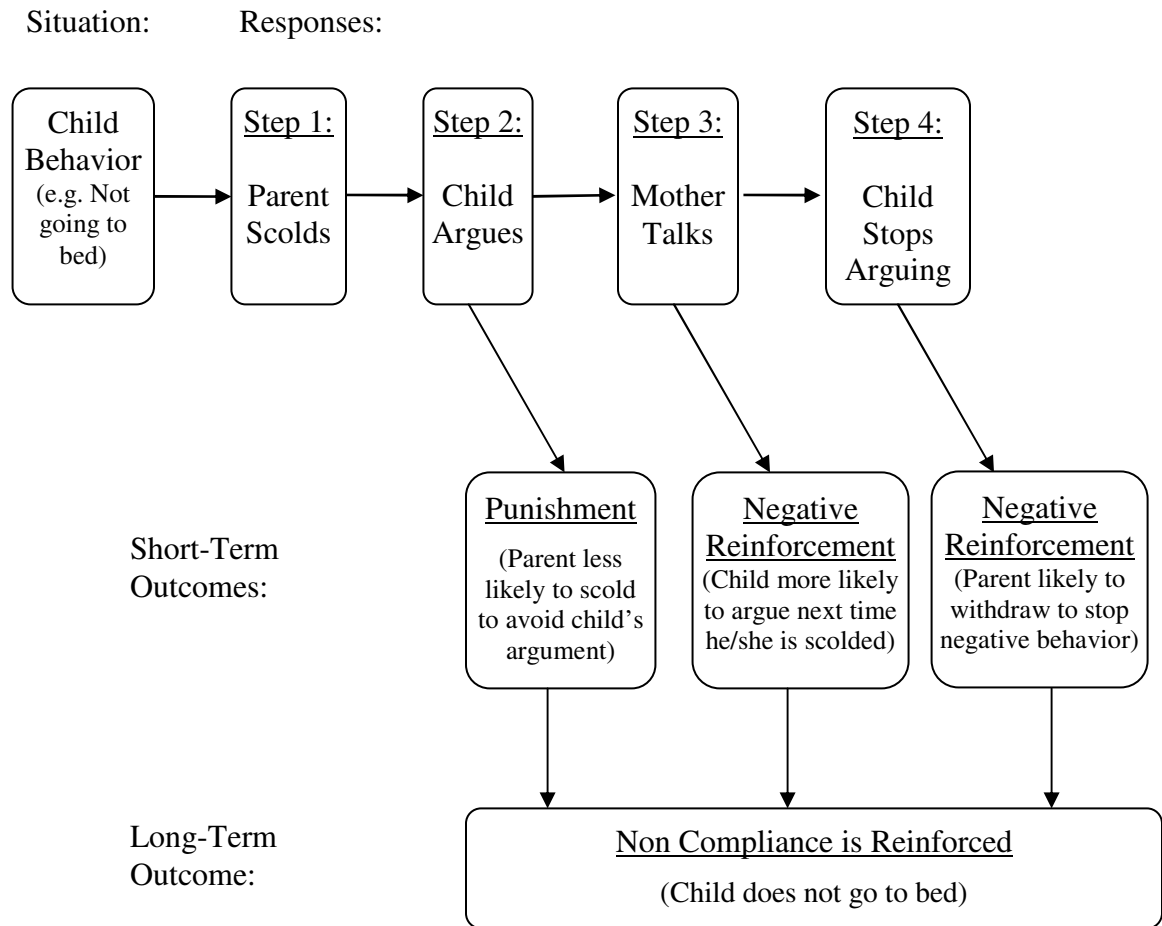


Figure 3. Ecological Model of Antisocial Behavior (Dishion, French & Patterson, 1995)

