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# Anxiety and Conduct Problems in Children and Adolescents: The Role of Executive Functioning in a Dual-Pathway Model

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# Anxiety and Conduct Problems in Children and Adolescents: The Role of Executive Functioning in a Dual-Pathway Model

**by**

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# Dedication

To my parents, Pam and Gary, who taught and encouraged me to work hard, work passionately, and always do my best. All of my accomplishments were made possible by the foundation you gave me.

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# Anxiety and Conduct Problems in Children and Adolescents: The Role of Executive Functioning in a Dual-Pathway Model

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Although anxiety disorders and conduct problems often co-occur in children and adolescents, literature describing the effects of such co-occurrence is mixed. There is evidence that symptoms of anxiety disorders may mitigate symptoms of conduct problems (buffering hypothesis) or may exacerbate symptoms of conduct problems (multiple problem hypothesis). A dual-pathway model has been proposed that suggests several possible etiological or risk processes that may differentiate these pathways (i.e., the buffering hypothesis or the multiple problem hypothesis) (Drabick, Ollendick, & Bubier, 2010). Executive functioning is one factor that has been identified that may differentially confer risk to the proposed pathways; however, little research has been done investigating its role. The purpose of the present study was to evaluate the dual-pathway model by determining whether executive functioning abilities contribute to differentiating those youth for whom anxiety exacerbates conduct problems from those for whom anxiety mitigates conduct problems. Specifically, the study sought to examine if executive functioning moderated the effect of anxiety symptom severity on conduct

problems. Latent variable structural equation modeling (SEM) was used to analyze the data of 221 youth aged 9 to 16 in a residential treatment center who completed a full neuropsychological evaluation. Results of the study failed to support the hypothesis that executive functioning moderates the effect of anxiety on conduct problems. Furthermore, a structural equation model without an interaction between executive functioning and anxiety was found to fit the data better than a model with an interaction between those variables. Overall, the study found that executive functioning abilities could not distinguish youth for whom anxiety exacerbates conduct problems from youth for whom anxiety mitigates conduct problems. Recommendations for future research in light of the limitations of the current study, as well as remaining gaps in the literature, are discussed.

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# Chapter I: Introduction

Anxiety disorders and conduct problems (including the diagnoses of Oppositional Defiant Disorder [ODD] and Conduct Disorder [CD]) are some of the most prevalent mental health problems affecting children and adolescents. It is estimated that over 30% of youth will experience an anxiety disorder and nearly 20% of youth will be diagnosed with ODD or CD during childhood or adolescence (Merikangas et al., 2010). Many children will experience both anxiety disorders and conduct problems as estimates of rates of anxiety disorders among youth with either CD or ODD range as high as 40% (Angold, Costello, & Erklani, 1999). Research suggests that such comorbidity may be linked to a variety of negative outcomes including more severe clinical presentations, increased academic problems, social impairment, family dysfunction, and elevated risk for additional psychological conditions both in childhood and adulthood (Drabick, Gadow, & Loney, 2008; Fergusson & Horwood, 1999; Greene et al., 2002; Kendall, Brady, & Verduin, 2001; Maser & Cloninger, 1990; Merikangas & Avenevoli, 2000).

Despite these findings, research examining the effect of anxiety on the expression of conduct problems is inconclusive. Studies have shown that anxiety serves to reduce the severity of conduct problems in some individuals (buffering hypothesis) (Hofmann, Richey, Kashdan, & McKnight, 2009; Walker et al., 1991) and increase the severity of conduct problems in others (multiple-problem hypothesis) (Garai, Forehand, Colletti, & Rakow, 2009; Lansford et al., 2008; Ollendick, Seligman, & Butcher, 1999). Factors that differentiate the two groups may be largely neurological; however, at this time, such factors have only been hypothesized indicating a large gap in research. Knowledge of these factors could have important consequences for the assessment and intervention of youth with comorbid anxiety disorders and conduct problems.

Several theories have been proposed to explain the etiology of co-occuring anxiety disorders and conduct problems. These theories generally propose that either one disorder precedes the other or that shared risk factors lead to comorbidity (Drabick, Ollendick, & Bubier, 2010). Longitudinal research on the emergence of anxiety disorders and conduct problems is mixed with some studies finding that conduct problems may precede anxiety disorders (Burke, Loeber, Lahey, & Rathouz, 2005; Lavigne et al., 2001; Roza, Hofstra, van der Ende, & Verhulst, 2003; Speltz, McClellan, DeKlyen, & Jones, 1999) and other studies showing that anxiety disorders may precede conduct problems (Foley, Pickles, Maes, Silberg, & Eaves, 2004; Last, Perrin, Hersen, & Kazdin, 1996). It appears that the association between these classes of disorders is not as straightforward and linear as researchers have assumed.

Anxiety disorders and conduct problems do share many risk factors which may contribute to their co-occurrence (Bubier & Drabick, 2009; Muris & Ollendick, 2005). Parental characteristics such as parental psychopathology (Weems & Silverman, 2008) and low levels of parental education (Merikangas et al., 2010) have been associated with increased risk for anxiety disorders and conduct problems. Rates of both classes of disorders have been found to be higher for adolescents whose parents are divorced or separated than for adolescents with cohabitating or married parents (Merikangas et al., 2010). Exposure to trauma and violence, including experiencing physical and sexual abuse, has been linked to the development of both anxiety disorders and conduct problems. Similarly, low levels of social support, poverty, and lower cognitive abilities, especially lower verbal intelligence, appear to be shared risk factors (Beitchman et al., 2001; Kristensen & Torgersen, 2008; Weems & Silverman, 2008). Additionally, there is evidence that non-Hispanic white youth are at decreased risk of developing both anxiety disorders and conduct problems (Cameron & Guterman, 2007; Ginsburg &

Silverman, 1996; Harden et al., 2009; Merikangas et al., 2010). These findings provide more consistent support for hypotheses suggesting that common risk factors may be responsible for the co-ocurrence of anxiety disorders and conduct problems; however, they fail to answer the primary question associated with the comorbidity of these disorders: Why is it that anxiety mitigates conduct problems in some individuals and exacerbates it in others?

Drabick, Ollendick, and Bubier (2010) have proposed an integrated dual-pathway model to explain the differential effects of anxiety on conduct problems. Their model is specific to children with anxiety disorders and ODD; however, it appears to be applicable to comorbid anxiety disorders and CD as well given the commonalities that exist between ODD and CD (e.g., risk factors, neurological processes). The authors hypothesize that what differentiates the subgroup of children for whom anxiety mitigates conduct problems from the subgroup of children for whom anxiety exacerbates conduct problems is primarily neurological functioning.

The limbic system, especially the amygdala, and the prefrontal cortex (PFC) have been implicated in playing important roles in the internal experience and external expression of both anxiety and conduct problems. The amygdala and the PFC have a reciprocal relationship as they work together to process information from the environment and produce behaviors in the individual. The amygdala evaluates the emotional significance of stimuli after receiving input from the cortex, hippocampus, and thalamus. It then transmits information to other parts of the brain including the PFC. The PFC is involved in behavior choice and uses the information from the amygdala to plan the individual's response to the stimuli.

Children and adolescents with anxiety disorders have been consistently found to display heightened levels of amygdala activity compared to normal controls (Guyer, et al., 2008; Thomas et al., 2001). In contrast, youth with non-comorbid conduct problems have been found to exhibit

amygdala hypoactivity (Davidson, Putnam, & Larson, 2000; Jones, Laurens, Herba, Barker, & Viding, 2009; Marsh et al., 2008). PFC dysfunction has been associated with both anxiety disorders and conduct problems (Finger et al., 2008; Isikawa & Raine, 2003; Lenroot et al., 2007; Monk et al., 2006; Raine, 2002).

The association between anxiety and conduct problems becomes clearer upon examining research regarding the co-activation of the amygdala and the PFC. Amygdala activation has been positively correlated with anxiety symptom severity in youth (Thomas et al., 2001), while PFC activation has been negatively correlated with anxiety symptom severity (Monk et al., 2006). Additionally, amygdala-PFC co-activation has been positively associated with the presence of anxiety disorders (Guyer et al., 2008), and it also has been linked to conduct problems. Specifically, greater levels of amygdala-PFC connectivity have been correlated with lower levels of ODD and CD symptom severity (Marsh et al., 2008). These findings suggest that developmentally appropriate PFC activity and amygdala-PFC connectivity may serve to modulate amygdala activity in children and adolescents with anxiety disorders and conduct problems, resulting in less severe symptoms. This hypothesized attenuation of overactive amygdala functioning in children with comorbid anxiety disorders and conduct problems is central to the dual-pathway model proposed by Drabick and colleagues (2010).

Drabick and colleagues (2010) hypothesized that the subgroup of youth for which anxiety mitigates conduct problems exhibits amygdala overactivity and high levels of negative emotionality, as these two things have consistently been found to be associated with anxiety disorders. For these individuals, however, it is hypothesized that age-appropriate PFC functioning modulates the heightened amygdala activity and negative emotionality resulting in

fewer impairments and less severe presentations of conduct problems than in youth in the other subgroup.

Youth in the second subgroup – the subgroup for which anxiety exacerbates conduct problems – are expected to exhibit decreased responsiveness in the PFC, which renders the PFC ineffective in modulating amygdala overactivity. This is hypothesized to result in higher levels of anger and reactive aggression; difficulties with social-information processing; lower levels of self-control; and poorer executive functioning abilities. Such vulnerabilities could, in turn, lead to problems with emotion regulation, planning, problem solving, self-monitoring, and behavioral selection, increasing the risk of these individuals engaging in the behaviors associated with conduct problems.

Although the literature supports the proposed dual-pathway model, research examining the model's accuracy is lacking. Research on the role of executive functioning in the model appears to be particularly necessary, as it is often considered to be an expression of PFC functioning (Baron, 2004). The construct of executive functioning is broad and variously defined. It is generally accepted that executive functioning refers to a variety of skills and abilities that are critical for engaging in goal-directed activity. Executive functioning abilities (also termed subdomains) are higher order in that they integrate more basic skills such as attention, memory, and perception (Baron, 2004). There is considerable variability in the literature as to the specific number and composition of executive functioning subdomains; however, the subdomains most frequently endorsed based on empirical research include planning, working memory, self-monitoring, cognitive flexibility, fluency, attention (sustained and divided), and response inhibition (Baron, 2004; Henry & Bettenay, 2010).

As previously mentioned, executive functioning has been consistently associated with functioning of the PFC (Baron, 2004). In children and adolescents, PFC dysfunction has been linked to impairments in cognitive flexibility, response inhibition, and working memory (Baron, 2004; Kim, Kim, & Kwon, 2001). Additionally, PFC activation has been reported during engagement in verbal fluency tasks (Ravnkilde, Videbech, Rosenberg, Gjedde, & Gade, 2002). These findings support the proposition that measures of executive functioning can reflect PFC functioning, and therefore be useful in testing the dual-pathway model of comorbid anxiety disorders and conduct problems.

Regarding the association between executive functioning and anxiety, research is lacking and that which does exist is inconclusive. One study failed to find a significant difference between adults with anxiety disorders and control participants on measures of executive functioning (Castaneda et al., 2011), while another found no correlation between anxiety symptom severity and executive functioning performance in an adult psychiatric sample (Smitherman, Huerkamp, Miller, Houle, & O'Jile 2007). Some studies with adults suggest that anxiety may be negatively associated with executive functioning abilities; however, only with the executive functioning subdomains of verbal fluency, divided attention, and spatial working memory (Gass, Ansley, & Boyette, 1994; Lautenbacher, Spernal, & Krieg, 2002; Purcell, Maruff, Kyrios, & Pantelis, 1998).

Research on children and adolescents is also limited and mixed. Several studies have failed to find differences between youth with anxiety disorders and normal controls on measures of inhibition, cognitive flexibility, performance monitoring, processing speed, and attention (Korenblum, Chen, Manassis, & Schachar, 2007; Kusché, Cook, & Greenberg, 1993; Shin et al., 2008). Other studies have found that anxious youth perform worse than normal controls on

measures of set-shifting, inhibition, cognitive flexibility, verbal fluency (Kusché et al., 1993; Shin et al., 2008; Tapasak, Roodin, & Vaught, 1978).

More research exists that examines the association between executive functioning and conduct problems, yet the results are also inconclusive. Research in young children, as well as older children and adolescents, has found poor inhibitory control to be associated with conduct problems and aggressive behaviors (Fisher, Barkley, Smallish, & Fletcher, 2005; Hughes & Ensor, 2011; Kusché et al., 1993; Raaijmakers et al., 2008; Utendale & Hastings, 2011; Utendale, Hubert, Saint-Pierre, & Hastings, 2011; Young et al., 2009; Youngwirth, Harvey, Gates, Hashim, & Friedman-Weieneth, 2007). Youth with conduct problems have also been found to display impairments in processing speed, attention, shifting, motor inhibition, cognitive flexibility, verbal fluency, working memory, and response execution (Hobson, Scott, & Rubia, 2011; Kim et al., 2001; Kusché et al., 1993; Närhi, Lehto-Salo, Ahonen, & Marttunen, 2010; Pajer et al., 2008; Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999).

In contrast to these findings, a number of studies have reported no significant differences between the performance of youth with conduct problems and control participants on measures of verbal fluency, attention, working memory, planning abilities, inhibition, and cognitive flexibility (Hobson et al., 2011; Kim et al., 2001; Kusché et al., 1993; Närhi et al., 2010; Oosterlaan, Scheres, & Sergeant, 2005; Pajer et al., 2008; Speltz et al., 1999; Thorell & Wåhlstedt, 2006). Clearly, the relationship between executive functioning and conduct problems requires further investigation.

The diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) appears to significantly affect the association between executive functioning and conduct problems. Two meta-analyses found significant associations between ADHD and executive functioning, with

subjects diagnosed with ADHD performing significantly worse than controls on measures of planning, interference control, response inhibition, motor inhibition, working memory, vigilance, set-shifting, planning, and processing speed (Pennington & Ozonoff, 1996; Willicutt, Doyle, Nigg, Faraone, & Pennington, 2005). Research has also found ADHD to be associated with CD and ODD in that a diagnosis of ADHD increased the odds of developing conduct problems and predicted the onset of ODD (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; van Lier, van der Ende, Koot, and Verjulst, 2007)

It appears as though only one study (Kusché, Cook, & Greenberg, 1993) has examined executive functioning abilities in children with co-occuring anxiety disorders and conduct problems, and its findings were mixed. Although children with comorbid anxiety and externalizing disorders displayed impairments in verbal fluency compared to control participants, no significant differences were found between the two groups on a measure of cognitive flexibility. Research on this topic is clearly inconclusive and leaves one of the most important questions raised by the dual-pathway model unanswered: Does executive functioning moderate the effect of anxiety on conduct problems?

The purpose of this study was to evaluate the dual-pathway model by determining whether executive functioning abilities contribute to differentiating those youth for whom anxiety exacerbates conduct problems from those for whom anxiety mitigates conduct problems. Specifically, this study examined the nature of the associations between anxiety symptom severity, executive functioning abilities, and conduct problem symptom severity using latent variable structural equation modeling. This method of analysis was utilized in order to simultaneously analyze the associations between the specified latent variables (Anxiety, Executive Functioning, and Conduct Problems), including the interaction between Anxiety and

Executive Functioning. Another advantage of using the latent variable approach was that measure invalidity and error was minimized in order to achieve a more accurate representation of the constructs of interest (Miyake et al., 2000). Examining the associations between these constructs not only provides a test of the validity of the dual-pathway model, but also contributes to furthering the understanding of the importance of considering executive functioning abilities when designing assessments and interventions for children suspected of having comorbid anxiety disorders and conduct problems.

## *Research Questions and Hypotheses*

### *Research Question 1*

Does executive functioning moderate the effect of anxiety on conduct problems?

*Hypothesis 1.* It is hypothesized that executive functioning will moderate the effect of anxiety on conduct problems as indicated by a statistically significant effect of the interaction latent variable, Executive Functioning x Anxiety, on the Conduct Problems latent variable.

### *Research Question 2*

Does an interaction model including the Executive Functioning x Anxiety latent variable better explain the associations between anxiety symptom severity, executive functioning abilities and conduct problems than a model that only examines the direct effects of anxiety and executive functioning on conduct problems?

*Hypothesis 2.* It is hypothesized that an interaction model including the Executive Functioning x Anxiety latent variable will better explain the associations between anxiety symptom severity, executive functioning abilities, and conduct problems than a model that only examines direct effect as indicated by more favorable model fit statistics for the interaction model than the no interaction model.

# Chapter II: Method

## *Participants*

This study will use an archival neuropsychological dataset of female and male children and adolescents between the ages of 9 and 16. Participants were obtained from a residential treatment center (RTC) in Texas where they received inpatient psychiatric treatment between the years 2000-2009. All participants were admitted to the RTC due to a history of severe emotional and behavioral disturbance. Upon admission to the RTC, each child and adolescent participated in a full neuropsychological evaluation as part of routine clinical care. Individuals were selected for inclusion in the current study if the following criteria were met: (a) they were between the ages of 9 and 16 at the time of neuropsychological evaluation; (b) they were administered the Multidimensional Anxiety Scale for Children (MASC; March, 1997) as part of the evaluation; and (c) they achieved a Verbal IQ (VIQ) score of 80 or greater on the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). Of the 667 participants in the dataset, 106 did not meet the age criteria. Of the remaining 561 participants, 300 were not administered the MASC. Of the remaining 261 participants, 40 did not meet the VIQ criteria. As a result, 221 participants were selected for inclusion in this study.

Demographic characteristics and relevant diagnosis-related information for the current sample are presented in Table 1. Participants' ages ranged from 9 to 16, with a mean age of 13.78 years (SD = 2.14). Males represented a greater proportion of the sample (54.3%; $n$ = 120) than females (45.7%; $n$ = 101). The majority of the participants were Caucasian (68.8%), followed by Other (11.3%), African-American (9.5%), Hispanic (9.0%), Asian/Pacific Islander (.5%), and Native American (.5%).

Most of the participants did not have a diagnosis of ADHD (no diagnosis = 81%; diagnosis = 19%). Eighty-one participants (36.7%) were diagnosed with Conduct Disorder and 37 (16.7%) participants were diagnosed with Oppositional Defiant Disorder. 90 (40.7%) participants has Legal Problems indicated on Axis IV of their five axis diagnosis, while 126 (57.0%) did not.

Table 1

*Demographic characteristics and relevant diagnosis-related information*

| **Variable** | ***M*** | **SD** | **Range** | ***n*** | **Percentage** |
|---|---|---|---|---|---|
| Age (years) | 13.78 | 2.14 | 9-16 | | |
| Sex | | | | | |
| Female | | | | 101 | 45.7 |
| Male | | | | 120 | 54.3 |
| Race/Ethnicity | | | | | |
| African-American | | | | 21 | 9.5 |
| Asian/Pacific Islander | | | | 1 | .5 |
| Caucasian | | | | 152 | 68.8 |
| Hispanic | | | | 20 | 9.0 |
| Native American | | | | 1 | .5 |
| Other | | | | 25 | 11.3 |
| Unavailable | | | | 1 | .5 |
| ADHD | | | | | |
| Diagnosis | | | | 42 | 19.0 |
| No diagnosis | | | | 179 | 81.0 |
| Conduct Disorder | | | | | |
| Diagnosis | | | | 81 | 36.7 |
| No diagnosis | | | | 140 | 63.3 |
| Oppositional Defiant Disorder | | | | | |
| Diagnosis | | | | 37 | 16.7 |
| No diagnosis | | | | 184 | 83.3 |
| Axis IV Legal Problems | | | | | |
| Diagnosis | | | | 90 | 40.7 |
| No Diagnosis | | | | 126 | 57.0 |
| Unavailable | | | | 5 | 2.3 |

*Instrumentation*

A subset of measures was chosen from the full neuropsychological test battery administered to participants in order to represent the latent constructs of interest in this study. These measures are described in the following section. Table 2 includes a list of the selected indicators organized by theoretical construct.

Table 2

*Latent constructs and associated indicators*

| **Latent Construct** | **Indicators** |
|---|---|
| IQ | WASI VIQ |
| | WASI PIQ |
| Anxiety | MASC PSS |
| | MASC SAS |
| | MASC HAS |
| | MASC SPS |
| Executive Functioning | COWAT |
| | Trails A |
| | Trails B |
| | WCST |
| Conduct Problems | MACI CC |
| | MMPI-A A-con |
| | M-PACI D |
| | CD/ODD |
| | Legal Problems |

*Measure of Intelligence*

*Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999).* The WASI is an individually administered intelligence test that provides a measure of general cognitive functioning for individuals aged 6 to 89. It provides a Verbal IQ (VIQ) score, Performance IQ (PIQ) score, and Full Scale IQ-4 (FSIQ-4) score that appears to be a valid and reliable estimate of global ability (Wechsler, 1999). The VIQ is derived from scores on the Vocabulary and

Similarities subtests, while the PIQ is derived from scores on the Block Design and Matrix Reasoning subtests. The FSIQ-4 utilizes all four subtests (Vocabulary, Block Design, Similarities, and Matrix Reasoning) that have high factor loadings (e.g., >.70) on *g*, or general intellectual ability (Wechsler, 1999).

