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DO FAMILY RISK FACTORS INFLUENCE ADHD VIA DISRUPTION OF NEUROCOGNITIVE FUNCTIONING?

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DO FAMILY RISK FACTORS INFLUENCE ADHD VIA DISRUPTION OF NEUROCOGNITIVE FUNCTIONING?

By

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ABSTRACT

DO FAMILY RISK FACTORS INFLUENCE ADHD VIA DISRUPTION OF NEUROCOGNITIVE FUNCTIONING?

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Family adversity has long been associated with maladaptive child behavior, yet few studies have examined mediators and moderators of family effects on Attention Deficit Hyperactivity Disorder (ADHD). The current study evaluated the influence of family risk factors on ADHD symptoms via mediating cognitive processes (response inhibition and reaction time variability). Participants, 394 children between the ages of 6-18 years, completed rating scales and cognitive tasks. Parents and teachers also provided ratings. The study replicated the effect of perceived interparental conflict on ADHD symptoms (Counts, Nigg, Stawicki, Rappley, & von Eye, 2005). The study validated a composite family risk construct that included perceived interparental conflict, maternal depression, and socioeconomic status. Cognitive regulation partially mediated the association between family risk and teacher-reported ADHD symptoms. The partial mediation of cognitive regulation was present in adolescents and not in children, in Caucasians and not in ethnic minorities. Further, ADHD symptoms partially mediated the relationship between family risk and adaptive functioning. Overall, the study supported the role of cognitive processes in the effect of family risk factors on ADHD, with moderated effects of developmental stage and race. Finally, family risk is associated with unique impairment related to ADHD symptoms. Clinical implications of these findings are discussed.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Do Family Risk Factors Influence ADHD via Disruption of Neurocognitive Functioning?

Attention deficit hyperactivity disorder (ADHD) is characterized by a persistent pattern of inattentive, hyperactive, and impulsive behaviors that begin in early childhood, often persist throughout development, and interfere with adaptive functioning (APA, 2000). Childhood ADHD affects between 3-7% of school-aged children in the United States (APA, 2000), and is more prevalent in males (APA, 2000; Carlson & Mann, 2000; Gaub & Carlson, 1997). Key domains of ADHD include the predominantly hyperactive/impulsive subtype (ADHD-H), predominantly inattentive subtype (ADHD-I), and combined type (ADHD-C) which includes both hyperactive/impulsive and inattentive symptoms. To meet DSM-IV criteria for ADHD, a child must evidence impairment from the symptoms, before age 7, in two or more settings (e.g. home and school) (APA, 2000). ADHD overlaps with disruptive behaviors including Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), such that approximately 30-45% of children with ADHD also have ODD and approximately 20% of children with ADHD also have CD (Acosta, Arcos-Burgos, & Muenke, 2004; Faraone & Biederman, 1998).

Whereas liability to ADHD is largely influenced by genetic factors (Faraone et al., 2005), ADHD expression is also associated with psychosocial risk (Biederman, Milberger, Faraone, Kiely, & et al., 1995a; Pressman et al., 2006; Scahill et al., 1999). To date, the latter is markedly under-investigated. ADHD is characterized by a heterogeneous behavioral profile, which may be explained by different interactions and correlations between genes and the environment. Researchers have therefore suggested that there are multiple causal pathways associated with ADHD (Nigg, Goldsmith, & Sachek, 2004).

One pathway involves the family environment, which may confer both genetic and environmental risk to children toward the development of ADHD. The potential effect of family risk on processes involved in cognitive development is of particular interest in the present investigation. Abnormalities in a range of cognitive operations have been identified in samples with ADHD. For example, deficits in executive functioning are common in groups of children with ADHD (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Sonuga-Barke, 2005). A key question is whether family risk factors operate through these problems. If family risk factors are associated with ADHD via cognitive mechanisms, another important question is whether this relationship changes at different stages of child development.

Historically, studies of ADHD examined Caucasian males (Barkley, Fischer, Smallish, & Fletcher, 2004; Loney, Kramer, & Milich, 1981; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993). Though that trend remains a common characteristic in the literature, studies have begun to incorporate cultural variation. Culture-specific effects have been observed in regard to childhood externalizing behavior problems (Deater-Deckard, Dodge, Bates, & Pettit, 1996), and associated risk factors (Lynch, 2003). However, little is known about differential cultural effects for ADHD, in particular. The present study, as a secondary aim, examined whether the relationship between family risk, cognitive regulation, and ADHD changed across racial groups (using race as a proxy for cultural variation).

A Theoretical Approach to How the Family May Influence ADHD

Developmental Psychopathology

A developmental psychopathology perspective (Rutter & Sroufe, 2000) suggests that maladaptive outcomes emanate from multiple risk and protective factors interacting over time (Sroufe, 1997). Mash and Johnston (2005) outline several pathways that connect ADHD to the family: biological predisposition (Faraone et al., 2005), shared genetic risk (Epstein et al., 2000), parenting and family environmental influences on biological/cognitive programming (O'Conner, 2003), family conflict due to ADHD (Biederman, Faraone, & Monuteaux, 2002; Lange et al., 2005; Pressman et al., 2006), family as a moderator and/or mediator of ADHD outcomes (Counts, Nigg, Stawicki, Rappley, & von Eye, 2005; Miller et al., 2006), and the family as a moderator and/or mediator of conditions that are commonly comorbid with ADHD such as conduct problems (Thapar, van den Bree, Fowler, Langley, & Whittinger, 2006).

This framework suggests that ADHD may be influenced by family factors that carry different levels of influence across children and over time (Johnston & Mash, 2001). Two points need to be considered in properly conceptualizing the family in relation to a high heritability disorder such as ADHD. First, causal processes likely involve both genetic and environmental factors via gene-environment interactions (individuals at genetic risk may be more susceptible to environmental risks) and gene-environment correlations (individuals at genetic risk may be more prone to exposure to environmental risk) (Plomin & Ruter, 1998). Although ADHD has high heritability of liability (in the range of .7 to .8 in parent ratings; Faraone et al., 2005), monozygotic twin concordance rates do not approach 100% (Kuntsi & Stevenson, 2000). In addition to direct environmental effects on behavior, this may be due to differences in the expression of genes through epigenetic variation (Rutter, Moffitt, & Caspi, 2006).

Epigenetic mechanisms such as methylation allow environmental influences to affect gene expression – not by altering the gene sequence, but by affecting the consequences of genes such that the genes are interpreted or expressed to differing degrees (Rutter, Moffitt, & Caspi, 2006). Thus, gene expression can be considered as a continuum such that an individual could exhibit behavioral variation due to environmental stress, mediated through variations in gene expression. Thus, events in the family environment could participate in epigenesis. Clarifying this possibility first requires additional mapping of the nature of family influences on ADHD. The present study examined family risk factors associated with ADHD in prior research, and clarified these relationships using a larger sample and more systematically than has been done previously.

Second, the family environment may be differentially important during critical developmental periods. For example, it may alter brain organization through a process called developmental programming, which occurs during sensitive periods of brain development wherein biological systems are thought to respond sensitively to environmental stimuli (O'Conner, 2003). It has long been recognized that experiences influence neural structures and processes that affect behavior (Hebb, 1949). These experiences include external as well as internal occurrences ranging from social events to psychological trauma, and affect short-term and long-term outcomes (Cicchetti & Curtis, 2006). A key question is whether young children evidence different behavioral effects of family risk than adolescents.

Mediating Cognitive Processes

Family experiences presumably can result in alterations in gene expression via modification of neuronal and synaptic patterns, which affect cognition and, by that route among others, behavioral change (Cicchetti & Curtis, 2006). There is growing recognition and empirical basis for the effects of parental attunement and caregiver interchange on early cognitive development. This is especially the case for language development (Appelbaum, 1977; LaParo, Justice, Skibbe, & Pianta, 2004), but it is also emerging for regulatory control (Dawson, Panagiotides, Klinger, & Spieker, 1997; Goodman & Gotlib, 1999). Thus, the argument here is that family risk factors can lead to subtle deficits in child cognition, which can in turn contribute to ADHD. However, the effects of experiences on cognitive processing are not well studied in relation to ADHD. The present study examined these effects to gain better understanding of how family context relates to cognitive effects in ADHD, which may help pinpoint neurological mechanisms that shape ADHD behaviors. We now turn to a description of cognitive processes of interest in this investigation.

Many cognitive functions have been implicated in ADHD (Barkley, 1997; Klorman et al., 1999; Tannock, Martinussen, & Frijters, 2000). Two of the most important theorized cognitive mechanisms involved in ADHD are response inhibition and response variability (Nigg, 2005). Both of these mechanisms are early emerging and develop throughout childhood, and so would be expected to be related to the development of regulatory control in the family context. Each construct will be discussed in turn.

Response Inhibition. Effortful response suppression is well associated with ADHD (Barkley, 1997; Crosbie & Schachar, 2001; Nigg, 1999). Also referred to as inhibitory control, this executive control mechanism involves restraining a planned action

based on a sudden change in the context (e.g. stopping oneself before running out into the street because a car has just appeared) (Schachar & Logan, 1990). The ability to inhibit follows developmental progressions of synaptic pruning and reinforcement of specific brain networks, namely, frontal striatal circuitry (Fuster, 2002). There is evidence for age-related change in inhibitory control across development (Booth et al., 2003; Rubia et al., 2006; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Response inhibition can be assessed experimentally in numerous ways. One very well accepted method is the stop-signal experimental paradigm (Logan, Schachar, & Tannock, 1997). Approximately 30 studies of ADHD have been conducted using this measure; based on that body of work, the main effect association of slower stop signal response with ADHD is well established (weighted effect size =.61; Willcutt et al., 2005). The present study investigated how performance on this task related to family risk as part of a potential process model involving family effects on neural and hence cognitive development.

