ABSTRACT

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                            OVERT/COVERT PATTERNS OF CONDUCT
                            DISORDER SYMPTOMS IN CHILDREN
                            WITH ADHD.

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       There are inconsistencies in findings exploring the relationship between

executive functions (EF), attention-deficit/hyperactivity disorder (ADHD), and

conduct disorder (CD) symptoms in children in adolescents. In order to reconcile

conflicting results in the existing literature, it is necessary to consider more carefully

how these constructs are measured and the theory underlying any expected

associations. The proposed study examined the EF correlates of overt and covert CD

symptoms in a high-risk sample of 6-14 year old children with ADHD and varying

levels of conduct problems. Several aspects of EF were examined, including shifting,

working memory, behavioral inhibition, and interference control, to examine their

relationship to both ADHD and overt and covert conduct problems. It was expected,

after ADHD was accounted for, that deficits in behavioral inhibition and working

memory would be related to both overt and covert CD symptoms, whereas deficits in

shifting and interference control would be uniquely related to overt CD symptoms.

Set shifting abilities were found to be significantly lower in children with co-occurring ADHD and CD in comparison to children with ADHD-only. Results failed to find consistent evidence for differential relationships between individual overt and covert behaviors and domains of EF, but an interaction between set shifting and interference control did significantly predict overt, but not covert symptoms. Potential reasons for these findings, as well as future directions for research are discussed.

EXECUTIVE FUNCTIONING AND OVERT/COVERT PATTERNS OF
CONDUCT DISORDER SYMPTOMS IN CHILDREN WITH ADHD

By

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Dedication

The contents of this document would not have been possible without the support of my family. My journey to, and through graduate school would not have been a success without the preparation and support provided to me by my mother, father, sister, and brother. They gave me the confidence and encouragement to pursue my dream. Most importantly, this work is dedicated to my husband, who through some of the most challenging times reminded me of the purpose of my efforts and gave me my vision and future dreams. It was only with this foundation that the motivation, creativity, and effort put forth in this project was possible.

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Introduction

CD symptoms in Children with ADHD: Definition of the Problem

Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by symptoms of inattention, hyperactivity, and/or impulsivity. ADHD is usually diagnosed early in the elementary school years (APA, 2000), with an onset which is typically prior to the onset of commonly co-occurring disruptive behavior problems, including conduct disorder (CD) and oppositional defiant disorder (ODD). There are two dimensions of ADHD symptoms: inattentive and hyperactive/impulsive. Inattentive symptoms include distractibility, difficulty concentrating, disorganization and forgetfulness. Hyperactive/Impulsive symptoms include fidgeting, excessive motor activity, and acting without thinking. Children with ADHD may have elevated levels of one or both of these symptom dimensions, and can therefore be diagnosed with one of three subtypes: the predominantly inattentive subtype, the predominantly hyperactive/impulsive subtype, and the combined subtype. ADHD is associated with impairment in academic, social, and familial domains, putting children with ADHD at risk for a multitude of co-occurring mood, anxiety, and disruptive behavior problems across development. Indeed, the comorbidity rate for ADHD and CD ranges from 13.7% (Romano, Tremblay, Vitarro, Zoccolillo, Pagani, 2005) to 93% (Jensen, Martin & Cantwell, 1997; Bird et al., 1993) in community samples of children with ADHD, and children are at 12 times increased risk of having ADHD or CD if they have the other disorder (Romano, et al., 2005).

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CD is defined by a persistent pattern of behavior in which the basic rights of others or societal norms are violated (APA, 2000). CD symptoms include aggression, destructiveness, deceitfulness, and general rule violations. Often, children with CD experience significant functional impairments in social relationships, academic functioning and familial relationships, and a portion of these children will go on to exhibit delinquent behaviors that will result in significant legal problems (Frick, 2001). Evidence indicates that the co-occurrence of ADHD and CD is particularly potent (Lynam, 1996). Children with co-occurring ADHD and CD have personality traits marked by higher negative emotionality and lower levels of constraint than children with only one of these diagnoses (Cukrowicz, Taylor, Schatschneider, Lacono, 2006). They are more impaired on indices of response modulation, cortical underarousal, and executive functioning (EF) than their non-comorbid counterparts (see Lynam, 1996, for a review). The presence of ADHD also contributes independently to the persistence of CD symptoms to antisocial behavior in adulthood, even after controlling for substance abuse (Gunter, Arndt, Riggins-Caspers, Wenman, Cadoret, 2006).

The majority of recent evidence appears to be consistent with an additive model of the co-occurrence of ADHD and CD (Waschbusch, 2002; Seguin, Nagin, Assaad, & Tremblay, 2004; King et al., 2005), meaning that children with ADHD/CD demonstrate a wider range of deficits on neuropsychological tests, behavioral symptoms at home and school, and social functioning than children with only one diagnosis. This model is such that the deficits are equal to a sum of the deficits associated with each disorder (Waschbush, 2002). Moreover, the additive model

suggests that a child with ADHD and CD will differ from a child with only one of these diagnoses because they demonstrate the associated symptoms, impairments, and endophenotypic characteristics of both of the disorders. In contrast, as synergistic model would find an interaction between deficits associated with each disorder resulting in even greater deficits that one would expect by a simple sum of deficits associated with each disorder. Importantly, while the existing research implicates the additive model, more research is needed to confidently conclude that the co-occurrence of ADHD and CD will result in additive, rather than synergistic effects.

The correlates of the ADHD/CD co-occurrence are important for a number of reasons. Specifically, they may play an important role in a child's inability to desist from behaviors associated with CD. For example, one study found that boys with ADHD-only, CD-only, and ADHD + CD, did not differ in their response to high interpersonal provocation from a peer, however the comorbid ADHD/CD group was more likely than the other two groups to *persist* in responsive aggression over time (Pelham et al., 1996). Furthermore, the same study found that the ADHD/CD group displayed higher rates of reactive aggression when they were only mildly provoked, as compared to the non-comorbid groups.

In addition to the great personal and familial detriment characteristic of children with ADHD and CD, these disorders, particularly when comorbid, are also associated with great cost to society. Incarceration and prevention of future delinquency involves significant monetary resources, and the involvement of delinquent children in the school system can create unsafe and unproductive learning environments for other students (Frick, 2001). Given these societal and personal

costs, considerable time and research has been dedicated to understanding the characteristics of children with comorbid ADHD and CD.

While ADHD is often considered a developmental precursor to conduct problems, CD symptoms in children are multiply determined (Lahey, Waldman, & McBurnett, 1999), making the etiological distinction of children with ADHD and CD more complicated. Researchers and theorists posit specific risk and protective factors as more salient than others; however, a general picture emerges of an inherently vulnerable child coupled with an inauspicious environment. This combination is reciprocal and interactive, such that child and environmental factors may combine to perpetuate a more chronic and severe developmental trajectory of conduct disordered behavior (for review see Lahey, Moffitt, & Caspi, 2003). These inherent child factors may be cognitive or executive function deficits (Nigg & Huang-Pollack, 2003), or negative or reactive temperamental dispositions (Lahey & Waldman, 2003) and often these factors go hand in hand with ADHD. When these child risk factors interact with contextual influences such as poor parenting or low socioeconomic status (Giancola et al., 1998), children may fail to develop adequate self-control or social skills to moderate the effects of the negative environment (Weisz, 2004). This confluence of internal and external child factors may help to explain the high comorbidity rate between ADHD and CD.

Another important contextual determinant that is related to both negative parenting and child CD is maternal depression. Importantly, the lifetime prevalence of maternal depression is around 40% in mothers of children with ADHD (Chronis et al., 2003) and maternal depression symptoms and associated depressive cognitions

have been implicated as a risk factor for later CD symptoms (Babinski, Hartsough, Lambert, 1999; Farrington, Loeber, van Kammen, 1990). Children with ADHD who have depressed mothers therefore, are burdened with a multitude of risk factors for the development of CD and thus typify a high-risk population for the emergence of childhood CD symptoms.

It is also interesting to consider neuropsychological correlates of hyperactivity and aggression in light of the high rates of co-occurrence between ADHD and aggression. Several studies have found that EF deficits are greatest in children with co-occurring ADHD and CD symptoms compared to children with one disorder or the other (Moffitt & Henry, 1989; Moffit, 1990). More specifically, when considered together, both hyperactivity and aggression demonstrate unique and independent relationships to deficits in working memory (Seguin, Nagin, Assaad, & Tremblay, 2004), and hyperactivity appears to have an additive effect on spatial working memory as measured by the Self Ordered Pointing Task (SOP; Petrides & Milner, 1982). Also, Seguin and colleagues (2004) found that these cognitive deficits are additive in children who exhibit both hyperactivity and aggression. Additional research has found additive deficits in measures of intelligence. Specifically, children with comorbid ADHD/CD, and children with ADHD-only had lower performance IQ relative to controls but did not differ from each other, whereas children with ADHD/CD had lower verbal IQ than groups of children with ADHD-only, CD-only, or no diagnosis (Waschbusch, 2002). Since no significant interaction effects were found, the evidence is suggestive of a non-synergistic model of co-occurrence.

Similar effects were found for social cognition. One study found that boys with comorbid ADHD/CD made more aggressive response decisions and encoded fewer social cues than boys with only one diagnosis or the other (Milich & Dodge, 1984). Direct examination of an interaction effect was not examined in this study, however an inspection of the means suggests that the weaknesses found in the comorbid group are a summation of weaknesses brought by each disorder individually (Waschbusch, 2002). Notably, in the Milich and Dodge study (which included ADHD/CD, CD-only, ADHD-only, and control groups), the group with the highest endorsement of a hostile attribution bias was the ADHD-only group. The findings cited here should be replicated, however; and the possibility of a synergistic effect of these disorders on EF abilities should not yet be eliminated.

While it is clear that the comorbid ADHD/CD subgroup exhibits more severe and persistent CD, less is known about the within-group variability in children with this diagnostic co-occurrence. Thus, breaking down further the construct of CD can be useful in deriving more specific subgroups of children with co-occurring ADHD and CD symptoms that may have differing developmental trajectories and patterns of deficits.

Subgroups and Dimensions of CD

Children with CD are a considerably heterogeneous population. The multiple developmental pathways to CD likely play a role in the diverse expression of these behaviors (Frick & Ellis, 1999). There are numerous ways to subtype children with CD, including dichotomies of proactive/reactive aggression (Card & Little, 2006), the presence of callous-unemotional traits (Frick & Ellis, 1999), or

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destructive/nondestructive behavior (Achenbach et al., 1989). The subtypes that exist in the DSM-IV diagnostic categories are based upon whether the onset of CD occurs during childhood or adolescence (APA, 2000), and children with early-onset CD symptoms have been found to be more severe and persistent in their symptomatology (Moffitt, 1993).

One such way of breaking down the diverse construct of CD is through distinguishing between overt and covert behaviors. Among the many ways of subtyping CD, this distinction is supported by decades of factor- analytic, cross-sectional, and longitudinal empirical evidence to support this distinction. Studies designed to assess the structure of CD through factor analyses of large samples of children have supported the distinction of overt and covert dimensions of behavior (Willoughby, Kupersmidt, & Bryant, 2001; Tackett et al., 2003). Furthermore, Achenbach's taxonomic studies of child symptom profiles led to a similar derivation of delinquent/covert CD symptoms (including stealing and running away) and aggressive/overt CD symptoms (including bullying and fighting; Achenbach, 1993), lending further empirical support to the notion that these dimensions should be considered separately. One example of this empirical classification is the Child Behavior Checklist (Achenbach and Rescorla, 2001). Thus, when factor analyzing DSM-IV symptoms of CD, as well as using a taxometric approach to classification of psychopathology, the construct of CD breaks down similarly.

There is also a logical distinction between overt and covert acts. Table 1 lists the distinction between overt and covert CD symptoms based upon several prior factor analyses (Tackett et al., 2003; Janson, Kjelsberg, 2006, Frick et al., 2003,

Tackett et al., 2005, Fergusson et al., 1994). Overt CD symptoms typically involve aggression or confrontation with others (e.g., fighting, bullying), while covert behaviors are purposefully non-confrontational (e.g., lying, stealing without confrontation of a victim). While both of these acts may be purposeful, the executive skills required to be successful in these behaviors will differ (Loeber, 1982). For example, a child who exhibits more covert behaviors may possess better developed social skills, planning abilities, or awareness of the social context that would enable them to conceal their behaviors from authority figures. Finally, the expression of a high degree of both overt and covert CD behavior may place a child at higher risk for impairment from his or her behavior and may also represent a more severe portion of children with CD. Thus, it seems likely that the different expression of overt and covert behaviors may be a telling aspect of a child's traits, background, or expected developmental outcome.

Both overt and covert CD behaviors are related to hyperactivity (Willoughby, Kupersmidt, Bryant, 2001; Clarke et al., 2007a), however the trajectories for these pathways appear to differ (Nagin & Tremblay, 1999). In fact, longitudinal studies examining trajectories of CD symptoms in children provide some of the most convincing support for the distinctiveness of these dimensions. Specifically, early oppositionality was found to be more longitudinally predictive of later covert behavior with aggression and hyperactivity held constant, while early physical aggression was more predictive of later overt behavior when oppositionality and hyperactivity were held constant (Nagin & Tremblay, 1999). Therefore, while overt

and covert behaviors often co-occur, there appear to be unique developmental

implications for the expression of each.

Additional longitudinal research in community samples has shown a general

rise in covert behavior and a concurrent decrease in overt behaviors as children

progress into adolescence (Loeber, 1982). Notably, findings from the Oregon Youth

Study indicate that children engaging in early overt CD behavior (i.e., by age 10) had

the greatest increases in covert behavior in adolescence, and were at greatest risk for

arrest at age 14 (Patterson & Yoerger, 2002). In this group of children, it may be that

overt and covert acts are different developmental manifestations of the same

antisocial propensity. In the same sample, there was a group of children who did not

display early overt behavior, but evidenced covert behavior in adolescence. These

children generally desisted from CD symptoms at the onset of adulthood and may

correspond to the DSM-IV adolescent-onset subgroup. A final group was the overt-

only group that never initiated covert acts, but evidenced overt aggression throughout

development. The latter two groups were at substantially lower risk of arrests,

pointing to the additive effect of both overt and covert behavior in risk for juvenile

delinquency. It should be noted that this particular study did not elucidate the

relationship of ADHD symptomatology to these developmental trajectories.

Interestingly, there is also some evidence to suggest that covert behavior is more

predictive of the persistence of CD and the development of antisocial personality

disorder in adulthood (Lahey et al., 2005). Notably, ADHD was not predictive of the

persistence of CD in this study as has been found in other studies. These findings

should be interpreted with caution, however, as this predictive capacity of covert

symptoms may be due in part to the higher degree of overlap between covert

symptoms and antisocial personality disorder (e.g., lying and manipulative behavior),

as opposed to the little overlap between overt symptoms and antisocial personality

disorder.

One source of the developmental increase in covert behaviors may be that the

increased independence given to adolescents gives rise to increased opportunity for

covert acts. Simultaneously, it may be that the aversive environmental contingencies

that follow easily-observed overt behavior serve to reduce these confrontational acts

in most older adolescents and adults (Snyder et al., 2003). The rapid increase in EF

and the corresponding cognitive capacities for deliberate, goal-directed behavior

aimed at avoiding negative contingencies may interact with these environmental

contingencies to produce a decrease in overt behavior in some children. Most

importantly, overt and covert behaviors may vary in what is considered normative or

aberrant across development (e.g., oppositionality during the "terrible twos" or mild

delinquency during adolescence), thereby adding further to the relevance of

examining these two dimensions of CD behaviors separately. This longitudinal

research provides evidence that the study of overt and covert acts separately may

provide unique information regarding the correlates of CD symptoms across

development and also supports the validity of the distinction of overt and covert

conduct- disordered behavior.

There is also evidence to suggest different etiological factors for overt and

covert behavior, including distinct genetic, biological, and environmental factors. For

example, the link between maternal smoking during pregnancy and CD appears to be

specific to overt child behavior; however, this study did not account for the influence of ADHD (Monuteaux, Blacker, Biederman, Fitzmaurice, Buka, 2006). Additionally, child maltreatment appears to be more strongly associated with overt, rather than covert, CD symptoms (Stouthamer-Loeber et al., 2001), and the lower cortisol levels often evident in individuals with CD have been found to be uniquely associated with overt/aggressive behaviors and not covert behaviors (McBurnett, Lahey, Capasso, & Loeber, 1996); however, neither of these studies accounted for ADHD in their analyses. Finally, studies exploring self-serving cognitive distortions in adolescent CD find fundamental differences in the thinking patterns related to overt and covert behaviors (Liau, Barriga, & Gibbs, 1998). Specifically, cognitive distortions with overt behavior as its referent (i.e., "people need to be roughed up once in a while") was associated with overt CD, while distortions that reference covert behaviors (i.e., "if someone is careless enough to lose a wallet, they deserve to have it stolen") was associated with covert CD symptoms. Taken together, there is substantial evidence from developmental and biological studies that it may be advantageous to consider child overt and covert CD symptoms separately. This distinction may be even more useful in further identifying subgroups of children with co-occurring ADHD and CD that may have different developmental trajectories.