Reliability estimates for both VIQ and PIQ were excellent with split-half reliability coefficients ranging from .92 to .95 in the children's standardization sample that included 1,100 youth ages 6 to 16 (Wechsler, 1999). Test-retest stability coefficients were .92 for VIQ and .88 for PIQ in a sample of 116 children (Wechsler, 1999).The WASI VIQ was found to be highly correlated with the Wechsler Intelligence Scale for Children – Third Edition (WISC-III; Wechsler, 1991) VIQ ($r$ = .82; Kamphaus, 2005) in a nonclinical sample of 176 children ages 6 to 16. Similarly, the WASI PIQ was found to be highly correlated with the WISC-III PIQ ($r$ = .76; Kamphaus, 2005) in the same sample suggesting good validity. The VIQ and PIQ will be used as indicators for the intelligence latent background variable to control for participants' level of intelligence in the structural equation model.

*Measures of Anxiety*

*Multidimensional Anxiety Scale for Children (MASC; March, 1997).* The MASC is a standardized 39-item self-report questionnaire intended to assess a cross-section of anxiety problems in individuals aged 8 to 19. The measure was standardized using a normative sample of 2,698 racially diverse male and female youth aged 8 to 19. Each item is rated on a 4-point Likert scale ranging from "never true about me" to "often true about me." The measure provides a Total Anxiety Index which appears to be a valid and reliable estimate of anxiety symptoms across clinically important symptom domains, as well as four scales (Physical Symptoms Scale, Social

Anxiety Scale, Harm Avoidance Scale, and Separation/Panic Scale), and two indices (Anxiety Disorders Index and Inconsistency Index).

The Physical Symptoms Scale (PSS) assesses how often youth experience physical symptoms typically associated with anxiety (e.g., feeling tense, dizzy, shaky, sweaty, etc.). The scale has two subscales that differentiate between tension symptoms (Tense Symptoms Subscale) and somatic symptoms (Somatic Symptoms Subscale). The Social Anxiety Scale (SAS) measures to what degree youth worry about others making fun of them, embarrassing themselves, or performing in front of others. The scale has two subscales that differentiate between fear of performance (Performance Fears Subscale) and fear of humiliation (Humiliation Fears Subscale). The Harm Avoidance Scale (HAS) assesses how often youth extend extra effort to do things correctly and follow rules and expectations. The scale has two subscales that differentiate between anxiety coping (Anxious Coping subscale) and perfectionism (Perfectionism Subscale). Lastly, the Separation/Panic Scale (SPS) measures to what degree youth become scared when they are alone, in unfamiliar situations, or separated from family members.

Scores for the MASC include raw scores and age-based *T*-scores for each index and scale. The internal reliability coefficients for the Total Anxiety Index ranged from .88 to .89 in the normative sample (March, 1997). The test-retest reliability coefficient for the Total Anxiety Index was found to be .93. The Total Anxiety Index was found to be moderately correlated with the Revised Children's Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1978) based on a sample of 24 children and adolescents suggesting adequate validity (March , Parker, Sullivan, Stallings, & Conners, 1997).

Internal reliability coefficients for the various MASC scales were determined using the normative sample (March, 1997). Internal reliability coefficients for the PSS ranged from .79 to .87, the SAS ranged from .79 to .86, the HAS ranged from .61 to .71, and the SPS ranged from .62 to .71. Test-retest reliability coefficients for the scales were in the satisfactory to excellent range (r = .70 to .93) in a sample of 24 children (March et al., 1997). Intercorrelations between the various MASC scales ranged from .16 to .56 in the normative sample (March, 1997). The weakest correlation ($r$ = .16) was between the PSS and the HAS using the female normative sample. The correlation between the PSS and the HAS was also the weakest ($r$ = .22) of those calculated using the male normative sample. The strongest correlation ($r$ = .56) was between the PSS and the SAS using the male normative sample. The correlation between the PSS and the SAS was also the strongest ($r$ = .45) of those calculated using the female normative sample

PSS, SAS, HAS, and SPS *T*-scores will be used as indicators for the anxiety latent variable in the hypothesized model.

*Measures of Executive Functioning*

*Controlled Oral Word Association Test (COWAT).*The COWAT is an individually administered test assessing the executive functioning subdomain of verbal fluency which incorporates the abilities to inhibit, self-monitor, and shift (Baron, 2004). The COWAT is a test of letter fluency which assesses the ability to produce words in response to a letter cue. There are multiple variations of this test: F-A-S, C-F-L, and P-R-W. The tests vary in the letters that are provided to the examinee as cues. For example, in the F-A-S variation, the examinee is asked to produce words beginning with the letters F, A, and S. In each version examinees are administered three 60-second trials – one trial for each letter. For each trial, the examinee is asked to generate as many words as possible in response to the letter cue. Inadmissible words

include proper names, intrusions, variations, and repetitions. The most common variation of the test, and the one used in this study, is the F-A-S form.

Scores on the COWAT include age-based *z*-scores based on the number of admissible words for each trial, age-based *z*-scores based on the total number of correct words for all three trials, and sum scores of intrusions and repetitions. Test-retest reliability coefficients ranged from .67 to .88 in various samples (Spreen & Strauss, 1998).The COWAT F-A-S form has been found to be equivalent to other forms of the COWAT, with correlations ranging from .87 to .94 (Lacy et al., 1996). Performance on verbal fluency tests has been found to be correlated with vocabulary, intelligence and ethnicity (O'Bryant & O'Jile, 2004). The *z*-score based on the total number of correct words for all three trails will be used in this study to assess the latent construct of executive functioning.

*Trail Making Test (TMT; Reitan, 1958).* The TMT is a timed individually administered paper-and-pencil test measuring the executive functioning subdomains of cognitive flexibility (i.e., sustain and shift) and inhibition, processing speed and attention (Kelly, 2000). The test consists of two parts: Trails A and Trails B. Trails A requires the child to connect a series of numbered targets scattered across a page in sequential order. Trails B is a more complex task in that it requires the child to alternate between connecting numbers and letters in sequence. Children age 14 and younger use a form called the Reitan Intermediate version with 15 numbers on Trails A and a total of 15 numbers and letters on Trails B. Examinees aged 15 or older complete the adult version with 25 numbers on Trails A and a total of 25 numbers and letters on Trails B. Examinees are instructed to complete the tasks as quickly as possible without making a mistake. During administration, examinee errors are immediately pointed out by the examiner

and correction is requested. The standard administration includes a practice trial for each condition.

Scores on this test include age-based *z*-scores based on completion time in seconds and sum scores of errors for each part. Although comprehensive normative data for the TMT is not available, norms for children ages 7-13, adolescents, and adults age 18-89 are available. Reported reliability coefficients ranged from the .60s to over .90 with the majority above .80 in a variety of samples (Lezak, Howieson, Loring, Hannay, & Fischer, 2004).

Trails A is recognized as a measure of processing speed and attention involving visual scanning and sequencing. In addition to processing speed and attention, Trails B involves divided attention, inhibitory control, and cognitive flexibility in that it requires examinees to simultaneously attend to two different task demands and continuously shift between them (Strauss, Sherman, & Spreen, 2006). *Z*-scores for Trails A and Trails B will be used as indicators for the executive functioning latent variable in the hypothesized model.

*Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993).* The WCST is recognized as a test of executive functioning measuring cognitive flexibility including the abilities of concept generation, cognitive set shifting, inhibition, abstract reasoning, hypothesis testing and problem solving, and sustained attention (Baron, 2004). During this individually administered test, four stimulus cards reflecting three stimulus parameters (Color, Form and Number) are placed in front of the examinee. Each response card displays figures of varying forms (circles, crosses, stars, or triangles), colors (blue, green, red, or yellow), and numbers of figures (one, two, three, or four). The examinee is asked to sequentially match response cards by placing each one in a pile beneath one of the four stimulus cards. Each response cards can be matched to a stimulus card based on one or a combination of multiple

stimulus patterns. Following each response, the examinee is given feedback regarding whether the response was "right" or "wrong;" however, the correct sorting principle or "category" is never revealed to the examinee.

The WCST provides a variety of scores based on test results including number of correct responses, number of errors, number of perseverative responses, number of non-perseverative responses, number of perseverative errors, and number of non-perseverative errors. The WCST manual provides normative data for children ages 6 to 17 years old based on a sample of 453 "normal" racially diverse male and female children and adolescents (Heaton et al., 1993). Generalizability coefficients for WCST scores based on a single test administration ranged from .39 to .72 in a subset of 46 children and adolescents from the standardization sample (Heaton et al., 1993). The WCST perseverative error score had a generalizability coeffeicient of .52 indicating moderate reliability in this sample.

Studies have found the correlation between specific WCST scores to be high ($r$ = .63 to.88; $M$ = .80), indicating that they share considerable common variance (O'Donnell, MacGregor, Dabrowski, Oestreicher, & Romero, 1994); however, factor structure studies have associated the total number of perseverative errors with the executive functioning domain of cognitive flexibility (Greve, Brooks, Crouch, Williams, & Rice, 1997). The total number of perseverative errors will be used in this study to assess the latent construct of executive functioning.

*Measures of Conduct Problems*

*Millon Pre-Adolescent Clinical Inventory (M-PACI; Millon, Tringone, Millon, & Grossman, 2005).* The M-PACI is a standardized 97-item self-report questionnaire designed to assess emerging personality characteristics and clinical symptoms in youth aged 9 to 12. The

measure includes 14 profile scales within two categories: Emerging Personality Patterns (7 scales) and Current Clinical Signs (7 scales). Additionally, there are two response validity indicators (Invalidity and Response Negativity). Within the Current Clinical Signs category is Scale D: Conduct Problems. This scale assesses the extent to which youth display an antisocial behavior pattern characterized by hostile, antisocial, and violent behaviors toward peers and others. Youth with high scores on this scale display a pattern of noncompliance, overt bullying, manipulative anger, and empathic deficits, as well as a lack of respect for the rights of others.

Scores for Scale D: Conduct Problems include raw scores and base rate scores. The internal consistency coefficients for the scale was found to be .79 based on a subsample of 100 children from the standardization sample that included 292 racially/ethnically diverse male and female children aged 9 to 12 (Millon, 2005). The scale was moderately correlated with the Behavior Assessment System for Children: Self-Report of Personality Form C (BASC SRP-C; Reynolds & Kamphaus, 1998) Attitude to School, Attitude to Teachers, and Relations with Parents scales, as well as the School Maladjustment composite indicating adequate validity. The Scale D: Conduct Problems base rate score will be used in this study as an indicator for the conduct problems latent variable. The M-PACI was administered to participants aged 9 to 12 in the existing dataset.

*Millon Adolescent Clinical Inventory (MACI; Millon, 1993).* The MACI is a standardized 160-item self-report questionnaire designed to assess personality characteristics and clinical syndromes in adolescents aged 13 to 19. The measure includes 31 scales within four categories: Personality Patterns (12 scales), Expressed Concerns (8 scales), Clinical Syndromes (7 scales), and Modifying Indices (4 scales). Within the Clinical Syndromes category is Scale CC: Delinquent Predisposition. This scale assesses the extent to which the examinee's behavior has

led or is likely to lead to situations in which the rights of others are violated, and societal norms and rule are broken. High scorers on this scale are likely to have engaged in a number of conduct problem behaviors including aggression to people and others, deceitfulness, theft, and truancy.

Scores for Scale CC: Delinquent Predisposition include raw scores and base rate scores. Internal consistency coefficients for the scale ranged from .76 to .77 in the standardization sample that included 1,017 racially/ethnically diverse male and female adolescents in a variety of treatment settings (Millon, 1993). The test-retest reliability coefficient for Scale CC: Delinquent Predisposition was found to be .80. The scale was moderately correlated with the Aggressive Behavior/Delinquency subscale of the Problem Oriented Screening instrument for Teenagers (POSIT; Rahdert, 1991) indicating adequate validity. The Scale CC: Delinquent Predisposition base rate score will be used in this study as an indicator for the conduct problems latent variable. The MACI was administered to participants aged 13 to 16 in the existing dataset.

*Minnesota Multiphasic Personality Inventory – Adolescent (MMPI-A; Butcher et al., 1992).* The MMPI-A is a standardized 478-item self-report questionnaire developed to measure personality characteristics, psychological symptoms, and behavior problems in adolescents aged 14 to 18. The measure is comprised of 7 Validity scales, 10 Clinical Scales, 31 Clinical Subscales, 15 Content Scales, 31 Content Component Subscales, and 11 Supplementary Scales. The Content Scale, A-con (Adolescent-Conduct Problems), measures the extent to which the examinee has engaged in a variety of conduct problem behaviors including stealing, lying, property destruction, and being oppositional.

Scores for A-con scale include raw scores and *T*-scores. Internal consistency coefficients for the scale ranged from .72 to .79 in the standardization sample that included 805 adolescents representing a variety of geographic regions and ethnicities (Butcher et al., 1992). The test-retest

reliability coefficient for the A-con scale was found to be .62. The A-con *T*-score will be used in the proposed study to assess the latent construct of conduct problems. The MMPI-A was administered to participants aged 14 to 16 in the existing dataset.

### *Procedure*

This study was conducted in compliance with the ethical principles and standards of research set forth by the American Psychological Association and The University of Texas at Austin. The study was approved by the Departmental Review Committee of the Department of Education Psychology at the University of Texas at Austin and by the Institutional Review Board at The University of Texas at Austin (IRB Protocol # 2012-03-0036).

Data for this study were obtained from an existing dataset of child and adolescent psychiatric inpatients who participated in a full neuropsychological evaluation as part of their routine care at a residential treatment center (RTC) in Texas. At admission, written consent was obtained in order to use the participants' de-identified data for research purposes. As part of a comprehensive neuropsychological evaluation, standardized tests measuring intelligence, executive functioning abilities, psychological symptoms, and behavior problems were administered to each participant individually. All testing was completed by a neuropsychologist or supervised doctoral student, trained in standardized administration and scoring procedures. Upon completion of the evaluation, each participant was diagnosed by a licensed psychologist and board certified child and adolescent psychiatrist according to the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision (DSM-IV-TR; American Psychological Association [APA], 2000) multi-axial assessment system. Demographic data including age, sex, and race/ethnicity were included in the preexisting dataset. Participant sex

will be used in the analyses in order to minimize potential confounds and to account for potential common causes.

### *Hypothesized Model*

Latent variable structural equation modeling (SEM) was used to determine the direct effects of anxiety symptom severity and executive functioning abilities on conduct problems, as well as the moderating effect of executive functioning abilities on conduct problems. An advantage of using latent variable SEM is it removes the effects of unreliability and invalidity when analyzing the effect of one variable on another, thereby reducing the problem of imperfect measurement (Keith, 2006). The removal of measurement error allows the constructs of interests to be more accurately represented and the effects of one variable on another to be more accurately estimated.

The hypothesized latent variable SEM model, shown in Figure 1, was developed to assess the theoretically-based constructs of executive functioning and conduct problems, and to investigate research-driven hypotheses regarding the relations among these constructs and anxiety. Measured variables (also termed indicator, observed, or manifest variables) are portrayed graphically in SEM with squares and rectangles. Latent variables (also referred to as factors, constructs, or unobserved variables) are portrayed graphically with ovals and circles.

As depicted in the model, a relevant demographic characteristic (sex), as well as psychometric intelligence (represented by the IQ latent variable), and ADHD diagnosis status were controlled statistically in the model. The indicators of the IQ latent variable are VIQ and PIQ. Sex and ADHD are categorical variables (sex = female or male; ADHD = no diagnosis or diagnosis).

The Anxiety latent variable was measured using the following indicator variables from the MASC: Physical Symptoms Scale (PSS), Social Anxiety Scale (SAS), Harm Avoidance Scale (HAS), and Separation/Panic Scale (SPS). The Executive Functioning latent variable was measured using the following indicator variables: COWAT, Trails A, Trails B, and WCST. The Conduct Problems latent variable was measured using the indicator variables M-PACI D, MACI CC and MMPI-A A-con, in addition to two categorical indicator variables: Conduct Disorder/Oppositional Defiant Disorder (CD/ODD) and Legal Problems. These two dummy-coded indicator variables represented whether each participant received a DSM-IV-TR diagnosis of Conduct Disorder or Oppositional Defiant Disorder and whether legal problems were indicated on Axis IV of their diagnosis. The Executive Functioning x Anxiety latent variable represents the interactive effect of anxiety symptom severity and executive functioning abilities.

The small circles labeled d1-d3 indicate disturbances (also called residuals), that represent all other sources of influence on the variables apart from those included in the model. The small circles labeled e1-e15 are error terms representing the effect of all other influences on the measured variable beside the latent construct it is intended to measure, including the effects of measurement error (unreliability and invalidity).

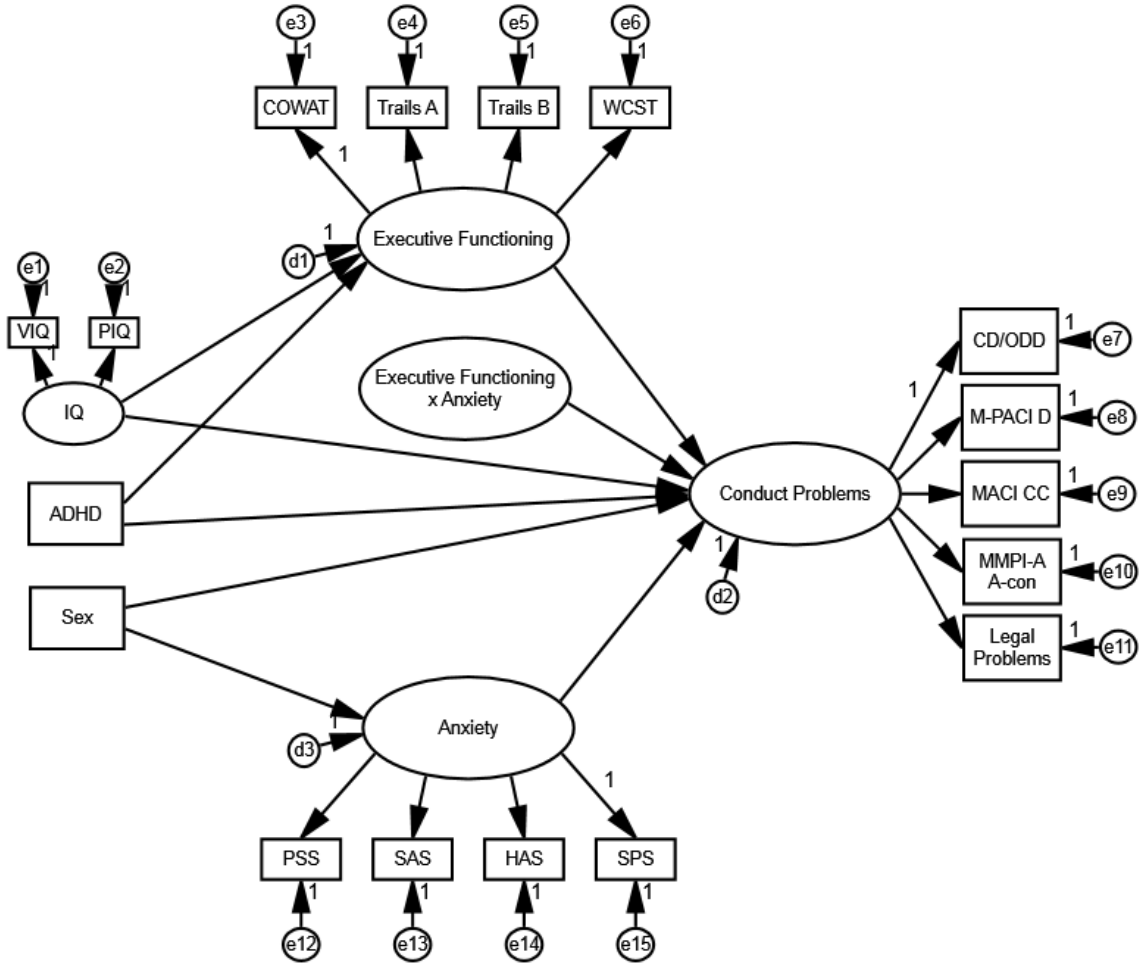


*Figure 1.* Hypothesized model of the interactive and direct effects of anxiety symptom severity and executive functioning abilities on conduct problems.

# Chapter III: Results

## *Preliminary Analyses*

### *Power and Sample Size*

A power analysis was performed to determine the appropriate sample size for the present study. Research has shown that power in confirmatory factor analysis (CFA) and structural equation modeling (SEM) is influenced by sample size, as well as by the degrees of freedom (i.e., the number of parameters in the model that are constrained and not freely estimated; Keith, 2006; MacCallum, Widaman, Zhang, & Hong, 1999). As degrees of freedom increase, so does power. Power has also been shown to be influenced by the number of indicators per latent variable, with a greater number of indicators increasing the stability of factors leading to greater power (Guadagnoli & Velicer, 1988; Keith, 2006). In accordance with the methods outlined by MacCallum, Browne, and Sugawara (1996), the CSMPOW program software was used to determine the sample size necessary with at least .80 power ($\alpha = .05$) and with 111 degrees of freedom in the hypothesized model (Figure 1). A sample size of 58 was determined necessary for the present study based on the power calculation. The current sample size of 221 should result in sufficient power to be able to reject an inadequate model.