Response Variability. The second cognitive mechanism targeted here, response variability, also has been associated with ADHD in several studies (Castellanos & Tannock, 2002; Epstein et al., 2006; Rapport, Chung, Shore, Denney, & Issacs, 2000). Greater response variability may be interpreted as corresponding to deficits in arousal (Parasuraman, Warm, & See, 1998), which are related to higher levels of inattention. The ability to maintain an alert state is linked to ascending noradrenergic networks in the brain, which are involved in arousal (Coull, 1998; Posner & Petersen, 1990). This ability develops through childhood (Akshoomoff, 2002; Booth et al., 2003; Levy, 1980), and is related to wakefulness, which is relevant to early caregiver attunement and parent-child interaction (Frolich & Lehmkuhl, 2004; Kim, Brody, & Murry, 2003). In the present

study, this construct was measured using response time variability on the Logan Stop Task to understand family effects on cognitive regulation. Response inhibition and response time variability are both key aspects of higher order regulatory operations, and were therefore aggregated in this study to create a composite cognitive regulation score.

Cognitive Functioning and Family Risk in ADHD. Despite the importance of family functioning, few studies have examined it in relation to cognitive functioning in ADHD. However, a handful of studies have examined the stop task and families – although not in the same way that the present study has. Crosbie and Schachar (2001) found that children with poor inhibition had families with higher ADHD prevalence than children with good inhibition or controls (N=80). However, they did not find group differences in psychosocial risk (which included factors such as living in overcrowded arrangements, subsidized housing, a single-parent family, or experience of parental separation before the age of 2) or neurobiological risk (including high blood pressure, seizures, nausea, perinatal bleeding, use of drugs or alcohol, or infection). Thus, the authors concluded that the increased risk of a positive family history of ADHD among the poor inhibition group was likely due to cognitive risk rather than psychosocial or neurobiological risk.

A small number of other kinds of cognitive risk have been examined in relation to families. Carlin Miller et al. (2006) examined family and cognitive risk factors for aggression in 7-11 year-old children with ADHD. A latent variable created to represent cognitive functioning included full scale IQ (FSIQ) assessed by the Wechsler Interview Schedule for Children, Revised Edition (WISC-R) or WISC, Third Edition (WISC-III), and general math and reading achievement scores measured by the Wide Range

Achievement Test, Revised Edition (WRAT-R) or the Wechsler Individual Achievement Test (WIAT). The family risk factors included demographic information such as number of siblings in the home, number of caregiving adults in the home, and history of aggression in biological parents of target child. Aggression was measured at home and school using the Children's Aggression Scale – Parent and Teacher Versions (CAS-P and CAS-T) to assess verbal, provoked physical, initiated physical, and object-directed aggression. Family risk had an effect on both home and school aggression, but cognitive risk was only related to school aggression. This suggests that the cognitive risk factors they assessed do not extend into the family domain to result in significant functional impairment observable by parents in the home setting. Alternatively, these results could suggest that school is associated with different cognitive and behavioral demands on children than home.

IQ and ADHD. In clinical samples, children with ADHD tend to have lower IQ scores than controls (Barkley, Karlsson, Pollard, & Murphy, 1985; McGee, Williams, & Feehan, 1992; Moffitt, 1990). The extent of the difference in IQ in these studies ranges from 7 to 10 points. ADHD severity and IQ were negatively associated in community (Hinshaw, Morrison, Carte, & Cornsweet, 1987; Peterson, Pine, Cohen, & Brook, 2001) and clinical samples (Sonuga-Barke, Lamparelli, Stevenson, Thompson, & Henry, 1994) with correlations ranging from r= -.25 to -.35. Thus, IQ was controlled in analyses herein to prevent a possible confound with ADHD effects.

Summary. A logical next step is to pursue family effects in relation to specific cognitive domains (i.e. response inhibition and vigilance) that are believed to be at the core of ADHD in how it develops. The present study examined mediational pathways to

better understand the relationship between family risk and ADHD symptom presentation. In this model, the cognitive component serves as an index of alterations in neural systems, which support functions such as response inhibition and vigilance. Family processes are hypothesized to influence ADHD symptoms. They are also hypothesized to influence cognitive development. In turn, the effects on cognition are hypothesized to predict ADHD symptoms. The development of self regulation, response control, and arousal are thought to depend on caregiver interchanges early in development and to mature throughout childhood (Kochanska & Aksan, 2006; Panzer & Viljoen, 2005; Tucker, Luu, & Derryberry, 2005). Thus, it is plausible that these cognitive operations mediate the relation of family distress to ADHD, potentially differently in elementary-aged children versus adolescents.

Conceptual Complexities

Genes and environments are keenly interrelated, and these relationships can be used to explore pathways to disordered behavior. Several important complications have to be considered, however. First, twin and adoption studies show that genes have an effect on measures of family environments (Plomin, 1994). Plomin and Rutter (1998) presented a conceptual model to illustrate that the relationship between genetic risk and ADHD could be mediated by psychosocial risk – an effect that would be independent of the direct relationship between genetic risk and ADHD. In this model, genetic risk was represented using a gene that has been associated with behavior. However, the correlation between genetic expression and variations in psychosocial environments that they suggested has now been described empirically (Goldsmith, Gottesman, & Lemery, 1997; Rutter, Moffitt, & Caspi, 2006). This supports the model underlying the current proposal.

However, it also introduces an important confound: environmental measures may be genetically influenced. Thus, measures of family risk factors may also carry genetic effects.

A second complication is that ADHD likely influences the family via effects on important subsystems (e.g. parent-child and marital relationships) (Mash & Johnston, 2005; Parke, 2004). For example, Gerdes and Hoza (2006) found that mothers of children with ADHD were more likely than comparison mothers to attribute their child's maladaptive behavior to unintentional and uncontrollable factors, and reported higher levels of negative affect and assertive parenting in response to the behavior. This suggests that parent-child relationships are strained when the child has ADHD. In another study, parents of children with ADHD were more distressed than parents of controls, and coping strategies were correlated with increased satisfaction (Podolski & Nigg, 2001). In other studies, disturbances in family boundaries (from a family systems perspective) as well as high levels of family adversity and discord have been associated with subsequent ADHD in children (Davies, Cummings, & Winter, 2004; Jacobvitz, Hazen, Curran, & Hitchens, 2004). However, direction of effects remains unclear. Genetic effects were not modeled, and parent ADHD, which may have contributed to these effects, does not appear to have been controlled in these studies.

Summary

In summary, it is important to note that family risk factors likely carry genetic effects, and that ADHD may have a reverse effect on family adversity. Nevertheless, the present study addressed the important need for further clarification of the effects of

family risk factors on ADHD, and the extent to which cognitive mechanisms may explain this relationship.

Specific Risk Factors at Issue

Specific Family Risk Factors Related to ADHD

Until this point family risk has been referred to generically, but it must be specified. When it comes to specific family correlates, a number of family risk factors have been associated with ADHD including adversity and conflict, in both community and clinic-referred samples of preschool and elementary school children (reviewed by Johnston & Mash, 2001). In addition, race has been identified as a possible moderator of family risk and ADHD. The major findings are briefly reviewed here.

Adversity in the Family Environment. It has long been established that adverse experiences in childhood are associated with maladaptive child behavioral outcomes (Rutter, Cox, Tupling, Berger, & Yule, 1975). However, applications to ADHD were more recent. Biederman et al. (1995b) examined family adversity as it relates to ADHD using parental psychopathology and exposure to family conflict (a variable that included six subscales of the Family Environment Scale (FES; Moos & Moos, 1974), and a severity rating based on FES scores) as indices of adversity in a sample of boys ages 6-17. Other measures included SES, birth order, family size, and intactness of the biological family. This method of deriving a family adversity variable began with Rutter et al. (1975) and is the most well recognized procedure for aggregating family risk in the field, having been used in various forms by subsequent investigators. Results of the Biederman et al. (1995b) study showed more adversity in the ADHD than control group. They also found that family conflict was a stronger predictor of ADHD and poorer

functioning than parental psychopathology. In a different sample, but using a similar adversity construct, Counts, Nigg, Stawicki, Rappley, and von Eye (2005) found that more adversity risk factors were associated with one ADHD subtype (ADHD-C) than another (ADHD-I) or controls. Again, perceived interparental conflict was the best predictor of increased inattention and hyperactivity after controlling for other risk factors.

Conversely, among a clinical sample of adolescents, Rey, Walter, Plapp, and Denshire (2000) found that poorer family environments (i.e. less emotional care, insecure attachment relationships, and punitive, inconsistent, and inappropriate limit setting) were related to ODD, CD, and increased functional impairment, but not associated with ADHD. Of most interest here is whether family adversity contributes to ADHD independent of these common comorbid conditions. Rey and colleagues concluded that the quality of the family environment was not related to a diagnosis of ADHD, but with CD. ADHD precedes CD along the developmental continuum (but may also interact with adversity to cause CD), thus their study of adolescents could indicate a differential effect of family environment at different stages of development. The present study examined whether family risk predicted ADHD beyond the effects of ODD and CD, and tested whether the influence of family environment on ADHD varied by age cohort (i.e., adolescents may have protective factors that younger children do not). That is, age would act as a moderator for the effects of family risk on ADHD.

Family Conflict. Pressman et al. (2006) found more impairment in children from families high in conflict and low in achievement and organization. Their study included 220 families consisting of two biological parents and two biological children between the ages of 5-18 who had been diagnosed with ADHD (ADHD-affected sibling pairs).

Family conflict accounted for about 40% of similarity in impairment across siblings, and this shared variance was not directly related to parent psychiatric diagnosis (Pressman et al., 2006). Impairment was determined using three indicators: a clinician's rating of the Children's Global Assessment Scale (C-GAS) of the K-SADS-PL, and t-scores from the internalizing and externalizing scales of the Achenbach CBCL. Family environment was measured using 6 subscales of the FES scale completed by both parents (cohesion, expressiveness, conflict, organization, control, and achievement/orientation). Additionally, family conflict was the mechanism that accounted for the relationship between parent psychiatric diagnosis and the sibling impairment score.