Both overt and covert CD symptoms exist at higher rates in children with ADHD relative to a normal comparison group (Hinshaw et al., 1997). Yet, there appears to be little research directly examining whether overt and covert behaviors are differentially related to ADHD. In general, the evidence suggests that, while ADHD is associated with both overt and covert behaviors, the magnitude of the

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association is somewhat higher with overt than covert behavior. Specifically, a study by Loeber, Keenan, and Zhang (1997) found a greater percentage of first- through seventh-grade boys with ADHD in a group that exhibited overt CD symptoms as compared to the group that evidenced a higher rate of covert CD symptoms, although it was unclear whether this difference was significant. Notably, ADHD was more prevalent in children with *persistent* CD (characterized by either overt and covert), but not with non-persistent CD. This study did not examine directly the rates of ADHD among the different overt and covert pathways. Another study similarly found that there was a unique relationship between both overt and covert behavior and hyperactivity, but the magnitude of the relationship between overt behavior and hyperactivity was stronger than for covert behavior and hyperactivity (Willoughby, Kupersmidt, Bryant, 2001). Furthermore, overt and covert behaviors appear to be primarily related to the hyperactive/impulsive ADHD symptoms, as opposed to inattentive symptoms (Clarke et al., 2007a). This finding is consistent with the generally- accepted view that CD symptoms are more highly related to hyperactivity as opposed to inattention (Eiraldi, Power, & Nezu, 1997; Faraone, Biederman, Weber, & Russel, 1998; Lahey, Schaughency, Hynd, Carlson, & Nieves, 1987). Most notably, <u>there is no known research study that has explored correlates of the overt and covert behavior distinction within a sample of children with ADHD.</u>

Executive Functions

Deficits in EF are a hallmark of ADHD (Barkley, 1997) and there is also evidence to suggest that additional, unique EF deficits are present in CD (Giancola, Mezzich, &

Tarter, 1998; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999). Importantly, the additive model of ADHD and CD also applies well within the domain of neuropsychological deficits (Seguin, Nagin, Assaad, & Tremblay, 2004).

It has been amply noted that the construct of EF is poorly defined and that the conceptualization and measurement of EF varies widely. The term EF subsumes a variety of functions such as planning, inhibition, set shifting, working memory, selective attention, and decision-making. Pennington and Ozonoff (1996) delineate several groups of EF measures based on reviewed literature that may be relevant to this discussion, including set shifting, planning, working memory, contextual memory, inhibition, and fluency (1996). They suggest both a unifying conceptualization of EF, in which all of these abilities represent the same general construct, while at the same time noting the distinctiveness of the multiple domains involved with EF. Correspondingly, the anatomy of the frontal lobe can be considered, as particular regions are associated with different functions. Specifically, the dorsolateral prefrontal cortex is linked closely with planning and inhibition, while the orbital and ventrolateral frontal cortex are associated with response contingency, extinction, and emotion regulation (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006; Blair & Frith, 2000). This distinction of frontal lobe regions supports the notion of multiple domains of EF at the neuroanatomical level.

Pennington and Ozonoff (1996, p. 55) present the idea that EF is the "context-specific action selection in the face of strongly competing, but context-inappropriate, responses." The relevance of this definition is clear when considering CD symptoms, as behavioral manifestations of CD can be considered context-inappropriate

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responses that compete with prosocial behavior. Thus, children with well-developed EF may have a greater ability to choose the prosocial option, despite a strong competing desire to be aggressive or manipulative. Furthermore, the idea of context-specific action selection is especially important when considering differences in overt and covert behaviors. Because covert behaviors are more generally outside of the view of adults, or designed to deceive adults, they imply a better sense of the environment and more careful action selection. Therefore EF deficits may be less related to covert behavior. It is because of this link between context-specific action selection and overt and covert behavior that the overt/covert distinction, above other CD distinctions, provides the most fertile ground for uncovering differences in EF.

Finally, with regard to ADHD, the logical relationship between EF deficits and ADHD behaviors is also evident. For example, a child with ADHD can be impulsive, intrusive or inappropriate in social contexts, indicating an impaired ability to select appropriate behavioral responses in that situation. Furthermore, a child with ADHD may be very distractible during class time, indicating that they are not able to filter out the context-inappropriate responses in order to focus on a lesson. (The evidence base for the association between ADHD and EF will be reviewed later in this section.)

Many factor analytic studies have delineated several basic EF capacities (Hedden & Yoon, 2006; Miyake et al., 2000) that may be relevant to consider with respect to both CD and ADHD, including working memory, inhibition, and set shifting. Before each of these functions is considered with regard to CD symptoms, another careful look should be taken to determine whether these constructs might be

broken down further. For example, working memory is commonly broken down into verbal and spatial components. This is consistent with both cognitive theory regarding working memory (Baddeley & Hitch 1974), and neuroimaging evidence implicating different neural structures in verbal and spatial working memory (Smith, Jonides, & Koeppe, 1996). It is argued here that a more specific delineation of EF will allow a more informative exploration into the relationship between CD and EF in children and may clarify some inconsistencies in the literature.

With this point, a closer examination of inhibition is necessary, in part because of its substantial implications for ADHD and CD, and in part because it has been the topic of careful examination in recent years. These examinations have revealed inhibition to be a complex, multifaceted construct and suggest that simple examinations of inhibition as a unitary construct may have been overextended. Friedman and Miyake (2004) outlined the role of inhibition in a cognitive process model. At the initial stage, relevant information must be selected out from a host of irrelevant information. At this time, resistance to interference of irrelevant information is most important. Once the appropriate information has entered working memory, cognitive inhibition is a principal factor. Here, the active suppression of *mental* contents aids in the successful processing of information. Finally, there is an output stage where behavioral inhibition or control of motor activity is vital for eliciting an appropriate response.

Nigg (2000) also took this multidimensional explanation of inhibition and linked it to roughly corresponding tasks that related to the inhibition component. For example, the Stroop task is a commonly-used measure of inhibition that requires the

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subject to suppress an inconsistent, dominant cue in order to respond appropriately to a non-dominant cue. This task likely pertains most to resistance to interference at the input stage and Nigg termed the type of inhibition required for the Stroop task as "interference control". There are several versions of the stop task and go/no-go tasks that are more representative of behavioral inhibition, or inhibition of a prepotent response at the output stage. Finally, Nigg proposes that directed ignoring tasks are best representative of cognitive inhibition. With regard to psychopathology, the evidence for behavioral inhibition (as opposed to other types of inhibition) in disorders such as ADHD and CD is probably best (Nigg, 2000), however the theoretical link between interference control and the inattention/distractibility component of ADHD is substantial as well, pointing to the need for further exploration of this topic.

Child Disruptive Behavior and Executive Functions

Developmental Considerations

The frontal lobes and EF undergo substantial changes between childhood and adulthood. EF emerges around the first year of life and develops quickly (Zelazo and Muller, 2002). A 7 to 8 month old infant can retain an object in working memory for 2 to 3 seconds, and by 12 months infants can hold an object in working memory during a 10-second delay (Diamond & Doar, 1989; Diamond & Goldman-Rakic, 1989). While EF continues to develop through adolescence, performance on some measures reaches adult levels by age 12. In adolescence, children experience a reduction in gray matter and synaptic pruning and concurrently the myelination of neural pathways is being refined. This process corresponds with the development of

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social cognition and abstract thought that we see in adolescence (Powell & Voeller, 2004) and thus has implications for higher-order cognition, reasoning, and judgment. It is important to remember that the frontal lobes, as well as EF capacities, are undergoing large changes throughout development. It is not clear at this point whether the association between EF and child disruptive behavior problems is due to a developmental *delay* in EF or a life-long *deficit* in EF (Zelazo & Muller, 2002). Importantly, studies of EF in antisocial adults also find deficits in comparison to normative groups, indicating that this association generally persists past childhood and adolescence into adulthood (Morgan & Lilienfeld, 2000).

The relation between the development of EF and disruptive behavior is complicated, especially when considering the neuroanatomical changes occurring in children. For example, consider the relationship between EF and attention-deficit/hyperactivity disorder (ADHD), a relationship that is well accepted among many researchers (Pennington & Ozonoff, 1996; Barkley, 1997). In a study examining developmental change in the relationship between ADHD and EF, Brocki and Bohlin (2006) found interesting age effects for the relation between verbal fluency, a measure of EF, and symptoms of inattention. In this study, inattention was related to verbal fluency more so in older children (ages 10 to 13), while behavioral inhibition was most clearly associated with ADHD symptoms in younger children (ages 6 to10). The authors suggest one possible reason for this is that inhibition may reach near adult levels by the ages of 8-12 years, and thus variability within older children may decrease. Another possibility is that the decrease in hyperactive symptoms that are seen as children age (Barkley, 1998; Biederman et al., 1996;

Faraone, Biederman, Feighner, & Monuteaux, 2000) may contribute to the change in relation between behavioral inhibition and ADHD. This study is important to consider when examining the literature looking at EF relations to CD symptoms, as age differences in samples may be an importance source of the variability in findings that are seen in this literature.

ADHD and EF

There are several theories relating EF deficits to ADHD (Barkley, 1997; Quay, 1997; Nigg, 2001), and empirical evidence examining group differences between ADHD and non-ADHD samples appears to support these theories (Pennington & Ozonoff, 1996; Willcutt, Doyle, Nigg, Faraone, Pennington, 2005). Additional support for implicating brain functions in ADHD comes from neuroimaging evidence finding reduced volume in the corpus callosum, frontal lobes, basal ganglia, and cerebellum, which are structures associated with EF (Swanson & Castellanos, 2002). Perhaps the most extensively studied theory of EF in ADHD is Barkley's theory of behavioral inhibition (1997). Barkley conceptualizes behavioral inhibition as the primary deficit that is involved with the lack of self-control often observed in individuals with ADHD, Combined Type. Quay (1997) also theorizes that behavioral inhibition is the primary deficit involved with ADHD. Quay goes on to explicate the involvement of the behavioral inhibition system, which comprises the connections between the septo-hippopampal regions and the frontal cortex. This system is involved with responding to rewards and punishments, as well as novelty. Quay notes that the biological base of his theory could provide a basis for understanding the genetic transmission of the disorder.

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With regard to the dimensions of ADHD, evidence appears inconclusive as to whether EF deficits are specific to one subtype or another (see Milich, Balentine, & Lynam, 2001 for review). Whereas some evidence suggests that behavioral inhibition deficits are isolated to children with the combined subtype compared to those with the inattentive type (Nigg, Blaskey, Huang-Pollock, Rappely, 2000), more recent evidence has not found support for these group differences on laboratory measures of inhibition (Riccio, Homack, Jarratt, Wolfe, 2006). In terms of other aspects of EF such as planning, working memory, and set shifting, the evidence is also inconsistent. Several studies have found no group differences between ADHD with and without hyperactivity (currently classified as ADHD predominantly inattentive type and ADHD combined type, respectively) on a broad range of EF measures, however these studies suffer from limitations of small sample sizes that may have restricted the ability to detect group differences (Geurts, Verte, Oosterlann, Roeyers, Sergeant, 2005; Riccio, Homack, Jarratt, Wolfe, 2006).

Importantly, Willcutt and colleagues (2005) note that, in order for behavioral inhibition to be a primary deficit in ADHD, the majority of individuals with ADHD should exhibit this deficit and the variability in behavioral inhibition should explain a large portion of the variability in ADHD symptoms. According to the meta-analysis conducted by Willcutt, this is not the case. Small effect sizes and the fact that the same EF deficit (i.e., behavioral inhibition) is not universally deficient in ADHD samples led the authors to conclude that EF deficits are neither necessary nor sufficient to explain the etiology of ADHD. Furthermore, a closer examination of the data reveals greater within-group variability in EF in ADHD samples than control

samples, suggesting that the group differences found may be driven by a subset of children with ADHD (Nigg et al., 2005). Thus, individuals who perform poorly on measures of EF are likely to have ADHD, however only a subset of children with ADHD exhibit clear deficits in EF.

Implications for this variability are significant. More generally, it speaks to the importance of examining within-group differences in a clinical sample, as these differences may be important indicators of differences in etiology or prognosis. Additionally, these within-group differences in ADHD samples may be important in the development of additional disruptive behavior problems or other co-occurring conditions. It may be that individuals with ADHD and EF deficits represent a subtype who have a shared etiology and who may benefit from treatments targeted at executive deficits (Nigg, Willcutt, Doyle, Sonuga-Barke, 2005). Another possibility is that the subgroup of children with ADHD who do indeed evidence deficits in EF may be at particular risk for the development of co-occurring behavior problems. Or more specifically, perhaps children who display deficits on a wider range of EF measures may be at the greatest risk for the development of CD. Indeed, a common finding is that children with co-occurring ADHD and CD have the greatest deficits in neuropsychological indices compared to their non-comorbid counterparts (Moffitt & Henry, 1989; Moffit, 1990).

Also important in this discussion are the specific facets of EF. The diverse expression of EF should be taken into account when examining the relationship between EF and ADHD. A recent meta-analysis provides important clues into the complex relationship between EF and ADHD (Willcutt et al., 2005). Across the 83

studies reviewed, evidence for inhibition deficits in all subtypes of ADHD is strongest relative to other measures of EF, however inhibition has been criticized as a broad and unspecific term that has been measured in a multitude of ways (Nigg, 2000). Deficits have also been found with planning abilities and working memory, although this evidence is less consistent. Finally, Willcutt concludes that set-shifting and Stroop effects are likely not good candidates to be considered as primary EF deficits in ADHD because of the weak effect sizes and very inconsistent evidence for these deficits being involved with ADHD. If this is the case, it is unclear why some studies have found evidence for such deficits in ADHD samples. One possibility is that the CD symptoms that often co-occur with ADHD may be the variable underlying the relationship that is sometimes observed between ADHD and set-shifting and Stroop effects. Indeed, CD is often not accounted for in such studies (Nigg, Hinshaw, Carte, & Treuting, 1998).

It appears that the EF deficits seen in ADHD are indeed specific to ADHD and are not due to comorbid conduct problems (Nigg, Hinshaw, Carte, Treuting, 1998; Speltz, DeKlyen, Calderon, Greenberg, Fisher, 1999; Pennington & Ozonoff, 1996). Given the overlap between ADHD and CD, it has been difficult to separate the distinct relationship between EF and CD. While studies statistically controlling for ADHD have demonstrated a unique relationship of EF to CD symptoms (Toupin, Dery, Pauze, Mercier, Fortin, 2000; Giancola, Mezzich, Tarter, 1998), one study comparing a CD-only group to a control group did not find a unique relationship between EF and CD in children with CD-only (Clark, Prior, & Kinsella, 2000). Until now, researchers have focused on the more general question of whether ADHD and

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disruptive behavior problems (including CD) are related to EF. The question of which aspects of EF might be related to more specific behavioral patterns (e.g. CD subtypes) has yet to be adequately explored.

Several well-designed studies have systematically examined the role of co-occurring disruptive behavior disorders in the relationship between EF and ADHD and have concluded that comorbid disorders do not, in fact, account for this relationship. In a clinical sample of 6-12 year old boys, ADHD was related to poor performance on effortful neuropsychological tasks, including the Porteus Maze Test (Porteus, 1973) and the Rey-Osterrieth complex figure (ROCF; Osterrieth, 1944) in comparison to a control group, and these findings remained when co-occurring CD symptoms were controlled (Nigg, Hinshaw, Carte, Treuting, 1998). In a clinic sample of preschool children with disruptive behavior disorders, the comorbid ODD/ADHD group had lower scores on the Verbal Fluency subtest of the McCarthy Scales of Children's Abilities (MCarthy 1972) and an adapted Motor Planning Task of the Luria-Nebraska Neuropsychological Battery (Golden, 1981) than a group of children with ODD alone (Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999). Therefore the relationship between ADHD and EF has been well established, independent of co-occurring disruptive behavior disorders.

Child CD Symptoms and EF

While the relationship between ADHD and EF is well-established, there is a strong theoretical basis for expecting a relationship between EF and CD. CD symptoms related to rule breaking and aggression have strong origins in impulse control and behavioral regulation that are associated with the functioning of the

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frontal lobes (Davidson, Putnam, & Larson, 2000). There are also social implications of EF abilities in that development of the frontal lobes is also associated with increased social cognition in the form of self-awareness (Ochsner, 2004), theory of mind (Carlson, Moses & Claxton, 2004), and perspective taking (Blakemore & Choudhury, 2006). These social-cognitive abilities are the vehicles by which external contingencies and responses are given meaning to a child and then transformed into a moral code of conduct. How the child encodes social cues and how they control their subsequent behavior in order to abide by internal rules or social norms may be aided by the complex behavioral and cognitive processes involved in EF. Awareness of contingencies, modification of behavior according to contingencies, inhibition of behaviors that are not considered prosocial, and maintaining these "rules" as accessible in working memory are all necessary to facilitate positive social interactions and EF is the foundation of all of these abilities. Furthermore, covert behavior implies an enhanced awareness of contingencies and social cues because of the concealed nature of these acts, and thus may be less closely linked to EF deficits than overt behavior.

The relationship between CD and EF cannot be explored without noting the remarkable confound with ADHD. Since ADHD is highly associated with both CD and EF, it is necessary to consider ADHD in order to establish that a unique relationship between EF and CD exists. Furthermore, it may be the case that EF deficits are more pronounced in co-occurring cases of ADHD and CD. Early studies in this area suffer from the limitation of not considering ADHD, however more recently there have been careful examinations of the EF deficits in children with CD

that consider the contributions of ADHD in the study design and/or analyses. The current review will focus on the latter studies that have considered the role of inattention and hyperactivity in their conclusions. Even more importantly, these findings will be considered in light of the potent expression of co-occurring ADHD and CD.