### *Data Screening*

All data were checked via examination of descriptive statistics, visual inspection of histograms, and review of skewness and kurtosis values using SPSS in order to ensure that variables were normally distributed. All model variables were reflective of reasonably normal distributions and skew and kurtosis values for all variables were acceptable with absolute values of less than two and seven, respectively (Curran, West, & Finch, 1996). Data were also examined for outliers, defined as scores greater than three standard deviations beyond the mean. Table 3

shows outliers which were detected from various variables and subsequently removed from analyses.

Table 3

*Outlier values removed from analyses*

| **Variable** | **Value** | **Standard deviations from mean** |
|---|---|---|
| WASI VIQ | 144 | 3.91 |
| MASC PSS | 0 | -4.48 |
| COWAT | -13.60 | -3.11 |
| | -13.60 | -3.11 |
| | 14.40 | 3.17 |
| | 16.41 | 3.62 |
| | 21.40 | 4.74 |
| Trails A | 8.08 | 3.17 |
| | 9.45 | 3.78 |
| | 12.37 | 5.08 |
| | 14.20 | 5.9 |
| Trails B | 10.49 | 3.26 |
| | 14.18 | 4.55 |
| | 20.39 | 6.72 |
| | 20.39 | 6.72 |
| WCST | 57 | 3.08 |
| | 72 | 4.18 |
| | 73 | 4.25 |
| | 86 | 5.21 |

A substantial number of values are missing from the dataset both by design and at random. The M-PACI, MACI, and MMPI-A were administered to participants only in specific age ranges, and therefore represent a large proportion of the missing values. The use of an archival dataset in this study rendered the recovery of other missing values unfeasible. Table 4 shows the number and percentage of each missing value for each model variable. Missing data were addressed via pairwise deletion in SPSS for the purpose of computing descriptive statistics. Table 5 shows the descriptive statistics for the scores of all measured variables.

Table 4

*Missing values*

| **Variable** | **Number of missing values** | **% missing values** |
|---|---|---|
| Sex | 0 | 0 |
| ADHD | 0 | 0 |
| WASI VIQ | 9 | 4.07 |
| WASI PIQ | 8 | 3.62 |
| MASC PSS | 1 | .45 |
| MASC SAS | 0 | 0 |
| MASC HAS | 0 | 0 |
| MASC SPS | 0 | 0 |
| COWAT | 7 | 3.17 |
| Trails A | 6 | 2.71 |
| Trails B | 7 | 3.17 |
| WCST | 7 | 3.17 |
| MACI CC | 145 | 65.61 |
| MMPI-A A-con | 148 | 66.97 |
| M-PACI D | 166 | 75.11 |
| CD/ODD | 0 | 0 |
| Legal Problems | 5 | 2.26 |

Scores for the variables WASI VIQ, WASI PIQ, MASC PSS, MASC SAS, MASC HAS, MASC SPS, WCST, MACI CC, MMPI-A A-con, and M-PACI D were each rescaled by dividing by ten after removal of outliers and calculation of descriptive statistics in order to increase consistency of variable scale and reduce variance differences. The rescaled values were retained and used in subsequent data analyses.

Table 5

*Descriptive statistics*

| Variable | Mean | Standard Deviation | Minimum | Maximum |
|---|---|---|---|---|
| Sex | .54 | .49 | 0 | 1 |
| ADHD | .19 | .39 | 0 | 1 |
| WASI VIQ | 98.51 | 11.18 | 80 | 133 |
| WASI PIQ | 98.52 | 14.50 | 60 | 135 |
| MASC PSS | 58.06 | 12.33 | 32 | 90 |
| MASC SAS | 59.24 | 13.00 | 32 | 90 |
| MASC HAS | 47.01 | 11.43 | 25 | 75 |
| MASC SPS | 59.92 | 13.72 | 33 | 90 |
| COWAT | .15 | 3.78 | -12.6 | 13.4 |
| Trails A | .80 | 1.76 | -2.33 | 7.06 |
| Trails B | .86 | 1.89 | -2.52 | 9.10 |
| WCST | 13.86 | 11.20 | 2 | 55 |
| MACI CC | 63.57 | 22.23 | 7 | 107 |
| MMPI-A A-con | 57.19 | 12.65 | 35 | 88 |
| M-PACI D | 63.05 | 20.77 | 6 | 88 |
| CD/ODD | .5 | .50 | 0 | 1 |
| Legal Problems | .42 | .49 | 0 | 1 |

*Note.* Sex: female = 0, male = 1. ADHD: no diagnosis = 0, diagnosis = 1. CD/ODD: no diagnosis = 0, diagnosis = 1. Legal Problems: no diagnosis = 0, diagnosis = 1.

*Model Estimation*

The hypothesized model was analyzed using the statistical software program Mplus (Version 6; Muthén & Muthén, 2010). A path from each latent variable to one indicator variable was constrained to 1 in order to set the scale of the latent variables. Figure 1 shows the factor loadings for VIQ, COWAT, PSS, and CD/ODD set to 1 to set the scale of their respective latent variables. For VIQ, PIQ, and COWAT, larger values indicated better performance. For Trails A, Trails B, and WCST, larger values indicated poorer performance. For all indicator variables of the Anxiety and Conduct Problems latent variables, larger values indicated greater problem severity. Intercorrelations among measured variables were estimated with Mplus using a

maximum likelihood (ML) estimation method with robust standard errors under missing at random (MAR) to deal with missing data. Although data for variables MACI CC, MMPI-A A-con, M-PACI D were missing by design, rather than truly missing at random, ML estimation under MAR addresses missing data both by design, as well as other missingness (Little & Rubin, 2002). All missing data were handled this way in subsequent analyses. Intercorrelations are presented in Table 6. The correlation (represented by the Phi coefficient) between the categorical indicators of the Conduct Problems factor, CD/ODD and Legal Problems, was calculated in Mplus using weighted least square estimation. The Phi coefficient for this correlation was .36 with a standard error of .10, indicating a moderate association between the categorical variables. Table 7 provides crosstabulation information for these variables.

As the normality assumption was met, the ML estimation method with robust standard errors was chosen to estimate the measurement and full latent variable structural equation models. To estimate structural equation models involving latent interactions, Mplus implements a version of the latent moderated structural equations (LMS)/quasi-maximum likelihood (QML) estimation method (Klein & Moosbrugger, 2000; Klein & Muthén, 2007; Kline, 2010). ML estimation is one of the most frequently used estimation methods and is considered robust to moderate violations of the normality (Weston, Gore, Chan, & Catalano, 2008). LMS uses a specific form of ML estimation that assumes normality for non-product variables; however, it takes into consideration the degree of non-normality inherent to latent interaction terms. LMS uses a form of the expectation-maximization (EM) algorithm in estimation. QML is similar to LMS, but utilizes a simpler algorithm that is less computationally complex than LMS. LMS/QML uses raw data, unlike covariance structure analysis, and does not require the calculation of products of indicator variables in order to estimate interactions (Klein &

Moosbrugger, 2000). LMS/QML may be considered the most precise method of estimation for latent variable interactions because it estimates and incorporates the form of non-normality into its analyses (Kline, 2010).

The approach to model estimation was consistent with Anderson and Gerbing's (1988) recommended two-step approach. The measurement portion of the model (i.e., the confirmatory factor model), which is comprised of the paths from the latent constructs to the measured variables was first estimated. The measurement model assessed whether the measured variables (also termed indicators) share enough variance to form the hypothesized latent variables. Respecifications based on modification indices and residual covariances that were consistent with theory were made to the measurement model in order to improve its fit. The final respecified measurement model was then used to evaluate the fit of the full structural model. The structural model included the path analysis of the latent variables with the estimated effects of one latent variable on another. Following estimation and respecification of the measurement model, the full structural equation model including both the measurement and structural portions was estimated. The full structural equation model included IQ, ADHD diagnosis, and sex as control variables in the model. The magnitude and statistical significance of specific paths of interest were examined and relevant competing theoretical models (i.e., interaction compared to no interaction) were tested using the final full structural equation model.

Table 6

*Full information maximum likelihood estimation with robust standard errors-derived correlation matrix of measured*

| **Variable** | **1** | **2** | **3** | **4** | **5** | **6** | **7** | **8** | **9** | **10** | **11** | **12** | **13** | **14** | **15** |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. Sex | 1.0 | | | | | | | | | | | | | | |
| 2. ADHD | .19 | 1.0 | | | | | | | | | | | | | |
| 3. WASI VIQ | .06 | -.09 | 1.0 | | | | | | | | | | | | |
| 4. WASI PIQ | .06 | -.03 | .49 | 1.0 | | | | | | | | | | | |
| 5. MASC PSS | .13 | .07 | .08 | .01 | 1.0 | | | | | | | | | | |
| 6. MASC SAS | .07 | .00 | .04 | .01 | .57 | 1.0 | | | | | | | | | |
| 7. MASC HAS | .34 | .20 | -.05 | -.06 | .31 | .29 | 1.0 | | | | | | | | |
| 8. MASC SPS | .14 | .24 | -.07 | -.05 | .45 | .42 | .47 | 1.0 | | | | | | | |
| 9. COWAT | -.13 | -.05 | .19 | .16 | .07 | -.08 | -.17 | -.16 | 1.0 | | | | | | |
| 10. Trails A | .04 | .03 | -.15 | -.33 | -.06 | -.01 | .16 | -.02 | -.19 | 1.0 | | | | | |
| 11. Trails B | -.03 | .07 | -.23 | -.27 | .00 | .01 | .10 | .11 | -.19 | .63 | 1.0 | | | | |
| 12. WCST | .11 | .19 | -.30 | -.37 | .14 | .04 | .21 | .25 | -.13 | .06 | .16 | 1.0 | | | |
| 13. MACI CC | .22 | .09 | .01 | .01 | -.24 | -.51 | -.30 | -.20 | .12 | -.01 | -.08 | .06 | 1.0 | | |
| 14. MMPI-A A-con | .06 | .25 | -.03 | .11 | .10 | .15 | -.07 | -.03 | .03 | -.18 | -.03 | .15 | -.01 | 1.0 | |
| 15. M-PACI D | -.24 | -.01 | -.11 | -.02 | .14 | .13 | -.25 | -.09 | .21 | .10 | .31 | -.02 | -.06 | .08 | 1.0 |
| *M* | .54 | .19 | 9.86 | 9.85 | 5.81 | 5.92 | 4.70 | 6.00 | .18 | .85 | .89 | 1.40 | 6.52 | 5.89 | 6.89 |
| *SD* | .50 | .99 | 1.12 | 1.44 | 1.23 | 1.30 | 1.14 | 1.37 | 3.77 | 1.78 | 1.89 | 1.13 | 2.17 | 1.26 | 2.21 |

*Note.* Correlations for categorical dependent variables (CD/ODD and Legal Problems) cannot be calculated in Mplus using maximum likelihood estimation.

Table 7

*Crosstabulation of CD/ODD and Legal Problems variables*

| | | CD/ODD | | Total |
|---|---|---|---|---|
| | | **No** | **Yes** | |
| **Legal Problems** | **No** | 74 | 52 | 126 |
| | **Yes** | 32 | 58 | 90 |
| | **Total** | 106 | 110 | 216 |

*Evaluation of Model Fit*

A variety of fit statistics exist to evaluate how well a specified model explains or "fits" the data. The root mean square error of approximation (RMSEA), standardized root mean square residual (SRMR), comparative fit index (CFI), and Tucker-Lewis index (TLI) were used to evaluate the measurement model. The RMSEA assesses the approximate fit of a model corrected for model complexity. RMSEAs below .05 suggest a close fit, values below .08 suggest a reasonable fit, and values over .10 suggest a poor fit (Browne & Cudeck, 1993). The SRMR is the standardized average difference between the actual covariance matrix in model estimation and the covariance matrix implied by the model. Simulation research suggests that the SRMR is among the best of the fit indexes with values below approximately .08 indicating a good fit to the data (Hu & Bentler, 1999). The CFI and TLI are two indexes that compare the fit of the estimated model to the fit of the null, or independence model. The CFI provides an estimate of the improvement in fit (based on the population) over the null model (Keith, 2006). The TLI is relatively independent of sample size and is slightly adjusted for parsimony (Tanaka, 1993). CFI and TLI values closer to 1.0 represent a better fit. Values over .95 suggest a good fit, while values less than .95 but greater than .90 suggest an adequate fit (Hu & Bentler, 1999; Keith, 2006). The chi-square difference test was used to compare nested measurement models (that is, models that can be derived from one another through the addition or removal of constraints). The

Akaike Information Criterion (AIC) and Bayes Information Criterion (BIC) were also used to compare nested, as well as non-nested, measurement and structural models. AIC and BIC are predictive fit indexes that are population-based and parsimony-adjusted (Kline, 2005). The BIC takes into account sample size, and also penalizes complexity more than the AIC (Kline, 2005). For both BIC and AIC, smaller values indicate a better fit to the data (Keith, 2006; Kline, 2005). Loglikelihood values were used to compare nested structural models (interaction compared to no interaction) by calculating the likelihood ratio test statistic (Klein & Moosbrugger, 2000).

## *Primary Analyses*

### *Measurement Model A*

The first phase of the structural equation modeling analysis required conducting a confirmatory factor analysis (CFA) to evaluate the fit of the measurement model to the data. This was performed in two steps: 1) analysis of fit of the measurement model including only latent variables with continuous indicators (i.e., IQ, Executive Functioning, and Anxiety; named Measurement Model A); 2) analysis of fit of the measurement model including all latent variables (i.e., IQ, Executive Functioning, Anxiety, and Conduct Problems; named Measurement Model B). The CFA was conducted in two steps because a variety of fit statistics very useful for making decisions for respecification (i.e., RMSEA, SRMR, TLI, and CFI) were not available through Mplus when categorical indicators were included in the analysis. By first examining the fit of Measurement Model A using the RMSEA, SRMR, TLI, and CFI, a more accurate analysis of fit could be preformed and more specific information indicating necessary respecifications were obtained than would have been possible had the entire measurement model initially been estimated.

An initial test of Measurement Model A suggested that the model was a poor fit for the data (see Table 8). Standardized factor loadings, modification indexes and standardized residual covariances were examined to determine whether any respecifications or modifications could be made that would result in a model that more accurately represented the data. Model modifications were only made if they were deemed theoretically meaningful or consistent with past research.

Examination of the standardized factor loadings, modification indices, and standardized residual covariances indicated three modifications to the hypothesized Measurement Model A that were consistent with theory. First, results suggested that the Wisconsin Card Sorting Test (WCST) variable was likely a source of model misfit. Specifically, the standardized factor loading for the WCST variable was small (-.18, $p$ = .031), and results also indicated that the variable, which was specified as an indicator of the Executive Functioning latent variable, was more related to some indicators of other latent variables (i.e, IQ and Anxiety) than accounted for in the model. The nature of the WCST appears to be factorially complex in that it may measure more than one factor included in the model. As a result, the WCST indicator was removed from the model.

Second, results also suggested that the Controlled Oral Word Association Test (COWAT) variable was likely another source of misfit. The standardized factor loading of the COWAT variable was small (.26, $p$ = .001), and the variable, an indicator of the Executive Functioning latent variable, was more related to indicators of other latent variables (i.e., IQ and Anxiety) than accounted for in the model. Similar to the WCST variable, the COWAT variable may measure more than one factor and, therefore, it was removed from the model.

The third modification suggested by the modification indices and standardized residuals suggested was to free (estimate) the correlation (covariance) between the error variances for the SAS variable and the PSS variable. This change makes sense in that these two scales were found to have higher correlations with one another than with other MASC scales both in this present study and in reliability and validity studies conducted by the creators of the MASC (March et al., 1997).

The three modifications to Measurement Model A were estimated one at a time in order to allow each adjustment to the model to be evaluated. Because the first two model adjustments involved removing indicator variables from the model, the models were not nested within the initial hypothesized model. In order to compare the non-nested modes, the AIC and BIC values were used along with the other fit indices. As shown in Table 8, the model with the WCST variable removed provided a better fit to the data than the hypothesized model as indicated by lower AIC and BIC values (AIC = 6902.05, BIC = 7003.99); however, RMSEA, TLI, and CFI values were still not considered adequate. This modification was retained in subsequent analyses. Removal of the COWAT variable resulted in additional fit improvement with lower AIC and BIC values (AIC = 5732.11, BIC = 5823.86); however, RMSEA and TLI values remained inadequate. This modification was also retained in subsequent analyses. The change in fit resulting from the third modification that involved freeing the correlation between the error variances for the SAS and PSS variables was evaluated using the chi-square difference test. The chi-square difference test can be utilized to measure this modification since the modification resulted in a nested model. As shown in Table 8, allowing the correlated error between the SAS and PSS variables resulted in a statistically significant improvement in fit, suggesting that the two scales do measure something in common beyond what is represented by the latent variable.

The smaller AIC and BIC values (AIC = 5715.36, BIC = 5810.51), and improvements in all other fit indices provided additional evidence that the third modified model provided a better fit to the data. As a result, this model modification was retained in subsequent analyses. The resulting final modified Measurement Model A achieved adequate levels of fit as suggested by all the fit indices examined, including SRMR (.038), RMSEA (.050), TLI (.955), and CFI (.974).

Table 8

*Fit statistics for measurement models with chi-square difference tests for nested models*

| **Measurement Model** | **$\chi^2$ (df)** | **$\Delta\chi^2$ ($\Delta$ df)[a]** | **AIC** | **BIC** | **SRMR** | **RMSEA** | **TLI** | **CFI** |
|---|---|---|---|---|---|---|---|---|
| Hypothesized Model A | 109.95 (32) | | 7558.29 | 7670.42 | .084 | .110 | .723 | .803 |
| Modified Model A 1 (WCST removed) | 60.51 (24) | | 6902.05 | 7003.99 | .058 | .085 | .846 | .897 |
| Modified Model A 2 (COWAT removed) | 43.38 (17) | | 5732.11 | 5823.86 | .049 | .086 | .869 | .920 |
| Modified Model A 3 (SAS ←→ PSS) | 24.63 (16) | 18.85 (1)** | 5715.36 | 5810.51 | .038 | .050 | .955 | .974 |
| Model B | | | 7117.25 | 7266.77 | | | | |
| Modified Model B 1 (MACI CC removed) | | | 6786.45 | 6925.77 | | | | |
| Modified Model B 2 (M-PACI D removed) | | | 6547.41 | 6676.54 | | | | |
| Modified Model B 3 (MMPI-A A-con removed) | | | 6305.14 | 6424.08 | | | | |

*Note.* Hypothesized Model A = latent variables with continuous indicators only; Hypothesized model B = all latent variables.

[a] Compared to the previous model.

***p* <.001

*Measurement Model B*

The second step of the confirmatory factor analysis was to analyze the fit of the measurement model including all latent variables (Measurement Model B). This model utilized the final modified Measurement Model A and added the latent variable Conduct Problems, along with its indicator variables. The initial test of Measurement Model B indicated that the model was a poor fit to the data based on the analysis of standardized factor loadings and residual covariances, as other indicators of fit were unavailable for this model. The standardized factor

loadings of both the M-PACI D and MMPI-A A-con variables were non-significant (.43, $p$ = .053 and .44, $p$ = .076, respectively), and the residual covariances for both of these variables, as well as the MACI CC variable, indicate that these three variables were likely sources of model misfit. These findings were consistent with the low correlations between these indicators as shown in Table 6. It is possible that these three scales measure disparate aspects of conduct problems (e.g., past behavior, beliefs about breaking social norms, difficult social interactions), that are not consistent with one another.

Each variable was removed from Measurement Model B one at a time and model fit was estimated after each removal to allow each model adjustment to be evaluated. The three modified models were not nested within the hypothesized Measurement Model B, therefore AIC and BIC values were used for model comparison. As shown in Table 8, each model modification resulted in a better fit to the data than the previous model as indicated by lower AIC and BIC values. Each model modification was retained in subsequent analyses. The resulting final modified Measurement Model B had an AIC of 6305.14 and a BIC of 6424.08. The final modified measurement model with standardized estimates is presented in Figure 2.