Pressman et al. (2006) also found apparent birth order or developmental effects such that the oldest sibling appeared to be vulnerable to a wider set of family environmental risk factors than younger siblings. Though family conflict predicted impairment in both siblings, low levels of family cohesion were associated with impairment only in the oldest child. This could be a birth order effect or an age effect; similar levels of impairment may yield different consequences in children across development.

Another form of family conflict, interparental conflict, is also characteristic of families of children with ADHD. Parents of children with ADHD have reported more marital conflict and less marital satisfaction than parents of children without ADHD (Murphy & Barkley, 1996). Harvey (2000) found that disagreement about childrearing among parents is related to more problematic child behavior (perhaps through a feedback loop as described by Patterson's (1982) model), and more marital conflict. Counts et al. (2005) found that child perception of interparental conflict carried most of the variance in

a family adversity model predicting inattention and hyperactivity that included SES, parental psychopathology, and stressful events.

Family conflict is likely associated with subsequent decrements in parenting skill and effectiveness that may play a role in the maintenance of ADHD. Maternal distress mediated the relationship between family conflict and child behavior problems (Kendall, Leo, Perrin, & Hatton, 2005). Satake, Yamashita, & Yoshida (2004) found that parents of children with ADHD and comorbid ODD/CD were more likely than parents of controls to have severe childhood ADHD symptoms and poorer adult mental health. Harel & Brown (2003) found that children referred for ADHD evaluations and those who were medicated for ADHD were more likely than controls to have parents that did not complete college, and have one stepparent. These findings suggest that parental distress is related to ADHD in offspring. Overall, it remains unclear how the interplay between these aspects of the family environment contribute to ADHD in children; effects on cognitive regulation are one potential pathway.

Several family risk variables appear to be related to ADHD, but interparental or family conflict may be among the most important. The next point to consider is whether these effects are driven by parent dysfunction.

Parent Risk Factors

Parent ADHD. ADHD is familial (Faraone & Biederman, 1994a, 1994b). This suggests that parents who themselves have ADHD may be implicated in the maintenance and/or exacerbation of ADHD symptomology in their children. That is, parent history of ADHD can be used as a proxy for genetic risk in their offspring (Crosbie & Schachar, 2001; Milberger, Biederman, Faraone, Guite, & Tsuang, 1997; Tsuang, Faraone, &

Lyons, 1993); although transmission may also occur via psychosocial mechanisms. Maternal ADHD limits signs of improvement shown by their preschool children following a parent training intervention (Sonuga Barke, Daley, & Thompson, 2002). Patterson et al. (2006) found that mother's ADHD status was more strongly related to child impairment in younger children, which suggests a developmental need for reliance on mothers for organization and supervision. Further, children with a parent with ADHD had a higher prevalence of mood, anxiety, oppositional, and conduct disorders (Minde et al., 2003).

Biederman, Faraone, & Monuteaux (2002) combined two case-control studies of nuclear families. They found that exposure to parental ADHD was linked to a disruptive family environment (i.e. increased levels of family conflict and decreased family cohesion), but did not independently increase ADHD risk in children beyond the genetic effects. Thus, this finding showed that exposure to parental ADHD did not increase the risk for ADHD in children beyond the level of genetic risk involved in parental ADHD (determined using the SCID for current symptoms and the KSADS-E for past symptoms). Nonetheless, parental ADHD was related to a disruptive family environment (operationalized using three FES dimensions: cohesion, expressiveness, and conflict). Overall, it remains unclear whether ADHD in parents confers genetic as well as psychosocial risk to their children. The mechanisms that underlie these processes remain to be revealed.

Maternal Distress/Depression. Negative emotion in mothers during pregnancy may be a risk factor for ADHD in children (Lee, Chang, & Lung, 2006). Also, mothers of children with ADHD have been found to show higher rates of depression than mothers of

controls (Cunningham, Benness, & Siegel, 1988). West, Houghton, Douglas, Wall, & Whiting (1999) showed that this may be specific to mothers with more than one child with ADHD or to mothers of children with ADHD-C. This research suggests that the more severe the behavior (i.e. multiple children with or a child with ADHD combined type which includes both inattentive and hyperactive/impulsive symptoms), the more likely the association with maternal distress and depression. Chi and Hinshaw (2002) suggested that mothers' perceptions of their parenting behaviors may be related to emotional distress and depressive symptoms rather than their actual behaviors as parents. Such distortions may compromise the parent-child relationship, which could introduce risk into the family environment that could in turn affect adaptive functioning in children as well as ADHD symptomology.

Kendall, Leo, Perrin, & Hatton (2005) modeled family relationships of children with ADHD and found that maternal distress may mediate the relationship between family conflict and child behavior problems. This study also showed that the age of the child with ADHD was associated with increased family conflict, an effect that was not driven by severity of child behavior problems. This finding contradicts age effects found in other research described earlier. However, it was supported by findings of Lewis-Abney (1993) who found that age was negatively correlated with family functioning in children with ADHD. This suggests that the longstanding collective negative effects of child behavior resulted in an increase in family conflict. These discrepancies in the literature suggest that age effects remain to be clarified.

Culture as a Moderator of Family Risk and ADHD

The relevant environment may extend beyond the family to include cultural influences. These influences represent key developmental contexts outside the child, such as ecological elements of culture, as well as identity characteristics of the child and family, and should be considered in analyses of environmental risk and protection (Bussing, Schoenberg, & Perwien, 1998; Milich, Balentine, & Lynam, 2001). As indicated, childhood studies of ADHD often involve predominately Caucasian male samples (Barkley, Fischer, Smallish, & Fletcher, 2004; Loney, Kramer, & Milich, 1981; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993); however, knowledge of ADHD derived on these children may not capture the disorder in children of color. There are examples of race and culture moderating family influences on child psychopathology (Angold et al., 2002; Deater-Deckard, Dodge, Bates, & Pettit, 1996; Lynch, 2003), however such effects are not well studied in ADHD, and less so with respect to cognitive influences.

As noted, the literature on ethnic differences in ADHD, as well as on how ADHD presents in different cultural contexts, is in its early development. Findings from the small literature on symptom severity suggest that ADHD in African Americans is associated with more symptoms (Arnold et al., 2003) and more comorbid pathology (Samuel et al., 1998). However, more investigation is needed to determine whether this suggests differential presentation of ADHD across race, or the presence of more complex moderating variables. Additionally, Bussing, Schoenberg, and Perwien (1998) reported that African American parents were less knowledgeable and had limited access to information about ADHD compared to Caucasian parents. In another study, conducted in Venezuela, Montiel-Nava, Montiel-Barbero, and Peña (2005) found significant

differences in cohesion, recreational activity orientation, and intellectual activity orientation between ADHD and controls. They also found increased ADHD symptom severity associated with greater levels of family conflict and lower levels of cohesion as measured by the FES. These studies suggest that an increased understanding of cultural effects on ADHD will help uncover potential differences in behavioral tolerance and interpretation, hence culture as a potential moderator of family risk.

Race, ethnicity, and culture are terms that are often used interchangeably in the literature as grouping variables based on assumed shared psychosocial characteristics that may influence behavior of individuals within these groups (Betancourt & Lopez, 1993; Okazaki & Sue, 1995). One of the most frequent critiques of race research in mainstream literature is that race is often measured as a demographic variable in and of itself, but that more measures should be used to determine the extent of the cultural contribution (Tyson, 2004). The inherent risk in focusing on such grouping variables is that diversity among individuals within the groups is often unrecognized (Tyson, 2004). Unfortunately, much of the literature on race and ADHD relies solely on demographic variables to measure racial/ethnic influences. As a possible solution to this problem, the present study incorporated measures of ethnic identity (Phinney, 1992). The Multigroup Ethnic Identity Measure (MEIM) measures one's sense of belonging, commitment, and active investment in their ethnic group. This construct may help assess a process responsible for cultural variation. Ethnic identity was covaried in race analyses, rather than utilizing the race and ethnic identity constructs as unique contributors, to more fittingly represent culture.

Overall, more clarity is needed to determine the influence of cultural constructs such as ethnic identity, which may moderate family environmental risk effects on ADHD.

An understanding of which underlying mechanisms account for observed cultural differences in ADHD may inform more appropriate diagnostic and risk and prevention practices that may better serve children from diverse backgrounds.

Global Adaptive Functioning

Impairment is used to describe deficits in functioning that include an inability to carry out appropriate tasks or interactions by oneself or with friends and family in home, school, and work domains. DSM-IV criterion D requires that children evidence impairment in functioning in at least two domains (i.e. home and at school or other places) in order to meet eligibility for an ADHD diagnosis (APA, 2000). Diagnostically, this construct is very important as it conveys whether the condition limits one's ability to function appropriately. However, its operational definition has been elusive, as it necessarily involves some degree of social value judgment (i.e. what is impairing to one individual may not be considered impairing to another), and some amount of overlap with ADHD symptoms themselves. Gordon et al. (2006) reported that the size of the ADHD group when classified by symptom count decreased by 77% when measures of impairment (based on criterion D) were required to be included in defining the group versus when they were not.

ADHD has been associated with high levels of impairment related to conduct, emotion, and peer interactions (Gaub & Carlson, 1997; Gershon, 2002; Strine et al., 2006), and difficulties related to impulsivity, hyperactivity, and inattention often result in difficulty at school that may result in low grades and disciplinary measures for affected children. There is also evidence that impairment can continue across domains for years after the initial problems arise (Hinshaw, Owens, Sami, & Fargeon, 2006).

For present purposes, the key issue is that it remains unclear which mechanisms are involved in the relationships between family risk (specifically developmental risk), ADHD, and adaptive impairment. Put another way, it remains to be determined whether adversity is a non-specific index of impairment in children, or whether it has a specific effect on ADHD symptoms. Ultimately, if family risk variables such as those described herein are linked to inattention, hyperactivity, and impulsivity, and these conditions affect adaptive functioning, then ADHD (a highly heritable condition) may mediate the relationship between family risk and functional impairment. On the other hand, family effects related to ADHD could be explained by overall deficits in family functioning, or by comorbid conditions.