Several studies found evidence for a unique relationship between EF and CD, with ADHD controlled (Toupin, Dery, Pauze, Mercier, & Fortin, 2000; Seguin, Harden, Tremblay, & Pihl, 1999; Giancola, Mezzich, & Tarter, 1998). Combined, these studies include children and adolescents between the ages of 7-18 and one study examined females only, indicating the potential robustness of this relationship. A study by Giancola and colleagues (1998) compared a large group of adolescent females with CD to a control group. They found lower scores on a factor-analyzed composite measure of EF (including Porteus Maze Test, Vigilance Task, Motor Restraint Task, and Stroop) in the CD group as compared to controls, even after controlling for ADHD symptoms. Importantly, this group of females had elevated rates of substance use that was not accounted for in the analyses and may have accounted for variance in EF task performance.

Another study of adolescent boys recruited from the community found a relationship between history of aggression and working memory, after accounting for ADHD (Seguin, Boulerice, Harden, Tremblay, Pihl, 1999). Unusually however, ADHD was not related to EF in this sample. This could perhaps be due to the procedures employed to diagnose ADHD in the study, which consisted of obtaining child report of ADHD symptoms[1]. Also, the ADHD diagnosis was not made until

adolescence, introducing difficulties discerning the age-of-onset criterion, particularly when obtaining retrospective reports from adolescents about their behavior prior to age 7. Finally, this study only examined overt (i.e., aggressive) behavior, leaving the question of the relationship between covert behavior and working memory (WM) open.

The third study finding a relationship between EF and CD after controlling for ADHD did so in a school-age sample of children (90% male) ages 7-12 (Toupin, Dery, Pauze, Mercier, & Fortin, 2000). Specifically, they found group differences between children with CD and a control group on the Wisconsin Card Sort Task (WCST), the Rey-Osterreith Complex Figure (ROCF), and the Stroop task.

Overall, findings of these studies indicated group differences between children with CD and a control group. Specifically, children with CD evidenced impaired performance on measures of working memory, inhibition, cognitive flexibility, and planning, which were not accounted for by ADHD symptoms.

While the evidence reviewed above can, in one respect, be considered robust and garner confidence in the CD-EF relationship, there are inconsistencies still. For example, some studies found group differences in specific aspects of EF (i.e., planning), while other studies did not examine this variable or pointedly did not find a relationship to CD. Also contributing to the inconsistency are the studies finding no unique relationship between EF on CD after controlling for ADHD (e.g., Clark, Prior, Kinsella, 2000). One study examined adolescent boys and girls using the Six Elements Test, a measure of planning, task-scheduling and performance monitoring (SET; Burgess et al., 1996), and the Hayling Sentence Completion Test (HSCT;

Burgess & Shallice, 1997), a test of one's ability to generate appropriate strategies to fulfill task requirements. Clark and colleagues found EF deficits on both measures to be specific to ADHD, but not CD in a sample with an ADHD-only group, ADHD+ODD/CD group, ODD/CD-only group, and a control group. Specifically, deficits in EF were evident in ADHD-only and ADHD+ODD/CD groups, but not the ODD/CD-only and control groups. Another study comparing adolescents with CD and a control group (statistically controlling for ADHD) found differences only in verbal skills and not EF using the Porteus Maze Test, Stroop, ROCF, Trailmaking test, and WCST (Dery, Toupin, Pauze, Marcier, & Fortin, 1999). So while it seems that there is, in fact, a relationship between CD and EF that is separable from comorbid attention deficits or hyperactivity, the remaining inconsistencies suggest that there is more to the picture that is not yet understood.

One possible explanation is that there are developmental age effects similar to the findings of Brocki and Bohlin (2006) regarding ADHD. In fact, two studies that failed to find relationships with EF to CD focused only on older adolescents (Clark, Prior, Kinsella, 2000; Dery et al., 1999), indicating that the relationship may change according to the child's developmental stage. As reviewed herein, there is variability in the expression of types of CD across development, and rapid changes in EF throughout development. It may be age-related changes related to either of these constructs that account for these discrepancies. Another possible reason for the inconsistencies is that, as described above, the construct of EF has been too broad. A single EF task often solicits more than one cognitive ability and there is also variability in which tasks are used to measure these abilities. Considering the

diversity of processes that are involved in EF, it may be the case that *specific* aspects

of EF are important in what is being explored, while others are not. There may be

some EF components that are driving the relationship with CD, while other EF

components are not related. It may be that through refining *both* the EF and CD

constructs that more reliable relationships will be generated through empirical study.

Considering the inconsistencies in findings of the relationship between EF and CD,

this method may be even more useful in outlining the precise EF deficits that could

contribute to affirmative findings of this relationship, as well as specific EF abilities

that may play a role in the negative findings.

Importantly, few of the studies reviewed herein examined how EF is

associated with comorbid ADHD/CD. Consistent with the idea that specific factors of

EF may be related to the disorders in question, it may be that certain aspects of EF are

related to ADHD and other aspects are related to CD. Considering the additive

conceptualization of the ADHD/CD co-occurrence, in this case there would be a

wider range of EF deficits present in comorbid children. Furthermore, as described

above, overt and covert dimensions of CD may be best examined separately with

regard to the exploration of correlates of CD, and thus may provide more information

into the nature of the relationship between EF and CD symptoms. The overt/covert

distinction also can be linked to the idea of context-specific action selection, in that

these behaviors occur within different contexts by definition, therefore this

delineation of CD symptoms may have differential relationships to EF.

There is some preliminary evidence that can guide hypotheses regarding the

relationship between EF and overt/covert dimensions of CD in children. There are

two studies that have looked at this question in an exploratory manner, and both studies have substantial limitations to their ability to make a conclusive statement about the relationship between EF and overt/covert behavior. The first study, a cross-sectional study by Giancola and colleagues (1998), found that an EF composite score mediated the relationship between aggressive CD symptoms (e.g., initiating fights, using weapons) and difficult temperament after controlling for age, SES, vocabulary abilities, and ADHD, but the same relationship was not found with non-aggressive CD symptoms (e.g., vandalism, stealing). Specifically, based on these findings it appears that EF deficits play an important role in aggression, but EF deficits may not be as closely linked to non-aggressive or covert CD symptoms. Importantly, this was a sample of adolescent females with a diagnosis of CD. Whether this finding would generalize to males is a crucial question, especially in light of large sex differences in the expression of aggressive CD symptoms (Tiet, Wasserman, Loeber, McReynolds, & Miller, 2001). Sex differences in EF are not as evident (Rucklidge & Tannock, 2002), although there is some evidence to suggest that the pattern of EF deficits among children with ADHD may differ according to sex (Nigg, Blaskey, Huang-Pollock, Rappley, 2002).

The other study that examined aggressive (overt) and non-aggressive (covert) CD symptoms separately (Dery, Toupin, Pauze, Marcier, & Fortin, 1999) did not find any relationship between EF and CD generally. Measures of EF were examined individually and included the Porteus Maze Test, Trail Making Test, Stroop, Wisconsin Card Sort Task, and Rey Osterreith Complex Figure Task. This study did not find any differences in this relationship between aggressive and non-aggressive

CD symptoms. Issues regarding this study include a small sample size that may not have been powered to examine group differences and, while these findings did account for ADHD within the sample, an ADHD-only group was not examined. Also, this sample included older adolescents, introducing possible additional variability related to substance-use that would preclude these findings from generalizing to younger samples.

An important consideration for CD is that the evidence appears to be more consistent for EF differences in younger children with CD than for adolescents (Barkley, 1992; Clark, Prior, Kinsella, 2000; Dery, Toupin, Pauze, Marcier, & Fortin, 1999; Toupin, Dery, Pauze, Mercier, & Fortin, 2000; Seguin, Harden, Tremblay, & Pihl, 1999; Giancola, Mezzich, & Tarter, 1998). One explanation for this is that there may be a delay in EF development in children with CD whereby younger children with CD are slower to develop these cognitive abilities, while older adolescents with CD may eventually "catch-up" to their peers. Another explanation for this with regard to CD symptoms is that childhood-onset CD is considered more severe than adolescent-onset (Moffit, 1993). Therefore samples of adolescents with CD likely include less severe subtypes that have a later onset and higher likelihood of desistence, whereas child samples will necessarily include only early-onset CD cases. Additionally, children with early-onset CD symptoms are more likely to have co-occurring ADHD, making them more likely to persist and demonstrate more severe CD. Yet another important consideration with adolescent samples is that CD is often comorbid with substance use and substance use might lead to EF deficits (Hanson & Luciana, 2004; Drafters, 2006; Verdejo-García, López-Torrecillas, Aguilar de Arcos,

Perez-Garcia, 2005). It is also possible that peer influences, as opposed to child factors, in the expression of CD may be more prominent as a child moves into adolescence, introducing additional variability into the causes of CD in older samples. Finally, there is also the developmental trend for covert behavior to increase in adolescence while overt behavior decreases (Loeber, 1982), leading to possible differences in prevalence rates of these behaviors for older and younger samples.

Therefore, existing research does not conclusively answer the question of whether EF is differentially related to overt and covert dimensions of CD. Furthermore, it may be beneficial to begin a coherent examination of this question in younger samples to bypass the above-stated issues involved with utilizing adolescent samples to explore the relationship between CD and EF.

Certainly, it is a worthwhile endeavor to explore possible neuropsychological correlates of ADHD and CD. There are clear parallels between the nature of EF deficits and inattentive, hyperactive, and disruptive behavior. Children with ADHD and disruptive behavior disorders often lack the ability to engage in goal-oriented behavior to the degree that their peers can (e.g., focusing on a lecture in school), or they may be less able to select the prosocial solution to a conflict on the playground. These behaviors typify children with ADHD and disruptive behavior patterns (i.e., CD). Moreover, goal-oriented behaviors solicit EF capacities, and it may indeed require adequately-developed EF in order to remedy disruptive behavior patterns. When viewed in this light, an understanding of EF deficits in this population may enhance the development of treatments for these children.

Bringing all of the reviewed evidence together, several points become apparent. First, the evidence for EF deficits in children with ADHD is robust, however this is more so true with regard to behavioral inhibition than other domains of EF, most notably shifting and interference control. In reviewing the research regarding co-occurring ADHD and CD, there seems to be an additive effect of these disorders, particularly with neuropsychological measures however, more evidence is needed for a confirmatory conclusion that the EF deficits in children with ADHD/CD are indeed additive and not synergistic. There is also a compelling evidence for a unique relationship between CD and EF independent of ADHD, although this is not fully established. One potential reason for this inconsistency is the diversity of behaviors included in the diagnosis of CD. The unique expression of overt and covert CD symptoms may provide additional information as to the nature of neuropsychological deficits in children who exhibit CD. However, few studies have examined this question, and those that have possessed substantial limitations. Taken together, the reviewed literature points to the need for clarification of both the additive nature of EF deficits in comorbid ADHD/CD, and the unique relationship between separate overt and covert CD symptoms and EF.

The Present Study

The present study took a more in-depth look at the relationship between ADHD, EF and CD symptoms through the exploration of more refined constructs. Specifically, this study adds to existing literature which examines associations between EF and overall CD symptoms. Additionally, this study was the first to test whether overt and covert dimensions of CD are differentially associated with

neuropsychological correlates in a sample of children with ADHD. Overt and covert

CD symptoms were measured in two ways: dividing DSM-IV CD symptoms into

overt and covert dimensions based on factor analytic studies, and using the Child

Behavior Checklist (CBCL) dimensions of aggression and delinquency as a proxy for

overt and covert behavior, respectively. The use of both DSM-IV symptom scales and

the empirically-derived scales of the CBCL satisfies arguments for both a categorical

and dimensional classification of psychopathology (Sonuga-Barke, 1998). We

utilized a sample of school-age children (age 6-14) who have ADHD and mothers

with elevated levels of depression. As reviewed herein, these children are at

heightened risk for the development of early conduct problems by virtue of both their

ADHD (Babinski, Hartsough, Lambert, 1999; Farrington, Loeber, van Kammen,

1990) and family adversity in the form of maternal depression (Chronis et al., 2007).

Preliminary Aim

As a preliminary aim, the relationship between overall CD symptoms and EF

was explored both categorically and continuously. Both an additive and synergistic

(i.e., interactive) model of the EF deficits in these disorders was considered.

Hypothesis 1. Whether the relationship between ADHD and CD is additive or

synergistic is not completely clear. It is possible that the severe and persistent

antisocial trajectory evident in children with co-occurring ADHD and CD may be a

result of a synergistic interaction between the endophenotypic characteristics of these

disorders. This synergistic relationship would be demonstrated empirically through a

significant interaction between ADHD and CD in predicting EF deficits. On the other

hand, consistent with the additive model of a broader range of neuropsychological

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deficits in children with co-occurring ADHD and CD, the deficits in shifting and interference control that are sometimes evidenced in ADHD populations may be a consequence of co-occurring CD symptoms often present in children with ADHD (Willcutt et al., 2005). This type of effect would be demonstrated by an individual contribution of differing EF deficits from both ADHD and CD in the form of two main effects. Therefore it was necessary to examine both additive and interactive models of the co-occurrence of ADHD and CD. Recent analyses of the question of whether the EF deficits associated with the ADHD/CD comorbidity is additive or synergistic support the additive model (Willcutt et al., 2005; Seguin et al., 2004). **Therefore, it was hypothesized that, CD symptoms would be negatively related to set shifting, interference control, working memory, and behavioral inhibition. The interaction between ADHD and CD was also tested and it was hypothesized that, consistent with the additive model, this interaction would not be significant, ruling out a synergistic effect.**

Hypothesis 2. By the same rationale outlined for hypothesis 1, it is likely that children with ADHD/CD would display a wider range of neuropsychological deficits, consistent with the additive model. **It was hypothesized that, in comparison to children with ADHD-only, children with co-occurring diagnoses of ADHD and CD would have deficits in set-shifting, interference control, working memory, and behavioral inhibition.**

Primary Aim

The primary aim was to determine the unique relationship between both covert and overt CD symptoms to the functions of behavioral inhibition, working

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memory, set shifting, and interference control within a sample of children with ADHD.

Hypothesis 1. The volatile and spontaneous nature of overt CD symptoms may have different EF correlates than the more purposeful or deliberate, covert CD symptoms. When considering the deliberate nature of covert behavior, it is possible to hypothesize that inhibition may be better developed in children that display more covert acts; however, upon consideration of the nature of covert CD symptoms, this does not appear to be the case. Specifically, covert acts such as lying and stealing often occur spontaneously as well (e.g., a child takes money that is lying around the house without thinking about the consequences), suggesting that deficits in inhibition are likely at play in covert behavior as well. In addition, both types of CD symptoms are related to hyperactivity and impulsivity (Willoughby, Kupersmidt, Bryant, 2001; Clarke et al., 2007a), and therefore both types of CD symptoms would be related to behavioral inhibition. Due to this relationship between ADHD symptoms and behavioral inhibition (Willoughby, Kupersmidt, Bryant, 2001; Clarke et al., 2007a), ADHD symptoms were expected to be related to Stop-Signal Task performance and thus were controlled for in analyses. **Therefore, it was hypothesized that, after controlling for ADHD symptoms, behavioral inhibition, as measured by the Stop-Signal Task would be negatively related to both overt and covert behavior.**

Hypothesis 2. Working memory enables an individual to update their representations in short-term memory to accommodate changing task demands (Hedden & Yoon, 2006). Working memory is also inconsistently related to ADHD (Wilcutt et al., 2005), suggesting that variability in working memory within the

diagnosis of ADHD may play a role in the expression of co-occurring behavior problems. One could hypothesize that children who engage in covert CD symptoms have better-developed working memory abilities that enable them to navigate the authority figures in their lives and commit antisocial acts when adults are not nearby. On the other hand, the executive component of working memory can be said to exert attentional control over a wide range of behaviors (Engle, 2002), which would suggest that working memory may be related to all types of CD symptoms. Furthermore, working memory capacity predicts a range of other cognitive processes, including memory and interference control, which may play a role in the expression of CD (Kane & Engle, 2003; Kane & Engle, 2000). Considering the overarching importance of working memory capacity in a wide range of cognitive and behavioral phenomena, it is unlikely that this factor would discriminate between covert and overt CD symptoms. **Therefore, it was hypothesized that verbal and spatial working memory, as measured by the Digit Span subtest of the WISC and the SOP, respectively, would be negatively related to both overt and covert CD symptoms.**

Hypothesis 3. Participants' performance on measures of shifting may be differentially related to overt and covert CD behavior. Shifting involves the coordination of multiple task demands, each with its own set of goals (Monsell, 2003). Considering that covert behavior is selective, in that it is engaged in during non-confrontational situations, it implicates a greater ability to modify goals according to the situational context. Children exhibiting overt behavior, on the other hand, may have deficits in shifting due to their presumed lack of ability to utilize contextual demands in determining acceptable behaviors and evaluating possible

consequences. Shifting is a construct that is very inconsistently related to ADHD (Willcutt et al., 2005). However, it may be that those children with ADHD that do have deficits in set-shifting may be those who have a propensity toward aggression as well. **Therefore, it was hypothesized that shifting, as measured by the WCST would be negatively related to overt CD symptoms, but not covert CD symptoms, after controlling for ADHD symptoms.**

Hypothesis 4. Interference control may have a unique relationship with type of CD symptom (i.e., overt or covert). Interference control pertains to the input stage of cognitive processing and refers to the ability to suppress a stimulus that calls for a competing response so that one can carry out a primary response (Nigg, 2000). Interference control has been only weakly associated with ADHD symptoms (Willcutt et al., 2005). Covert CD symptoms have been notoriously difficult to measure because it is, in fact covert and children intentionally engage in this behavior when adults are not nearby (Hinshaw, Simmel, & Heller, 1995; Willoughby, Kupersmidt, & Bryant, 2001). This indicates that these children, despite having a propensity toward CD behaviors, are somehow able to suppress stimuli that would elicit more overt behavior thereby implicating greater ability to resist interference. On the other hand, children who engage in overt behavior commit more observable antisocial acts, indicating that the primary response is exhibited despite stimuli (i.e., adults or authority figures) calling for a competing response. **Therefore, it was hypothesized that interference control, as measured by the Stroop task, would be negatively related to overt behavior only (i.e., and not covert behavior) after controlling for ADHD symptoms.**

Method

Participants

Participants included 49 6-14 year-old children who are at heightened risk for conduct problems. All participants were part of a larger NIH-funded treatment development study of an integrated treatment for depressed mothers of children with ADHD. Recruitment took place through mailings to local pediatricians, mental health providers, community organizations, summer camps, and public and private schools. This population was defined as "at-risk for CD" based on the presence of two documented risk factors: a DSM-IV diagnosis of ADHD in the child (Babinski, Hartsough, Lambert, 1999; Farrington, Loeber, van Kammen, 1990) and the presence of elevated levels of depression in the mother (Chronis et al., 2007). Thus, inclusion criteria for the child involved the presence of a DSM-IV diagnosis of ADHD. Children who had an IQ below 70, or who had been diagnosed with a pervasive developmental disorder were excluded. Elevated levels of depression in the mother were determined by two administrations of the Beck Depression Inventory- II (BDI-II; Beck, Steer, & Brown, 1996) with a score of 10 or above. In addition, due to the aims of the larger study, mothers were excluded if they met criteria for current substance abuse, psychosis, or bipolar disorder.