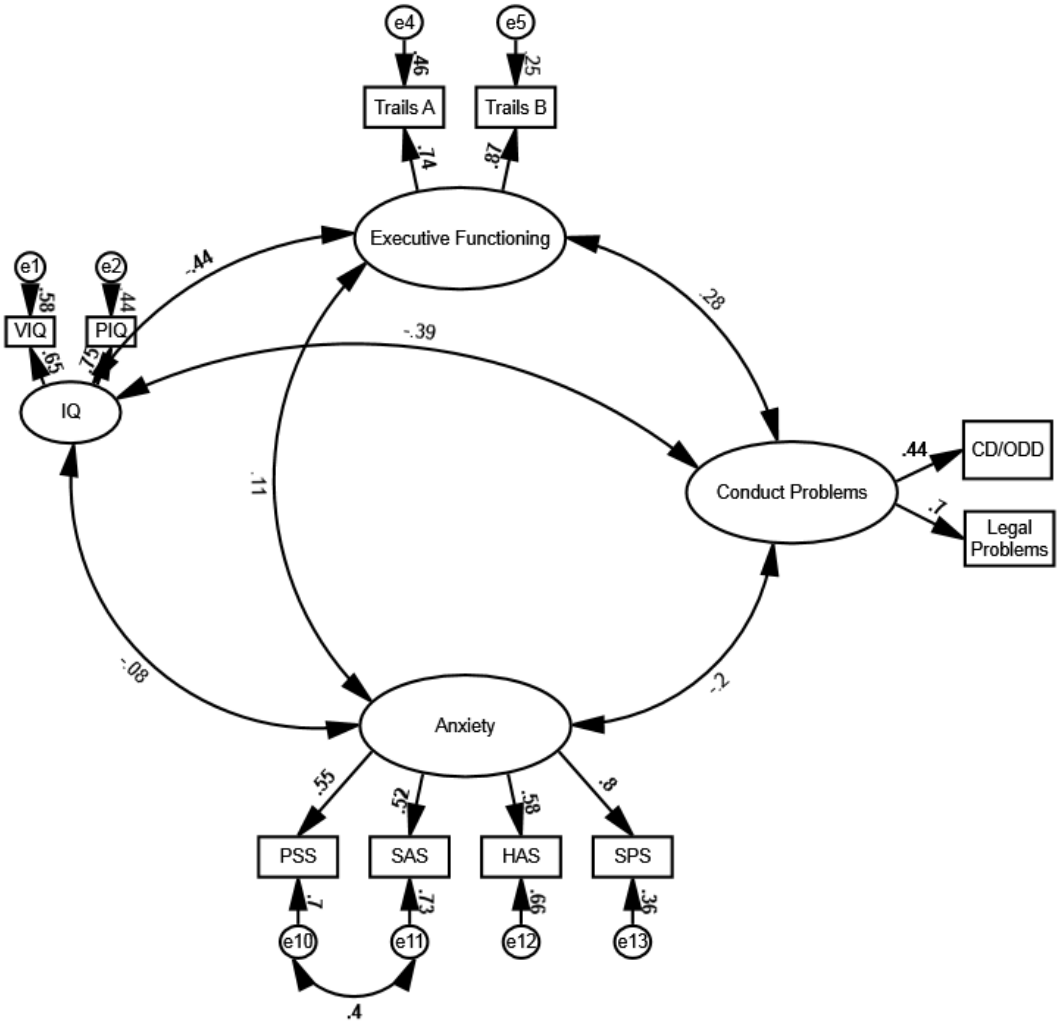


*Figure 2.* Final measurement model with modifications (standardized estimates).

*Full Latent Variable Models*

Once the measurement model was developed, the full structural models (interaction and no interaction) were tested with the hypothesized relationships among the latent variables specified. In addition to the latent variable IQ, ADHD diagnosis and sex were included in the models as background variables to minimize potential confounds and account for potential common causes. Fit statistics for both the interaction and no interaction models are summarized

in Table 9. Unstandardized results for the final full structural interaction model are presented in Figure 3, while unstandardized results for the final full structural model without an interaction are presented in Figure 4. With a sample size of 221 and 47 degrees of freedom, the estimated power for the final full interaction model was .995. The estimated power for the final full model without an interaction was .996 with a sample size of 221 and 49 degrees of freedom. Specific paths between the latent variables were examined and interpreted in relation to the proposed research questions and hypotheses.

Table 9

*Fit statistics for full latent variable models*

| **Model** | **AIC** | **BIC** | **Loglikelihood (df)** | **-2(Δ Loglikelihood) (Δ df)** |
|---|---|---|---|---|
| Interaction | 6296.57 | 6425.70 | -3110.28 (47) | |
| No Interaction | 6292.50 | 6418.23 | -3109.25 (49) | 2.06 (2)* |

*$p$ = .36

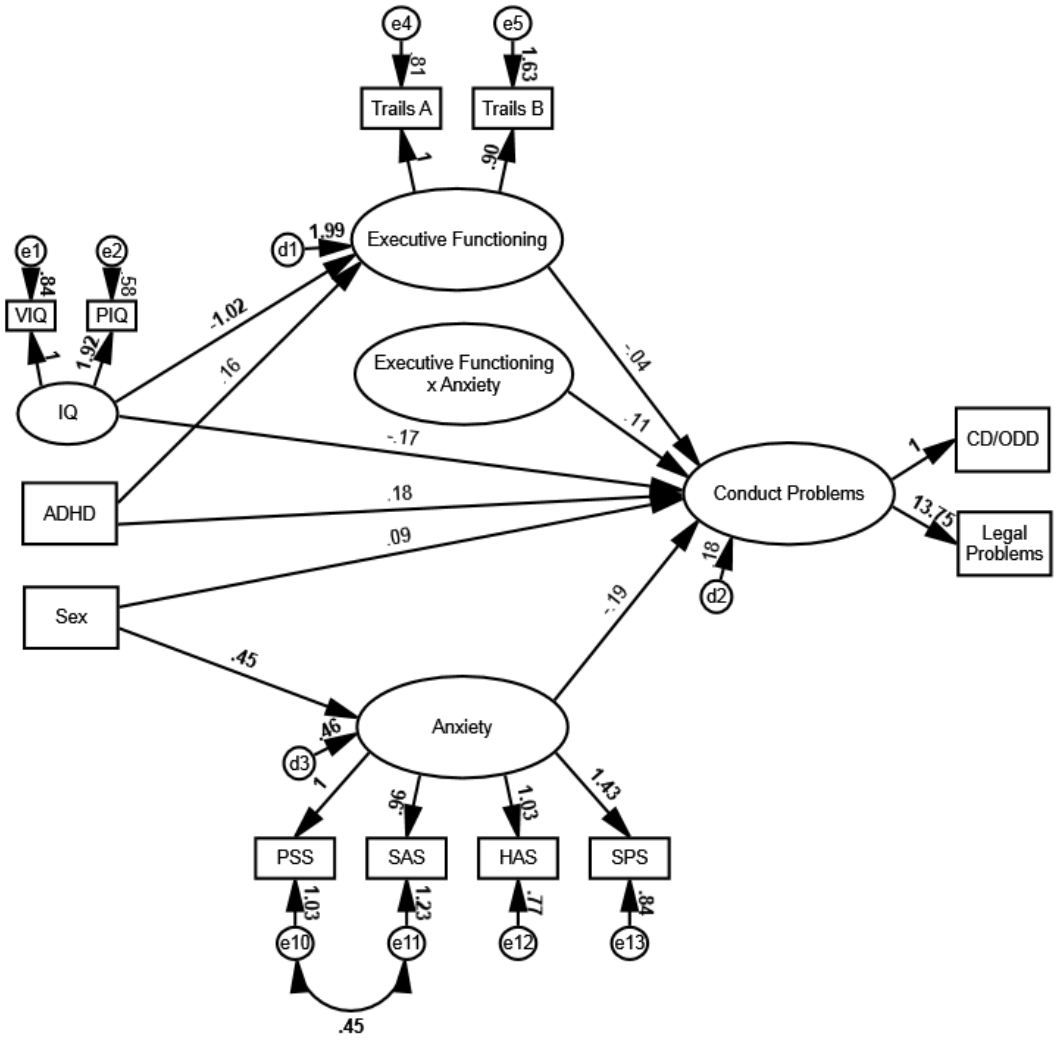


*Figure 3.* Unstandardized estimates for the final full interaction model.

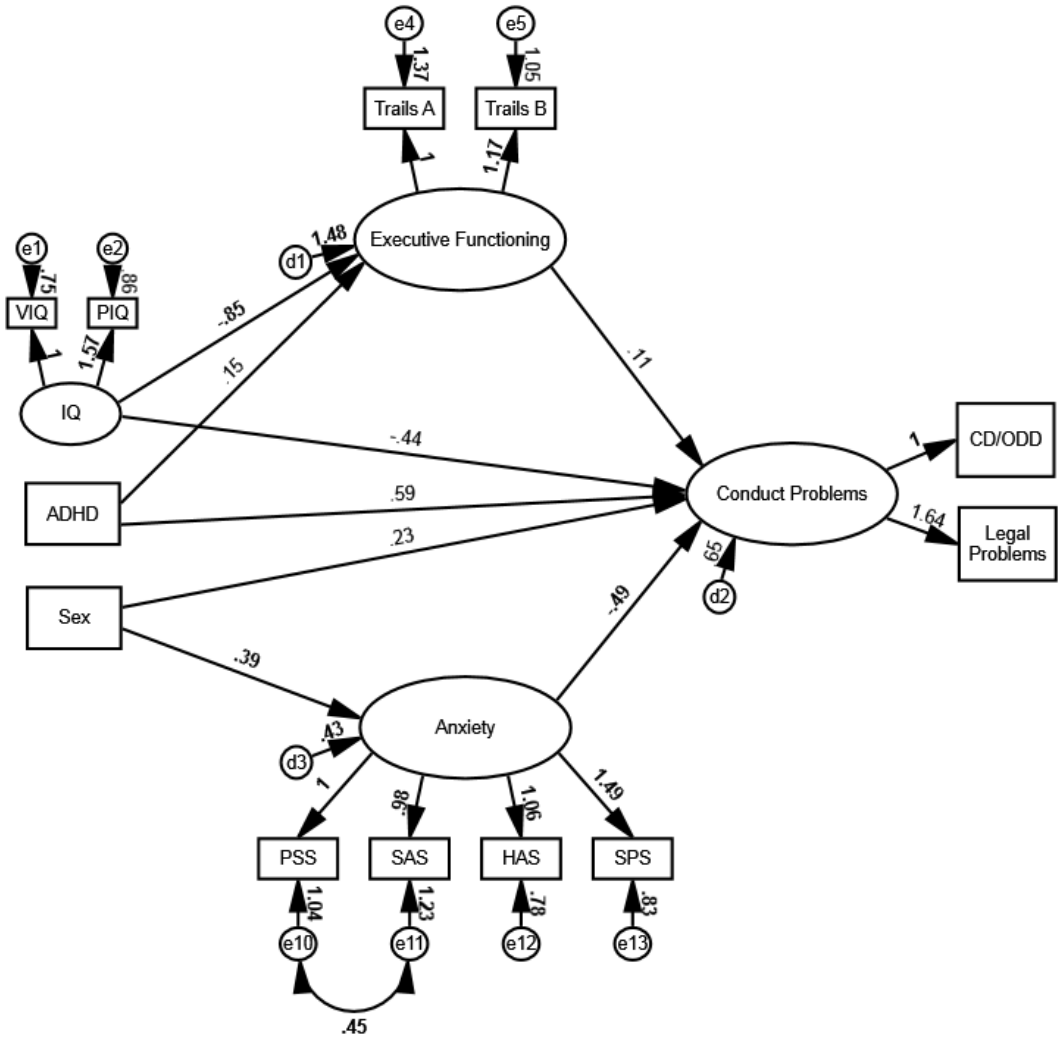


*Figure 4.* Unstandardized estimates for the final full model without an interaction.

## *Tests of Hypotheses*

### *Hypothesis 1*

Hypothesis 1 was that executive functioning would moderate the effect of anxiety on conduct problems. To determine the presence of an interaction, the unstandardized direct effect of the interaction latent variable, Executive Functioning x Anxiety, on the Conduct Problems latent variable was examined. As shown in Table 10, the unstandardized direct effect of the

interaction latent variable was statistically nonsignificant ($p$ = .11). Therefore, executive functioning was not found to moderate the effect of anxiety on conduct problems and Hypothesis 1 was not supported. Additionally, neither the direct effects of the Anxiety latent variable or the Executive Functioning latent variable were statistically significant ($p$ = .13 and $p$ = .54, respectively) indicating that anxiety symptom severity and executive functioning abilities did not influence conduct problems when the data were analyzed using the interaction model.

Table 10

*Unstandardized direct effects of latent variables on conduct problems*

| **Model** | **Latent Variable** | ***b*** | **SE** | ***p*** |
|---|---|---|---|---|
| Interaction | Anxiety | -.19 | .13 | .13 |
| | Executive Functioning | -.04 | .06 | .54 |
| | Executive Functioning x Anxiety | .11 | .07 | .11 |
| No Interaction | Anxiety | -.49 | .25 | .05 |
| | Executive Functioning | .11 | .13 | .40 |

*Note.* Unstandardized estimates are represented by *b*.

*Hypothesis 2*

Hypothesis 2 predicted that the interaction model would better explain the associations between anxiety symptom severity, executive functioning abilities, and conduct problems than a model that only examines the direct effects of anxiety and executive functioning on conduct problems. To determine how well each model explained the data, applicable fit statistics for the interaction and no interaction model were examined (see Table 9). The difference between the models was tested for statistical significance by performing a chi-square difference test based on loglikelihood values. As shown in Table 9, AIC, BIC, and loglikelihood fit statistics were all more favorable for the model without an interaction indicating that it explained the data better; however, the chi-square difference test based on loglikelihood values indicated that the difference in fit between the two models was not significant ($p$ = .36). Results indicated that the

interaction model and the no interaction model were equivalent in their ability to "fit" the data and estimate the effects between variables. Because the two models fit equally well, and because parsimony is valued when evaluating models (Keith, 2006), the less parsimonious interaction model was rejected in favor of the more parsimonious no interaction model, thus Hypothesis 2 was not supported

# Chapter IV: Discussion

## *Overview of Findings*

The goal of this study was to examine the association between anxiety and conduct problems. Specifically, this study evaluated whether executive functioning abilities contribute to differentiating those youth for whom anxiety exacerbates conduct problems from those for whom anxiety mitigates conduct problems. A latent variable structural equation model was proposed and tested to examine the associations between anxiety symptom severity, executive functioning abilities, and conduct problems. This method of analysis was utilized in order to simultaneously analyze the associations between the specified latent variables, including the interaction between anxiety and executive functioning. The model was estimated using data collected from 221 youth aged 9 to 16 at a residential treatment center. Although previous studies have examined the comorbidity of anxiety and conduct problems, and studies have examined executive functioning abilities in relation to anxiety and conduct problems independent of one another, this study is unique in that it is the first known to the author that examined how executive functioning moderates the effect of anxiety on conduct problems.

Results of this study failed to support the hypothesis that executive functioning would moderate the effect of anxiety on conduct problems. Furthermore, a structural equation model with an interaction between executive functioning and anxiety was not found to fit the data better than a model without an interaction. Overall, findings did not support the hypothesis that executive functioning abilities could distinguish youth for whom anxiety exacerbates conduct problems from youth for whom anxiety mitigates conduct problems.

*Association between Anxiety, Executive Functioning, and Conduct Problems*

Drabick , Ollendick, and Bubier (2010) proposed several factors that could explain the differential effects of anxiety on conduct problems. Along with negative emotionality, social information processing abilities, the function of aggression, level of effortful control, and limbic system activity, executive functioning abilities were identified as a potential moderating construct. Executive functioning was hypothesized as a moderator based primarily on neurological findings linking dysfunction in the prefrontal cortex (PFC) and the limbic system to both anxiety disorders and conduct problems. Specifically, research suggests that heightened amygdala and attenuated PFC activity may contribute to anxiety symptom severity and conduct problems in youth (Bubier & Drabick, 2009; Guyer et al., 2008; Monk et al., 2006). The presence of PFC deficits in youth with conduct problems (as indicated by deficits in executive functioning) has been supported by several studies (Drabick et al., 2007; Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999); however, research testing the hypothesis the executive functioning moderates the effect of anxiety on conduct problems is lacking.

The present study tested this moderation hypothesis. Results indicated that the interaction effect of anxiety and executive functioning on conduct problems was not significant, and that an interaction model did not explain the association between anxiety, executive functioning, and conduct problems better than a model without an interaction. Based on the data used and model proposed in this study, executive functioning does not appear to contribute to any differential effect anxiety may have on conduct problems. Therefore, results suggest that the direct effects of executive functioning and anxiety on conduct problems should be examined in order to better understand the association between these three constructs.

The direct effect of executive functioning on conduct problems was not found to be significant in either of the proposed models. These results are consistent with previous research that found no significant differences between the performance of youth with externalizing disorders and control participants on tasks evaluating various executive functioning subdomains including verbal fluency, inhibition, cognitive flexibility, attention, working memory, and planning abilities (Hobson et al., 2011; Kim et al., 2001; Kusché et al., 1993; Närhi et al., 2010; Oosterlaan et al., 2005; Pajer et al., 2008; Speltz et al., 1999; Thorell & Wåhlstedt, 2006). However, results of the present study fail to support other studies that have found executive functioning abilities, particularly inhibition, to be negatively associated with conduct problems (Fisher et al., 2005; Hughes & Ensor, 2011; Kusché et al., 1993; Utendale & Hastings, 2011; Utendale et al., 2011; Young et al., 2009; Youngwirth et al., 2007). Of perhaps greater relevance to the present study, youth with conduct problems have been previously found to display impairments in processing speed, attention, and shifting as indicated by their performance on Trails A and Trails B, the same two indicators of executive functioning utilized in this study (Kusché et al., 1993; Närhi et al., 2010). One explanation for these disparate findings is that the studies finding differences on Trails A and Trails B did not control for ADHD diagnosis as the present study did, making it difficult to interpret the association between participants' performance on these measures and conduct problems independent of ADHD. Findings from the present study suggest that the differences found by Kusché's and Närhi's groups may be due to ADHD and that further analyses of their data may be warranted.

Regarding the direct effect of anxiety on conduct problems, a significant negative effect was found in the model that was found to best fit the data (the model without an interaction). These findings are consistent with previous research; specifically, the buffering hypothesis that

suggests anxiety mitigates conduct problems in youth (Hofmann et al., 2009; Walker et al., 1991). Anxiety has been found to reduce the risk of substance use, aggression, and police contact in youth with comorbid conduct problems (Hofmann et al., 2009; Walker et al., 1991). The present study offers additional evidence that anxiety serves to reduce impairment in youth with conduct problems, rather than exacerbate it as hypothesized by the multiple problem hypothesis (Garai et al., 2009; Lansford et al., 2008; Ollendick et al., 1999).

### *Limitations and Future Directions*

There are several potential limitations of the present study that should be recognized when considering the results and their implications. First, there are a number of limitations related to the characteristics of the sample used. In particular, it is important to note that the sample was comprised of youth with serious emotional and behavioral symptoms requiring residential treatment. Due to the high level of emotional and behavioral impairment of the individuals in this sample, issues of restriction of range appear applicable to the data in that the data may not have represented the variation in scores one would expect from a community sample, even though the data for each variable was found to meet the normality assumption. This limitation is supported by descriptive statistics calculated from the data which found that mean *z*-scores for Trails A and Trails B to not be equal to zero as would be expected, but rather .80 and .86, respectively (Table 5). Due to the specificity of the sample, findings from the study may lack generalizability to less acute populations of children and adolescents. Future research utilizing data from outpatient clinical samples and community samples should yield more widely applicable results that should provide a more complete understanding of the association between anxiety, executive functioning, and conduct problems.

A second potential limitation to the study concerns the indicator variables chosen to represent the latent constructs of interest. Indicator variables were selected to represent latent constructs based on both theory and previous research; however, there are many other measures not included in this study that have been used to assess anxiety symptom severity, executive functioning abilities, and conduct problems (e.g., RCMAS, Stroop Color-Word Test, POSIT). Of particular relevance to the anxiety and conduct problems indicators, are the problems inherent to the use of self-report measures. Social desirability and fear of reprisal, as well as comprehension difficulties, are factors that have been identified that may contribute to validity problems with self-reported data, especially when assessing alcohol and drug use and aggressive behavior (Brener, Billy, & Grady, 2003). Similarly, youth may tend to underestimate or underreport anxiety symptoms on self-report measures in order to present themselves more favorably and to avoid treatment (Silverman, 1987). Gender and cultural differences may also affect accuracy of self-reporting of anxiety symptoms and behaviors commonly associated with conduct problems (Brener et al., 2003; Ollendick, Matson, & Helsel, 1985). Problems with questionnaire item comprehension due to reading ability and other cognitive abilities also impact the validity of self-report measures for a variety of reasons (March et al., 1997). For those individuals who ask for adult help to complete self-report measures, a response bias may be present based on the youth's perceptions of the adult's expectations, thereby affecting validity of the data obtained (March et al., 1997). In order to mitigate these threats to validity, future studies should examine data from a variety of respondents such as guardians and teachers, as well as the participants, in order to obtain the most accurate information regarding the social, emotional, and behavioral functioning of participants.

Although the indicators chosen to represent the anxiety latent variable were found to represent the construct well, significant respecifications to the executive functioning and conduct problems latent variables were required in order to improve the fit and accuracy of the model. These respecifications were required because the indicators initially chosen to represent the constructs of executive functioning and conduct problems did not adequately load on these factors. As a result, the executive functioning and conduct problems latent variables had two indicators each. This is a potential limitation to the study, as only having two indicators for each of these latent variables increases the risk that the latent variables represent more narrow constructs than the broadly defined executive functioning and conduct problems constructs that were originally intended to be represented. For example, the indicators for the executive functioning latent variable in the final models were Trails A and Trails B. These two measures have been found to assess the executive functioning subdomains of processing speed, attention, inhibition, and shifting (Kelly, 2000); however, other subdomains of executive functioning such as abstract reasoning, concept formation, planning, and working memory are not assessed by these measures. In order to gain a better understanding of the moderating effect of the broad construct of executive functioning on the effect of anxiety on conduct problems, measures assessing the wide range of subdomains encompassed by the construct of executive functioning should be included in future research. Alternatively, subsequent studies may wish to focus more narrowly on the moderating effect of specific executive functioning subdomains, such as attention or inhibition. This approach may be beneficial in that results from these studies could have implications for the development and implementation of very focused recommendations for enhancing specific executive functioning abilities.