Possible Confounds

Health Risks. Several risk factors associated with ADHD in children, in particular low birth weight, maternal alcohol use, and maternal tobacco use during pregnancy, are also associated with high levels of family adversity. If they are not considered, spurious conclusions may result. Key confounds are therefore considered in this section.

With regard to low birth weight, in a longitudinal study by McGrath et al. (2005), 188 infants (39 full-term and 149 pre-term) were tested for medical and neurological status at age 18 months and 30 months. Behavior at age 4 was rated by parents and trained evaluators on inattention and activity level. A number of significant associations were found including low birth weight and gestational age, which predicted hyperactivity and inattention at age 4. Low birth weight was associated with increased risk for ADHD (among other conditions) in a study of a sizeable sample of children ages 0-12 years (N=7817) (Stein, Siegel, & Bauman, 2006).

With regard to alcohol use and smoking during pregnancy, in a study that examined children of monozygotic and dizygotic female twins at risk for alcohol use disorder, Knopik et al. (2006) found evidence for a genetic relationship of increased risk for ADHD. Children of twins with a history of alcohol use disorder and children of mothers with no history of alcohol use disorder but with a co-twin with an alcohol use disorder were more likely to show ADHD than children of controls. This study also found significant effects for prenatal smoking; the alcohol use disorder results remained significant when statistical adjustment was made for prenatal smoking. Thus, it appeared that genetic risk for alcohol use disorder may also influence risk for ADHD.

Prenatal smoking appears to be a well-studied risk factor for ADHD (Button, Thapar, & McGuffin, 2005; Rodriguez & Bohlin, 2005), and its effects appear to be related to influences during early brain development (Ernst, Moolchan, & Robinson, 2001). In a study of response to preventive intervention, children exposed to prenatal cigarette smoking by mothers displayed higher levels of ADHD symptoms and did not respond to the intervention – they were even more likely to experiment with smoking themselves at an early age (Vuijk, van Lier, Huizink, Verhulst, & Crijnen, 2006). Further, inattentive symptoms were higher in children whose mothers smoked over 10 cigarettes per day versus controls (after adjusting for confounds including maternal ADHD, birth weight, and prenatal alcohol use) (Schmitz et al., 2006). Linnet et al. (2003) reviewed the literature on the risk of prenatal exposure to psychosocial stress, alcohol, nicotine, and caffeine on ADHD behavior to that time. They concluded that prenatal cigarette exposure conferred the clearest risk, whereas results of studies of other risk factors were

contradictory and inconclusive. However, studies on psychological stress during pregnancy tended to show a small relationship to ADHD in the child.

Overall, prenatal and perinatal factors appear to be associated with increased risk for ADHD. Yet these are likely to co-occur with family adversity, taking place in lower income, less organized families with more parent psychopathology. Therefore, it is appropriate to attempt to account for these effects when investigating pathways between family risk and ADHD.

Hypotheses

The study examined the extent to which family risk factors influence ADHD via disruption of neurocognitive functioning. Preliminary hypotheses posited that 1a) selected family measures would statistically predict ADHD symptoms independent of parent ADHD, one another, ODD, and CD; 1b) the same family measures would predict inattentive and hyperactive symptoms. Next, it was hypothesized that 2a) the family measures could be consolidated to form a latent family risk construct; 2b) ADHD would be significantly related to the latent family risk construct; 2c) family risk would predict ADHD when items were used to create latent factors for ADHD symptom domains. With the preliminary analyses completed, the focus shifted to the primary hypotheses: 3a) cognitive regulation would partially mediate the relationship between family risk and ADHD; and child age (3b) and race (3c) would moderate the partial mediation described in 3a. Finally, it was hypothesized that 4) ADHD would partially mediate the relationship between family risk and adaptive functioning.

METHOD

Participants

The sample included 394 girls and boys between the ages of 6-18 years old – 53% of whom met DSM-IV criteria for ADHD[1] and 47% of whom were typically developing comparison children. Data were collected using the Michigan State University Attention Study protocol for children and adolescents. Participants were recruited widely from the local community via advertisements placed in the community newspaper, on local radio broadcasts, and during previews at a local movie theater, and through letters and pamphlets mailed to parents of children in several of the local school districts. Volunteers were evaluated for study eligibility through a standard multistage screening process, as follows.

Sample and Recruitment

Procedure overview. First, prospective participants contacted the project office for an explanation of the study, and a brief phone screen to check for rule-outs (age 6-18, no sensory-motor handicap, no neurological illness, medication rule outs, and native English-speaking). Second, eligible participants were scheduled for a 3 hour diagnostic visit wherein the visiting parent gave written informed consent (in compliance with the MSU Internal Review Board) and completed semi-structured clinical interviews and normative rating scales to assess ADHD and other Axis I disorders in the child. During this visit, the child gave a written assent to participate in the study and completed

[1] A diagnostic team of certified mental health professionals reviewed information from the semi-structured interview and parent, teacher, and youth-report rating scales to arrive at a categorical "best estimate" diagnosis of ADHD present or absent (Faraone, 2000). Inter-clinician agreement was adequate (from k = .864 to .967). This categorical diagnosis was used in Table 1a to describe the sample; however continuous Conners' Teacher Rating Scale scores were used to address study hypotheses.

academic and IQ screening in addition to normative rating scales and a brief clinical interview to assess ADHD and other Axis I disorders.

After the screening visit, some families were screened out and paid for their time whereas other families were asked to return to the laboratory to complete another 3 hour visit during which the cognitive measures were obtained. During this visit the parent completed rating scales regarding behavior, mood, personality, and relationships regarding their child, family, and self. The child completed a series of cognitive tasks and rating scales regarding their perceptions of their parents and families as well as their own habits and preferences. Thus, by design cognitive and family measures were available on all 394 families in the sample selected for study.

The second parent, when available, completed 30-45 minutes of questionnaires and returned them to the laboratory by mail. The child's current teachers (main teacher for elementary aged children; teacher selected by family for adolescents) received a 30-minute packet of questionnaires regarding the child's behavior at school.

Diagnostic Measures Pertaining to the Child

Diagnostic Dimensions of ADHD in the child. To obtain normative, standardized dimensional ratings of attention problems as well as other current symptoms, teachers completed the Conners' et al. Teacher Rating Scale – Revised: Short (CTRS; Conners, Sitarenios, Parker, & Epstein, 1998). This 28-item questionnaire involved rating the child's behavior in the classroom and in school on a 4-point Likert scale (0 = not true at all; 3 = very much true). Two of the subscales were of interest: hyperactivity (α = .94) and cognitive problems (α = .85; used in this study as a proxy for inattention). High scores on the hyperactivity scale indicated trouble sitting still or remaining at the same

task for very long, more restlessness and impulsivity than most individuals their age, and a continuous need to be "on the go" (Conners et al., 1998). High scores on the cognitive problems (or inattention) scale indicated slower learning than most individuals their age, difficulty organizing schoolwork, trouble finishing tasks or schoolwork, and problems concentrating on tasks that required constant mental effort. The subscales displayed appropriate factorial validity (N=1,897, all loadings ranged between .436 and .657, $p<.05$) with adequate fit (GFI=.91, AGFI=.88, RMS=.062) and assessed relatively distinct dimensions (Conners et al., 1998). Test-retest reliability coefficients ranged from .47-.86, ($p<.05$) indicating stability of the subscales (Conners et al., 1998).

Assessment of other Axis I disorders in the child. The Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS-E; Puig-Antich & Ryan, 1986) for DSM-IV was administered by a trained master's level clinician after extensive training in the interview and checkout of taped interviews for validity reviewed by our staff social worker. Diagnoses examined in the current study included ODD, CD, major depressive disorder, dysthymic disorder, bipolar disorder, substance abuse and dependence, psychotic disorders, obsessive-compulsive disorder, panic disorder, agoraphobia, simple phobia, social phobia, elimination disorders, sleep disorders, and eating disorders. Autistic disorder was screened by added symptom questions and was a rule out as noted. The K-SADS has exhibited acceptable validity and reliability (Ambrosini, 2000). To assess reliability of the interview procedures in our sample, 20 K-SADS interviews were videotaped and coded by two qualified interviewers. Inter-interviewer reliability for Axis I disorders was acceptable ($k = .798$).

Assessment of Parent Psychopathology

Parent ADHD. Parent ADHD was a key control variable — it could represent genetic risk and/or could be a driver of family adversity. Therefore, its control was considered crucial to understanding family findings. To assess parent ADHD, both parents were asked to complete self-report ratings of ADHD symptoms in childhood (ages 5-12, which precedes child ADHD in time sequence) and currently (past 6 months). These included the Conners' Adult ADHD Rating Scale: Long Version (1999), and ADHD Rating Scale (Barkley & Murphy, 1998), which have acceptable reliability and validity. The primary parent (usually the mother) was also asked to rate their child's other biological parent using the Barkley & Murphy ADHD Rating Scale. I combined this information via a composite ADHD score algorithm that involved (1) converting the scales into a uniform rubric (z-scores), and (2) taking the self score for each symptom whenever both ratings were available. Higher symptom ratings indicated more problems associated with attention, hyperactivity, and/or impulsivity. Adults with a history of ADHD in childhood may under-endorse childhood symptoms (Barkley, Fischer, Smallish, & Fletcher, 2002), thus parent's current ADHD symptoms were considered in analyses. Reliability was acceptable for the parent 1 (α = .85) and parent 2 (α = .76) composite scores in this sample.