Measures

Child symptoms and behavior

Schedule for Affective Disorders for School-Aged Children- Present and Lifetime Version. The Schedule for Affective Disorders for School-Aged Children-

Present and Lifetime Version (K-SADS-PL; Kaufman, Birmaher, Brent, Rao &Ryan, 1996) is a semi-structured clinical interview assessing DSM-IV symptoms of Separation Anxiety Disorder, ODD, CD, ADHD, Post-Traumatic Stress Disorder, Major Depression, Panic Disorder, Obsessive Compulsive Disorder, and Generalized Anxiety Disorder in children. Each symptom was rated on a three-point scale, ranging from 1 (not present) to 3 (threshold: definitely present), and symptoms were counted as clinically significant on this measure if the clinician rated it as a three.

Disruptive Behavior Disorders Rating Scale. The Disruptive Behavior Disorder (DBD) symptom checklist (Pelham et al., 1992) was completed by the child's mother as well as the child's teacher. The DBD includes all DSM-IV symptoms of ADHD, ODD, and CD. Symptoms endorsed as occurring "pretty much" or "very much" in either the school setting or at home were considered clinically significant and were included in the symptom count. In prior studies, the DBD has demonstrated internal consistency of .96, .96, and .81 for the ADHD, ODD, and CD subscales respectively (Pelham et al., 1992).

Children's Impairment Rating Scale. To determine the nature of the child's impairment, the Children's Impairment Rating Scale (CIRS; Fabiano et al., 2006) was completed by the child's mother and the child's teacher. The CIRS contains ratings of impairment on a 7-point scale across multiple domains including peers, parents, siblings, academic, self-esteem, family, teacher, classroom, and overall. One-year temporal stability of this measure ranges from .54 - .76 for the parent measure and .40 - .67 for the teacher measure. Cross-informant reliability from parent and teacher is

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.64, which is typical in the literature considering the differing contextual demands of the home and school environment (De Los Reyes & Kazdin, 2005).

Child Behavior Checklist. The Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001) is a self-administered questionnaire designed to assess a wide range of child internalizing and externalizing problems in children, as rated by parents. DSM-IV is often criticized for a lack of sensitivity to the differing behavioral manifestations of disorders across development; however, the CBCL empirically-derived, age- and gender-normed scales address many of these concerns. The CBCL was chosen due to the empirically-derived narrowband dimensions of Aggression and Rule-Breaking that are both age and gender normed. These dimensions map on well to the conceptualizations of overt and covert CD symptoms and may therefore be examined as alternative measures of child overt and covert behavior.. The test-retest item reliabilities for the broadband internalizing, externalizing, and total problems scales are .91, .92, and .94, respectively. The internal consistency of these measures is also relatively high, with alpha coefficients of .90, .94, and .97, for the internalizing, externalizing, and total problems scales, respectively.

Behavioral Inhibition

Stop-Signal Task. This is a computerized version of the stop signal task (SST; Logan, 1994; Logan, Schachar, Tannock, 1997) that is designed to measure a child's inhibitory control. On primary trials, participants were presented with either an X or an O on a computer screen and they are instructed to press the key on the keyboard that corresponds with the stimulus. Stop-signal trials consisted of the computer presenting either and X or and O on the screen and p articipants were instructed to

only press the corresponding X or O key on the keyboard if they did not hear an auditory tone. Inhibition was computed by subtracting the average stop-signal delay at which the child was able to inhibit their response 50% of the time. Thus, higher scores would be indicative of greater deficits in inhibition. Split-half reliability for this task has been found to be as high as .945 in college samples (Logan, Schachar, Tannock, 1997) and in this sample it was estimated at .745, perhaps due to the age difference in the sample. The stop-signal task has been used frequently in studies of children with behavior problems (Oosterlaan, Logan, Sergeant, 1998) and deficits in stop-signal task performance have been found in children with ADHD (Nigg, Blaskey, Huang-Pollock, Rappeley, 2002) and CD (Oosterlaan, Logan, Sergeant, 1998).

Interference control

Stroop test. The Stroop effect is a widely-studied measure of an individual's ability to suppress a dominant cue (reading a written color word) in order to accomplish the task of naming the ink color that the word is printed in. For example, subjects with deficits in interference control typically take longer to name the ink color of a word that spells a different color (i.e. saying the word "blue" when presented with the word "yellow" printed in blue), than to name the color ink when a neutral word is printed in the same color (e.g., cat). Presumably the word is processed faster than the color is (Nigg, 2000) and therefore would be representative of a dominant cue. A participant's ability to respond to the non-dominant cue of color would be indicative of his/her ability to resist interference. The stroop color-word score (Stroop-CW) is an age-normed score of the total number of items completed

when the color name was printed in a different color ink. Raw scores are response time to name words or read colors, and these were converted into age-normed t-scores for analysis. Reliability coefficients for Stroop-like tasks in children range from .82 to .93 (Archibald & Kerns, 1999). Split-half reliability in this sample was .861. Evidence for deficits in Stroop effects have been found in children with CD (Toupin, Dery, Pauze, Mercier, & Fortin, 2000; Giancola, Mezzich, & Tarter, 1998) and ADHD (Nigg, Blaskley, Huang-Pollock, Rappeley, 2002).

Shifting

Wisconsin Card Sort Task. The Wisconsin Card Sort Task (WCST; Heaton, 1983) is a measure of abstract reasoning and the ability to maintain and shift a cognitive set (i.e., flexibility). In the computerized version used in this study, children were asked to sort through a deck of cards that have different shapes and colors according to an unstated rule set (e.g., all cards that have red on them are to be placed in the same pile), and they received feedback as to whether they sorted them according to the proper criterion. After the participants had sorted several cards correctly (indicating that they are aware of the criterion), the criterion changed (e.g., all cards that have the same shape are to be placed in the same pile). The number of perseverative errors (i.e., continuing to respond with the prior criterion when a new criterion is in effect) on this task is an indicator of the set shifting ability that the measure taps. This score was transformed into a T-score that was age-normed for analysis. Intraclass correlation coefficients for this measure ranged from .828 to 1.000. In this sample, split-half reliability was .876. This task was selected because it is one of the most commonly-used indicators of EF (Pennington & Ozonoff, 1996)

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and has been shown to discriminate between control groups and groups of children with ADHD (Shue & Douglas, 1992) and CD (Brewer et al., 2001) individually.

Working Memory

Digit Span Subtest of the WISC-IV. The digit span subtest (Wechsler, 2003) is commonly considered a verbal working memory task or a short-term memory task. During the administration of the digits forward portion of the test, the experimenter read the child a list of 2-9 numerical digits and the child was instructed to repeat the digits back in the same order. During the digits backward portion of the task, the child was instructed to repeat the digits in reverse of how the experimenter stated them. This digits backward portion requires the child to retain the digits in memory while simultaneously rearranging their order so that they may be repeated back. For the purposes of this study, the digits backward score (DSB) was considered representative of verbal working memory. This score was converted into a standardized score based upon the child's age. Digit span split-half reliability coefficients range from .79 to .87 for the age ranges in this study and test-retest reliability ranges from .67 to .75. In this sample, split-half reliability for this measure was .828. This task has been shown to discriminate between children with ADHD and controls (Martinussen & Tannock, 2006; Thorrell & Whalstedt, 2006), and has been shown to be related to both aggression and hyperactivity (Seguin et al., 2004).

Self-Ordered Pointing Task. The Self-Ordered Pointing task (SOP; Petrides & Milner, 1982) is commonly considered a spatial working memory task which has been linked to frontal lobe activity both theoretically and empirically (Pennington & Ozonoff, 1996). Archibald and Kerns (1999) adapted this task for children, and this

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adapted version was used in this study. Participants were presented with a binder in which each page had a set of drawings of common object representations. They first viewed a set of 6 pictures on a page and they were instructed to select one picture. The next five pages contained the same 6 images, but in different order and the child pointed to a different image on each page. Thus, children were instructed to point to each of the 6 images only once, and were not allowed to point to the same spatial position on each page. This process was administered 3 times. Once completed, the same process was repeated, but with 8 images per page, then 10 images per page, and finally, 12 images per page. All errors, where the child points to the same object in the same trial, were recorded and used as the dependent variable. Test-retest reliability for the SOP is .76, and it correlates with other measures of working memory (Archibald & Kerns, 1999). In this sample, split-half reliability of this measure was .79. The SOP has been utilized in several studies of both normative and clinical samples of children ages seven to adolescence (Archibald & Kerns, 1999; Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999) and both hyperactivity and aggression have been related to SOP performance (Seguin, Nagin, Assaad, & Tremblay, 2004).

Child IQ

 Vocabulary and Block Design Subtests of the WISC-IV. The vocabulary and block design subtests of the WISC-IV (Wechsler, 2003) represented an estimated IQ score. This method of estimating IQ has been used to control for IQ in several studies reviewed in the introduction (e.g., Seguin, Nagin, Assaad, & Tremblay, 2004), and has been shown to be distributed in the same way as full scale IQ estimates in

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samples of children with ADHD (Kaplan, Crawford, Dewey, & Fisher, 2000).

Furthermore, this method of estimating IQ was determined to be the most appropriate

in a clinical sample of children, out of seven commonly-used IQ estimation

procedures, with a correlation of .92 with full scale IQ (Campbell, 1998). The scaled

scores of both of these subtests were averaged to create the total estimated IQ.

Administration of the vocabulary subtest involved having the tester read words aloud

to the child while also pointing to written version of the same word. The child was

then asked what the word means. Reliability for this scale ranges from .79 to .89 for

children ages 6 to 12. The block design subtest involved showing the child a picture

of a block arrangement and asking them to replicate it within a specific time frame.

Reliability for this subtest ranges from .77 to .89 for children ages 6 – 12.

Maternal Depression

    BDI-II. The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) is a 21-

item self-report instrument that assesses depressive symptomatology continuously in

the mothers of children in the present study. A total score was obtained by summing

over items, with greater scores indicating a greater degree of depression.

Psychometric data indicate coefficient alphas of .92 for an outpatient sample and .93

for a college sample, demonstrating high reliability (Beck et al., 1996) and test-retest

reliability as high as .96 (Sprinkle, Lurie, Insko, Atkinson, Jones, Logan, Bissada,

2002).

Procedure

      Interested participants were screened over the telephone to determine preliminary eligibility for the larger study. Screening included the first administration of the BDI-II and questions pertaining to the child's diagnosis and behavior. Families who remained eligible after the phone screen were invited to come in for an assessment. Participants were then be mailed all forms that the mother was required to complete (i.e., DBD, CIRS) and brought the completed forms to the assessment session. At the assessment, participants completed a separate consent form to consent to the procedures involved with the present study. Teachers were also mailed the DBD rating scale and the CIRS to assess for symptoms and impairment in the academic environment.

      Mothers were interviewed about their child's behavior using the K-SADS. Clinicians were doctoral students and post-doctoral fellows in the clinical psychology program at the University of Maryland, College Park. They were closely supervised by a licensed, doctoral-level clinical psychologist. Children ages 10 and older were also interviewed with specific sections of the K-SADS (mood, anxiety) and this information was combined with maternal report of symptoms using the "or rule." Any discrepancies between mother and child report were reconciled through discussion with both the mother and child, and a consensus rating was made by the interviewer. Furthermore, all children in the study were interviewed individually with the conduct disorder section of the K-SADS. Their reports of CD symptoms were not subject to reconciliation with the parent report in order to obtain the most truthful reports from the children. However, children were informed that any information that they

provided that indicated that they may be putting themselves or others in harm was considered an exception to confidentiality and may be addressed with their parents. Evidence indicates that children as young as five years of age can report through structured interview on their CD symptoms reliably (Arseneault, Kim-Cohen, Taylor, Caspi, Moffitt, 2005). Specifically, children's report correlates with known correlates of CD symptoms such as IQ, hyperactivity, and family variables, and children's report is also predictive of a research diagnosis of CD made though reports of alternative informants . Child symptoms of ADHD, ODD, and CD were calculated by counting a symptom as clinically significant if it was endorsed on either the parent DBD, teacher DBD, parent K-SADS interview, or child K-SADS interview (Piacentini et al., 1992).

To determine which CD symptoms would be included in the overt and covert dimensions, a literature review was conducted to find factor analytic studies of DSM-III or DSM-IV CD symptoms that attempted to determine an underlying factor structure of CD symptoms. Five studies meeting such criteria were found and are summarized in Table 1. Some inconsistencies were found regarding which factor specific symptoms loaded on, which may be attributable to the variability in sample age, gender, and referral source. For the purposes of this study, a symptom was included in either the overt or covert dimension if: 1) at least 3 of the 5 studies found definitive support for one dimension or the other and 2) no study found definitive results to contradict the results of criterion number 1. The final overt and covert dimensions used in this study are presented in Table 2.

When analyses were conducted using the CBCL, overt and covert behavior were defined as the empirically-derived, age-normed aggression and rule-breaking subscales. These subscales are highly related to the overt and covert dimensions of behavior, although there are some variations between the scales (Tacket et al., 2003). For example, there is no direct equivalent on either the aggression or rule-breaking subscales for the symptoms of using a weapon, being physically cruel to people, staying out at night, breaking into a house, car, or building, and forcing someone into sexual activity.

Whenever possible, during the initial assessment for the larger study, children also completed the neuropsychological battery. However, treatment with stimulant medications can affect performance of EF tasks involving focus and attention (Arsten, 2006); therefore, children who attended the initial assessment while actively medicated were scheduled for a second neuropsychological assessment session during which they were not actively being treated with stimulant medication (i.e., during the weekend or times when the child was not normally medicated). Neuropsychological tests were administered by the author, as well as by trained, closely-supervised advanced undergraduate research assistants. The administration of neuropsychological measures by trained graduate and undergraduate students is a practice that has been implemented elsewhere (Nigg et al., 1999; Toupin et al., 2000).

Experimental Design Considerations

The sample of 6-14 year old high-risk children with ADHD was selected for several reasons. First, it was important to include an age range that was not too large, as age-related increases in EF are an important factor to consider (Brocki & Bohlin,

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2004; 2006). On the other hand, due to the large increases in EF during this age range, age was accounted for in the analyses through both the use of age-based norms in scoring some of the EF measures and statistical control of the child age variable when EF scores were not age-normed. Additionally, it was important to allow for the detection of a relationship between EF and CD symptoms prior to the possible initiation of substance use, as substance use has been shown to impact EF (Hanson & Luciana, 2004; Drafters, 2006; Verdejo-García, López-Torrecillas, Aguilar de Arcos, Perez-Garcia, 2005) and is more common among youth with ADHD and CD (Elkins, McGue, Iacono, 2007). According to the American Academy of Child and Adolescent Psychiatry, the average age for the onset of marijuana use is 14 years old, and onset of earliest alcohol use begins around age 12 (2004), so inclusion of children ages 6-14 allows for an inquiry into the CD-EF relationship with minimal risk of substance use as a confounding factor. Indeed, findings from one study suggesting a unique relationship between EF and CD symptoms in an adolescent sample were qualified by the authors, noting that it cannot be determined that the relationship between EF and CD is not due to the high rate of substance use in that particular sample (Giancola et al., 1998). In addition, the age range within the current study allowed for exploration of the CD-EF relationship before peer influences become a large part of the emergence of CD symptoms (i.e., most of the children necessarily have childhood-onset CD symptoms). Finally, the utilization of a sample of children at high-risk for CD by virtue of both being diagnosed with ADHD and having mothers with depression symptoms allowed us to look continuously at children with a

wide range of CD symptoms. Thus, we were able to explore important within-group variability in children with ADHD and CD.