Similarly, only having two indicators (CD/ODD and Legal Problems) for the conduct problems latent variable in the final structural model is a potential limitation of the study. The CD/ODD variable was a dichotomous variable that only indicated whether a participant had been diagnosed with CD or ODD or had no diagnosis. Similarly, the Legal Problems variable was dichotomous and indicated whether the diagnosing psychologist or psychiatrist was aware of any legal problems the participant may have had. Being dichotomous variables, neither of these indicators assessed to what degree or with what frequency participants had engaged in aggressive, oppositional, destructive, or disrespectful behavior and they also failed to assess attitudes and beliefs often associated with conduct problems. In order to achieve a more comprehensive representation of the construct of conduct problems, three continuous indicators were included in the hypothesized model (MMPI-A A-con, M-PACI D, and MACI CC); however, these indicators were not found to adequately load on the conduct problems latent variable. Although data on the convergent validity of these three measures is lacking, it was assumed that they would all load on the conduct problems latent variable based on the descriptions provided by the measures' authors. It may be that they each assess diverse facets of conduct problems (e.g., past behavior, attitudes toward social norms, associations with negative peer groups), but the greater source of model misfit may have been that each participant was administered only one of these measures resulting in a large number of missing values that were then estimated by the statistical software during model estimation. Future studies should utilize better indicators for the construct of conduct problems that are administered to a high percentage of the sample to reduce missing values. More appropriate indicators would assess various components of conduct problems on a continuous scale, and, in light of limitations previously

noted, would include self-report measures, as well as measures completed by other respondents with knowledge of a youth's social, emotional, and behavioral functioning.

Lastly, a limitation of the current study is that it did not take into account the heterogeneity of anxiety and conduct problems. In future studies, the specificity of the dual-pathway model could be tested for different types of conduct problems (e.g, CD vs. ODD) and anxiety disorders (e.g., Generalized Anxiety Disorder vs. Social Phobia). Future research should also evaluate and potentially control for the effects of comorbid disorders other than ADHD, such as depression, to better understand the influence of comorbidity. Additionally, future studies should examine whether the association between anxiety, executive functioning, and conduct problems is consistent across various ages and developmental periods. Given that all three of these constructs have been found to vary by age, the influence of age and development on the association between the three is important to consider (Baron, 2004; Lahey, 2008; Merikangas et al., 2010).

### *Conclusions and Implications*

This study has provided several contributions to the literature examining the comorbidity of anxiety disorders and conduct problems. Not only did this study help to clarify the dual-pathway model proposed by Drabick, Ollendick, and Bubier (2010), but it also filled a large gap in the research examining the moderating influence of executive functioning on the effect of anxiety on conduct problems. More specifically, results found that executive functioning did not moderate the effect of anxiety on conduct problems. Furthermore, the study showed that anxiety symptom severity is inversely related to conduct problems when the association between the three constructs of interest was examined.

This study has important implication s for the future of research examining the comorbid anxiety disorders and conduct problems. Further investigation into the possible moderating effect of executive functioning on the association between anxiety and conduct problems appears warranted given the limitations of the current study. Future studies utilizing indicator variables that sufficiently represent the breadth and depth of the constructs of interest, that include self-report and guardian/teacher-report data, and that investigate and potentially control for the influence of comorbid disorders and development would greatly enhance the understanding of the role of executive functioning in the dual-pathway model.

Results of the current study also support the investigation of other processes that may explain the relationship between anxiety and conduct problems. Negative emotionality, social information processing abilities, aggression function, level of effortful control, and limbic system activation are processes that may better account for the differential effect of anxiety on conduct problems. Undeniably, ongoing research on the factors that contribute to anxiety mitigating conduct problems in some youth while exacerbating conduct problems in other youth will be necessary.

# Appendix A: Literature Review

This appendix will provide a review of the literature regarding the comorbidity of anxiety disorders and conduct problems in children and adolescents focusing on their common neurological underpinnings. The first section will provide an overview of the clinical presentation, epidemiology, risk factors, developmental progression, and neurological findings related to anxiety disorders. The second section will summarize the research on these same topics as they relate to conduct problems. Next, a summary of the literature relevant to the comorbidity of these disorders is presented including research surrounding the leading hypotheses proposed to explain the nature of the association between anxiety and conduct problems. The review concludes with an examination of the role of executive functioning in anxiety disorders, conduct problems, and comorbid presentations.

## *Anxiety in Children and Adolescents*

### *Clinical Presentation*

Anxiety is a higher-order feeling state that is a product of brain mechanisms responsible for basic emotion (Weems & Silverman, 2008). A result of a multifaceted response system involving affective, behavioral, physiological, and cognitive components, anxiety can serve an adaptive role in preparing an individual to anticipate future danger. Anxiety problems arise when dysregulation of this normal response system occurs. Such dysregulation may present as intense and disabling worry or fear that is not accurate in predicting true future danger or threat. Impairment, distress, and accompanying negative emotional states frequently result from the dysregulation. Impairment and distress can be expressed behaviorally (e.g.,withdrawal), cognitively (e.g., concentration problems), physiologically (e.g., sweating), and socially (e.g., avoidance). Dysregulation of the anxiety response system and the corresponding impairment and

distress are primary features of anxiety problems (Weems & Silverman, 2008). These primary features are not specific to any particular anxiety diagnosis, but instead are common to all anxiety disorders represented in the *Diagnostic and Statistical Manual of Mental Disorders* (4th edition, text revision) (DSM-IV-TR; American Psychological Association [APA], 2000). In contrast to primary features, secondary features are characteristics of anxiety problems such as fears of certain situations or objects that differentiate specific anxiety disorders.

The American Psychological Association (2000) recognizes 16 anxiety disorders that occur in children, adolescents, and adults. Table 11 provides a brief description of each of these disorders. All of these disorders can appear at any age except for Separation Anxiety Disorder which can only be diagnosed in individuals age 18 and younger. The most common anxiety disorders in children and adolescents include Agoraphobia, Generalized Anxiety Disorder (GAD), Social Phobia, Specific Phobia, Panic Disorder, Posttraumatic Stress Disorder (PTSD), and Separation Anxiety Disorder (SAD) (Merikangas et al., 2010).

Table 11

*DSM-IV-TR anxiety disorders*

| **Anxiety Disorder** | **Description** |
|---|---|
| Panic Attack | Discrete period during which there is a sudden onset of terror or fearfulness accompanied by physical symptoms such as shortness of breath, palpitations, chest pain, and fear of losing control |
| Agoraphobia | Anxiety about, or avoidance of, places or situations from which escape may be difficult |
| Panic Disorder Without Agoraphobia | Recurrent unexpected Panic Attacks about which there is persistent concern |
| Agoraphobia Without History of Panic Disorder | Presence of Agoraphobia and panic-like systems without a history of Panic Attacks |

| | |
|---|---|
| Specific Phobia | Significant anxiety caused by exposure to a specific feared object or situation |
| Social Phobia | Significant anxiety caused by exposure to certain types of social or performance situations |
| Obsessive-Compulsive Disorder | Presence of obsessions (causing anxiety and distress) and/or compulsions (serving to reduce anxiety) |
| Posttraumatic Stress Disorder | Reexperiencing of a traumatic event accompanied by symptoms of increased arousal and by avoidance of stimuli associated with the trauma |
| Acute Stress Disorder | Symptoms similar to Posttraumatic Stress Disorder that occur immediately after a traumatic event |
| Generalized Anxiety Disorder | At least six months of persistent and excessive anxiety and worry |
| Anxiety Disorder Due to a General Medical Condition | Anxiety symptoms that are determined to be a direct physiological consequence of a general medical condition |
| Substance-Induced Anxiety Disorder | Anxiety symptoms that are determined to be a direct physiological consequence of a drug of abuse, a medication, or toxin exposure |
| Anxiety Disorder Not Otherwise Specified | Symptoms of anxiety or phobic avoidance that do not meet diagnostic criteria for any specific Anxiety Disorder |
| Separation Anxiety Disorder | Anxiety symptoms that are related to separation from a paternal figure |
| Sexual Aversion Disorder | Phobic avoidance that is limited to genital sexual contact with a sexual partner |

*Epidemiology*

*Prevalence.* Anxiety disorders are amongst the most prevalent psychological conditions in children and adolescents. A study conducted in 2010 of over 10,000 adolescents in the United States found that anxiety disorders were the most frequently reported condition with a lifetime prevalence rate of 31.9% (Merikangas et al., 2010). Of all participants surveyed, 19.3% met criteria for Specific Phobia, 9.1% met criteria for Social Phobia, 7.6% met criteria for SAD, and

5.0% met criteria for PTSD. The rate of severe anxiety disorder was found to be 8.3%. Another study of American children and adolescents ages 9 to 16 found that 9.9% of children meet criteria for an anxiety disorder (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003). Three-month prevalence for any anxiety disorder in the sample was found to range from 0.9% to 4.6% depending on age.

*Sex differences.* Significant gender differences exist in the diagnosis of anxiety disorders. Females experience anxiety and related symptoms at higher rates than males (Silverman & Carter, 2006), and girls report having more fears than boys (Ginsburg & Silverman, 2000). The predicted cumulative prevalence of any anxiety disorder by age 16 has been reported as 12.1% for girls compared to 7.7% for boys (Costello et al., 2003). In a nationally representative sample of adolescents, all anxiety disorder subtypes were found to be more frequent in females with the greatest sex difference being observed in PTSD (Merikangas et al., 2010). Little is known about the reason for such sex differences; however, females show higher heritability estimates for anxiety than males (Eley, 2001), and girls may be differentially willing to report anxiety symptoms (Ginsburg & Silverman, 2000).

*Cultural and ethnic differences.* The limited literature that exists on the topic indicates that there may be some ethnic differences in the prevalence of anxiety symptoms. Non-Hispanic black adolescents have been found to have higher rates of anxiety disorders than non-Hispanic white adolescents (Merikangas et al., 2010). Similarly, Latino children appear to have higher levels of internalizing symptoms (i.e., anxious and somatic complaints) than non-Latino white children (Ginsburg & Silverman, 1996).

*Risk Factors*

Parental characteristics have been related to the prevalence of anxiety symptoms and disorders. Specifically, maternal anxiety has been associated with child anxiety (Costa & Weems, 2005), as has maternal overcontrol and overinvolvement, and parental psychopathology (Weems & Silverman, 2008). Twin studies have found that approximately 33% of the variance in anxiety symptoms in childhood can be attributed to genetic influences (Eley, 2001). Rates of anxiety disorders appear to be higher for adolescents whose parents are divorced or separated than for adolescents with cohabitating or married parents (Merikangas et al., 2010). Additionally, adolescents whose parents are not college graduates have been found to be at increased risk for all mental disorders, including anxiety disorders (Merikangas et al., 2010).

Attachment style and child temperament have been associated with the development of anxiety disorders. Longitudinal research has shown that children classified as anxious/resistant when assessed at 12 months of age are more likely to have anxiety disorders at age 17 (Warren, Huston, Egeland, & Sroufe, 1997). Researchers have also found that temperamental fear/sadness as an infant predicts internalizing problems (i.e., anxiety and depression) in childhood (Blair, 2002; Rydell, Berlin, & Bohlin, 2003). Additional factors that have been found to exacerbate vulnerability to anxiety disorders include low verbal intelligence, poverty, low social support, and exposure to trauma and violence (Kristensen & Torgersen, 2008; Weems & Silverman, 2008).

*Developmental Progression*

Research suggests that anxiety disorders appear earlier than other mental disorders with a median age of onset of 6, compared to 11 for behavior disorders, 13 for mood disorders, and 15 for substance use disorders (Merikangas et al., 2010). The age-specific incidence curve for

anxiety disorders begins with a steep slope in early childhood and levels off after age 12 (Merikangas et al., 2010). Childhood anxiety disorders appear to be associated with adult anxiety and depressive disorders; however, stability estimates appear to vary widely ranging from 4% to 80% (Weems & Silverman, 2008). The prevalence of adolescents having any anxiety disorder appears to be stable across age groups; however, the prevalence of specific anxiety disorders has been found to vary by age with rates of GAD, panic disorder, PTSD, and social phobia increasing , and rates of SAD decreasing, as age increases (Merikangas et al., 2010; Weems, Silverman, Saavedra, Pina, & Lumpkin, 1999).

Some authors have tied age-related differences in the expression of anxiety disorders to developmental stage theories (Warren & Sroufe, 2004; Westenberg, Siebelink, & Treffers, 2001). Specifically, Westenberg et al. (2001) and Warren and Sroufe (2004) propose that children ages 6 to 9 primarily express their anxiety through separation anxiety and animal phobias, whereas children ages 10 to 13 express anxiety with generalized anxiety symptoms and fears related to danger and death, and adolescents ages 14 to 17 predominately express social anxiety symptoms and fears related to performance and social situations. This model of heterotypic continuity (Moffitt, 1993) has been supported by multiple studies (Weems & Costa, 2005; Westenberg, Drewes, Siebelink, & Tressfers, 2004) indicating the importance of considering differences across childhood and adolescence in the expression of anxiety.

*Neurological Findings in Anxiety*

Anxious emotion is the result of complex interactions between multiple related neurological symptoms; however, the limbic system, especially the amygdala, and the prefrontal cortex (PFC) have been identified as having particularly important roles in the experience and expression of anxiety. The amygdala, which is found in the anterior portion of the temporal

lobes, evaluates the emotional significance of incoming stimuli relayed from the cortex, hippocampus, and thalamus. After receiving such input, the amygdala projects to the other brain structures involved in the anxiety response system including the PFC, the hippocampus, the striatum, the hypothalamus, and the brain stem. These structures are responsible for unique processes in the anxiety response system. The PFC is involved in planning and behavior choice, the hippocampus in memory consolidation, the striatum in approach/avoidance responses, and the hypothalamus and brain stem in autonomic responses, startle, and corticosteroid response (Gordon & Hen, 2004; LeDoux, 2000).

Research regarding the functioning of the amygdala in individuals with anxiety disorders consistently shows a positive association between heightened amygdala activity and anxiety disorders. For example, children with anxiety disorders respond with greater levels of amygdala activation when shown angry and fearful faces as compared to children without anxiety disorders (Thomas et al., 2001). In the study conducted by Thomas and colleagues, amygdala activation was found to be positively associated with anxiety symptom severity. Similarly, youth with anxiety disorders appear to display heightened amygdala activation when anticipating evaluation from peers (Guyer et al., 2008).

PFC activation has also been associated with anxiety and anxiety symptom severity. Functional brain studies suggest that the PFC is involved in the assessment of threat and emotional learning – two constructs relevant to anxiety (Baron, 2004). Three specific regions of the PFC have been identified as important in the processing of emotions: the dorsolateral, ventrolateral, and ventromedial regions (Drabick, Ollendick, & Bubier, 2010). The dorsolateral region has been associated with planning and higher-order cognitions; the ventrolateral region

with stimulus-response learning (specifically, when response modulation is necessary); and the ventromedial with positive and negative affective consequences.

Electroencephalography (EEG) research has found that negative emotion, which is associated with avoidance and fear, leads to increased right PFC activation (Davidson, 1998). Greater relative levels of right PFC activation have been found in adults, school-age children, and infants with elevated levels of anxiety and anxiety disorders (Baving, Laucht, & Schmidt, 2002; Davidson & Fox, 1989; Davidson, Marshall, Tomarken, & Henriques, 2000). Youth with GAD appear to exhibit greater activation in the ventrolateral PFC in response faces displaying negative emotions as compared to control participants; however, this activity appears to be negatively associated with GAD symptom severity (Monk et al., 2006). Guyer and colleagues (2008) found that amygdala-PFC co-activation was positively associated with the presence of an anxiety disorder. These findings suggest that amygdala-PFC co-activation has an important role in the anxiety response system in that the PFC may serve to modulate amygdala activity in youth with anxiety disorders.

Certain neurotransmitters and their corresponding systems have been related to anxiety and anxiety-related behavior. Gamma aminobutyric acid (GABA) and serotonin (5-HT) are two such neurotransmitters. Benzodiazepines and selective serotonin reuptake inhibitors (SSRIs) are psychopharmacological drugs that reduce anxiety by modulating the neurotransmission of GABA and serotonin (Weems & Silverman, 2008). The norandrenergic system has also been implicated in anxiety disorders, especially those with a strong physical component, such as Panic Disorder (Gordon & Hen, 2004). The hypothalamic-pituitary-adrenal (HPA) axis is another system that has been associated with anxiety. It is often activated following fight-or-flight reaction in response to fear and stress, and it's responsible for the increase of cortisol secretions

that results from fear (Gunnar, 2001). It has been suggested that prolonged exposure to glucocorticoids such as cortisol may be neurotoxic and related to anxiety problems (Weems & Silvernan, 2008).

## *Conduct Problems in Children and Adolescents*

### *Clinical Presentation*

Conduct problems are exhibited by children and adolescents as negativistic, defiant, disobedient and hostile behavior toward authority figures and as behaviors which violate the basic rights of others, major age-appropriate social norms, or rules (APA, 2000). Children and adolescents display varying degrees of conduct problems; in fact, some behaviors may be considered developmentally appropriate. For children and adolescents who display persistent and repetitive patterns of conduct problems, the DSM-IV-TR (APA, 2000) recognizes two relevant diagnoses: Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD).

ODD refers to engaging in at least four of the following behaviors in the last six months: actively defying or refusing to comply with the requests or rules of adults, arguing with adults, being angry and resentful, being irritable, being spiteful and vindictive, blaming other for one's own mistakes or misbehaviors, deliberately doing things that will annoy other people, or losing one's temper (APA, 2000). In order to receive the diagnosis of ODD, the behaviors most occur more frequently than is typical for same-age peers and they must lead to significant impairment in academic, social, or occupational functioning. Additionally, a diagnosis of ODD cannot be given if the behaviors occur exclusively during the presence of a Psychotic or Mood Disorder or if the individual meets criteria for CD or Antisocial Personality Disorder (APD; for individuals age 18 and older).

The diagnosis of CD is similar to ODD; however, it includes a persistent pattern of more serious behaviors that violates basic rights of others, rules, or social norms (APA, 2000). To meet criteria for CD, an individual must display 3 or more behaviors that would fall into the following categories over the past 12 months: aggressive conduct that causes or threatens physical harm to people or animals (e.g., bullying, initiating physical fights, physical cruelty to animals), nonaggressive conduct that causes property loss or damage (e.g., vandalism, smashing car windows), deceitfulness or theft (e.g., "conning," shoplifting, forgery), and serious violations of rules (e.g., elopement, truancy). These behaviors must cause significant impairment in academic, social, and occupational functioning in order for an individual to receive the diagnosis of CD and criteria cannot be met for APD (for individuals age 18 and older). There are three subtypes of CD based on the age of onset: Childhood-Onset Type, Adolescent-Onset Type, and Unspecified Onset. The Childhood-Onset Type diagnosis is given when one criterion characteristic of CD is met prior to the age of 10. The Adolescent-Onset Type diagnosis is defined by the absence of any criteria prior to the age of 10. Unspecified Onset is assigned when the age of onset of CD is unknown.

*Epidemiology*

*Prevalence.* The prevalence of conduct problems has appeared to increase over the last few decades with ODD and CD being two of the most frequently diagnosed mental disorders amongst children and adolescents (APA, 2000). Reported prevalence rates range from 2% to 16% for ODD and less than 1% to over 10% for CD (APA, 2000). Merikangas and colleagues (2010) estimated the lifetime prevalence rates of ODD and CD to be 12.6% and 6.8%, respectively. They found approximately one half of those diagnosed with ODD and one third of those diagnosed with CD to be experiencing severe impairment. ODD has been found to have a

predicted cumulative prevalence rate by age 16 of 11.3% and a three-month prevalence rate of 2.7% (Costello et al., 2003). CD was found to have a similar three-month prevalence rate (2.7%) and a slightly lower cumulative prevalence rate by age 16 (9.0%) (Costello et al., 2003).

*Sex differences.* Research suggests significant sex differences in the prevalence and presentation of conduct problems. The majority of research suggests that conduct problems are considerably more common in males (Lahey et al., 2006). Females age 9 to 16 were found to experience significantly lower three-month prevalence rates of CD compared to same-age male peers (females = 1.2%; males = 4.2%) (Costello et al., 2003). Additionally, girls were found to have significantly lower cumulative prevalence rates by age 16 of both ODD (girls = 9.1%; boys = 13.4%) and CD (girls = 3.8%; boys = 14.1%) (Costello et al., 2003). A recent study by Merikangas and colleagues (2010), however, failed to find a significant difference between the lifetime prevalence rates of CD in males and females.