Parent Depression. Maternal depression was one of the variables used in the Rutter index of family adversity. To assess it, the visiting parent (usually the mother) completed the 13-item depression subscale of the Symptom Checklist-90-R (SCL-90-R; Derogatis, 1994). The SCL-90-R is a 90-item measure of psychological distress during the past week, which is rated on a 5-point Likert scale (0 = not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit, 4 = extremely). Thus, higher ratings indicated more

depressive symptomology. Some items include: feeling low in energy, crying easily, feeling lonely, feeling blue, and feeling no interest in things. This scale was been found to be statistically reliable in this sample ($\alpha = .94$).

Child Cognitive Regulation

Cognitive measures were selected so as to capture two core functions thought to be involved in ADHD: response inhibition and response variability. Full scale IQ was be covaried.

Logan Stop Task. (Logan, 1994). The stop task is a computerized choice-reaction time task that measures executive response inhibition. During the task, the participant was instructed to press the "X" or the "O" key as quickly as they can when an "X" or "O" appears on the computer screen. On 25% of trials, a warning tone sounds, indicating they should interrupt their response. The timing of the tone was varied in a stochastic tracking procedure to maintain a successful inhibition rate of 50%. The amount of warning needed (stop signal reaction time (SSRT)) was then computed by subtracting average stop signal latency (warning time) from average go response time (Logan, 1994). Data cleaning procedures were identical those described by Nigg (1999). The response time (RT) variability score is the standard deviation of go reaction time, and an index of arousal such that greater variability indicates deficits in arousal (Parasuraman, Warm, & See, 1998). Larger values for SSRT and RT variability indicate deficits in regulatory control. Thus, a cognitive regulation composite variable was generated using the mean of the SSRT and RT variability z-scores ($r = .35, p < .001, \alpha = .89$).

Full scale IQ. Child participants completed a 3-subtest short form of the WISC-IV (Wechsler, 2003) or 5-subtest short-form of the WISC-III (Wechsler, 1991). The full-

scale IQ (FSIQ) score was estimated based on established norms for the respective age groups, with WISC-IV reliability = .97 and validity = .87 (Sattler & Dumont, 2004) and WISC-III reliability = .96 and validity = .89 (Sattler, 2001). For 17 year-olds, a 5 or 3-subtest short-form of the WAIS-III (Wechsler, 1997) was used, and FSIQ was estimated using norms established for this age group, with reliability >.93 and validity = .88 (Sattler, 2001).

Remaining Measures Related to Family Adversity

Demographics. The visiting parent was asked to provide information about each parent's education level, occupation, employment status, and yearly household income on a family background questionnaire. Household income was used in analyses as a proxy for SES in the family risk model. Parents also indicated their ethnicity and the ethnicity of the participating child(ren). In the event that there was disagreement among reporters of ethnicity, the primary parent's report was used in analyses of ethnic moderation of family effects described later.

Family Environment Scale (FES; Moos & Moos, 1974). The visiting parent (usually but not always the mother) completed the FES. It is a 90-item questionnaire comprised of true/false questions related to the participant's general perceptions of their family. Moos, Insel, & Humphrey (1974) used a sample of 814 family members in 240 families and found three reliable and valid dimensions that encompass 10 subscales: Relationship (Cohesion, Expressiveness, Conflict), Personal Growth (Independence, Achievement Orientation, Intellectual-Cultural Orientation, Active-Recreational Orientation, and Moral-Religious Emphasis), and System Maintenance (Organization and

Control). Internal consistencies ranged between $r = .64$ to $r = .78$, which is acceptable. Test-retest reliability was also acceptable and ranged between .68 and .86.

FES Data Reduction. The FES and several additional family factors should create a latent family risk variable for use in study analyses. In order to reduce the large number of FES subscales into a smaller and more manageable number of factors, an Exploratory Factor Analysis (EFA) was conducted. Analyses were conducted in M*plus* using Full Information Maximum Likelihood (FIML). SPSS produced similar results (with raw data) to those generated using FIML. This supports the utility of the FIML method in this dataset, as the imputation process did not significantly alter the results. M*plus* was used to test the fit of the 10 FES scales to 1, 2, 3, and 4 factor models (Appendix B). The fit indices suggested an unacceptable fit for 1 and 2 factor models. A 3-factor model would not converge. A 4-factor model suggested good fit, however 2 of the 4 factors contained only 1 subscale. When the 4-factor model was examined in a Confirmatory Factor Analysis (CFA), it was under-identified and standard errors could not be computed. Together, these findings suggest that a 2-factor model would be the most promising.

An examination of the factor loadings in the 2-factor EFA analysis revealed that several subscales loaded similarly on both factors. The subscale with the smallest difference between factor scores (Achievement) was removed from the model and it was re-run to determine whether the fit improved. An improvement was observed, but not enough to support an acceptable fit. The Conflict, Cohesion, and Active-Recreational subscales were systematically removed from the 2-factor model (in the manner described above), until the fit indices suggested that the model fit the data well (Muthén & Muthén, 1998-2007). The promax loadings were interpreted because the factors were correlated

(Muthén & Muthén, 1998-2007). Appendix B also shows a CFA of the 6-subscale 2-factor solution with overall good fit (χ^2 (5, N = 374) = 10.84, p > .05, CFI = .99, RMSEA = .06, RMR = .03.). The correlation between the two factors was also significant (r = .55, p < .05), as were the variance components (p < .001), suggesting that each factor accounts for an amount of variance that is significantly different from zero. It is also notable that a 1-factor model of the 6 subscales did not result in good fit, and a 3-factor model had worse fit and accounted for less variance when compared to the 2-factor model.

The two theoretically meaningful and statistically reliable factors were labeled Values and Expressiveness. The Values factor was comprised by the Control, Organizational, Independence, and Moral-Religious subscales, which indicate the extent to which the family has set rules and procedures, clear organization and structure, independence, and ethical and religious values. The Expressiveness factor includes the Intellectual-Cultural and Expressiveness subscales, indicating the extent to which the family is interested in political, social, cultural, and intellectual activities, and encourages open actions and expression of feelings.

Children's Perception of Interparental Conflict Scale (CPIC; Grych, Seid, & Fincham, 1992). The CPIC is a reliable and valid 48-item questionnaire completed by the target child assessing their appraisals of and reactions to parental conflict. The items were rated T = true, ST = sort of true, or F = false. Grych et al. initially proposed that there be 9 subscales that assess frequency, intensity, resolution, content, perceived threat, coping efficacy, self-blame, triangulation, and stability related to parental conflict. However, Nigg et al. (under review) conducted the first empirical study of the 48 items and

completed a cross-replicated EFA-CFA design superior to analyses used in the Grych et al. study. The Nigg et al. study included the present study's sample, and therefore provided an appropriate solution for use with the present sample. The resulting 4 factors, shown in Appendix C, were used in analyses herein: Conflict Quality (5 items, including "My parents hardly ever argue", "When my parents have an argument they yell a lot"), Marital Unhappiness (7 items, including "My parents have arguments because they are not happy together", "My parents have pushed or shoved each other during an argument"), Self Blame (9 items, including "My parents' arguments are usually about something I did", "My parents blame me when they have arguments"), and Perceived Threat (5 items, including "I get scared when my parents argue", "When my parents argue I worry that they might get divorced"). The scale was reliable in the current dataset (α = .89).

Multigroup Ethnic Identity Measure (MEIM; Phinney, 1992). This 12-item scale measures ethnic identity search (a developmental and cognitive component) as well as affirmation, belonging, and commitment (an affective component). Examples of ethnic identity search items include: "I think a lot about how my life will be affected by my ethnic group membership", "I am active in organizations or social groups that include mostly members of my own ethnic group." Examples of affirmation, belonging, and commitment items include: "I understand pretty well what my ethnic group membership means to me", "I have a lot of pride in my ethnic group." Items were rated on a 4-point Likert scale (1 = strongly disagree, 2 = disagree, 3 = agree, 4 = strongly agree). The measure was reliable in the current dataset (α = .88). A total score was used in analyses.

Developmental History. To assess perinatal and health risk factors that could account for apparent family adversity effects, the visiting parent (usually the mother) completed a developmental history of the child across three domains: (1) pregnancy history, (2) birth, and (3) growth and development. The pregnancy history section included 9 items (e.g. mother use of tobacco, alcohol, mother illness or pregnancy complications, medications during pregnancy, severe emotional distress during pregnancy, and exposure to x-ray shortly before or during pregnancy). The birth measure was comprised of 6 items (e.g. timing of the birth, child's birth weight, type and normalcy of delivery, baby medical difficulty at birth, and whether breast fed). Finally, the growth and development section included 12 questions that referred to significant developmental milestones (e.g. difficulty during baby's first month at home, age at first step, first single word, bed wetting, etc.) and health information (e.g. medications, psychotherapy history, serious injury, allergies, medical illness). Information for the three scales was aggregated by assigning a point for each endorsed risk incident, resulting in a count measure of developmental wellbeing in each of the three domains (prenatal risk, perinatal risk, and early delay). The yes/no coding revealed very low endorsement rates, which limited the ability of the items to correlate, and reliability of the three individual scales was not adequate ($\alpha < .51$). Therefore, the mean of the standardized (z) scores was taken in order to create a reliable and meaningful composite total risk score. To ensure that the composite was sound, the inter-item correlations and scale internal reliability were examined. The 27 items were entered into SPSS to test reliability, and items were systematically removed based on highest alpha if item deleted score until the scale demonstrated acceptable reliability. Six items were retained ($\alpha = .700$): low birth weight

(below 2500g), tobacco use during pregnancy, use of alcohol during first trimester, second trimester, and third trimester, and number of drinks consumed per week during pregnancy.

Adaptive Functioning. Adaptive functioning was assessed by two measures: clinician rated Global Assessment of Functioning (GAF; APA 2000), and parent ratings of Total Competency on the Child Behavior Checklist (CBCL; Achenbach, 1991). The interviewing clinician assigned GAF scores at the end of the structured clinical interviews. This score of overall functional adjustment was an index of impairment from 0 to 100 with high scores indicating better functioning and low scores indicating more impairment. Inter-rater reliability for GAF scores (ICC $r = .714$) was determined using the same coding procedure used for K-SADS interviews (20 interviews were videotaped and coded by two qualified interviewers). The CBCL Competence Scale is a parent rating that measures the child's participation and efficacy in activities in social, school, and home domains. It is scored using T-scores ranging from 0 to 100 with high scores indicating better functioning and low scores indicating clinical impairment. To create a single score, the GAF and CBCL scores ($r = .478, p < .001$) were standardized and averaged into a single adaptive functioning composite ($\alpha = .70$).