As noted above, children with ADHD and early emerging CD symptoms are an important subgroup of the population at heightened risk for more severe and persistent CD. Children with both ADHD and CD are more likely to have childhood-onset CD symptoms (Speltz, DeKlyen, Calderon, Greenberg & Fisher, 1999). Specific to neuropsychological correlates, several studies have found that children with comorbid ADHD and CD have the greatest deficits in EF (Moffitt & Henry, 1989; Moffit, 1990) and that these deficits appear to be additive (Seguin, Nagin, Assaad, & Tremblay, 2004). Therefore EF deficits are likely to be more strongly associated with early appearing, severe and persistent CD symptoms than with CD symptoms that are more likely to desist. Implications for this are important as there may be a relationship between EF deficits and the inability to recover from a pattern of CD behavior. Indeed, evidence indicates that adult psychopaths are four times more likely to have a history of ADHD ymptoms and conduct problems in childhood (Johansson, Kerr, Andershed, 2005), supporting the notion that this comorbidity is often associated with more severe and persistent CD. Considering the substantial societal cost of long-term, severe criminal behavior, developing a more refined understanding of early expressions of an antisocial propensity may help in prevention efforts. The use of a sample of children with ADHD allowed for statistical control of ADHD severity to isolate the unique EF deficits related to CD, while also enabling the exploration of children with co-occurring ADHD and CD.

Another important design consideration is the timing of the administration of the neuropsychological battery to the child with regard to the mother's participation in the treatment group. Every effort was made to administer the neuropsychological battery prior to the initiation of behavioral parent training with the mother, however due to time constraints, some children were not administered the tests during the initial assessment. However, it is not expected that children's performance on EF measures would change as a result of their mothers' participation in a parent training group. Indeed, prior studies examining the relationship between child CD and EF included children in various stages of treatment, and this did not inhibit their ability to uncover meaningful relationships (e.g., Toupin, Dery, Pauze, Mercier, & Fortin, 2000). Furthermore, measures of child behavior (parent-reported KSADS, DBD, CBCL) were collected prior to treatment to ensure that any treatment-related changes in child behavior were not introduced into the analyses.

Finally, a decision was made to use the overt/covert CD symptom distinction as opposed to alternative distinctions of CD. This classification was chosen primarily due to its relevance in examining the executive functions explored in this study. The Pennington and Ozonoff definition of EF involves the ability for context-specific action selection in spite of strongly competing alternatives (1996). Considering EF in this manner, overt behaviors are not context-specific, and are, in effect, an expression of strongly competing but context-inappropriate responses. For example, aggressive acts are sanctioned within society under only specific circumstances (e.g., certain athletic activities). Thereby, should aggression occur within contexts that are not designated as accepting of aggression or overt acts (e.g., the school playground), it

implies a deficiency in EF. However, covert acts occur specifically outside of the presence of authority figures and are designed to avoid negative consequences. This awareness of contextual surroundings and the forethought involved in these behaviors seems to imply better developed EF in children displaying covert CD behaviors. Therefore the overt/covert distinction of CD lends itself well to the study of EF characteristics of children. In addition to this theoretical reason for selecting the overt/covert distinction, as reviewed in the introduction, there is a convincing body of evidence for these dimensions. Numerous factor analyses, longitudinal developmental investigations, and etiological studies have replicated this distinction. Since the overt/covert breakdown of CD symptoms maps on to the aggressive/delinquent subscales of the CBCL (Tackett et al., 2003), it reflects a synthesis of the DSM-IV theoretical approach to conceptualizing CD, and the data-driven factor analytical work of Achenbach and colleagues. In sum, considering the substantial evidence accumulated over a wide range of studies, overt and covert behaviors can be considered distinct, and are indeed relevant to the constructs of interest in the present study.

Finally, as a compliment to the examination of the overt and covert dimensions of DSM-IV symptoms, the CBCL was used as an alternative conceptualization of these dimensions. DSM-IV criteria include several CD symptoms that may not be considered developmentally appropriate for younger children (i.e., forcing someone into sexual activity, running away from home overnight). In fact, these symptoms have been excluded from analyses in many studies due to low base rates in younger samples (Tackett et al., 2003). The CBCL is

a taxonomic scale that contains age and gender norms for the aggression and rule-breaking scales. Since these scales map on well to the overt and covert dimensions of primary interest in this study (Tackett et al., 2003), they were included as a more developmentally-sensitive index of CD-like behaviors in younger children.

Analytic Strategy

As noted above, all child behavior variables, including ADHD and CD symptoms (both covert and overt), were computed using the "or rule." Thus, these predictor variables represented the synthesis of information across all informants (mother, teacher, and child).

Child estimated IQ, child sex, age, and SES were entered as predictors of each measure of EF and variables that were significantly predictive of EF at $p < .05$ were included in subsequent analyses as covariates. With regard to child age, it was only considered as a potential control variable for EF measures that were not already age-normed.

Preliminary Aim. The preliminary aim determined whether there was an additive or synergistic/interactive effect of ADHD and CD on neuropsychological deficits in children with ADHD. MANOVA analyses were conducted to determine whether a broader range of differences exists in children with co-occurring categorical diagnoses of ADHD and CD (*n*=16), in comparison to children with ADHD-only (*n*=33). Due to the small group sizes that were present for these analyses, these results were considered exploratory. The potentially synergistic effect of comorbid ADHD/CD was evaluated by looking at the ADHD x CD interaction with regression analyses. Overall CD symptoms were also examined continuously to

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explore the relationship between the number of CD symptoms and the domains of EF, with the continuous number of ADHD symptoms controlled.

Primary Aim. The primary aim was to determine the relationship between overt and covert child CD symptoms and behavioral inhibition (measured by the Stop-Signal Task), verbal working memory (measured by the digit span subtest of the WISC-IV), spatial working memory (measured by the SOP), set-shifting (measured by the WCST), and interference control (measured by the Stroop Task). Separate linear regression analyses were conducted for each EF measure.

Related variables from the preliminary analyses were entered on the first step of the regression as covariates in each analysis predicting EF deficits on a given measure. For the purpose of the primary aim, child ADHD symptoms (a single score of inattention symptoms + hyperactivity symptoms) were entered as a covariate on the second step. Both covert and overt CD behaviors were entered on the next step as predictor variables and thus, significant results took into account the covariation between overt and covert behaviors.

All analyses were then followed up with similar analyses, with one exception. Covert and overt CD symptoms were replaced with the delinquency and rule-breaking subscales of the CBCL. Therefore, the question of whether covert and overt dimensions of child CD symptoms are differentially related to components of EF was asked by measuring covert and overt CD dimensions in two similar, yet distinct ways. Findings that were replicated across measurement type were considered especially robust.

Results

Preliminary Analyses

Demographic characteristics are presented in Table 3. Prior to analyses, all independent and dependent variables were examined for skewness, and data suggested that they were normally distributed. Means and variability for the main variables of interest are presented in Table 4. Prior to conducting analyses, the relationship between independent and dependent variables were examined with correlation analyses. Results are presented in Table 5. Notable findings include increases in child age being related to better performance on the SST, SOP, and DSB subtests. Child ADHD symptoms were also related to poor performance on the SST.

Child age, gender, total family income (SES), and WISC-IV average IQ scores were entered into a regression analysis for each dependent variable to determine which variables to control. Of note, the only EF measures that were age-normed were the DSB, WCST, and Stroop scores. None of the control variables were significantly related to the SOP, DSB, WCST perseverative errors (WCST-PE) or Stroop-CW scores (all $ps > .05$). Child age was significantly predictive of stop signal scores (SST; β=-.427, p=.022), such that scores were higher for children of lower ages, indicating better task performance as age increases.

The total estimated IQ score (the average of WISC-IV vocabulary and WISC-IV block design scaled scores) was not significantly related to any EF measures. The further explore this relationship, correlations were examined between each WISC-IV subtest scaled score individually and all dependent variables. No significant

correlations were found between subtest scores and measures of EF (all $ps > .05$): therefore, estimated IQ was not controlled in any of the subsequent analyses.

Although children treated with stimulant medication were not medicated on the day of the neuropsychological assessment, it was possible that whether a child takes stimulant medication at all would be related to test performance. To explore this possibility, the relationship between stimulant medication status and performance on EF measures was also examined. Medication status was significantly related to DSB scores ($F = 10.181$, $p = .003$), such that children who usually take ADHD medication performed worse on the DSB subtest. Medication was unrelated to all other dependent variables.

Preliminary Aim

Total CD symptoms were examined continuously using linear regression analysis and controlling for ADHD symptoms. These analyses did not result in any significant findings ($ps > .05$) relating CD symptoms to EF.

Linear regression analysis was conducted to explore the potentially interactive effect of the combination of ADHD and CD. After controlling for the effects of each of these variables independently, the ADHD/CD interaction did not significantly predict any EF variables (Table 6).

Children with a diagnosis of CD and ADHD ($n = 16$) were compared to children without a diagnosis of CD ($n = 33$) to examine group differences in EF. After controlling for age, there was a trend for WCST-PE to be lower in children with a diagnosis of CD relative to those without comorbid CD ($F = 3.496$, $p = .071$, *eta* $= .098$). No other measures of EF were significantly different between groups.

Primary Aim

Behavioral inhibition

SST scores were significantly predicted by ADHD symptoms, but overt and covert CD symptoms were not related to SST after controlling for ADHD and child age (Table 7). When the CBCL was examined, SST was unrelated to Rule Breaking and Aggression (Table 8).

Spatial Working Memory

Regression analyses indicated that overt and covert CD symptoms were not significantly related to SOP scores after controlling for ADHD (Table 7). ADHD symptoms were not significantly related to SOP scores. When the CBCL was examined, again there were no significant relationships between Rule Breaking or Aggression (Table 8) and SOP scores.

Short Term Memory

DSB scores were not related to overt or covert CD symptoms, after controlling for medication status and ADHD symptoms (Table 7). ADHD symptoms did not predict DSB scores. Similarly, there were no significant relationships between DSB and CBCL Rule Breaking or Aggression (Table 8).

Set Shifting

WCST scores were not related to ADHD symptoms, nor were they significantly related to overt and covert CD symptoms (Table 7). When dimensions of CD were considered using the CBCL, WCST was also not significantly related to either Rule Breaking or Aggression (Table 8) scales.

Interference Control

After accounting for ADHD symptoms, overt and covert CD symptoms were not significantly related to Stroop-CW performance (Table 7). However, ADHD symptoms were significantly negatively related to Stroop-CW performance. Similarly, when conducting these analyses using the CBCL in place of DSM-IV CD symptoms, Rule Breaking and Aggression were unrelated to Stroop-CW after controlling for ADHD (Table 8).

Multivariate Analysis

Multivariate analyses examining the primary aim were also conducted to conserve power and account for collinearity between EF measures. Overt and covert behaviors were entered as independent variables, with Stoop-CW, SST, WCST-PE, DSB, and SOP entered as dependent variables. After accounting for ADHD, neither overt nor covert behavior significantly predicted any EF measure (all $ps > .05$).

Additional Analyses

Social Impairment

Given that both overt and covert CD symptoms often occur in the context of social interactions (i.e., lying, aggression to others), social impairment was examined in relation to the variables of interest. The social impairment question from the teacher- and parent-rated IRS scales were averaged to create a social impairment score. Correlations were conducted to determine the relationship between social impairment and ADHD, and overt and covert CD symptoms. Social impairment was significantly related to ADHD symptoms ($r=.322$, $p=.029$), but not to overt or covert CD behavior. When the CBCL was examined, social impairment was not significantly related to aggression ($r=.230$, $p=.142$), but exhibited a non-significant

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trend with rule breaking behavior (r=.256, p=.097). After controlling for ADHD symptoms in a regression analysis, this relationship was not significant (β=.127, $R^2\Delta$=.013, p=.443).

Pairwise correlation analyses were then completed examining social impairment as related to the dependent EF measures used in this study in order to clarify understanding of how EF might relate to the social development of the child. There was a significant relationship between social impairment and WCST-PE (r=.311, p=.048). A regression analysis was then done, controlling for ADHD symptoms to examine the relationship between social impairment and WCST. Social impairment significantly predicted WCST-PE (β=.466, $R^2\Delta$=.187, p=.004) when ADHD sx were controlled.

Interactions between measures of EF

The interaction between WCST and Stroop-CW scores was examined to explore whether simultaneous deficits in multiple domains of EF might have a more powerful relationship to behavior than a deficit in a single domain of EF. The interaction between these particular EF domains was chosen since WCST-PE and Stroop-CW scores were both hypothesized to be related to overt, and not covert behavior. Findings indicate that the interaction between these two t-scores yielded a significant negative relationship to overt CD symptoms, such that children with low scores on both the WCST and Stroop-CW had higher levels of overt symptoms ($R^2\Delta$=.117, β=-1.941, p=.031) after the effects of each of these scores individually was controlled for. The same relationship was not found for covert symptoms ($R^2\Delta$=.004, β=-.380, p=.673). After controlling for ADHD symptoms, the same

pattern remains with the WCST-PE and Stroop-CW interaction significantly predicting overt ($R^2\Delta$=.132, β=-2.069, p=.019), but not covert symptoms ($R^2\Delta$=.006, β=-.438, p=.630). The interactions between Stroop-CW and SST, and SOP and DSB were also explored and did not demonstrate significant relationships to overt or covert behavior.

Age groups

As reviewed above, child age and developmental considerations may play an important role in the relationship between EF and CD symptoms. Therefore, the relationships proposed for the primary aim were examined after using a median split to divide the sample into older (10-14 years; $n = 26$) and younger (6-9 years; $n = 23$) children to explore potential differences in the relationship in younger and older children due to developmental changes described above. Pairwise correlations were conducted within each age range. No significant relationships were found between overt or covert CD symptoms and EF measures in the younger age group, nor were relationships between CBCL Aggression and Rule Breaking and EF found. When these relationships were explored in the older children, however, covert CD symptoms demonstrated a negative relationship with WCST-PE scores (r=-.468, p=.024), suggesting that poor performance on the WCST is related to higher levels of covert behavior. Additionally, a positive relationship was found between CBCL Rule Breaking and SOP errors (r=.495, p=.037), suggesting that children who performed poorly on the SOP had higher levels of rule breaking behavior. When total CD symptoms were examined in relation to EF measures, there were no significant

correlations between total CD symptoms and any EF measure in both younger and older age groups.

Discussion

This study examined the relationship between CD symptoms and EF in a high-risk sample of 6-14 year old children with ADHD who have mothers with elevated levels of depression. Existing literature is enhanced by this study through its use of refined constructs of specific conduct disorder symptoms, as well as the systematic examination of these symptoms in relation to a multitude of EF dimensions that are theoretically related to overt and covert behaviors. Analyses also considered the role of ADHD symptoms when exploring these relationships in order to isolate the unique relationship between EF and CD. It was hypothesized that a wider range of EF deficits would be associated with CD symptoms as a whole, and that an additive, rather than synergistic, association with EF deficits would be found in children with co-occurring ADHD and CD. Primary hypotheses included a negative relationship between overt CD symptoms and set-shifting and interference control. Conversely, it was theorized that covert CD symptoms would not be related to these EF variables after the relationship to overt symptoms was accounted for. Behavioral inhibition and working memory were hypothesized to be equally negatively related to both overt and covert CD symptoms.

Significant findings of the preliminary aim indicated that children with ADHD/CD have deficits in set shifting, as compared to children with ADHD without CD; however, no other EF deficits were noted to be more pronounced in the comorbid group. Therefore there was no support for the synergistic model of

ADHD/CD, but there was partial support for the hypothesis that children with

ADHD/CD have a wider range of neuropsychological deficits than children with

ADHD without CD. Specifically, children with ADHD/CD demonstrated poorer

performance on a measure of set shifting, suggesting that their ability to incorporate

contextual cues into modifications in response tendencies is limited. Considering the

definition of set shifting as the coordination of multiple task demands (Monsell,

2003), one can imagine that conflicting demands such as avoiding negative or

punitive consequences for behavior, but also seeking the satisfaction of a desired

outcome, may be difficult for children with CD. For example, a child may want to

take a piece of candy that they are not allowed to have, but also have been told they

will be punished for this infraction. These multiple task demands present a

challenging scenario, and children who are better able to coordinate these demands

may choose to ask a caregiver for the candy, instead of taking it. However, a child

with CD who has impaired set shifting abilities may fail to remember being punished

for a similar act previously and take the candy in the hopes of attaining their goal of

having a treat. Thus, the link between set shifting abilities and CD behaviors is a

logical one, and one that is consistent with prior evidence suggesting a relationship

between WCST and CD (Toupin et al., 2000). Given that the comorbid children were

compared to an ADHD-only group, it is possible that this finding is independent of

the effects of impulsivity or inattention that one would expect from a child with

ADHD.

ADHD symptoms measured continuously were significantly associated with

behavioral inhibition and interference control. These findings are consistent with

literature suggesting that the broad construct of inhibition is the most consistent EF deficit found within the diagnosis of ADHD (Willcutt et al., 2005; Barkley, 1997). However, these results should be interpreted with caution, since this was found within a sample consisting solely of children with ADHD and no non-disordered control group was utilized. On the other hand, the consistency of this finding with previous literature highlights the robustness of this relationship, given that this is a sample of all ADHD children. To our knowledge, this relationship between ADHD and inhibition has never been demonstrated within a sample consisting exclusively of children with ADHD. Furthermore, the fact that there were clear relationships between ADHD and EF, and the relationships between CD and EF were less clear, suggests that in fact the EF deficits sometimes found to be related to CD might indeed be better accounted for by ADHD.