*Cultural and ethnic differences.* Research on the association between culture, race, and ethnicity and conduct problems is limited, yet suggests that non-Hispanic white youth experience lower rates of conduct problems than youth of other races/ethnicities. Harden and colleagues (2009) found that African-American and Hispanic ethnicity predicted mother-reported conduct problems in a nationally representative sample of mothers and their children. A study of nearly 2,500 children and adolescents in residential treatment facilities across the United States found significant differences between the rates of CD in Hispanic, African-American, and non-Hispanic white youth with the CD diagnosis being most prevalent in Hispanic youth and less prevalent in Caucasian youth (Cameron & Guterman, 2007).

*Risk Factors*

Several characteristics of behavior in early childhood have been linked to conduct problems in later childhood and adolescence. Temperament-related factors including impulsivity and lack of persistence (Henry, Caspi, Moffitt, & Silva, 1996), low levels of prosocial behavior (Coté, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002), a tendency to resist control by adults (Keily, Bates, Dodge, & Pettit, 2001), and a tendency to respond with negative emotions to threats and frustration (Gilliom & Shaw, 2004) appear to increase the risk of developing conduct problems. In longitudinal studies of young children, temperamental anger/frustration was found to predict externalizing problems (i.e., aggression, noncompliance) (Blair, 2002; Rydell et al., 2003). Lower cognitive ability scores along with lower executive functioning and memory abilities have been associated with conduct problems, especially early onset conduct problems (Lahey, 2008). Similarly, lower verbal intelligence and slower language development have been correlated with the development of conduct problems (Beitchman et al., 2001).

Parent and family characteristics have also been found to exert a strong influence on conduct problems. A meta-analytic review conducted by Rhee and Waldman (2002) found that 50% of the variance in those diagnosed with CD was attributable to genes. Inappropriate methods of parenting including coercive, harsh, inconsistent, and rejecting parenting behaviors have also been associated with conduct problems in children (Patterson, Reid, & Dishion, 1992). Additionally, inadequate supervision and punitive discipline – including abuse and neglect – are correlated with conduct problems (Lahey, Miller, Gordon, & Riley, 1999). Similar to anxiety disorders, rates of conduct problems were found to be higher for adolescents whose parents are divorced or separated rather than cohabitating or married, and also higher for adolescents whose parents are not college graduates (Merikangas et al., 2010). Lower familial income has been

associated with increased conduct problems (Coté, Vaillancourt, Le Blanc, Nagin, & Tremblay, 2006; Lahey et al., 1999); perhaps because circumstances that promote conduct problems are created by poverty (Lahey, 2008). Indeed, conduct problems are more prevalent in youth who live in impoverished and socially disorganized neighborhoods (Loeber, Farrington, Stouthamer-Loeber, & Van Kammen, 1998).

Additional risk factors that have been correlated with conduct problems include prenatal and perinatal factors and peer influence. Low birth weight (Brennan, Grekin, & Mednick, 2003), birth complications (e.g., hypoxia) (Raz, Shah, & Sander, 1996), and maternal cigarette smoking and substance use during pregnancy have been correlated with future conduct problems (Wakschlag, Pickett, Cook, Benowitz, & Leventhal, 2002). Evidence from longitudinal studies suggest that children who associate with peers who exhibit conduct problems are more likely to develop conduct problems of their own (Keenan, Loeber, & Zhang, 1995).

*Developmental Progression*

Conduct problems may be best understood by examining their different developmental trajectories (Lahey, 2008). Moffitt (1993, 2003) has proposed that youth with conduct problems follow two different trajectories. The childhood-onset (or life-course persistent) trajectory is followed by a relatively small number of youth (5% to 14%) who first exhibit conduct problems in childhood and continue to display symptoms during adolescence and throughout adulthood. The adolescent-onset (or adolescence-limited) trajectory is followed by a larger group of youth (10% to 21%) who display few conduct problems in childhood or adulthood. Moffit has hypothesized that the combination of neurodevelopmental deficits, inadequate parenting, and adverse social influences cause the conduct problems of youth on the childhood-onset trajectory, while adolescent-onset conduct problems are due to peer influences during the transition to

adulthood. Studies have shown that sex differences exist between the trajectories with equal numbers of males and females on the adolescent-onset trajectory and a male to female ratio of 3:1 on the childhood-onset trajectory (Lahey et al., 2006; Moffitt, Caspi, Rutter, & Silva, 2001).

Research on ODD and CD supports Moffit's (1993, 2003) theory that most youth with CD – as opposed to youth with ODD or subclinical levels of conduct problems – follow the childhood-onset trajectory. Lahey and colleagues (1998) found that most clinic-referred adolescents who met criteria for CD reported that they began exhibiting conduct problems in childhood. Only a small percentage of the sample reported an adolescent onset of conduct problems. A longitudinal study of a representative sample of girls found that almost every adolescent girl who met criteria for CD began displaying symptoms in childhood (Coté, Zocolillo, Tremblay, Nagin, & Vitaro, 2001).

The prevalence of ODD and CD has been found to vary with age (Lahey, 2008). ODD is more prevalent than CD during early childhood; however, prevalence rates of the disorders are nearly equal by adolescence (Lahey et al., 1999; Loeber, Burke, Lahey, Winters, & Zera, 2000; Maughan, Rowe, Messer, Goodman, & Meltzer, 2004). Although rates of ODD appear to remain stable between the ages of 13 and 18, Merikangas and colleagues (2010) found that rates of CD peak at 9.6% in late adolescence (ages 17 and 18). The increase in the prevalence of CD related to age is significantly greater for boys than for girls (Maughan et al., 2004; Moffitt et al., 2001).

Mental health outcomes of children and adolescents with conduct problems are important to acknowledge. Children with ODD and CD have an increased risk of developing other serious mental disorders in adolescence and adulthood (Lahey, 2008). For example, ODD and CD have both been associated with later depressive disorders (Burke, Loeber, Lahey, & Rathouz, 2005). Children diagnosed with CD are also at increased risk for engaging in criminal behavior

(Fergusson, Horwood, & Ridder, 2005; Kjelsberg, 2002) and abusing drugs and alcohol (Marshal & Molina, 2006) in adolescence and adulthood, as well as developing Antisocial Personality Disorder (Washburn et al., 2007).

*Neurological Findings in Conduct Problems*

Many of the same neurological structures and systems associated with anxiety have also been implicated in conduct problems, particularly the limbic system (i.e., amygdala) and the PFC. For example, grey matter reductions in limbic brain structures including the amygdala have been correlated with an increase in CD symptoms (Huebner et al., 2008). Impaired amygdala functioning has been related to conduct problems (Davidson, Putnam, & Larson, 2000), perhaps due to resulting difficulties in learning stimulus-incentive associations and in processing negative expressions of others (Drabick et al., 2010). A study conducted by Marsh and colleagues (2008) found that youth diagnosed with CD and ODD displayed lower levels of amygdala activation in response to fearful expressions when compared to control participants and participants with Attention-Deficit/Hyperactivity Disorder (ADHD). A similar pattern of hypoactivity was found by Jones, Laurens, Herba, Barker, and Viding in their 2009 study.

Structural and functional deficits of the PFC have also been associated with conduct problems (Isikawa & Raine, 2003; Raine, 2002). Boys with conduct problems, as compared to a control group, have exhibited increased grey matter concentration in the orbitofrontal cortex, a region of the PFC associated with decision making (De Brito et al., 2009). This finding may indicate that children with conduct problems experience a delay in cortical maturation, as normally developing boys experience a loss of frontal cortex grey matter starting between the ages of 10 and 11 years (Lenroot et al., 2007). Finger and colleagues (2008) found that children with diagnoses of ODD and CD displayed abnormal PFC activity when punished (i.e., losing

points) for making errors on a reversal learning task. Additionally, greater amygdala-PFC connectivity has been correlated with lower levels of CD and ODD symptom severity (Marsh et al., 2008).

The neurotransmitters, GABA and serotonin, along with the HPA axis appear to be important in the development of conduct problems, as well as anxiety disorders. Decreased levels of serotonin have been associated with aggression in non-clinical, clinical, and antisocial samples of adults (Eichelman, 1995; Limson, et al, 1991; Virkkunen, Nuutila, Goodwin, & Linnoila, 1987); however, findings in child and adolescent samples have been mixed (Hanna, Yuwiler, & Coates, 1995; Unis et al, 1997). The HPA axis seems to have the opposite effect on conduct problems than it does on anxiety in that lower levels of activation have been associated with increased levels of conduct problems. Specifically, aggression and other conduct problems have been correlated with several indicators of depressed HPA axis functioning including lower cortisol levels, lower heart rate, and lower skin conductance (Brennan et al., 1997; Raine, 1996; Raine, Venables, & Mednick, 1997).

## *Comorbidity of Anxiety and Conduct Problems*

### *Prevalence*

The comorbidity of conduct problems and anxiety is a significant problem with important implications for many children and adolescents. Estimates of rates of comorbidity of anxiety disorders and CD or ODD generally range from 3% to 13% (Costello, Egger, Angold, 2004), with one meta-analytic review reporting the rate of anxiety disorders among youth with either CD or ODD as approximately 40% (Angold, Costello, & Erkanli, 1999). In boys, the comorbidity odds ratios of having any anxiety disorder and CD or any anxiety disorder and ODD were found to be 1.6 and 0.8, respectively (Costello et al., 2003). The same study found

comorbidity odds ratios of having any anxiety disorder and CD or any anxiety disorder and ODD of 0.3 and 2.4, respectively, in girls. These analyses controlled for all other possible comorbidities.

*Implications*

Research suggests a variety of negative consequences are associated with the comorbidity of conduct problems and anxiety disorders. Studies show that the co-occurence of anxiety disorders and conduct problems results in more severe clinical presentations and increased academic problems, social impairment, and family dysfunction (Drabick, Gadow, & Loney, 2008; Greene et al., 2002; Maser & Cloninger, 1990). The presence of conduct problems and anxiety disorders have also been found to elevate risk for additional psychological conditions and negative sequelae including Major Depressive Disorder, affiliation with defiant peers, and substance use and abuse (Drabick et al., 2008; Fergusson & Horwood, 1999; Kendall, Brady, & Verduin, 2001; Merikangas & Avenevoli, 2000).

*Theories of Comorbidity*

A variety of theories have been proposed to explain the co-occurrence of anxiety disorders and conduct problems. These theories generally fall into one of two categories: 1) those that propose that one disorder precedes the other and 2) those that hypothesize that shared risk factors or etiological processes lead to comorbidity (Drabick et al., 2010). Some longitudinal studies of children and adolescents have found that conduct problems may precede anxiety disorders in clinical and community samples (Burke, Loeber, Lahey, & Rathouz, 2005; Lavigne et al., 2001; Roza, Hofstra, van der Ende, & Verhulst, 2003; Speltz, McClellan, DeKlyen, & Jones, 1999). Research also exists, however, suggesting that anxiety disorders may precede conduct problems (Foley, Pickles, Maes, Silberg, & Eaves, 2004; Last, Perrin, Hersen, &

Kazdin, 1996). There is also evidence that shared risk factors including exposure to neighborhood violence, parent-child processes, social information processing biases, and temperament may lead to the comorbidity of anxiety disorders and conduct problems (Bubier & Drabick, 2009; Muris & Ollendick, 2005).

One reason for the variety of explanations and mixed findings may be that research has not been sensitive to the complexity of the comorbidity of anxiety disorders and conduct problems. For example, studies often examine a combination of externalizing disorders including ADHD with CD and ODD when testing longitudinal trajectories making it difficult to understand the specific associations between symptoms and diagnoses (Drabick et al., 2010). Furthermore, research may not take developmental periods into account. Some anxiety disorders (e.g., SAD) may be more likely to precede conduct problems, while others (e.g., GAD) may be more likely to develop after the onset of conduct problems (Bubier & Drabick, 2009; Costello et al., 2003). These important factors may not be adequately accounted for in research that is not longitudinal and research that uses the broad definitions of internalizing and externalizing disorders to describe anxiety disorders and conduct problems. Despite the apparent weaknesses in the research, it is clear that a variety of explanations for the comorbidity of anxiety disorders and conduct problems are possible.

In addition to there being multiple explanations of the etiology of comorbid anxiety disorders and conduct problems, there appear to be multiple patterns that characterize the association between anxiety disorders and conduct problems. Specifically, anxiety may mitigate or reduce conduct problems or anxiety may exacerbate or increase conduct problems.

*Buffering hypothesis (Hofmann, Richey, Kashdan, & McKnight, 2009; Walker et al., 1991).* The buffering hypothesis suggests that anxiety serves to lessen conduct problems in

children and adolescents with comorbidity. Findings from several studies support the hypothesized buffering effect of anxiety. Hofmann and colleagues found that co-occurring anxiety disorders moderated the association between conduct problems and substance use disorders in adults with disruptive behavior disorders (CD, ODD, and/or ADHD). Specifically, comorbid anxiety disorders were associated with lower odds for substance use problems in these individuals when compared to individuals without co-occurring anxiety disorders in a nationally representative sample. Hofmann and colleagues (2009) postulated that this moderating effect may be due to fear of bodily symptoms preventing individuals with conduct problems from engaging in drug-seeking behaviors. Similarly, Walker and colleagues (1991) found lower levels of impairment (e.g., less aggression, reduced frequency of police contact) in youth with comorbid anxiety and conduct problems when compare to youth with conduct problems alone.

*Multiple problem hypothesis (Garai, Forehand, Colletti, & Rakow, 2009; Lansford et al., 2008; Ollendick, Seligman, & Butcher,1999).* There is also research that supports the multiple problem hypothesis; that is, that anxiety exacerbates conduct problems. Co-occurring conduct problems and internalizing problems have been associated with higher rates of co-occurring substance use disorders, with substance use peaking during adolescence (Lansford et al., 2008). Anxiety has also been shown to attenuate levels of risk taking behaviors, age of first offense, or the frequency or severity of delinquent acts for youth meeting diagnostic criteria for CD (Ollendick et al., 1999).

*Dual-pathway model.* It is clear that neither the buffering hypothesis nor the multiple problem hypothesis provide a comprehensive explanation of the association between anxiety and conduct problems. In an attempt to create a more accurate developmental model of comorbidity of ODD and anxiety disorders, Drabick, Ollendick, and Bubier (2010) have proposed an

integrated dual-pathway model (see Table 12). Although the model specifically looks at the comorbidity of ODD and anxiety disorders, its utility in understanding the comorbidity of more broadly defined conduct problems and anxiety disorders appears appropriate given the commonalities (i.e., risk factors and clinical presentation) between CD and ODD. The model suggests that there are two groups of youth with co-occurring ODD and anxiety disorders: one group for which the multiple problem hypothesis is more accurate and a second group for which the buffering hypothesis is more accurate.

Table 12

*Dual-pathway model of comorbid ODD and anxiety disorders in children and adolescents*

| **Proposed process** | **Subgroup 1: Multiple problem hypothesis (anxiety exacerbates ODD)** | **Subgroup 2: Buffering hypothesis (anxiety mitigates ODD)** |
|---|---|---|
| Negative emotionality | High anger/frustration<br>Low fear | Low anger/frustration<br>Moderate to high fear |
| Social information processing | Poor | Age appropriate; biases attenuated by PFC |
| Aggression function | Reactive | Proactive |
| Effortful control | Low levels | Age-appropriate levels |
| Limbic System | Overactive | Overactive but attenuated by PFC |
| Executive functioning | Poor | Age-appropriate |

(Drabick et al., 2010)

What differentiates the two groups is largely neurological. According to Drabick and colleagues (2010), youth in the first subgroup are expected to exhibit decreased responsiveness in the ventromedial and ventrolateral PFC and increased responsiveness in neural regions associated with threat assessment, such as the amygdala. The authors propose that this would result in dysregulation of the limbic system leading to impaired transmission of information from

the ventral PFC to the dorsolateral PFC resulting in impaired PFC functioning. PFC impairments could lead to problems with planning, problem-solving, behavioral selection, emotion control, and self-monitoring. Additionally, such impairments could render the PFC ineffective in modulating amygdala over-activity. As a result of these effected neurological processes, individuals in this subgroup are hypothesized to exhibit higher levels of negative emotionality, reactive aggression, and emotion dysregulation; difficulties with social-information processing; lower effortful control (i.e., temperamental self-regulation); and poorer executive functioning abilities.

The second subgroup of youth – the group for which the buffering hypothesis is more accurate – is also expected by the authors to exhibit high levels of negative emotionality (e.g., fear/sadness) and overactive limbic system functioning in response to angry, sad, and threatening stimuli. For this group, however, it is hypothesized that age-appropriate PFC functioning would attenuate the limbic system's overactivity and the expression of negative emotionality. Modulated limbic system activity would facilitate reciprocal limbic system-PFC connections resulting in developmentally appropriate levels of effortful control, social information processing and executive functioning abilities.

### *Anxiety, Conduct Problems, and Executive Functioning*

#### *What is Executive Functioning?*

Executive functioning is a heterogeneous construct that has been defined in a variety of ways. Anderson (1998) broadly defined executive functions as "those skills necessary for purposeful, goal-directed activity" (p. 319). Willicutt, Doyle, Nigg, Faraone, and Pennington (2005) provided a similar yet slightly more specific definition of executive function as "neurocognitive processes that maintain an appropriate problem-solving set to attain a later goal"

(p. 1336). Baron's (2004) definition is even more specific and emphasizes the "metacognitive capacities that allow an individual to perceive stimuli from his or her environment, respond adaptively, flexibly change direction, anticipate future goals, consider consequences, and respond in an integrated or common-sense way, utilizing all these capacities to serve a common purposive goal" (p. 135). Although definitions of executive functioning vary, it is generally accepted that executive functions are higher order functions that integrate other, more basic functions such as attention, memory, and perception (Baron, 2004). These higher order functions include a myriad of abilities or subdomains, with some being more consistently endorsed based on empirical research than others. The subdomains most frequently associated with executive functioning include planning, working memory, self-monitoring, cognitive flexibility, fluency, attention, and response inhibition (Baron, 2004; Henry & Bettenay, 2010). A list of common executive functioning subdomains is presented in Appendix B.

The heterogeneity of executive functioning has resulted in difficulties developing sensitive and specific measures of the construct (Archibald & Kerns, 1999). One significant problem has been that executive functioning subdomains and abilities are often also subsumed under other cognitive domains (Baron, 2004). Due to the nature of executive functions being higher order functions, many of its subdomains cannot be dissociated from constructs such as attention, information processing speed, and memory, therefore making it difficult to measure "pure" executive functioning abilities. Nevertheless, there are many tests that have been found to be reliable and valid measures of specific subdomains of executive functioning (Baron, 2004; Henry & Bettenay, 2010). Of relevance to this study, the Controlled Oral Word Association Test (COWAT) has been found to assess verbal fluency (also incorporating inhibition, self-monitoring, and shifting abilities; Baron, 2004); the Trail Making Test (TMT; Reitan, 1958) to

assess attention, processing speed, and cognitive flexibility (also incorporating inhibition and self-monitoring; Kelly, 2000); and the Wisconsin Card Sorting Test (WSCT; Heaton, 1993) to assess cognitive flexibility (also incorporating abstract reasoning and attention; Baron, 2004).

Executive functioning in children and adolescents has been associated with multiple neural networks including those involving the basal ganglia and thalamus, as well as the frontal cortex (Anderson, 1998; Willicutt et al., 2005). The frontal lobes, especially the PFC, dorsolateral regions, and subcortical connections, appear to be particularly important for performing tasks associated with the most common executive functioning abilities (Baron, 2004). PFC dysfunction has been associated with impaired executive functioning abilities such as cognitive flexibility and inhibition as measured by tests such as the WCST and Stroop Color-Word Test in adolescents (Baron, 2004; Kim, Kim, & Kwon, 2001). Similarly, positron emission tomography (PET) research with adolescents has found left inferior frontal cortex and left dorsolateral PFC activation to be significantly associated with performance on tests of verbal fluency (Ravnkilde, Videbech, Rosenberg, Gjedde, & Gade, 2002). The PFC has been widely recognized as important for intact working memory abilities, both verbal and non-verbal (Baron, 2004). In adults, correlations have been found between specific activation of the dorsolateral PFC (Baker et al., 1996) and the left PFC (Morris, Ahmed, Syed, & Toone, 1993) when engaging in planning tasks such as the Tower of London; however, this correlation has not been found in children likely due to immaturity of prefrontal circuitry (Luciana & Nelson, 1998). Studies examining the neuroanatomical underpinnings of attention in children have highlighted the importance of the inferior parietal lobe, orbital frontal region, limbic system, subcortical systems, and midbrain systems (Baron, 2004). The PFC appears to be especially important for alerting or sustained attention in both children and adults (Posner & Peterson, 1990). Although

many structures and complex neurological processes are involved in executive functioning, it is clear that the PFC plays a particularly important role in a variety of relevant subdomains and abilities.

*Executive Functioning and Anxiety*

Relatively little is known about the nature of executive functioning abilities related to anxiety disorder, especially in children and adolescents. The research that does exist is mixed. Castaneda and colleagues (2011) found no significant differences between young adults (ages 21 to 35 years) with anxiety disorders and control participants on measures of verbal and visual working memory, processing speed, cognitive flexibility, ability to shift attention, and strategy. Similarly, no correlation has been shown between anxiety symptom severity and performance on several measures of executive functioning (WCST, Trail Making Test, COWAT, and the Letter-Number Sequencing subtest of the Weschler Adult Intelligence Scale-III [WAIS-III]) in an adult psychiatric sample (Smitherman, Huerkamp, Miller, Houle, & O'Jile, 2007).