Data Analysis

Missing data that occurred at random due to changes in the protocol, administrative or computer error, child refusal or invalid response on some measures were imputed using full information maximum likelihood (FIML) in *Mplus* using the Type = complex command (Muthén & Muthén, 1998-2007). This imputation procedure

follows the guidelines in McCartney, Burchinal, and Bub (2006), and is considered superior to listwise deletion.

Data collected for this study are nested to account for non-independence of observations, as they include data from siblings within the same family. M*plus* was used to cluster the data in order to correct for the nestedness in all analyses (unless otherwise indicated).

A priori decision criteria for factor analyses, and structural equation models are outlined in this section. Hu and Bentler (1999) suggest that Root Mean Square Error of Approximation (RMSEA) values below .06 and Root Mean Square Residual (RMR) values below .08 indicate satisfactory model fit, with lower values indicating better model fit. Chi-square values are indicative of best model fit when they are closest to zero with no degrees of freedom, and the model fit worsens as the chi-square value increases (Kline, 2005). Further, a normed chi-square value can be obtained to reduce its sensitivity to sample size, such that $\chi^2/df < 2$ can indicate acceptable fit (Kline, 2005). M*plus* Version 4 outputs provide unstandardized coefficients (Est.), standard errors (S.E.), estimates divided by their standard errors (Est./S.E.), and standardized coefficients (StdYX) based on latent and observed variables' variances (representing the amount of change in Y per standard deviation unit X). The Est./S.E. value is a Z statistic, and tests the null hypothesis that the population parameter estimate is zero. The Z values are significant at the $p < .05$ level when they exceed ± 1.96. The StdYX values are the regression weights shown in the figures herein. The regressions captured between as well as within group variation.

RESULTS

Sample Description

Demographic information for the sample is shown in Tables 1a and 1b. ADHD diagnoses were established using the Best Estimate Diagnosis, and these assignments are further validated by ADHD symptom rating scales that show significant elevations in the ADHD sample. Table 1a shows a greater proportion of male participants with ADHD; this is common in ADHD studies and may reflect the male preponderance of ADHD in the population (APA, 2000; Gaub & Carlson, 1997). Each hypothesis that involved ADHD was checked for confounding effects of gender, and none were found. In addition, Table 1a shows a greater proportion of children under age 13 in the ADHD group. Age was covaried in analyses that involved ADHD, and results were not significantly affected. The percentage of minority participants was similar across groups. The full sample was 74% Caucasian, 12% African American, 5% Latino, 2% other, and 7% multiracial, representative of the ethnic makeup of the local county (US Census Bureau, 2000). Consistent with other studies of ADHD, participants with ADHD were more likely to have symptoms of ODD, CD, greater global impairment (GAF), and ADHD and depressive symptoms in the primary parent than the non-ADHD comparison group.

Annual household income was significantly lower in the ADHD group (M = 62k, SD = 34k) than in the non-ADHD comparison group (M = 71k, SD = 35k; $p < .05$). This is consistent with research that showed an increased prevalence of ADHD among individuals with lower socioeconomic status (Barkley, 1998). Table 1b shows that the average income for the full sample was 66k (SD = 35k), which reveals socioeconomic variability in the sample with a relatively normally distributed range from approximately

6k to 180k US dollars annually. Group comparisons revealed that more households in the child (under age 13) and ethnic minority groups were below the poverty level compared to households of target children in the adolescent and Caucasian groups. Percentages closely mirrored the poverty rates in the local county (14.6%; US Census Bureau, 2000). No significant differences were found in regards to parent's education level among the child and adolescent groups; however, parents in the Caucasian group attained higher levels of education than those in the ethnic minority group. Similarly, no significant age group differences were found related to the child's living situation; however, children in the Caucasian group were significantly more likely to live with both parents than children in the ethnic minority group.

Power Analyses

For multiple regression analyses a priori power analyses required a sample of at least N=172 to reliably detect a medium effect size (f^2 = .15) with up to 10 predictors with power (b) = .95 and alpha = .05 (Cohen, 1977, 1992; Faul & Erdfelder, 1992). All analyses were at this power level or better, and with N = 394, there was ample power to detect medium sized effects. In analyses with 2 predictors, there was sufficient power to detect a small effect size (f^2 = .02) at a power of b = .714 (a = .05) (Cohen, 1977, 1992; Faul & Erdfelder, 1992). In the SEM analyses, there were at least 10 participants for every 1 estimated variable, 5 participants for every parameter (Bentler, 1990; Kline, 2005).

Hypothesis 1a. Selected family measures would statistically predict ADHD symptoms independent of parent ADHD, one another, ODD, and CD.

To test this preliminary hypothesis, a hierarchical multiple regression was conducted in SPSS[2] with teacher-rated ADHD symptoms as the dependent variable[3]. Developmental risk factors (i.e. low birth weight, prenatal smoking, and prenatal alcohol use) as recalled by the mother were entered into Step 1 of this model to account for potential confounds. Next, parent 1 ADHD and parent 2 ADHD were entered into Step 2. Finally, FES factors, CPIC factors, parent depression, income, ODD, and CD were entered into Step 3. This analysis helps clarify whether family risk factors contribute to ADHD independent of common comorbid conditions (Rey, Walter, Plapp, & Denshire, 2000). Results of this analysis are presented in Table 2. Step 3 brought a significant gain in r^2 ($\Delta r^2 = .175$, $p < .001$), which justified pursuing the corresponding effects. The CPIC Self Blame factor was uniquely related to ADHD apart from the other family measures (including Developmental Risk factors), parent ADHD, ODD, and CD ($\beta = .143$, $p = .042$)[4].

Hypothesis 1b. The same family measures would predict inattentive and hyperactive symptoms.

Two additional preliminary multiple regression analyses were conducted in SPSS using the predictors and the procedures outlined in Hypothesis 1a. However, in this case the dependent variables were (1) the number of ADHD inattentive symptoms and (2) the number of ADHD hyperactive/impulsive symptoms defined by teacher report of current symptoms on the Conners'. In each analysis, there was a significant gain in r^2 ($\Delta r^2 =$

[2] Results were similar when I ran these analyses in M*plus* using FIML.

[3] Ratings on the Conners' Teacher Rating Scale were significantly correlated with parent report ratings on the KSADS-E semi-structured interview ($r = .65$, $p < .001$), and on the Conners' Parent Rating Scale ($r = .61$, $p < .001$). Teacher ratings were used as the dependent variable to limit reporter bias, as several independent variables were obtained via parent report.

[4] As an additional check on the effect of covariates, ODD and CD were removed from the model and results are shown in Appendix D. Another analysis was run with ODD and CD in Step 2, and findings were similar to those reported in Hypothesis 1a. Results of this analysis are also shown in Appendix D.

.196, $p < .001$ for inattentive symptoms, and $\Delta r^2 = .187$, $p < .001$ for hyperactive/impulsive symptoms).

In the analysis of inattentive symptoms, the CPIC Conflict Quality factor (β = .177, $p = .037$), and the CPIC Self Blame factor ($\beta = .269$, $p < .001$) were significant beyond the effects of the other family measures (including Developmental Risk factors), apart from parent ADHD, ODD, or CD. However, in the analysis of hyperactive/impulsive symptoms, there was only a significant effect of ODD symptoms ($\beta = .226$, $p = .004$). This illustrates a difference in the predictability of inattentive and hyperactive/impulsive symptoms of ADHD.

The CPIC Self Blame factor was a consistently significant predictor of ADHD symptoms. A supplementary regression analysis was conducted to determine whether ODD, inattentive symptoms, or hyperactive/impulsive symptoms influence the CPIC Self Blame factor. The regression model was significant, $F = 12.55$, $p < .001$, $R^2 = .095$. However, the inattentive symptom variable was the only significant predictor ($\beta = .270$, $p < .001$). These results suggest that the significant effect of CPIC Self Blame was not carried by ODD or hyperactive/impulsive symptoms.

Hypothesis 2a. The family measures could be consolidated to form a latent family risk construct.

A Confirmatory Factor Analysis (CFA) was conducted to evaluate the fit of our a priori single factor model of "family risk" (comprised by FES factors, CPIC factors, parent depression, and income). The initial model fit was unacceptable (χ^2 (14, $N = 394$) = 212.15, $p < .001$, Comparative Fit Index (CFI) = 0.29, Root Mean Square Error of Approximation (RMSEA) = 0.19, and Standardized Root Mean Square Residual (SRMR)

= 0.10). FES Values and FES Expressiveness had poor loadings on the family risk factor (FES Values StdYX = -.14, $p > .05$; FES Expressiveness StdYX = -.15, $p > .05$), and were therefore eliminated from the model. The means and standard deviations of the FES factors suggested that most families endorsed sub-clinical levels of these phenomena (clinical cut-off = $T = 65$; mean T score = 47.92, mean SD = 10.84), reflecting similarities in value-orientation and expressiveness across the sample.

Separate analyses were conducted to determine whether ADHD was related to FES independent of ODD. First, FES Values and FES Expressiveness were regressed onto CTRS ADHD symptoms and ODD. ADHD significantly predicted FES Expressiveness ($F = 6.56$, $p < .01$, $R^2 = .036$), but not FES Values beyond the effects of ODD ($F = 2.46$, $p = .09$, $R^2 = .014$). Next, CTRS ADHD was regressed onto FES Expressiveness and ODD. FES Expressiveness was significantly related to ADHD ($F = 24.16$, $p < .001$, $R^2 = .121$), even when ODD was in the model. Nevertheless, the previous CFA model showed that neither FES factor contributed to the family risk variable.