Related to the fact that an ADHD sample was used, there was a weak correlation between the Stroop-CW and SST scores, which is curious given that both tasks measure aspects of inhibition and previous literature suggests a correlation in children (Archibald & Kerns, 1999). This may be due to the fact that this was an exclusively ADHD sample and there may have been lack of variability in scores on these EF measures in relation to variability one might expect in non-clinical samples. This may have weakened the relationship between these variables. Given that it is difficult to compare the variability in the current sample with existing literature using a sample of similar age range and the same EF measures, future research should explore whether correlations among inhibition tasks are weakened within purely ADHD samples. With regard to the primary aim exploring differential relationships

between EF and overt and covert CD symptoms, both overt and covert symptoms (measured using both DSM-IV and empirically-derived measures) did not significantly predict any measure of EF used in this study. Importantly, follow-up analyses revealed a significant interaction effect, such that children with low scores on both the set shifting and interference control measures had greater levels of overt CD symptoms. The same relationship was not found for covert behaviors. This finding is consistent with the hypothesis that these EF factors would be negatively related to overt symptoms, and may indicate that having intact abilities in one of these two EF domains is protective against the manifestation of overt CD symptomatology. The exploratory nature of this finding highlights the need for replication of this result.

Additionally, due to the large age range of the sample, the primary aim was examined within groups of older and younger children in the sample. There were no significant correlations between CD symptoms and EF in younger children (i.e., between the ages of six to nine). This implies that perhaps the EF-CD relationship does not become evident until later in childhood, when the gap may begin to widen between disordered children and their peers. Alternatively, the measures used in this study may have been more sensitive to relevant EF differences in older children, or older children may have been a more appropriate population for use of these measures and therefore results may be more valid. In older children, covert behavior was found to be associated with poor set shifting abilities, while overt behavior was not. This finding is in contrast to the hypotheses of this study. Some evidence suggests that covert behavior is persistent and is more predictive of antisocial behavior in adulthood than overt CD behavior (Lahey et al., 2005). Perhaps this

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persistence in CD behavior is linked to deficient abilities to shift behavioral responses. Consistent with this possibility, rule breaking behavior (but not aggression) was also found to be related to poor spatial working memory performance in the older children. Due to the small sample sizes for these follow-up/exploratory analyses, these results should be replicated to determine whether these relationships are consistently generated in other samples.

Deficits in set shifting were also related to social impairment, lending more credibility to the importance of this variable in a child's social-emotional functioning. Indeed social skills and success in social contexts involves the utilization of contextual cues such as signals that a child welcomes another child's advances to play, monitoring of peer responses to different attempts at engagement, and modification of behavior according to these cues. These findings are consistent with studies linking EF to social skills in children with fetal alcohol exposure (Schonfeld, Paley, Frankel, O'Conner, 2006). Perhaps this vulnerability in social domains is one potential mediator of the complex relationship between EF deficits and susceptibility to CD. Indeed, longitudinal research does link early EF deficits to later problematic social and emotional behaviors in children (Wahlsttedt, Thorell, Bohlin, 2008).

The failure to find other relationships between CD symptoms examined in total, and broken down into overt and covert behavior deserves careful consideration. Reasons for these results can be summarized as problems with the theory, and problems with the measures used in the study. Beginning with problems in the theory, it is possible that the inconsistent evidence reviewed in the introduction is in fact suggestive of a lack of association between dorsolateral EF abilities (e.g., inhibition,

working memory, etc.) and CD symptoms. Indeed, as pointed out in the introduction, the existing evidence does not present a clear picture defining this relationship. The argument was made herein that breaking down the constructs of EF and CD might elucidate these relationships more effectively; however, this refined examination of these constructs in the manner proposed herein did not yield any consistent links. Indeed, there is a lack of evidence at the neuroanatomical level that would support a distinction between overt and covert CD symptoms, indicating that the selection of this breakdown may have been a flawed aspect of the theory as well. For example, some literature suggests that the core deficit in CD is an emotion processing deficit linked to an amygdale circuit (Blair & Frith, 2000). The overt and covert distinction is less amenable to an emotional processing deficit explanation. There is additional evidence that CD is associated with the functions of alternative neuroanatomical regions, such as the orbitofrontal cortex, amygdala, or the ventromedial prefrontal cortex. There is accumulating theory and evidence implicating poor decision making and impaired reversal learning in severe CD and psychopathy (Finger et al., 2008). For instance, recent neuroimaging evidence links abnormal functioning of the amygdala and orbitofrontal cortex in youth with aggressive CD (Decety, Michalska, Akitsuki, and Lahey, 2008). Future research should specifically examine alternative distinctions of CD that have been clearly defined (i.e., callous-unemotional traits, proactive and reactive aggression) and have support at the biological level. These distinctions should be examined in relation to constructs like reversal learning that have demonstrated clear relationships to behavior problems in children.

In addition, it is important to remember that CD is multiply determined by factors at a variety of levels in the child's environment. Contextual factors such as poor socioeconomic conditions, harsh or ineffective parenting, association with deviant peer groups, as well as biological factors such as prenatal exposure to teratogens, temperament, or genetic vulnerabilities all have demonstrated relationships to CD (Lahey, Moffit, and Caspi, 2003). It may be that the multidetermined nature of CD may be best examined as an interaction between individual (e.g., EF) and environmental variables. Perhaps a simplistic examination of neuropsychological function solely would not create a powerful effect amongst all the variables at play.

At this point, a discussion of the potential impact of the measures used in the study is useful. In terms of the independent variables, the breakdown between overt and covert CD symptoms was measured in two ways, using both DSM-IV symptom profiles, and the empirically-derived dimensions from the CBCL which are very well validated and replicated (Achenbach and Rescorla, 2001). This was designed to eliminate any potential problems with the delineation of overt and covert symptoms. Neither measurement of these dimensions resulted in differential relationships to EF, therefore the next consideration should be the EF measures themselves.

The measurement of EF in school-age children is a challenging task. There is wide variability in the development of EF simply through developmental maturation. Furthermore, many EF measures, including the ones used in this study (specifically the Stroop) rely on rapidly developing academic skills such as reading. Yet other tasks require extended periods of seated concentration and are designed to challenge

children's attentional capabilities (i.e, WCST, SST), which can be problematic for young children who are rarely exposed to non-interactive challenges such as these. The sample used in this study was comprised solely of children with ADHD, which additionally highlights this point. Furthermore, EF tasks for children are often created as downward extensions or modifications of EF tasks used with adults (i.e., SOP, Stroop), when in fact these abilities may be better conceptualized from the skill and maturational level of a child. Due to these constraints, there is a notable lack of developmentally-sensitive EF tasks for young children, and thus the measures selected for this study may not be as precise at measuring EF in young children. In fact, analyses discussed above that were separated by age failed to generate relationships in the younger age ranges. This suggests that perhaps the measures were indeed not developmentally sensitive enough in children of younger ages.

Finally, an important limitation of this study is its sample. Children in this study were part of an existing study that comprised treatment-seeking families. It is possible that these children differ from the more general population of children with ADHD and CD. The sample size also may have limited our ability to uncover relationships. The exploration of overt and covert CD symptoms warrants replication with a larger sample size designed to detect the nuances of the potentially complex relationship between EF and CD. Notably, the low range of overt and covert symptoms (0-3 symptoms) may have contributed to the need for greater power. However, prior samples gathered in our laboratory have generated variability in overt/covert symptom dimensions and have demonstrated relationships between these dimensions of CD and parental personality and parenting (Clarke et al., 2007: Clarke

et al., 2007b). The present sample was relatively small and underpowered which greatly limited our ability to uncover relationships. Therefore, additional research with larger samples should be conducted to allow an improved examination of the relationship between EF and CD.

The questions asked of this study might best be asked using a group design utilizing an ADHD-only group, a CD-only group, an ADHD/CD group, and a control group. For the purposes of this study, an ADHD sample was used because this is a group at increased risk for CD, which allowed for greater variability in CD symptoms. Furthermore, understanding correlates of CD within an ADHD sample is important due to the severity and stability of CD symptoms within this group. It was believed that ADHD would contribute EF deficits most specific to inhibition, and that CD would contribute EF deficits most closely linked to shifting and interference control. Therefore, while the selection of an ADHD sample was based in a theoretical rationale, the fact that this study only examined children with ADHD limits the generalizability of the findings, as well as the ability to truly understand the potentially unique relationships between EF and ADHD and CD.

The aims of this study would also best be served with a study that is better designed to address the developmental considerations discussed. Alternative measures of EF that have been normed and developed for children of younger ages are critical to ensure the validity of measurement for the variables of interest. Longitudinal research on this topic can clarify the relationship between age and EF, and could answer the question of whether the relationship between EF and CD changes across development. For example, there are many changes in the expression

of overt and covert behaviors over development and there are symptoms that may not present themselves until adolescence (i.e., using a weapon, truancy, breaking and entering). Trajectory analyses may be useful to examine children with different longitudinal patterns of overt and covert behavior, and perhaps there may be a different pattern of EF deficits among children who express overt-only, covert-only, and both overt and covert behaviors. Also, the currently study only included children with early-onset CD symptoms, whereas the correlates of adolescent-onset CD may be very different. This sample, while it comprises children with early-onset CD, may also include children who will eventually desist from these behaviors. Because CD symptoms are constantly fluid and in flux throughout development, a single snapshot of their correlates simply may not be adequate. Indeed, there are also substantial changes in development of EF abilities throughout childhood, and psychological implications of each variable of consideration across development were not adequately considered in this cross-sectional study.

Future research should continue to refine our understanding of the complex causes of CD. From a neuropsychological perspective, continued efforts in developing appropriate EF measures for children of all ages will assist in clarifying the inconsistencies in existing research and allow more confidence in both significant and null findings of associations between EF and child disruptive behavior. With regard to the study of CD, a more multi-faceted conceptualization of the correlates of CD symptoms will be necessary given the numerous potential causal factors. The study of integrative concepts such as "executive emotion" or "hot" executive functions that consider both the executive and emotional neuroanatomical pathways

that may interplay in the development of CD is a promising direction. Additionally, movement to the simultaneous examination of multiple levels of analysis, from biological to environmental, would allow for a more sophisticated understanding of the causes of CD. Specifically, examination of social cognition and social skills may help to outline a pathway between EF and the expression of CD and assist in the development either of targeted interventions or prevention programs. The importance of innovative and rigorous research into the underpinnings of CD cannot be understated. Aggression and delinquency result in prominent and longstanding impairments in the lives of affected children and the individuals with whom they are in contact. Therefore, the clinical implications of obtaining a better understanding of the etiological causes of CD are considerable.

Table i. Literature review of factor analytic studies of CD symptoms

CD Symptom	Frick et al, 1993 O	C	Tackett et al., 2003 O	C	Tackett et al., 2005 O	C	Janson et al., 2006 O	C	Fergusson et al., 1994 O	C	# of studies in support O	C
Bullies others	X		X		-	-	X		-	-	*3*	*0*
Physical fights	X		X		X		X		X		*5*	*0*
Used a weapon	-	-	X		X		X		X		*4*	*0*
Cruel to people	X		X		X		X		X		*5*	*0*
Cruel to animals		X	X		-	-	-	-	X		2	1
Steal with confront	-	-	X	X	-	-	-	-	X		1	0
Forced sex	-	-	-	-	-		-	-	-	-	0	0
Set fire		X	-	-		X	X	X	X		1	3
Vandalism		X	X	X	X		X		X		1	3
Break in	-	-		X		X		X		X	*0*	*4*
Lies		X	X	X		X		X		X	*0*	*4*
Steals with no confront	-	-		X		X		X		X	*0*	*4*
Stay out late	-	-	X		-	-	-	-	-	-	0	1
Run away		X	X		-	-	-	-		X	*0*	*3*
Truant		X	X		X		-	-		X	*0*	*4*

Note. O=Overt Symptoms. C=Covert Symptoms. - = No information because symptom was not assessed, had a low base rate in the sample, or loaded onto another factor.

Table ii. Conduct Disorder Symptom Dimensions

Overt Symptoms	Covert Symptoms
Often bullies, threatens, or intimidates others	Has run away from home
Often initiates physical fights	Has broken into someone else's house, building, or car
Has used a weapon that can cause serious physical harm to others	Often lies to obtain goods or favors or to avoid obligations
Has been physically cruel to people	Has stolen items of a nontrivial value without confronting a victim
	If often truant from school, beginning before age 13 years

Table iii. Demographic Characteristics (*N*=49)

Variable	Child Characteristics	
	Mean (SD)	% (*n*)
Age (years)	9.59 (2.3)	
Sex (Male)		71 (33)
WISC Verbal (Scaled Score)	11.20 (3.9)	
WISC Performance (Scaled Score)	9.61(3.8)	
Race/Ethnicity		
Caucasian		32.7 (16)
African-American		30.6 (15)
Hispanic		8.7 (4)
Asian		2.2 (1)
Bi-racial		17.4 (8)
Other		410.2 (5)
ADHD Diagnosis		
Combined Type		67.4 (33)
Inattentive Type		30.6 (15)
Hyperactive/Impulsive Type		2 (1)
Comorbidity		
Oppositional Defiant Disorder		28.6 (14)
Conduct Disorder		32.7 (16)
Medication Status (on medication)		55 (27)

Note. WISC= Weschler Intelligence Scale for Children; ADHD=Attention Deficit Hyperactivity Disorder

Table iv. Means and Standard Deviations (*n*=49)

	M	SD	Range
ADHD Symptoms	14.37	3.04	9-18
CD Symptoms	2.04	2.04	0-9
Overt Symptoms	.76	1.01	0-3
Covert Symptoms	.86	.79	0-3
SOP Errors	10.37	6.59	1-35
Digit Span Backwards	9.33	3.27	4-16
Stop signal task	295.33	115.70	89-666
WCST Perseverative Errors	40.81	17.71	5-65
Stroop Color Word	41.57	7.72	34
PRR Reversal Errors	49.9	11.5	26-60
PRR Reaction Time	1048.1	366.4	289-1764
Callous Unemotional Traits	4.63	2.13	0-10
Proactive Aggression	3.86	1.17	3-7
Reactive Aggression	6.24	1.75	3-9
CBCL Aggression	65.95	10.42	50-86
CBCL Rule Breaking	62.65	9.13	50-84

Note. ADHD=Attention Deficit Hyperactivity Disorder; CD= Conduct Disorder; CBCL=Child Behavior Checklist; PRR=Probabilistic Response Reversal

Table v. Relationship between independent and dependent variables (*n*=49)

	1	2	3	4	5	6	7	8	9	10	11	12
1. Child age	-	-.214	-.035	.200	-.320*	-.345*	-.045	.182	-.423**	.094	-.106	-.171
2. ADHD sx		-	.240	.204	.109	-.176	-.362*	-.237	.396*	.313*	.394**	.451**
3. Overt sx			-	.294	-.028	.000	-.114	-.094	-.043	.752**	.427**	.491**
4. Covert sx				-	.013	-.256+	-.059	-.252	-.015	..701**	.366*	.548**
5. SOP errors					-	-.012	-.100	-.008	-.165	.022	.100	.213
6. DSB						-	.287	-.210	.105	-.124	-.140	-.170
7. Stroop CW							-	-.108	-.137	-.122	-.233	-.271+
8. WCST PE								-	-.171	-.277+	-.082	-.055
9. Stop signal									-	.024	-.001	.217
10. CD sx										-	.408**	.530**
11. Aggress											-	.807**
12. Rules												-

+ indicates p < .10 * indicates p < .05 ** indicates p < .01

Note. ADHD=Attention Deficit Hyperactivity Disorder; Sx= Symptoms; Stroop CW=Stroop Color Word; WCST- PE=Wisconsin Card Sort Task Perseverative Errors T-Score; CD=Conduct Disorder; Aggress=CBCL Aggression Scale; Rules=CBCL Rule Breaking Scale

Table vi

ADHD x CD Interaction									
	df	*F*	$R^2\Delta$	*β*		*df*	*F*	$R^2\Delta$	*β*
SOP					**Stroop-CW**				
Step 1	2, 40	.246	.012		Step 1	2, 44	3.339	.132*	
CD sx				-.016	CD sx				.024
ADHD sx				.114	ADHD sx				-.371*
Step 2	3, 39	1.011	.060		Step 2	3, 43	3.256	.053	
CD sx				-1.299	CD sx				-1.309
ADHD sx				-.134	ADHD sx				-.607**
ADHD x CD				1.405	ADHD x CD				1.455
Stop Task					**Digit Span**				
Step 1	2, 38	3.865	.169		Step 1	2, 46	.863	.036	
CD sx				-.119	CD sx				-.076
ADHD sx				.435**	ADHD sx				-.152
Step 2	3, 37	2.521	.001		Step 2	3, 45	.580	.001	
CD sx				-.258	CD sx				.101
ADHD sx				.408**	ADHD sx				-.119
ADHD x CD				.152	ADHD x CD				-.193
WCST-PE									
Step 1	2, 40	2.256	.101						
CD sx				-.225					
ADHD sx				-.165					
Step 2	3, 39	1.510	.003						
CD sx				-.507					
ADHD sx				-.221					
ADHD x CD				.309					

+ indicates p < .10   * indicates p < .05   ** indicates p < .01

Note. CD sx= Conduct Disorder Symptoms; ADHD sx=Attention Deficit Hyperactivity Disorder Symptoms; ADHD x CD= Interaction of ADHD and CD symptoms

Table vii

	df	F	$R^2\Delta$	β		df	F	$R^2\Delta$	β
SOP					**Stroop-CW**				
Step 1	1,42	.495	.012		Step 1	1, 45	6.801*	.131	
ADHD				.109	ADHD				-.362*
Step 2	3,42	.195	.003		Step 2	3, 43	2.180	.001	
ADHD				.121	ADHD				-.361*
Overt				-.055	Overt				-.024
Covert				.022	Covert				.023
Stop Task					**Digit Span**				
Step 1	1,39	8.94	.187**		Step 1	1, 46	5.529	.107	
Age				-.432**	Meds				-.328*
Step 2	2, 38	7.79	.104*		Step 2	2, 45	3.131	.015	
Age				-.373*	Meds				-.296*
ADHD				.328*	ADHD				-.126
Step 3	4, 36	4.29	.032		Step 3	4, 43	2.216	.049	
Age				-.406*	Meds				-.292
ADHD				.355*	ADHD				-.089
Overt				-.194	Overt				.068
Covert				.074	Covert				-.234
WCST-PE									
Step 1	1, 41	2.435	.056						
ADHD				-.237					
Step 2	3, 39	1.431	.043						
ADHD				-.196					
Overt				.014					
Covert				-.215					

+ indicates $p < .10$ * indicates $p < .05$ ** indicates $p < .01$

Note. ADHD=Attention Deficit Hyperactivity Disorder; Meds=Child Medication Status

Table viii

	df	F	$R^2\varDelta$	β		df	F	$R^2\varDelta$	β
CBCL Aggression/Rule Breaking									
SOP					**Stroop-CW**				
Step 1	1,36	.357	.010		Step 1	1, 40	3.395	.076	
ADHD				.099	ADHD				-.276+
Step 2	3,34	.654	.045		Step 2	3, 38	1.367	.021	
ADHD				-.003	ADHD				-.210
Rule Break				.379	Rule Break				-.086
Aggression				-.213	Aggression				- .082
Stop Task					**Digit Span**				
Step 1	1,36	3.412	.087+		Step 1	1, 41	2.851	.065	
Age				-.294+	Meds				-.255+
Step 2	2, 35	1.662	.000		Step 2	2, 40	2.081	.029	
Age				-.290+	Meds				-.205
ADHD				.013	ADHD				-.178
Step 3	4, 33	1.234	.043		Step 3	4, 38	1.111	.010	
Age				-.288	Meds				-.222
ADHD				.087	ADHD				-.145
Rule Break				.372	Rule Break				-.177
Aggression				-.207	Aggression				.119
WCST-PE									
Step 1	1, 37	2.135	.055						
ADHD				-.234					
Step 2	3, 35	.778	.008						
ADHD				-.261					
Rule Break				.156					
Aggression				-.096					

+ indicates p < .10   * indicates p < .05   ** indicates p < .01

Note. ADHD=Attention Deficit Hyperactivity Disorder; Meds=Child Medication Status; Rule Break=CBCL Rule Breaking T-Score; Aggression=CBCL Aggression T-Score

Bibliography

Achenbach, T. M. (1993). Taxonomy and comorbidity of conduct problems:

Evidence from empirically based approaches. *Development and*

Psychopathology, 5, 51-64.