Other research with adults suggests that anxiety may be negatively associated with executive functioning abilities. In contrast to the findings of Smitherman and colleagues (2007), Gass, Ansley, and Boyette (1994) found that verbal fluency performance, as measured by the COWAT, was moderately correlated with levels of generalized anxiety in a sample of adult male psychiatric patients. Lautenbacher, Spernal and Krieg (2002) found that adults with panic disorder exhibited divided attention impairments when compared to controls; however, Gladsjo and colleagues (1997) failed to find the same effect in a similar sample. A study by Purcell, Maruff, Kyrios, and Pantelis (1998) found spatial working memory deficits in patients with Obssessive-Compulsive Disorder (OCD), but no delays in attentional set shifting or planning were observed.

Research on children and adolescents is similarly mixed and very limited. A study of children ages 6 to 14 found that participants with anxiety disorders exhibited inhibition deficits compared to normal controls; however, this association disappeared once analyses controlled for comorbid ADHD (Korenblum, Chen, Manassis, & Schachar, 2007). No significant differences in performance monitoring were found between the groups. No differences were found between groups of 6- to 10-year-old anxious children and control children on one measure of processing speed and attention (Trails A), but there was a significant difference in group performance on a more complex task involving shifting and inhibition (Trails B) (Kusché, Cook, & Greenberg, 1993). Specifically, anxious children took significantly longer to complete the task than children in the control group. Shin and colleagues (2008) also found executive function deficits in cognitive flexibility as measured by performance on the WCST in children diagnosed with OCD. Interestingly, no significant difference between groups in performance on another measure of cognitive flexibility and inhibition (Stroop Color-Word Test) was found (Shin et al., 2008). On a test of verbal fluency (Verbal Fluency subtest of the McCarthy Scales of Children's Abilities), Kusché and colleagues (1993) found that anxious children exhibited impaired performance when compared to normal controls. Anxiety was also found by Tapasak, Roodin, and Vaught (1978) to impair verbal fluency performance in a sample of children ages 8 to 18, but only for females.

*Executive Functioning and Conduct Problems*

The quantity of research examining the association between executive functioning and conduct problems is significantly greater than that looking at executive functioning and anxiety; however, the findings are similarly inconsistent. Longitudinal research in young children has shown that slower gains in executive functioning across the transition to elementary school predict increased teacher-rated conduct/peer problems (Hughes & Ensor, 2011). Specifically,

diminished inhibitory control has been linked with externalizing problems in children as young as four years old, and the association has been found to persist into the elementary school-age years (Kusché et al., 1993; Utendale & Hastings, 2011; Utendale, Hubert, Saint-Pierre, & Hastings, 2011; Youngwirth, Harvey, Gates, Hashim, & Friedman-Weieneth, 2007). Another study examining executive functioning in preschool children found that aggressive behaviors were associated with inhibition deficits with boys exhibiting more impairment than girls (Raaijmakers et al., 2008). Impairments in response inhibition have also been associated with the behavioral disinhibition exhibited in children with conduct problems in a sample of youth ages 12 to 17 (Young et al., 2009), as well as with CD in young adults (Fisher, Barkley, Smallish, & Fletcher, 2005).

In addition to impairments in response inhibition, youth with externalizing disorders have also been found to display impairments in processing speed, attention, and shifting as evidenced by longer completion times for Trails A and Trails B compared to normal controls (Kusché et al., 1993; Närhi, Lehto-Salo, Ahonen, & Marttunen, 2010). Hobson, Scott, and Rubia (2011) reported that adolescents with conduct problems showed increased risky decision-making and decreased motor inhibition compared to control participants and participants with ADHD, and impaired sustained attention and response execution abilities compared to control participants. Deficits in cognitive flexibility, as measured by the WCST, have been evidenced in adolescents with CD (Kim et al., 2001; Pajer et al., 2008; Närhi et al., 2010), and have been associated with an increased risk of engaging in violent behaviors (Miura, 2009). Youth with conduct problems have also been reported to exhibit verbal fluency and working memory ability impairments (Närhi et al., 2010; Speltz, DeKlyen, Calderon, Greenberg, & Fisher, 1999), and working memory abilities have been negatively correlated with physically aggressive behaviors (Séguin,

Boulerice, Harden, Trembly, & Pihl, 1999). Both working memory abilities and task-set shifting have been inversely associated with behavioral disinhibition (Young et al., 2009).

In contrast to the findings just presented, a number of studies have reported no significant differences between the performance of youth with externalizing disorders and control participants on measures of verbal fluency (Kusché et al., 1993; Oosterlaan, Scheres, & Sergeant, 2005; Pajer et al., 2008; Thorell & Wåhlstedt, 2006). No significant differences have also been reported between youth with externalizing disorders and controls on measures of inhibition (Kusché et al., 1993; Pajer et al., 2008; Thorell & Wåhlstedt, 2006). Hobson and colleagues (2011) failed to find differences between adolescents with conduct problems and control participants on a measure of cognitive flexibility (Wilding Monster Sorting Task). Kim and colleagues (2001) reported no differences between adolescents with CD and controls on measures of attention and working memory, and Närhi and colleagues (2010) failed to find impairments in planning abilities. Similarly, Oosterlaan and colleagues (2005) found no significant differences between children with CD or ODD and controls on measures of working memory and planning. Speltz and colleagues (1999) also failed to find planning deficits and Thorell and Wåhlstedt (2006) found no impairments in working memory abilities in samples of preschool boys with ODD.

When examining the relationship between executive functioning and conduct problems, it is important to acknowledge the influence of ADHD, as ADHD has been linked to executive functioning impairments, as well as conduct problems. One review of executive functioning literature found significant differences between ADHD subjects and controls on one or more measures of executive functioning in fifteen out of eighteen studies, with the ADHD group performing significantly worse than controls (Pennington & Ozonoff, 1996). Executive

functioning measures included in the review assessed various dimensions of executive functioning including planning, interference control, response inhibition, motor inhibition, working memory, vigilance, and processing speed. A more recent meta-analysis found similar results reporting significant group differences between subjects with and without ADHD on thirteen executive functioning tasks measuring response inhibition, vigilance, set-shifting, planning, verbal working memory, and spatial working memory (Willicutt et al., 2005).

ADHD has also been associated with CD and ODD, both in clinical and community samples. In boys, the comorbidity odds ratios of having ADHD and CD or ADHD and ODD were found to be 1.9 and 6.6, respectively (Costello et al., 2003). The same study found comorbidity odds ratios of having any ADHD and CD or any ADHD and ODD of 3.9 and 56.3, respectively, in girls. These analyses controlled for all other possible comorbidities. Loeber, Green, Keenan, ,and Lahey (1995) found that nearly 50% of clinic referred boy aged 7 to 12 developed CD within one to five years. In a longitudinal community study, Costello and colleagues (2003) found that ADHD diagnosis predicted the onset of ODD. Similarly, van Lier, van der Ende, Koot, and Verjulst (2007) found the that ADHD predicted the onset of CD in a sample of females.

*Executive Functioning in Youth with Comorbid Anxiety and Conduct Problems*

A thorough search of the literature revealed only one study (Kusché et al., 1993) that has examined executive functioning abilities in children with comorbid anxiety and conduct problems, indicating a large gap in research. Kusché and colleagues (1993) found no significant differences between controls and children with comorbid anxiety and externalizing disorders in cognitive flexibility and inhibition as measured by performance on the Stroop Color-Word Test. Children with comorbid anxiety and externalizing disorders were found to exhibit impairments in

verbal fluency performance when compared to control participants and children with noncomorbid externalizing disorder.

Additional research examining general and specific executive functioning abilities in children and adolescents with comorbid anxiety and conduct problems would contribute greatly to the field. Not only would such research serve to fill a large gap in the literature, but it may also help clarify the nature of the association between anxiety and conduct problems. Specifically, the dual-pathway model of anxiety and conduct problems may be supported if it is found that executive functioning abilities moderate the effect of anxiety on conduct problems.

## Appendix B: Common Executive Functioning Subdomains

Abstract Reasoning

Anticipation

Attentional Control

Behavioral Regulation

Cognitive Flexibility

Concept Formation

Estimation

Fluency

Goal Setting

Hypothesis Generation

Initiative

Organization

Planning

Problem Solving

Response Inhibition

Self-control

Self-monitoring

Set Shifting

Working Memory

(Baron, 2004)

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.). Washington, DC: Author.

Anderson, V. (1998). Assessing executive functions in children: Biological, psychological and developmental considerations. *Neuropsychological Rehabilitation, 8,* 319-349.

Anderson, J. C., & Gerbing, D. W. (1988). Structural equation modeling in practice: A review and recommended two-step approach. *Psychological Bulletin, 103,* 411-423.

Angold, A., Costello, E. J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40,* 57-87.

Archibald, S., & Kerns, K. A. (1999). Identification and description of new tests of executive functioning in children. *Child Neuropsychology, 5,* 115-129.

Baker, S. C., Rogers, R.. D., Owen, A. M., Frith, C., D., Dolan, R. J., Frackowiak, R. S. J., & Robbins, T. W. (1996). Neural systems engaged by planning: A PET study of the Tower of London task. *Neuropsychologia, 34,* 515-526.

Baron, I. S. (2004). *Neuropsychological Evaluation of the Child.* New York, NY: Oxford University Press.

Baving, L., Laucht, M., & Schmidt, M. H. (2002). Frontal brain activation in anxious school children. *Journal of Child Psychology and Psychiatry, 43,* 265-274.

Beitchman, J. H., Wilson, B., Johnson, C. J., Atkinson, L., Young, A., Adlaf, E.,…Douglas, L. (2001). Fourteen-year follow-up of speech/language-impaired and control children: Psychiatric outcome. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 75-82.

Blair, C. (2002). Early intervention for low birth weight, preterm infants: The role of negative emotionality in the specification of effects. *Development and Psychopathology, 14,* 311-322.

Blair, R. J. R. (2004). The roles of the orbital frontal cortex in the modulation of antisocial behavior. *Brain and Cognition, 55,* 198-208.

Brener, N. D., Billy, J. O. G., & Grady, W. R. (2003). Assessment of factors affecting the validity of self-reported health-risk behavior among adolescents: Evidence from the scientific literature. *Journal of Adolescent Health, 33,* 436-457.

Brennan, P. A., Grekin, E. R., & Mednick, S. (2003). Prenatal and perinatal influences on conduct disorder and serious delinquency. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and serious delinquency* (pp. 319-344). New York: Guilford.

Brennan, P. A., Rain, A., Schulsinger, F., Kirkegaard-Sorensen, L., Knop, J., Hutchings, B.,…Mednick, S. A. (1997). Psychophysiological protective factors for male subjects at high risk for criminal behavior. *American Journal of Psychiatry, 154,* 853-855.

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136-162). Newbury Park, CA: Sage.

Bubier, J., & Drabick, D. A. (2009). Co-occurring anxiety and disruptive behavior disorders: The roles of anxious symptoms, reactive aggression, and shared risk processes. *Clinical Psychology Review, 29,* 658-669.

Burke, J. D., Loeber, R., Lahey, B. B., & Rathouz, P. J. (2005). Developmental transitions among affective and behavioral disorders in adolescent boys. *Journal of Child Psychology and Psychiatry, 46,* 1200-1210.

Butcher, J. N., Williams, C. L., Graham, J. R., Archer, R. P., Tellegen, A., Ben-Porath, Y. S., & Kaemmer, B. (1992). *Minnesota Multiphasic Personality Inventory – Adolescent Version (MMPI-A).* Minneapolis, MN: University of Minnesota Press.

Cameron, M., & Guterman, N. B. (2007). Diagnosing conduct problems of children and adolescents in residential treatment. *Child & Youth Care Forum, 36*(1), 1-10.

Castaneda, A. E., Suvisaari, J., Marttunen, M., Perälä, J., Saarni, S. I, Aalto-Setälä,…Tuulio-Henriksson, A. (2011). Cognitive functioning in a population-based sampled of young adults with anxiety disorders. *European Psychiatry, 26,* 346-353.

Costello, E. J., Egger, H. L., & Angold, A. (2004). Developmental epidemiology of anxiety disorders. In T. H. Ollendick & J. S. March (Eds.), *Phobic and anxiety disorders in children and adolescents: A clinician's guide to effective psychosocial and pharmacological interventions* (pp. 61-91). New York: Oxford University Press.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., & Angold, A. (2003). Prevalence and development of psychiatric disorders in childhood and adolescence. *Archives of General Psychiatry, 60,* 837-844.

Coté, S., Tremblay, R. E., Nagin, D. S., Zoccolillo, M., & Vitaro, F. (2002). Childhood behavioral profiles leading to adolescent conduct disorder: risk trajectories for boys and girls. *Journal of the American Academy of Child and Adolescent Psychiatry, 41,* 1086-1094.

Coté, S. M., Vaillancourt, T., Le Blanc, J. C., Nagin, D. S., & Tremblay, R. E. (2006). The development of physical aggression from toddlerhood to pre=adolescence: A nationwide longitudinal study of Canadian children. *Journal of Abnormal Child Psychology, 34,* 71-85.

Coté, S., Zoccolillo, M., Tremblay, R. E., Nagin, D., & Vitaro, F. (2001). Predicting girls' conduct disorder in adolescence from childhood trajectories of disruptive behaviors. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 678-684.

Curran, P. J., West, S. G., & Finch, J. (1996). The robustness of test statistics to nonnormality and specification error in confirmatory factor analysis. *Psychological Methods, 1,* 16-29.

Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition and Emotion, 12,* 307-320.

Davidson, R. J., & Fox, N. A. (1989). Frontal brain asymmetry predict infants' response to maternal separation. *Journal of Abnormal Psychology, 98,* 127-131.

Davidson, R. J., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: regional brain electrical and autonomic activity in social phobics during anticipation of public speaking. *Biological Psychiatry, 47,* 85-95.

Davidson, R., Putnam, K., & Larson (2000). Dysfunction in the neural circuitry of emotion regulation: A possible prelude to violence. *Science, 289,* 591-594.

De Brito, S. A., Mechelli, A., Wilke, M., Laurens, K. R., Jones, A. P., Barker, G. J.,…Viding, E. (2009). Size matters: Increased grey matter in boys with conduct problems and callous-unemotional traits. *Brain, 132,* 843-852.

Drabick, D. A. G., Gadow, K. D., & Loney, J. (2008). Co-occuring ODD and GAD symptom groups: Source-specific syndromes and cross-informant comorbidity. *Journal of Clinical Child and Adolescent Psychology, 37,* 314-326.

Drabick, D. A. G., Ollendick, T. H., & Bubier, J. L. (2010). Co-occurrence of ODD and Anxiety: Shared Risk Processes and Evidence for a Dual-Pathway Model. *Clinical Psychology Science and Practice, 17,* 307-318.

Eichelman, B. (1995). *Animal and evolutionary models of impulsive aggression: Impulsivity and aggression.* Oxford: John Wiley & Sons.

Eley, T. C. (2001). Contributions of behavioral genetics research: Quantifying genetic, shared environmental and nonshared environmental influences. In M. W. Vasey and M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 45-59). London: Oxford University Press.

Fergusson, D. M., & Horwood, L. J. (1999). Prospective childhood predictors of deviant peer affiliations in adolescence. *Journal of Child Psychology and Psychiatry, 40,* 581-592.

Fergusson, D. M., Horwood, L. J., & Ridder, E. M. (2005). Show me the child at seven: The consequences of conduct problems in childhood for psychosocial functioning in adulthood. *Journal of Child Psychology and Psychiatry, 46,* 837-849.

Finger, E.C., Marsh, A. A., Mitchell, D. G. V., Reid, M. E., Sims, C., Budhani, S.,…Blair, J. R. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of General Psychiatry, 65,* 586-594.

Fischer, M., Barkley, R. A., Smallish, L., & Fletcher, K. (2005). Executive functioning in hyperactive children as young adults: Attention, Inhibition, Response Perseveration, and the Impact of Comorbidity. *Developmental Neuropsychology, 27,* 107-133.

Foley, D. L., Pickles, A., Maes, H. M., Silberg, J. L., & Eaves, L. J. (2004). Course and short-term outcomes of separation anxiety disorder in a community sample of twins. *Journal of the American Academy of Child and Adolescent Psychiatry, 43,* 1107-1114.

Garai, E. P., Forehand, R., Colletti, C. J. M., & Rakow, A. (2009). The role of anxiety/depression the development of youth high risk behaviors: An examination of two competing hypotheses in a sample of African-American, single mother families. *Journal of Psychopathology and Behavioral Asssessment, 31,* 340-346.

Gass, C. S., Ansley, J., & Boyette, S. (1994). Emotional correlates of fluency test and maze performance. *Journal of Clinical Psychology, 50,* 586-590.

Gilliom, M., & Shaw, D. S. (2004). Codevelopment of externalizing and internalizing problems in early childhood. *Development and Psychopathology, 16,* 313-333.

Ginsburg, G. S., & Silverman, W. K. (1996). Phobic and anxiety disorder in Hispanic and Caucasian youth. *Journal of Anxiety Disorders, 10,* 517-528.

Ginsburg, G. S., & Silverman, W. K. (2000). Gender role orientation and fearfulness in children with anxiety disorders. *Journal of Anxiety Disorders, 14,* 57-68.

Gladsjo, J. A., Rapaport, M. H., McKinney, R., Lucas, J. A., Rabin, A., Oliver, T.,…Judd, L. L. (1998). A neuropsychological study of panic disorder: negative findings. *Journal of Affective Disorders, 49,* 123-131.

Gordon, J. A., & Hen, R. (2004). The serotonergic system and anxiety. *Neuromolecular Medicine, 5,* 27-40.

Greene, R. W., Biederman, J., Zerwas, S., Monuteaux, M., Goring, J. C., & Faraone, S. V. (2002). Psychiatric comorbidity, family dysfunction, and social impairment in referred

youth with oppositional defiant disorder. *American Journal of Psychiatry, 159,* 1214-1224.

Greve, K. W., Brooks, J., Crouch, J., Williams, M., & Rice, W. (1997). Factorial structure of the Wisconsin Card Sorting Test. *British Journal of Clinical Psychology, 36,* 283-285.

Guadagnoli, E., & Velicer, W. F. (1988). Relation of sample size to the stability of component patterns. *Psychological Bulletin, 103,* 265-275.

Gunnar, M. (2001). Cortisol and anxiety. In M. W. Vasey & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety.* London: Oxford University Press.

Guyer, A. E., Lau, J. Y. F., McClure-Tone, E. B., Parrish, J., Shiffrin, N. D., Reynolds, R. C.,…Nelson, E. E. (2008). Amygdala and ventrolateral prefrontal cortex function during anticipated peer evaluation in pediatric social anxiety. *Archive of General Psychiatry, 65,* 1303-1312.

Hanna, G., Yuwiler, A., & Coates, J., (1995). Whole blood serotonin and disruptive behaviors in juvenile obsessive-compulsive disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 28-35.

Harden, K. P., D'Onofrio, B. M., Van Hulle, C., Turkheimer, E., Rodgers, J. L., Waldman, I. D., & Lahey, B. B. (2009). Population density and youth antisocial behavior. *Journal of Child Psychology and Psychiatry, 50*(8), 999-1008.

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, Inc.

Henry, L. A., & Bettenay, C. (2010). The assessment of executive functioning in children. *Child and Adolescent Mental Health, 15,* 110-119.

Henry, B., Caspi, A., Moffitt, T. E., & Silva, P. A. (1996). Temperamental and familial predictors of violent and nonviolent criminal convictions: Age 3 to age 18. *Developmental Psychology, 32,* 614-623.

Hobson, C. W., Scott, S., & Rubia, K. (2011). Investigation of cool and hot executive function in ODD/CD independently of ADHD. *Journal of Child Psychology and Psychiatry, 52,* 1035-1043.

Hoffman, S. G., Richey, J. A., Kashdan, T. B., & McKnight, P. E. (2009). Anxiety disorders moderate that association between externalizing problems and substance use disorders: Data from the National Comorbidity Survey – Revised. *Journal of Anxiety Disorders, 23,* 529-534.

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6,* 1-55.

Huebner, T., Vloet, T. D., Marx, I., Konrad, K., Fink, G. R., Herpertz, S. C., & Herpertz-Dahlmann, B. (2008). Morphometric brain abnormalities in boys with conduct disorder. *Journal of the Academy of Child and Adolescent Psychiatry, 47,* 540-547.

Hughes, C., & Ensor, R. (2011). Individual differences in growth in executive function across the transition to school predict externalizing and internalizing behaviors and self-perceived academic success at 6 years of age. *Journal of Experimental Child Psychology, 108,* 663-676.

Isikawa, S. S., & Raine, A. (2003). Prefrontal deficit and antisocial behavior: A causal model. In B. B. Lahey, T. E. Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 277-304). New York: Guilford.

Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G., Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry, 166,* 95–102.

Kamphaus, R. W. (2005). *Clinical Assessment of Child and Adolescent Intelligence (Second Edition).* New York, NY: Springer Science + Business Media, Inc.