The CFA model was computed again without the FES factors, and resulted in a significant improvement in fit, $\Delta \chi^2 (5, N = 394) = 27.29$, $p < .01$. Modification indices suggested that CPIC Perceived Threat be allowed to correlate with CPIC Self Blame (modification index = 18.69). The resulting model is shown in Figure 1. It resulted in good overall fit, $\chi^2 (8, N = 394) = 4.97$, $p > .05$, CFI = 1.00, RMSEA = 0.00, and SRMR = 0.02. This family risk latent factor was used in all subsequent analyses.

Hypothesis 2b. ADHD would be significantly related to the latent family risk construct.

The analyses from Hypothesis 1 were conducted again using the family risk latent variable in an SEM analysis predicting ADHD total symptoms (measured henceforth using the Conners' Teacher Rating Scale) as well as teacher-reported inattentive symptoms and teacher-reported hyperactive/impulsive symptoms (also measured using the CTRS).

When covarying family risk with developmental risk, ODD, CD, and parent ADHD, most of the fit indices were indicative of unacceptable fit: χ^2 (36, N = 394) = 126.27, $p < .001$, CFI = .80, RMSEA = .08, and SRMR = .06. In this model, family risk was appropriately indicated by the CPIC, depression, and income factors as in Hypothesis 2a, and ADHD total symptoms were significantly regressed onto family risk ($\beta = .607$, $p < .05$). However, ODD also significantly predicted ADHD total symptoms ($\beta = .202$, $p < .05$), and ODD was also significantly correlated to family risk ($r = .279$, $p < .05$). The significant effects of ODD are consistent with findings of frequent comorbidity with ADHD.

An additional analysis revealed that the relation of ADHD to family risk was not due to the relation of ODD to family risk. The model shown in Figure 2 provided a good overall fit: χ^2 (16, N = 394) = 21.31, $p > .05$, CFI = .98, RMSEA = .03, and SRMR = .03. When ODD was removed from the model, ADHD's estimate became StdYX = .37, $p < .05$. Analyses have shown that the covariates (developmental risk, parent ADHD, ODD, and CD) do not account for the relationship between ADHD and family risk, therefore remaining analyses focused solely on the role of family risk factors in predicting ADHD outcomes (without covariates).

The model shown in Figure 3 shows the relationship between the latent family risk variable and ADHD, and provided a good overall fit: χ^2 (12, N = 394) = 20.47, p > .05, CFI = .97, RMSEA = .04, and SRMR = .03. A single manifest dependent variable was used once again to represent ADHD total symptoms (CTRS), and all path estimates were significant[5]. The model for inattentive symptoms (CTRS) was similar, however all fit indices except the χ^2 were indicative of good fit: χ^2 (12, N = 394) = 24.04, p = .02, CFI = .96, RMSEA = .05, and SRMR = .03. The model for hyperactive/impulsive symptoms (CTRS) was indicative of overall good fit: χ^2 (12, N = 394) = 11.94, p > .05, CFI = 1.0, RMSEA = .00, and SRMR = .03. These findings consistently show a significant relationship between family risk and ADHD, with Figure 3 representing the most comprehensive best-fitting model across both components of the ADHD construct measured by teacher report on the CTRS.

Hypothesis 2c. Family risk would predict ADHD when items were used to create latent factors for ADHD symptom domains.

An SEM analysis was conducted to determine whether the latent family risk variable was related to ADHD as represented by two latent variables – inattention and hyperactivity/impulsivity – indicated by CTRS items. The purpose of this was to evaluate specificity of effects to ADHD symptom domains, as well as to help confirm that findings were not an artifact of how ADHD was measured. The model shown in Figure 4 resulted in a good overall fit: χ^2 (123, N = 394) = 147.80, p > .05, CFI = .99, RMSEA = .02, and SRMR = .04. Following the suggested modification indices allowed items within the same scale to correlate with one another. The correlated items were theoretically (as well as empirically) related.

[5] Similar fit and significant path loadings were obtained when age was covaried.

The preceding hypotheses involved preliminary analyses in order to set the stage for the primary focus of investigation, which now follows.

Hypothesis 3a. Cognitive regulation would partially mediate the relationship between family risk and ADHD.

Cognitive regulation was, as indicated earlier, a manifest variable comprised of the average of standardized scores for Stop Task SSRT and response variability. FSIQ was covaried to check for effects on results. The model, shown in Figure 5, provided a good overall fit: χ^2 (23, N = 394) = 27.45, p > .05, CFI = .98, RMSEA = .02, SRMR = .02, and Bayesian Information Criterion[6] (BIC) = 9079.18. The indirect effect for this model was significant (StdYX = .03, p < .05)[7]. When the mediation analysis shown in Figure 5 was conducted using bootstrapping (rather than clusters), the findings were similar (see Appendix E). Thus, cognitive regulation did partially mediate the relationship between family risk and ADHD.

Hypothesis 3b. Child age would moderate the partial mediation described in 3a.

An M*plus* multiple group analysis was used to test this relationship. This was done by adding age (at assessment of outcome) as a grouping variable to the model in Figure 5. The child group was comprised by children ages 6-12 years old (N = 166), and the adolescent group included youth ages 13-18 years old (N =228). Although alternative models were not formally tested, the resulting fully constrained models, shown in Figures 6 and 7, suggest that the factor loadings and the factor variance-covariance structures were not equivalent for children and adolescents: χ^2 (56, N = 394) = 121.36, p < .001, CFI = .84, RMSEA = .08, SRMR = .07, and BIC = 8956.92. In the child group, the indirect

[6] The model with the lowest BIC value is preferable to models with higher values (Kline, 2005).
[7] The results for model fit, significant path loadings, and indirect effect were similar when age was covaried.

effect was non-significant (StdYX = .02, $p > .05$), whereas the indirect effect was significant (StdYX = .05, $p < .05$) in the adolescent group. The lack of an indirect effect in the child group is due to the non-significant path between cognitive regulation and family risk. Qualitative differences were observed between the models in the child and adolescent groups. For example, FSIQ was not a significant predictor of ADHD in the child group. Thus, family risk appears to have a significant effect on cognitive regulation in adolescents, though not in children. However, family risk was significantly related to ADHD in both groups.

Hypothesis 3c. Race would moderate the partial mediation described in 3a.

An M*plus* multiple group analysis was again used to test this relationship. In this case, race was used as a grouping variable (Caucasian $N = 291$, ethnic minority $N = 103$), and results on the Multigroup Ethnic Identity Measure (MEIM) were covaried to prevent sole reliance on the demographic race variable. Again, although alternative models were not formally tested, the resulting fully constrained models, shown in Figures 8 and 9, suggest that the factor loadings and the factor variance-covariance structures were not equivalent for Caucasian and ethnic minority groups: χ^2 (68, $N = 394$) = 123.33, $p < .001$, CFI = .87, RMSEA = .06, SRMR = .07, BIC = 9947.08. The indirect effect was significant in the Caucasian group (StdYX = .04, $p < .05$), but was not significant in the ethnic minority group (StdYX = .01, $p > .05$). This is apparent in Figure 9, as family risk did not predict cognitive regulation and cognitive regulation did not predict ADHD in the ethnic minority group. FSIQ and MEIM were not significantly related to ADHD in either group. The effect of family risk on ADHD was not accounted for by cognitive regulation in the ethnic minority group.

Given the similarity of the race effects to the age findings, an additional check was run to ensure that results were not an artifact of the sample distribution of race and age. There was a proportional distribution of Caucasian to ethnic minority participants in the child and adolescent age groups: child Caucasian $N = 115$ (69%), child ethnic minority $N = 51$ (31%), adolescent Caucasian $N = 176$ (77%), adolescent ethnic minority $N = 52$ (23%).

Hypothesis 4. ADHD would partially mediate the relationship between family risk and adaptive functioning.

ADHD should partially mediate the relationship between family risk and adaptive functioning if family risk is specific to ADHD, and not merely representative of global impairment. To test this, an M*plus* mediation analysis was conducted (as in Hypothesis 3) using a composite adaptive functioning score that represented parent and clinician report.

The model, shown in Figure 10, resulted in good overall fit: $\chi^2 (17, N = 394) = 29.72, p < .05$, CFI = .97, RMSEA = .04, and SRMR = .04. The indirect effect was significant (StdYX = -.13, $p < .05$), resulting in a partial mediation[8]. This showed that the effect of family risk on participants' adaptive functioning was significantly partially mediated by ADHD.

[8] Results and significant path loadings were similar when age was covaried.

DISCUSSION

Adversity in the family environment has been established as a risk factor for maladaptive child behavioral outcomes (Rutter et al., 1975); however the mediating mechanisms associated with this relationship were under-researched. The present investigation had as its main aim to examine whether cognitive processes accounted for the relationship between family risk and ADHD. Via preliminary analyses, the present study replicated existing research showing that family risk factors predict ADHD and that these measures were able to be fit as valid indicators of a latent composite family risk construct using confirmatory factor analysis. To the main hypothesis, cognitive regulation did partially explain the relation between family risk and ADHD, although this relationship depended upon age and (a secondary focus) race group. Finally, ADHD partially explained the relationship between family risk and impairment. Each of these findings is considered in sequence.

Preliminary Findings

First, it will be useful to discuss the preliminary findings that helped determine the building blocks for use in the study's main hypothesis regarding neurocognitive functioning. Consistent with Hypothesis 1a, family measures (Conflict Quality, Marital Unhappiness, Self Blame, Perceived Threat, Maternal Depression, and Income) were collectively associated with ADHD symptoms (measured using CTRS Total Symptoms) even when covaried with one another, developmental risk factors, parent ADHD, ODD, and CD. However, only Self Blame was uniquely related to ADHD above and beyond the other family measures. In a related study that used a different sample, but similar family risk measures as those used in the present study (Counts et al., 2005), summary score of

child perception of interparental conflict was the best predictor of ADHD after controlling for other risk measures. Thus, it is important to note that the present study replicated the Counts et al. findings.