Achenbach, T. M., Conners, C. K., Quay, H. C, Verhulst, F. C., & Howell, C. T.

(1989). Replication of empirically derived syndromes as a basis for taxonomy

of child/adolescent psychopathology. *Journal of Abnormal Child Psychology,*

17, 299-323.

Achenbach, T. M. & Rescorla (2001). *Manual for the ASEBA School-Age Forms and*

Profiles. Burlington, VT: University of Vermont, Research Center for

Children, Youth, & Families.

American Academy of Child and Adolescent Psychiatry. (2004). Teens: Alcohol

And Other Drugs Fact Sheet.

American Psychiatric Association. (2000). *The diagnostic and statistical manual of*

mental disorders (4th ed., text rev.). Washington, DC.

Applegate, B., Lahey, B.B., Hart, E.L., Biederman, J., Hynd, G.W., Barkley, R.A.,

Ollendick, T., Frick, P.J., Greenhill, L., McBurnett, K., Newcorn, J.H.,

Kerdyk, L., Garfinkel, B., Waldman, I., & Shaffer, D. (1997). Validity of the

age-of-onset criterion for ADHD: a report from the DSM-IV field trials.

Journal of the American Academy of Child & Adolescent Psychiatry, 36(9),

1211-1221.

Archibald, S.J., & Kerns K.A. (1999). Identification and description of new tests of

executive function in children. *Child Neuropsychology, 5*, 115-129.

Arsten, A. F. (2006). Stimulants: Therapeutic actions in ADHD.

    Neuropsychopharmacology, 31, 2376-2383.

Arseneault, L, Kim-Cohen, J., Taylor, A, Caspi, A., & Moffitt, T. (2005).

    Psychometric Evaluation of 5- and 7-Year-Old Children's Self-Reports of

    Conduct Problems. *Journal of Abnormal Child Psychology, 33*, 537-550.

Babinski, L. M., Hartsough, C. S., & Lambert, N. M. (1999). Childhood conduct

    problems, hyperactivity-impulsivity, and inattention as predictors of adult

    criminal activity. *The Journal of Child Psychology and Psychiatry and Allied*

    Disciplines, 40, 347-355.

Baddeley, A.D. (1986). Working memory. Oxford: Clarendon Press.

Baddeley, A.D. & Hitch, G.J. (1974). Working memory. In Bower, G.H. (ed.), *Recent*

    Advances in Learning and Motivation (Volume 8), New York: Academic

    Press.

Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. (1992). Frontal lobe functions in

    attention deficit disorder with and without hyperactivity: A review and

    research report. *Journal of Abnormal Child Psychology, 20*, 163-188.

Barkley, R. A. (1997). Behavioural inhibition, sustained attention, and executive

    functions: Constructing a unifying theory of AD/HD. *Psychological Bulletin,*

    121, 65-94.

Barkley, R. A. (1998). *Attention deficit hyperactivity disorder. A handbook for*

    diagnosis and treatment. New York: Guilford.

Barkley, R. A., Edwards, G., Laneri, m., Fletcher, K., & Metevia, L. (2001).

    Executive functioning, temporal discounting, and sense of time in adolescents

with attention- deficit/hyperactivity disorder (ADHD) and oppositional defiant

disorder (ODD). *Journal of Abnormal Child Psychology, 29*, 541-556.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction

in social psychological research: Conceptual, strategic and statistical

considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression

Inventory-II.* San Antonio TX: Psychological Corporation.

Bird, H. R., Gould, M., & Staghezza, B. M. (1993). Patterns of diagnostic

comorbidity in a community sample of children aged 9 through 16 years.

Journal of the American Academy of Child and Adolescent Psychiatry, 32,

361-368.

Birmaher, B., Brent, D., Rao, U. & Ryan, M. (1996). Kiddie-Schedule for Affective

Disorders- Present and Lifetime Version.

http://www.wpic.pitt.edu/ksads/default.htm.

Blair RJ, Colledge E, & Mitchell DG. (2001). Somatic markers and response reversal:

is there orbitofrontal cortex dysfunction in boys with psychopathic

tendencies? *Journal of Abnormal Child Psychology, 29,* 499-511.

Blair J. & Frith, U. (2000). Neurocognitive explanations of the antisocial personality

disorders. *Criminal Behavior and Mental Health, 10,* S66-S81.

Blair, R.J.R., Peschardt, K.S., Budhani, S., Mitchell, D.G. & Pine, D.S. (2006). The

development of psychopathy. *Journal of Child Psychology and Psychiatry,

47,* 262-276.

Blakemore, S., & Choudhury, S. (2006). Development of the adolescent brain:

Implications for executive function and social cognition. *Journal of Child Psychology and Psychiatry, 47*, 296-312.

Budhani, S., & Blair, R.J.R. (2005). Response reversal and children with psychopathic tendencies: Success is a function of salience of contingency change. *Journal of Child Psychology and Psychiatry, 46,* 972-981.

Budhani, S., Richell, R.A., & Blair, R.J. (2006). Impaired reversal but intact acquisition: probabilistic response reversal deficits in adult individuals with psychopathy. *Journal of Abnormal Psychology, 115,* 552-8.

Burgess, P.W., Alderman, N., Evans, J.J., Wilson, B.A., Emslie, H., & Shallice, T. (1996). Modified six elements test. In B.A. Wilson, N. Alderman, P.W. Burgess, H. Emslie, & J.J. Evans (Eds), *Behavioral assessment of the dysexecutive syndrome.* Bury St. Edmunds: Thames Valley Test Company.

Burgess, P.W., & Shallice, T. (1997). *Hayling Sentence Completion Test*. Suffolk, England: Thames Valley Test Co. Ltd.

Brocki, K.C., & Bohlin, G. (2004). Executive functions in children age 6-13: a dimensional and developmental study. *Developmental Neuropsychology, 26*, 571-593.

Brocki, K.C., & Bohlin, G. (2006). Developmental change in the relation between executive functions and symptoms of ADHD and co-occurring behavior problems. *Infant and Child Development, 15*, 19-40.

Campbell, J. M. (1998). Internal and external validity of seven Wechsler Intelligence Scale for Children--Third Edition short forms in a sample of psychiatric inpatients. *Psychological Assessment, 10*, 431-434.

Card, N. A., & Little, T. D. (2006). Proactive and reactive aggression in childhood and adolescence: A meta-analysis of differential relations with psychosocial adjustment. *International Journal of Behavioral Development, 30*, 466-480.

Chronis, A. M., Lahey, B. B., Pelham, W. E., Kipp, H., Baumann, B, & Lee, S. S. (2003). Psychopathology and substance abuse in parents of young children with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry, 42,* 1424-1432.

Chronis, A. M., Lahey, B.B., Pelham, W.E., Williams, S.H., Baumann, B.L., Kipp, H., & Jones, H.A. (2007). Parental psychopathology and maternal positive parenting predict future conduct problems in young children with attention-deficit/hyperactivity disorder. *Developmental Psychology, 42,* 70-82.

Clark, C., Prior, M., & Kinsella, G. J. (2000). Do executive function deficits differentiate between adolescents with ADHD and oppositional defiant/conduct disorder? A neuropsychological study using the six elements test and hayling sentence completion test. *Journal of Abnormal Child Psychology, 29,* 403-414.

Clarke, T.L., & Chronis, A.M. (2005). Book Review: Causes of Conduct Disorder and Juvenile Delinquency. [Review of the book *Causes of Conduct Disorder and Juvenile Delinquency*]. The Behavior Therapist: AABT.

Clarke, T. L., Chronis, A. M., Raggi, V. L., Diaz, Y., & Ali, M. (2007a). Parenting and overt/covert patterns of ASB in children with ADHD. Abstract accepted for presentation at the annual meeting for the Association for Psychological Science, Washington D.C.

Clarke, T.L., Chronis, A.M., Raggi, V.L., Diaz, Y. (2007b). Maternal Personality and

    its Relationship to Child Overt and Covert Behavior in Children with ADHD:

    The Mediating Role of Parenting. Abstract accepted for presentation at the

    annual meeting of the Association for Behavioral and Cognitive Therapies,

    Philadelphia, PA.

Conners, C. K. (1993). *The Conners Continuous Performance Test*. North

    Tonawanda, NY: Multi-Health Systems.

Cukrowicz, K.C., Taylor, J., Schatschneider, C., & Lacono, W.G. (2006). Personality

    differences in children and adolescents with attention-deficit/hyperactivity

    disorder, conduct disorder, and controls. *Journal of Child Psychology and*

    Psychiatry, 47, 151-159.

Decety, J., Michalska, K.J., Akitsuki, Y., & Lahey, B.B. (2008). Atypical empathic

    responses in adolescents with aggressive conduct disorder: a functional MRI

    investigation. *Biological Psychology*, Epub ahead of print.

De Los Reyes, A., & Kazdin, A.E. (2005). Informant discrepancies in the assessment

    of childhood psychopathology: A critical review, theoretical framework, and

    recommendations for further study. *Psychological Bulletin*, *131*, 483-509.

Dery, M., Toupin, J., Pauze, R., Mercier, H., & Fortin, L. (1999). Neuropsychological

    characteristics of adolescents with conduct disorder: Association with

    attention-deficit/hyperactivity and aggression. *Journal of Abnormal Child*

    Psychology, 27, 225-236.

Diamond, A., & Doar, B. (1989). The performance of human infants on a measure of

    frontal cortex function, the delayed response task. *Developmental*

    Psychobiology, 22, 271-94.

Diamond, A., & Goldman-Rakic, P. S. (1989). Comparison of human infants and

    rhesus monkeys on Piaget's A-not-B task: Evidence for dependence on

    dorsolateral prefrontal cortex. *Experimental Brain Research, 74*, 24-40.

DuPaul, G.J., McGoey, K.E., Eckert, T.L., & VanBrakle, J. (2001). Preschool

    children with attention-deficit/hyperactivity disorder: Impairments in

    behavioral, social, and school functioning. *Journal of the American Academy*

    of Child and Adolescent Psychiatry, 40, 508-515.

Engle, R. W. (2002) Working Memory Capacity as Executive Attention. *Current*

    Directions in Psychological Science 11, 19–23.

Eiraldi, R. B., Power,T. J.,& Nezu, C.M. (1997). Patterns of comorbidity associated

    with subtypes of attention-deficit/hyperactivity disorder among 6- to 12-year-

    old children. *Journal of the American Academy of Child and Adolescent*

    Psychiatry, *36*(4), 503–514.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis

    program. *BehaviorResearch Methods, Instruments, & Computers*, *28*, 1-11.

Faraone, S. V., Biederman, J., Weber, W., & Russel, R. L. (1998). Psychiatric,

    neuropsychological, and psychosocial features of DSM-IV subtypes of

    attention-deficit/hyperactivity disorder: Results from a clinically referred

    sample. *Journal of the American Academy of Child and Adolescent*

    Psychiatry, *37*(2), 185–193.

Farrington, D.P., Loeber, R., & Van Kammen, W.B. (1990). Long-term criminal

outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood. *In:* Robins, L. N., Rutter, M. (Eds*.) Straight and devious pathways from childhood to adulthood.* New York, NY: Cambridge University Press, pp. 62-81.

Fabiano, G. A., Pelham, W. E., Waschbusch, D. A., Gnagy, E., Lahey, B. B., Chronis, A. M., Onyango, A. N., Kipp, H., Lopez-Williams, A., & Burrows-MacLean, L. (2006). A Practical Measure of Impairment: Psychometric Properties of the Impairment Rating Scale in Samples of Children With Attention Deficit Hyperactivity Disorder and Two School-Based Samples. *Journal of Clinical and Adolescent Psychology*, 35, 369-385.

Faraone, S. V., Biederman, J., Feighner, J. A., & Monuteaux, M. C. (2000). Assessing symptoms of attention deficit/hyperactivity disorder in children and adults: Which is more valid? *Journal of Consulting and Clinical Psychology*, *68*(5), 830-832.

Fergusson, D.M., Horwood, L.J., & Lynskey, M.T. (1994). Structure of DSM-II-R Criteria for Disruptive Childhood   Behaviors: Confirmatory Factor Models. *Journal of the American Academy of Child and Adolescent Psychiatry, 33*, 1145-1157.

Finger EC, Marsh AA, Mitchell DG, Reid ME, Sims C, Budhani S, Kosson DS, Chen G, Towbin *KE, Leibenluft E, Pine DS, & Blair JR. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning.* Archives of General Psychiatry, 65(*5*), 586-94.

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and

interference control functions: A latent-variable analysis. *Journal of Experimental Psychology: General, 133,* 101-135.

Fite PJ, Colder CR, Lochman JE, & Wells KC. (2008). Developmental trajectories of proactive and reactive aggression from fifth to ninth grade. *Journal of Clinical Child and Adolescent Psychology, 37*, 412-421.

Frick, P.J. (2001). Effective interventions for children and adolescents with conduct disorder. *Canadian Journal of Psychiatry, 46,* 597-608.

Frick, P.J. & Ellis, M. (1999). Callous-unemotional traits and subtypes of conduct disorder. *Clinical Child and Family Psychology Review, 2,* 149-168.

Frick, P.J. & Hare, R.D. (2001). The Antisocial Process Screening Device (APSD). Toronto: Multi-Health Systems. Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L. E., Van Horn, Y, Christ, M. A. G., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review, 13,* 319-340.

Frick, P.J., Stickle, T.R., Dandreaux, D.M., Farrell, J.M., & Kimonis, E.R. (2005). Callous-unemotional traits in predicting the severity and stability of conduct problems and delinquency.   *Journal of Abnormal Child Psychology, 33,* 471-487.

Frick, P.J. & White, S.F. (2008). Research review: The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry, 49*(4), 359-375.

Geurts, H.M., Verte, S., Oosterlann, A., Roeyers, H., & Sergeant, J.A. (2005). ADHD

subtypes: Do they differ in their executive functioning profile? *Archives of Clinical Neuropsychology, 20*, 457-477.

Giancola, P.R., Mezzich, A.C., & Tarter, R.E. (1998). Executive cognitive functioning, temperament, and ASB in conduct-disordered adolescent females. *Journal of Abnormal Psychology, 107*, 629-641.

Golden, C.J. (1981). A standardized version of Luria's neuropsychologicl tests. In S. Filskov & T.J. Boll (Eds.), *Handbook of clinical neuropsychology* (pp. 608-642). New York: Wiley-Interscience.

Hawes, D.J., & Dadds, M.R. (2005). The treatment of conduct problems in children with callous-unemotional traits. *Journal of Consulting and Clinical Psychology, 73,* 737-741.

Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin card sorting test manual: Revised and expanded*. Odessa, FL: Psychological Assessment Resources.

Hedden, T., & Yoon, C. (2006). Individual differences in executive processing Predict susceptibility to interference in verbal working memory. *Neuropsychology, 20*, 511-528.

Hinshaw, S. P., Simmel, C., & Heller, T. L. (1995). Multimethod assessment of covert ASB in children: Laboratory observations, adult ratings, and child self-report. *Psychological Assessment, 7*, 209-219.