Keenen, K., Loeber, R., & Zhang, Q. (1995). The influence of deviant peers on the development of boys' disruptive and delinquent behavior: A temporal analysis. *Development and Psychopathology, 7,* 715-726.

Keith, T. Z. (2006). *Multiple Regression and Beyond.* Boston, MA: Pearson Education, Inc.

Keily, M., Bates, J., Dodge, K., & Pettit, G. (2001). Effects of temperament on the development of externalizing and internalizing behaviors over 9 years. In F. Columbus (Ed.), *Advances in psychology research* (Vol. 6, pp. 255-288). Hauppauge, NY: Nova Science Publishers.

Kelly, T. (2000). The development of executive function in school-aged children. *Clinical Neuropsychological Assessment, 1,* 38-55.

Kendall, P. C., Brady, E. U., & Verduin, T. L. (2001). Comorbidity in childhood anxiety disorders and treatment outcome. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 787-794.

Kim, M-S., Kim, J-J., & Kwon, J. S. (2001). Frontal P300 decrement and executive dysfunction in adolescents with conduct problems. *Child Psychiatry and Human Development, 32,* 93-106.

Klein, A., & Moosbrugger, A. (2000). Maximum likelihood estimation of latent interaction effects with the LMS method. *Psychometricka, 65,* 457-474.

Klein, A. G., & Muthén, B. O. (2007). Quasi-maximum likelihood estimation of structural equation models with multiple interaction and quadratic effects. *Multivariate Behavioral Research, 42,* 647-673.

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York: Guilford.

Kline, R. (2010). *Principles and practice of structural equation modeling* (3rd ed.). New York: Guilford.

Kjelsberg, E. (2002). *DSM-IV* conduct disorder symptoms in adolescents as markers of registered criminality. *European Child and Adolescent Psychiatry, 11,* 2-9.

Korenblum, C. B., Chen, S. X., Manassis, K., & Schachar, R. J. (2007). Performance monitoring and response inhibition in anxiety disorders with and without comorbid ADHD. *Depression and Anxiety, 24,* 227-232.

Kristensen, H., & Torgersen, S. (2008). Is social anxiety disorder in childhood associated with developmental deficit/delays? *European Child and Adolescent Psychiatry, 17,* 99-107.

Kusché, C. A., Cook, E. T., & Greenberg, M. T. (1993). Neuropsychological and cognitive functioning in children with anxiety, externalizing, and comorbid psychopathology. *Journal of Clinical Child Psychology, 22,* 172-195.

Lacy, M. A., Gore, Jr., P. A., Pliskin, N., Henry, G. K., Heilbronner, R. L., & Hamer, D. P. (1996). Verbal fluency task equivalence. *The Clinical Neuropsychologist, 10,* 305-308.

Lahey, B. B. (2008). Oppositional Defiant Disorder, Conduct Disorder, and Juvenile Delinquency. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and Adolescent Psychopathology* (pp. 335-369). Hoboken, NJ: John Wiley & Sons, Inc.

Lahey, B. B., Loeber, R., Quay, H. C., Applegate, B., Shaffer, D., & Waldman, I. (1998). Validity of *DSM-IV* subtypes of conduct disorder based on age of onset. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 435-442.

Lahey, B. B., Miller, T. L., Gordon, R. A., & Riley, A. (1999). Developmental epidemiology of the disruptive behavior disorders. In H. Quay & A. Hogan (Eds.), *Handbook of the disruptive behavior disorders* (pp. 23-48). New York: Plenum.

Lahey, B. B., Van Hulle, C. A., Waldman, I. D., Rodgers, J. L., D'Onofrio, B. M., Pedlow, S.,…Kennan, K. (2006). Testing descriptive hypotheses regarding sex differences in the development of conduct problems and delinquency. *Journal of Abnormal Child Psychology, 34,* 737-755.

Lansford, J. E., Erath, S., Yu, T., Pettit, G. S., Dodge, K. A., & Bates, J. E. (2008). The developmental course of illicit substance use from age 12 to 22: Links with depressive, anxiety and behavior disorders at age 18. *Journal of Child Psychology and Psychiatry, 49,* 877-885.

Last, C., Perrin, S., Hersen, M., & Kazdin, A. E. (1996). A prospective study of childhood anxiety disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 35,* 1502-1510.

Lautenbacher, S., Spernal, J., Krieg, J-C. (2002). Divided and selective attention in panic disorder. A comparative study of patients with panic disorder, major depression and healthy controls. *European Archives of Psychiatry and Clinical Neuroscience, 252,* 210-213.

Lavigne, J. V., Cicchetti, C., Gibbons, R. D., Bimms, H. J., Larsen, L., & DeVito, C. (2001). Oppositional defiant disorder with onset in preschool years: Longitudinal stability and

pathways to other disorders. *Journal of the American Academy of Child and Adolescent Psychiatry, 40,* 1393-1400.

LeDoux, J. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23,* 155-184.

Lenroot, R. K., Gogtay, N., Greenstein, D. K., Wells, E. M., Wallace, G. L., Clasen, L. S.,…Giedd, J. N. (2007). Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *NeuroImage, 36,* 1065-1073.

Limson, R., Goldman, D., Roy, A., Lamparski, D., Ravitz, B., Adinoff, B., & Linnoila, M. (1991). Personality and cerebrospinal fluid monoamine metabolites in alcoholics and controls. *Archives of General Psychiatry, 48,* 437-441.

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data, 2nd ed.* New York, NY: Wiley.

Loeber, R., Burke, J. D., Lahey, B. B., Winters, A., & Zera, M. (2000). Oppositional defiant and conduct disorder: A review of the past 10 years, Part I. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1468-1484.

Loeber, R., Farrington, D. P., Stouthamer-Loeber, M., & Van Kammen, W. (1998). *Antisocial behavior and mental health problems.* Mahway, NJ: Erlbaum.

Loeber, R., Green, S. M., Kennan, K., & Lahey, B. B. (1995). Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry, 34,* 499-509.

Lezak M. D., Howieson, D. G., Loring, D. W., Hannay, J. H., & Fischer, J. S. (2004). *Neuropsychological Assessment, (Fourth Edition).* New York, NY: Oxford University Press.

Luciana, M., & Nelson, C. A. (1998). The functional emergence of prefrontally-guided working memory systems in four- to eight-year-old children. *Neuropsychologia, 36,* 273-293.

MacCallum, R. C., Widaman, K. F., Zhang, S., & Hong, S. (1999). Sample size in factor analysis. *Psychological Methods, 4,* 84-99.

March., J. S. (1997). *Multidimensional Anxiety Scale for Children.* North Tonawanda, NY: Multi Health Systems.

March, J. S., Parker, J. D. A., Sullivan, K., Stallings, P., & Conners, K. (1997). The Multidimensional Anxiety Scale for Children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*(4), 554-565.

Marsh, A. A., Finger, E. C., Mitchell, D. G. V., Reid, M. E., Sims, C., Kosson, D. S.,…Blair, R. J. R. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry, 165,* 712-720.

Marshal, M. P., & Molina, B. S. G. (2006). Antisocial behaviors moderate the deviant peer pathway to substance use in children with ADHD. *Journal of Clinical Child and Adolescent Psychology, 35,* 216-226.

Maser, J. D., & Cloninger, J. R. (1990). Comorbidity of anxiety and mood disorders: Introduction and overview. In J. D. Maser & J. R. Cloninger (Eds.), *Comorbidity of mood and anxiety disorders* (pp. 3-12). Washington, DC: American Psychiatric Press.

Maughan, B., Rowe, R., Messer, J., Goodman, R., & Meltzer, H. (2004). Conduct disorder and oppositional defiant disorder in a national sample: Developmental epidemiology. *Journal of Child and Adolescent Psychology, 45,* 609-621.

Merikangas, K. R., & Avenevoli, S. (2000). Implications of genetic epidemiology for the prevention of substance use and disorders. *Addictive Behaviors, 25,* 807-820.

Merikangas, K. R., He, J., Burstein, M., Swanson, S. A., Avenevoli, S., Cui, L.,…Swendsen, J. (2010). Lifetime prevalence of mental disorders in U.S. adolescents: Results from the National Comorbidity Survey Replication – Adolescent supplement (NCS-A). *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(10), 980-989.

Millon, T. (1993). *Millon Adolescent Clinical Inventory.* Minneapolis, MN: NCS Pearson, Inc.

Millon, T., Tringone, R., Millon, C., & Grossman, S. (2005). *Millon Pre-Adolescent Clinical Inventory.* Minneapolis, MN: NCS Pearson, Inc.

Miura, H. (2009). Differences in frontal lobe function between violent and nonviolent conduct disorder in male adolescents. *Psychiatry and Clinical Neurosciences, 63,* 161-166.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (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.

Moffit, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674-701.

Moffitt, T. E. (2003). Life-course persistent and adolescence-limited antisocial behavior: A research review and a research agenda. In B. B. Lahey, T. E., Moffitt, & A. Caspi (Eds.), *Causes of conduct disorder and juvenile delinquency* (pp. 49-75). New York: Guilford.

Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. (2001). *Sex differences in antisocial behavior.* Cambridge, UK: Cambridge University Press.

Monk, C. S., Nelson, E. E., McClure, E. B., Mogg, K., Bradley, B. P., Leibenluft, E.,…Pine, D. S. (2006). Ventrolateral prefrontal cortex activation and attentional bias in response to

angry faces in adolescent with generalized anxiety disorder. *American Journal of Psychiatry, 163,* 1091-1097.

Morris, R. G., Ahmed, S., Syed, G. M., & Toone, B. K. (1993). Neural correlates of planning ability: Frontal lobe activation during the Tower of London test. *Neuropsychologia, 31,* 1367-1378.

Muris, P., & Ollendick, T. H. (2005). The role of temperament in the etiology of child psychopathology. *Clinical Child and Family Psychology Review, 8,* 271-289.

Muthén, L. K., & Muthén, B. O. (2010). Mplus User's Guide. Sixth Edition. Los Angeles, CA: Muthén & Muthén.

Närhi, V., Lehto-Salo, P., Ahonen, T., & Marttunen, M. (2010). Neuropsychological subgroups of adolescents with conduct disorder. *Scandinavian Journal of Psychology, 51,* 278-284.

O'Bryant, S. E., & O'Jile, J. R. (2004). Attenuating demographic influences on verbal fluency and animal naming in a psychiatric sample. *Applied Neuropsychology, 11,* 208-212.

O'Donnell, J. P., MacGregor, L. A., Dabrowski, J. J., Oestreicher, J. M., & Romero, J. J. (1994). Construct Validity of Neuropsychological Tests of Conceptual and Attentional Abilities. *Journal of Clinical Psychology, 50,* 596-600.

Ollendick, T. H., Matson, J. L., & Helsel, W. J. (1985). Fears in children and adolescents: Normative data. *Behavior Research and Therapy, 23,* 465-467.

Ollendick, T. H., Seligman, L. D., & Butcher, A. T. (1999). Does anxiety mitigate the behavioral expression of severe conduct disorder in delinquent youths? *Journal of Anxiety Disorders, 13,* 565-574.

Oosterlaan, J., Scheres, Anouk, & Sergeant, J. A. (2005). Which executive functioning deficits are associated with AD/HD, ODD/CD and comorbid AD/HD+ODD/CD? *Journal of Abnormal Child Psychology, 33,* 69-85.

Pajer, K., Chung, J., Leininger, L., Wang, W., Gardner, W., & Yeates, K. (2008). Neuropsychological function in adolescent girls with conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 47,* 416-425.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys.* Eugene, OR: Castalia.

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37,* 51-87.

Posner, M. I., & Peterson, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25-42.

Purcell, R., Maruff, P., Kyrios, M., & Pantelis, C. (1998). Neuropsychological deficits in obsessive-compulsive disorder. A comparison with unipolar depression, panic disorder, and normal controls. *Archives of General Psychiatry, 55,* 415-423.

Raaijmakers, M. A., Smidts, D. P., Sergeant, J. A., Maassen, G. H., Posthumus, J. A., van Engeland, H., & Matthys, W. (2008). Executive functions in preschool children with aggressive behavior: Impairments in inhibitory control. *Journal of Abnormal Child Psychology, 36,* 1097-1107.

Rahdert. E. R. (Ed.). (1991). *The Adolescent Assessment/Referral System Manual* (DHHS Publication No. 91-1735). Washington, DC: US Department of Health and Human Services, Alcohol, Drug Abuse, and Mental Health Administration.

Raine, A. (1996). Autonomic nervous system activity and violence. In D. Stoff & R. Cairns (Eds.), *Aggression and violence: Genetic, neurobiological, and biosocial perspectives* (pp. 165-168). Mahwah, NJ: Lawrence Erlbaum Associates.

Raine, A. (2002). The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry, 43,* 417-434.

Raine, A., Venables, P., & Mendick, S. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Evidencce from the Mauritius Child Health Project. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 1457-1464.

Ravnkilde, B., Videbech, P., Rosenberg, R., Gjedde, A., & Gade, A. (2002). Putative tests of frontal lobe function: A PET-Study of brain activation during Stroop's test and verbal fluency. *Journal of Clinical and Experimental Neuropsychology, 24,* 534-547.

Raz, S., Shah, F., & Sander, C. J. (1996). Differential effects of perinatal hypoxic risk on early developmental outcome: A twin study. *Neuropsychology, 10,* 429-436.

Reitan, R. (1958). Validity of the trail making test as an indicator of organic brain damage. *Perceptual Motor Skills, 8,* 271-276.

Reynolds, C. R., & Kamphaus, R. W. (1998). *Behavior Assessment System for Children manual.* Circle Pines, MN: American Guidance Service, Inc.

Reynolds, C. R., & Richmond, B. O. (1978). Factor structure and construct validity of "What I Think and Feel". The revised Children's Manifest Anxiety Scale. *Journal of Personality Assessment, 43,* 281-283.

Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin, 128,* 490-529.

Roza, S. J., Hofstra, M. B., van der Ende, J., & Verhulst, F. C. (2003). Stable prediction of mood and anxiety disorders based on behavioral and emotional problems in childhood: A 14-year follow-up during childhood, adolescence, and young adulthood. *American Journal of Psychiatry, 160,* 2116-2121.

Rydell, A. M., Berlin, L., & Bohlin, G. (2003). Emotionality, emotion regulation, and adaptation among 5- to 8-year-old children. *Emotion, 3,* 30-47.

Séguin, 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.

Shin, M. S., Choi, H., Kim, H., Hwang, J. W., Kim, B. N., & Cho, S. C. (2008). A study of neuropsychological deficit in children with obsessive-compulsive disorder. *European Psychiatry, 23,* 512-520.

Silverman, W. K., & Carter, R. (2006). Anxiety disturbance in girls and women. In J. Worell & C. Goodheart (Eds.), *Handbook of girls' and women's psychological health* (pp. 60-68). New York: Oxford University Press.

Smitherman, T. A., Huerkamp, J. K., Miller, B. I., Houle, T. T., & O'Jile, J. R. (2007). The relation of depression and anxiety to measures of executive functioning in a mixed psychiatric sample. *Archives of Clinical Neuropsychology, 22,* 647-654.

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.

Speltz, M. G., McClellan, J., DeKlyen, M., & Jones, K. (1999). Preschool boys with oppositional defiant disorder: Clinical presentation and diagnostic change. *Journal of the American Academy of Child and Adolescent Psychiatry, 38,* 838-845.

Spreen, O., & Benton, A. (1977*). Neurosensory centre comprehensive examination for aphasia. Manual of instructions.* Victoria, B.C.: University of Victoria.

Spreen, O., & Strauss, E. (1998). *A compendium of neuropsychological tests: Administration, norms, and commentary* (2nd ed.). New York: Oxford University Press.

Strauss, E., Sherman, E., & Spreen, O. (2006). *A compendium of neuropsychological tests: Administration, norms, and commentary* (3rd ed.). New York: Oxford University Press.

Tapasak, R. C., Roodin, P. A., & Vaught, G. M. (1978). Effects of extraversion, anxiety and sex on children's verbal fluency and coding task performance. *The Journal of Psychology, 100,* 49-55.

Tanaka, J. S. (1993). Multifaceted conceptions of fit in structural equation models. In K. S. Bollen & J.S. Long (Eds.), *Testing structural equation models* (pp. 10-39). Newbury Park, CA: Sage.

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

Thomas, K. M., Drevets, W. C., Dahl, R. E., Ryan, N. D., Birmaher, B., Eccard, C. H.,…Casey, B. J. (2001). Amygdala response to fearful faces in anxious and depressed children. *Archives of General Psychiatry, 58,* 1057-1063.

Utendale, W. T., & Hastings, P. D. (2011). Developmental changes in the relations between inhibitory control and externalizing problems during early childhood. *Infant and Child Development, 20,* 181-193.

Utendale, W. T., Hubert, M., Saint-Pierre, A. B., & Hastings, P. D. (2011). Neurocognitive development and externalizing problems: the role of inhibitory control deficits from 4 to 6 years. *Aggressive Behavior, 37,* 476-488.

Unis, A. S., Cook, E. H., Vincent, J. G., Gjerde, D. K., Perry, B. D., Mason, C., & Mitchell, J. (1997). Platelet serotonin measures in adolescents with conduct disorder. *Biological Psychiatry, 42,* 553-559.

van Lier, P. A. C., van der Ende, J., Koot, H. M., & Verhulst, F. C. (2007). Which better predicts conduct problems? The relationship of trajectories of conduct problems with ODD and ADHD symptoms from childhood into adolescence. *Journal of Child Psychology and Psychiatry, 48,* 601-608.

Virkkunen, M., Nuutila, A., Goodwin, F. K., Linnoila, M., (1987), Cerebrospinal fluid monoamine metabolites in male arsonists. *Archives of General Psychiatry, 44,* 241-247.

Wakschlag, L. S., Pickett, K. E., Cook, E., Benowitz, N. L., & Leventhal, B. L. (2002). Maternal smoking during pregnancy and severe antisocial behavior in offspring. Are they causally linked? *American Journal of Public Health, 92,* 966-974.

Walker, J. L., Lahey, B. B., Russo, M. F., Frick, P. J., Christ, M. A., McBurnett, K.,…Green., S. M. (1991). Anxiety, inhibition, and conduct disorder in children: I. Relations to social impairment. *Journal of the American Academy of Child and Adolescent Psychiatry, 30,* 187-191.

Warren, S. L., Huston, L., Egeland, B., & Sroufe, L. A. (1997). Child and adolescent anxiety disorders and early attachment. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 637-644.

Warren, S. L., & Sroufe, L. A. (2004). Developmental issues. In T. H. Ollendick & J. S. March (Eds.), *Phobic and anxiety disorders in children and adolescents: A clinician's guide to effective psychosocial and pharmacological interventions* (pp. 91-115). New York: Oxford University Press.

Washburn, J., Romero, E., Welty, L., Abram, K., Teplin, L., McClelland, G., & Paskar, L. D. (2007). Development of antisocial personality disorder in detain youths: The predictive value of mental disorders. *Journal of Consulting and Counseling Psychology, 75,* 221-31.

Wechsler, D. (1999). *Wechsler Abbreviated Scale of Intelligence*. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale – Third Edition.* San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children – Third Edition.* San Antonio, TX: The Psychological Corporation.

Weems, C. F., & Costa, N. M. (2005). Developmental difference in the expression of childhood anxiety symptoms and fears. *Journal of the American Academy of Child and Adolescent Psychiatry, 44,* 656-663.

Weems, C., & Silverman, W. (2008). Anxiety Disorders. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and Adolescent Psychopathology* (pp. 447-476). Hoboken, NJ: John Wiley & Sons, Inc.

Weems, C. F., Silverman, W. K., Saavedra, L. S., Pina, A. A., & Lumpkin, P. W. (1999). The discrimination of children's phobias using the Revised Fear Survey Schedule for Children. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35,* 941-952.

Westenberg, P. M., Drewes, M. J., Siebelink, B. M., & Treffers, P. D. A. (2004). A developmental analysis of self-reported fears in late childhood through mid-adolescence: social-evaluative fears on the rise? *Journal of Child Psychology and Psychiatry, 45,* 481-496.

Westenberg, P. M., Siebelink, B. M., & Treffers, P. D. A. (2001). Psychosocial developmental theory in relation to anxiety and its disorders. In W. K. Silverman & P. D. A. Treffers (Eds.), *Anxiety disorders in children and adolescents: Research, assessment and intervention* (pp. 72-89). Cambridge, UK: Cambridge University Press.

Weston, R., Gore, P. A., Chan, F., & Catalano, D. (2008). An introduction to using structural equation models in rehabilitation psychology. *Rehabilitation Psychology, 53,* 340-356.

Willicutt, 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.

Young, S. E., Friedman, N. P., Miyake, A., Willicutt, E. G., Corley, R. P., Haberstick, B. C., & Hewitt, J. K. (2009). Behavioral disinhibition: Liability for externalizing spectrum disorders and its genetic and environmental relation to response inhibition across adolescence. *Journal of Abnormal Psychology, 118,* 117-130.

Youngwirth, S. D., Harvey, E. A., Gates, E. C., Hashim, R. L., & Friedman-Weieneth, J. L. (2007). Neuropsychological abilities of preschool-aged children who display

hyperactivity and/or oppositional-defiant behavior problems. *Child Neuropsychology, 13*, 422-443.