Hinshaw S. P., Zupan, B. A., Simmel, C., Nigg, J.T., & Melnick, S. (1997). Peer status in boys with and without attention-deficit hyperactivity disorder: Predictions from overt and covert ASB, social isolation, and authoritative

parenting beliefs. *Child Development, 68*, 880-896.

Janson, H., & Kjelsberg, E. (2006). Factor structure and individual patters of DSM-IV
conduct disorder criteria in adolescent psychiatric inpatients. *Nordic Journal
of Psychiatry, 60,* 168-175.

Jensen, P. S., Martin, D., & Cantwell, D.P. (1997). Comorbidity in ADHD:
Implications for research, practice, and DSM-V. *Journal of the American
Academy of Child and Adolescent Psychiatry, 36*(8), 1065-1079.

Johansson, P., Kerr, M., & Andershed, H. (2005). Linking Adult Psychopathy with
Childhood Hyperactivity-Impulsivity-Attention Problems and Conduct
Problems Through Retrospective Self-reports. *Journal of Personality
Disorders, 19*, 94-101.

Kane, M. J., & Engle, R. W. (2000). Working memory capacity, proactive
interference, and divided attention: Limits on long-term memory retrieval.
Journal of Experimental Psychology: Learning, Memory, and Cognition, 26,
236-358.

Kaplan, B.J., Crawford, S.G., Dewey, D.M., & Fisher, G.C. (2000). The IQs of
children with ADHD are normally distributed. *Journal of Learning
Disabilities, 33*, 425-342.

Lahey, B.B., & Waldman, I.D. (2003). A developmental propensity model of the
origins of conduct problems during childhood and adolescence. In B.B.
Lahey, T.E. Moffit, and A. Caspi, (Eds.), *Causes of Conduct Disorder and
Juvenile Delinquency*. The Guilford Press, New York, NY. pp. 76-117.

Lahey, B.B., Waldman, I.D., & McBurnett, K. (1999). Annotation: The development

of ASB: An integrative causal model. *Journal of Child Psychology and*

Psychiatry, 40, 669-682.

Lahey, B. B., Schaughency, E. A., Hynd, G., Carlson, C. L.,& Nieves, N. (1987).

Attention deficit disorder with and without hyperactivity: Comparison of

behavioral characteristics of clinic-referred children. *Journal of the American*

Academy of Child and Adolescent Psychiatry, *26*(5), 718–723.

Liau, A.K., Barriga, A.Q., & Gibbs, J.C. (1998). Relations between self-serving

cognitive distortions and overt vs. covert ASB in adolescents. *Aggressive*

Behavior, 24, 335-346.

Lochman, J. E., & Wells, K. C. (2002). The coping power program at the middle

school transition: Universal and indicated prevention effect. *Psychology of*

Addictive Behaviors, 16, S40–S54.

Loeber, R. (1982). The stability of antisocial and delinquent child behavior: A review.

Child Development, 53, 1431-1446.

Loeber, R., Green, S. M., Lahey, B. B., Christ, M. A. G., & Frick, P. J. (1992).

Developmental sequences in the age of onset of the disruptive behavior

disorders. *Journal of Child and Family Studies*, *1*(1), 21–41.

Loeber, R., Keenan, K., Lahey, B. B., Green, S. M., & Thomas, C. (1993). Evidence

for developmentally based diagnoses of oppositional defiant disorder and

conduct disorder. *Journal of Abnormal Child Psychology*, *21*(4), 377–410.

Loeber, R., Keenan, K., & Zhang, Q. (1997). Boys' experimentation and persistence in developmental pathways toward serious delinquency. *Journal of Child and Family Studies, 6*, 321-357.

Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In T. H. Dagenbach & D. Carr (Eds.), Inhibitory processes in attention, memory, and language (pp. 189-239). San Diego: Academic Press.

Logan, G.D., Schachar, R.J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science, 8*(1), 60-64.

Lynam, D.R. (1996). Early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin, 120,* 209-234.

Martinussen, R. & Tannock, R. (2006) 'Working Memory Impairments in Children with Attention-Deficit Hyperactivity Disorder With and Without Comorbid Language Learning Disorders', *Journal of Clinical and Experimental Neuropsychology, 28:*7, 1073 - 1094

McBurnett, K., Lahey, B.B.., Capasso, L., & Loeber, R. (1996). Aggressive symptoms and salivary cortisol in clinic-referred boys with conduct disorder. *Annals of the New York Academy of Sciences, 794*, 169-179.

McCarthy, D. (1972). *Manual for the McCarthy Scales for Children's Abilities*. New York: Psychological Corporation.

Milich, R. & Dodge, K. A. (1984). Social information processing in child psychiatric populations. *Journal of Abnormal Child Psychology, 12,* 471–490.

Milner, B. & Petrides, M. (1984). Behavioral effects of frontal lobe lesions in man. *Trends Neuroscience 7*, 403–407.

Milner, B., Petrides, M., & Smith, M. L. (1985). Frontal lobes and the temporal organization of memory. *Human Neurobiology, 4,* 137-142.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41,* 49-100.

Moffitt, T. E. (1990). Juvenile delingquency and attention-deficit disorder: Developmental trajectories from age 3 to 15. *Child Development, 61*, 893-910.

Moffitt. T.E. (1993). Life-course-persistent and adolescence-limited ASB: A developmental taxonomy. *Psychological Review, 100*, 674-701.

Moffitt T. E., & Henry B. (1989). Neuropsychological assessment of executive functions in self-reported delinquents. *Development and Psychopathology, 1*, 105-118.

Monuteaux, M. C., Blacker, D., Biederman, J., & Buka, S. L. (2005). Symptomatic subtypes of conduct disorder: A critical review. In J. P. Morgan (Ed). *Psychology of Aggression*, New York: Nova Science Publishers.

Monuteaux, M.C., Blacker, D., Biederman, J., Fitzmaurice, G., Buka, S.L. (2006). Maternal smoking during pregnancy and offspring overt and covert conduct problems: a longitudinal study. *Journal of Child Psychology and Psychiatry 47*, 883–890.

Monsell, S. (2003). Task switching. *Trends in Cognitive Sciences, 7,* 134–140.

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation

    between ASB and neuropsychological measures of executive function.

    Clinical Psychology Review, 20, 115-136.

Nagin, D., & Tremblay, R. E. (1999). Trajectories of boys' physical aggression,

    opposition, and hyperactivity on the path to physically violent and nonviolent

    juvenile delinquency. *Child Development, 70*, 1181-1196.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology:

    Views from cognitive and personality psychology and a working inhibition

    taxonomy. *Psychological Bulletin, 126*, 220-246.

Nigg, J. T. (2001). Is ADHD a disinhibitory disorder? *Psychological Bulletin, 127*,

    571-598.

Nigg, J.T., Blaskey, L.G., Huang-Pollock, C.L., & Rappely, M.D. (2000).

    Neuropsychological executive functions and DSM-IV ADHD subtypes.

    Journal of the American Academy of Child and Adolescent Psychiatry, 41, 59-

    66.

Nigg, J. T., & Huang-Pollock, C.L. (2003). An early-onset model of the role of

    executive functions and intelligence in conduct disorder/delinquency. In B.B.

    Lahey, T.E. Moffit, and A. Caspi, (Eds.), *Causes of Conduct Disorder and*

    Juvenile Delinquency. The Guilford Press, New York, NY. pp. 227-253.

Norman, D., & Shallice, T., 1986: Attention to action: Willed and automatic control

    of behavior. In Davidson, R., Schwartz, G., and Shapiro, D., (eds.)

    Consciousness and Self Regulation: Advances in Research and Theory,

    Volume 4. Plenum, New York, NY. pp. 1-18.

Orvaschel, H., & Puig-Antich, J. (1995). *Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Epidemiologic 5th Version.* Ft. Lauderdale, FL: Nova University.

Patterson G.R., & Yoerger, K. (2002). A developmental model for early and late onset delinquency. *ASB in children and adolescents: A developmental analysis and model for intervention.* In J.B. Reid, Patterson, G.R., Snyder, J. (Eds.), (pp. 147-172). Washington DC: American Psychological Association.

Pelham, W. E., Gnagy, E. M., Greenslade, K. E., & Milch, R. (1992). Teacher ratings of DSM-III-R symptoms of the disruptive behavior disorders. *Journal of the American Academy* Pelham, W. E., Waschbusch, D. A., Greiner, A. R., Jennings, J. R., Tarter, R. E., & Moss, H. B. (1996, January). *Reactive aggression in boys with disruptive behavior disorders* Poster presented at the International Society for Research in Child and Adolescent Psychopathology, Santa Monica, CA.

Pennington, B.F. & Ozonoff, S. (1997). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37*, 51-87.

Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal and temporal-lobe lesions in man. *Neuropsychologia, 20*, 249-262.

Piacentini, J. C., Cohen, P., & Cohen, J. (1992). Combining discrepant diagnostic information from multiple sources: Are complex algorithms better than simple ones? *Journal of Abnormal Child Psychology, 20,* 51-63.

Porteus, S.D. (1973). *Porteus Maze Test: Fifty years application.* Palo Alto: Pacific Books.

Powell, K. B., & Voeller, K. S. (2004). Prefrontal executive function syndromes in children. *Journal of Child Neurology, 19*, 765-797.

Quay, H. C. (1997). Inhibition and attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 25*, 7-13.

Oosterlaan, J., Logan, G.D., & Sergeant, J.A. (1998). Response Inhibition in ADHD, CD, Comorbid ADHD-CD, Anxious, and Control Children: A Meta-analysis of Studies with the Stop Task. *Journal of Child Psychology and Psychiatry, 39*, 411-425.

Osterrieth, P.A. (1944). A test of copying of a complex figure. *Archive de Psychologie, 30*, 206-256.

Riccio, C.A., Homak, S., Jarratt, K.P., & Wolfe, M.E. (2006). Differences in academic and executive function domains among children with ADHD Predominantly Inattentive and Combined Types. *Archives of Clinical Neuropsychology, 21*, 657-667.

Riggs, N. R., Blair, C. B., & Greenberg, M. T. (2003). Concurrent and 2-year longitudinal relations between executive function and the behavior of 1st and 2nd grade children. *Child Neuropsychology, 9*, 267-276.

Rosen, V. M., & Engle, R. W. (1997). The role of working memory capacity in retrieval. *Journal of Experimental Psychology, General, 126*, 211-227.

Santor, D.A., Ingram, A., & Kusumakar, V. (2003). Influence of executive functioning difficulties on verbal aggression in adolescents: Moderating effects of winning and losing and increasing and decreasing levels of provocation. *Aggressive Behavior, 29*(6), 475-488.

Schwartz D., Dodge K.A., Coie J.D., Hubbard J.A., Cillessen A.H., Lemerise E.A., & Bateman H. (1998). Social-cognitive and behavioral correlates of aggression and victimization in boys' play groups. *Journal of Abnormal Child Psychology, 26*, 431-440.

Seguin, J. R., Boulerice, B., Harden, P. W., Tremblay, R. E., & Pihl, R. O. (1999). Executive functions and physical aggression after controlling for attention deficit hyperactivity disorder, general memory, and IQ. *Journal of Child Psychology and Psychiatry, 40*, 1197-1208.

Seguin, J.R., Nagin, D., Assaad, J., & Tremblay, R. (2004). Cognitive-Neuropsychological Function in Chronic Physical Aggression and Hyperactivity. *Journal of Abnormal Psychology, 113*, 603-613.

Seguin, J.R., Pihl, R.O. and Harden, P.W., Tremblay, R.E., & Boulerice, B. (1995). Cognitive and neuropsychological characteristics of physically aggressive boys. *Journal of Abnormal Psychology, 104*, 614-624.

Schonfeld, A.M., Paley, B. Frankel, F., & O'Connor, M.J. (2006). Executive functioning predicts social skills following prenatal alcohol exposure. *Child Neuropsychology, 12,* 439-452.

Shue, K. L., & Douglas, V. I. (1992). Attention deficit hyperactivity disorder and the frontal lobe syndrome. *Brain and Cognition, 20*, 104-124.

Smith, E.E., Jonides, J., & Koeppe, R.A. (1996). Dissociating Verbal and Spatial

Working Memory Using PET. *Cerebral Cortex, 6*, 11-20.

Smith, B. H., Pelham, W. E., Gnagy, E., Molina, B., & Evans, S. (2000). The reliability, validity, and unique contributions of self-report by adolescents receiving treatment for attention-deficit/hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, *68*(3), 489-499.

Snyder, J., Reid, J., Patterson, G. (2003). A social learning model of child and adolescent ASB. In B.B. Lahey, T.E. Moffit, and A. Caspi, (Eds.), *Causes of Conduct Disorder and Juvenile Delinquency*. The Guilford Press, New York, NY. pp. 27-48.

Sonuga-Barke, E. J. S. (1997). Categorical models of childhood disorder: A conceptual and empirical analysis. *Journal of Child Psychology and Psychiatry, 39*, 115-133.

Speltz, M. L., DeKlyen, M., Calderon, R., Greenberg, M. T., & Fisher, P. A. (1999). Neuropsychological characteristics and test behaviors of boys with early onset conduct problems. *Journal of Abnormal Psychology, 108*, 315-325.

Sprinkle, S. D., Lurie, D., Insko, S. L, Atkinson, G., Jones, G. L., Logan, A. R., & Bissada, N. N. (2002). Criterion validity, severity cut scores, and test-retest reliability of the Beck Depression Inventory-II in a university counseling center sample. *Journal of Counseling Psychology, 49*(3), 381-385.

Stouthamer-Loeber, M., Loeber, R., Homish, D. L., Wei, E. (2001). Maltreatment of boys and the development of disruptive and delinquent behavior. *Development and Psychopathology,13*, 941-955.

Tackett, J. L., Krueger, R. F., Sawyer, M. G., & Graetz, B. W. (2003). Subfactors of

DSM-IV conduct disorder: Evidence and connections with syndromes from the child behavior checklist. *Journal of Abnormal Child Psychology, 31,* 647-654.

Thorrell, L.B., & Whalstedt, C. (2006). Executive functioning deficits in relation to symptoms of ADHD and ODD in preschool children. *Infant and Child Development, 15*, 503-518.

Tiet, Q. Q., Wasserman, G. A., Loeber, R., McReynolds, L. S., & Miller, L. S. (2001). Developmental and sex differences in types of conduct problems. *Journal of Child and Family Studies, 10,* 181-197.

Toupin, J., Dery, M., Pauze, R., Mercier, H., & Fortin, L. (2000). Cognitive and familial contributions to conduct disorder in children. *Journal of Child Psychology and Psychiatry, 41*, 333-344.

Viding, E., Blair, J.R., Moffitt, T.E., & Plomin, R. (2005). Evidence for substantial genetic risk for psychopathy in 7-year olds. *Journal of Child Psychology and Psychiatry, 46,* 592-597.

Vitaro F, Brendgen M, & Tremblay RE. (2002). Reactively and proactively aggressive children: antecedent and subsequent characteristics. *Journal of Child Psychology and Psychiatry, 43,* 495-505.

Wahlsttedt, C., Thorell, L.B., & Bohlin, G. (2008). ADHD Symptoms and Executive Function Impairment: Early Predictors of Later Behavioral Problems. Developmental Neuropsychology, 33, *160-178.*

Waschbusch, D. A. (2002). A Meta-Analytic Examination of Comorbid Hyperactive–Impulsive–Attention Problems and Conduct Problems. *Psychological Bulletin,*

128, 118–150.

Waschbusch, D.A., Pelham, W.E., Jennings, J.R., Greiner, A.R., Tarter, R.E., &
Moss, H.B. (2002). Reactive aggression in boys with disruptive behavior
disorders: Behavior, physiology, and affect. *Journal of Abnormal Child
Psychology, 30*(6), 641-656.

Waschbusch, D.A. & Willoughby, M. T. (2008). Attention-deficit/hyperactivity
disorder and callous-unemotional traits as moderators of conduct problems
when examining impairment and aggression in elementary school children.
Aggressive Behavior, 34(2), 139-153.

Webster-Stratton, C. & Hammond, M. (1997). Treating children with early-onset
conduct problems: A comparison of child and parent training interventions.
Journal of Consulting and Clinical Psychology, 65, 93-109.

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children – Fourth Edition:
Manual.* San Antonio: The Psychological Corporation.

Weisz, J.R. (2004). *Psychotherapy for children and adolescents.* New York:
Cambridge University Press.

Welsh, M.C., & Pennington, B.F. (1988). Assessing frontal lobe functioning in
children: Views from developmental psychology. *Developmental
Neuropsychology, 4*(3), 199-230.

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005).
Validity of the executive function theory of attention-deficit/hyperactivity
disorder: A meta-analytic review. *Biological Psychiatry, 57*, 1336-1346.

Willoughby, M., Curran, P.J., Costello, E.J., & Angold, A. (2000). Implications of

early vs. late onset of attention deficit hyperactivity disorder symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 1512-1519.

Willoughby, M., Kupersmidt, J., & Bryant, D. (2001). Overt and covert dimensions of antisocial behavior in early childhood. *Journal of Abnormal Psychology, 29*, 177-187.

Zelazo, P. D. & Mueller, U. (2002). Executive functions in typical and atypical development. In U. Goswami (Ed.), *Handbook of Childhood Cognitive Development* (pp. 445-469). Oxford: Blackwell.

Zelazo, P. D., Mueller, U., Frye, D., & Marcovitch, S. (2003). The development of executive function in early childhood. *Monographs of the Society for Research in Child* Development, 68(3), 93-119.