One could speculate that the effect of Self Blame, in particular, may have been prominent because it is the only risk factor that involves perceived culpability of the child for the family adversity. Another explanation for fewer unique predictors is that the other family risk measures were related to aspects of ADHD that overlap with ODD, which was also significant in the model. According to this supposition, Self Blame evidenced an observably significant effect on ADHD beyond the other covariates because it may not overlap with ODD. A supplementary analysis confirmed that Self Blame does not predict ODD when ADHD was covaried.

In a more robust test, two analyses were conducted to determine the relationship between the selected family risk measures (Conflict Quality, Marital Unhappiness, Self Blame, Perceived Threat, Maternal Depression, and Income) and (1) ADHD inattentive and (2) ADHD hyperactive symptom domains (measured using the CTRS). These findings were consistent with Hypothesis 1b and suggest that the family risk measures were collectively associated with ADHD beyond possible confounds, even when it was measured in terms of its two major symptom domains.

The individual family risk measures had different effects on inattentive versus hyperactive symptoms. In regard to inattentive symptoms, CPIC Self Blame and Conflict Quality factors emerged as uniquely predictive above and beyond the other measures and confounds. One interpretation of these results is that children experience inattentive symptoms due to impairment associated with perceiving that interparental conflict is the

child's own fault and the frequency and intensity of parents' arguments. Further study is needed to determine whether inattention mediates the relationship between Self Blame and Conflict Quality, and impairment. This would show that impairment specific to inattention is related to these risk factors. In regard to hyperactive/impulsive symptoms, family risk measures were not unique predictors beyond confounds. It appears that ODD contributed significantly to the effect, which is consistent with research that showed that hyperactivity/impulsivity is more readily associated with comorbid externalizing problems (including ODD) than the ADHD inattentive subtype (Eiraldi et al., 1997; Milich et al., 2001).

The present study showed that the effect of Self Blame on ADHD was not due to its relation with hyperactivity or to co-occurring ODD, but to its relation with the inattention domain in particular. These Self Blame findings may be consistent with research that showed that parental disagreement about childrearing was related to more marital conflict as well as more problematic child behavior (Harvey, 2000). In summary, whereas several studies have identified family conflict as an important risk factor associated with ADHD (Biederman et al., 1995b; Counts et al., 2005; Lange et al., 2005; Murphy & Barkley, 1996; Pressman et al., 2006); the present study extended these findings by adding information on specific interparental conflict factors as perceived by the child, and their relationship to the ADHD inattentive symptom domain.

After looking at the individual family risk measures, the present study validated a latent family risk construct that included most hypothesized family risk measures (Hypothesis 2a). Measures that were significant indicators of family risk include four variables related to child perception of interparental conflict (Conflict Quality, Marital

Unhappiness, Self Blame, and Perceived Threat), as well as maternal depression, and income. These findings are consistent with previous research on family adversity (Counts et al., 2005; Kendall et al., 2005; Minde et al., 2003; Pressman et al., 2006; Satake, Yamashita, & Yoshida, 2004; West et al., 1999). Contrary to hypotheses, the FES factors (Values and Expressiveness) did not load on the family risk latent factor; however this may be due to floor effects in the data that were more evident on these factors than on others. Supplementary findings confirmed a relationship between FES and ADHD (when other variables were excluded), however FES did not lend significantly to the family risk construct as in previous studies (Biederman, Milberger, Faraone, Kiely, & et al., 1995b; Pressman et al., 2006). This weak result for the parent-reported FES measure may be due to the prominent effects of child perception of interparental conflict as a predictor of ADHD in the present study. Child perceived interparental conflict was not included in models used in the other studies.

As anticipated in Hypothesis 2b, the significant relationship between the family risk latent variable and ADHD symptoms remained independent of ODD effects on ADHD. These findings are consistent with prior studies that included children and adolescents, assigned diagnoses using structured clinical interviews, and derived family adversity variables using the most well recognized procedures for aggregating family risk in the field (Biederman et al., 1995b; Counts et al., 2005). However, Rey et al. (2000) found no relationship between ADHD and the quality of the family environment in clinically referred adolescents. Several methodological differences between the Rey et al. and present (and previous) studies may account for the dissimilar results. First, the Rey et al. study was limited to adolescents. Second, clinicians in the Rey et al. study used

information from patient charts to assign ADHD diagnoses, rather than using structured clinical interviews. Third, Rey et al. measured family factors using different constructs the Global Family Environment Scale (GFES; Rey et al., 1997). The GFES consisted of clinician ratings of quality of the family environment (i.e. punitive, inconsistent, and inappropriate limit setting, insecure attachment relationships, and emotional care) based on chart reviews reflecting the lowest family environment score that occurred for a 12 month duration, before the child was 12 years of age. These methodological differences may account for differences in findings.

The present study showed that family risk predicted ADHD even when ADHD was evaluated as a latent variable, and items were used to create latent factors to represent inattentive and hyperactive/impulsive symptom domains. This was consistent with predictions in Hypothesis 2c. The most important finding here was that family risk was not differentially associated with either inattention or hyperactivity. This means that the latent variable did not predict one symptom domain significantly more strongly than the other, which suggests that family risk is relevant across ADHD symptoms, and permits the use of an ADHD total symptom variable in subsequent analyses. These findings further confirm the utility of the family risk variable, as it has been shown to predict ADHD using a range of operational measures.

Main Findings

Second, and coming to the main finding in this dissertation, consistent with Hypothesis 3a, the relationship between family risk and ADHD was partially mediated by cognitive regulation. That is, family risk factors were related to cognitive processes, which were in turn associated with ADHD deficits – all independent of IQ. One way to

interpret this is that it supports the possible role of family events in epigenetic processes (i.e. effects on gene expression) (Rutter et al., 2006). The present study identified cognitive mechanisms (response inhibition and vigilance) known to shape ADHD behaviors (Barkley, 1997; Crosbie & Schachar, 2001; Nigg, 1999), and demonstrated their connection to family risk. It may be speculated that, among other possibilities, the model described by Cicchetti and Curtis (2006) wherein family effects on gene expression can manifest behavioral change via effects on cognition – might map onto the mediation described in Figure 5.

Another interpretation of these findings is based on established relationships between family risk factors and cognitive and behavioral functioning in children. The cognitive model used by many psychotherapists theorizes that an individual's emotions and behaviors are influenced by their perception of events (Beck, 1964; Ellis, 1962). This model appears to explain the relationships found in Figure 5, and is also consistent with findings of other relevant studies. Grych and Fincham (1993) found that children's appraisals of interparental conflict (i.e. their cognitive interpretations of the stressor) were affected by its intensity, content, and cause, which thereby determined the extent of the stressor's effect on the child's emotions and behaviors. Nigg, Nikolas, Kriderici, Park, and Zucker (2007) found that strong response inhibition capability was a protective factor against ADHD when moderate to high levels of family adversity were present. The partial mediation found in the present study may therefore have important implications for clinical practice in support of the cognitive model.

Third, a child's developmental stage was hypothesized to moderate the mediation of family risk on ADHD via cognitive regulation, due to the influence of experiences on

brain development and behavior (Cicchetti & Curtis, 2006; Hebb, 1949; O'Conner, 2003). Consistent with Hypothesis 3b, age groups showed a differential effect of risk on cognitive regulation which suggests that risk, though it predicts ADHD in both groups, affects cognitive regulation in adolescents and not in younger children. These findings are consistent with Richmond and Stocker's (2007) results which indicated that children's appraisals of marital conflict declined significantly from childhood to adolescence in terms of perceived threat; however, little change was observed over time in appraisals of self-blame.

An alternative explanation for the age moderation is a possible group difference in early caregiver interchanges that are thought to facilitate the development of self regulation, response control, and arousal (Kochanska & Aksan, 2006; Panzer & Viljoen, 2005; Tucker et al., 2005). Yet another explanation is that inhibitory control and arousal mechanisms that are related to family risk in the adolescent group have not fully developed to evidence a significant effect in the younger child group (Akshoomoff, 2002; Booth et al., 2003; Levy, 1980; Rubia et al., 2006; Williams et al., 1999). IQ was negatively associated with ADHD in the adolescent group (Hinshaw et al., 1987; Peterson, et al., 2001), but this path may not have been evident in the child group due to differences in cognitive development or good matching of groups. Further study is needed to determine other mechanisms that account for the effect of family risk on ADHD behavior in young children.

Fourth, as a secondary focus, race was also hypothesized to moderate the mediated relationship between family risk and ADHD via cognitive regulation. Consistent with Hypothesis 3c, differences in the mediation model were observed

between the Caucasian and ethnic minority groups. Family risk predicted ADHD in both groups. However in ethnic minorities, cognitive regulation did not partially mediate the relationship between risk and ADHD. This suggests that a different mechanism may explain the relationship in ethnic minorities. Neuropsychological deficits are known to be associated with ADHD (Nigg, Blaskey, Huang-Pollock, & Rappley, 2002), and controlling for acculturation has been shown to account for ethnic differences in neuropsychological test performance (Manly et al., 1998). It can be speculated that the relationship between family risk and ADHD may change over levels of race due to differences in neuropsychological functioning. In addition, ethnic identity did not appear to contribute significantly to the model in either group, though it was significantly correlated with family risk in the ethnic minority group. That is, increased ethnic identity was a protective factor for ADHD. These findings are consistent with group differences in race salience (Sellers, Smith, Shelton, Rowley, & Chavous, 1998), and suggest the need for future studies to clarify factors that may account for the observed race differences such as acculturation, multidimensional racial identity, or parent explanatory models (Bussing et al., 1998; Sellers et al., 1998; Sullivan et al., 2007).

Finally, as predicted in Hypothesis 4, ADHD partially mediated the relationship between family risk and adaptive functioning. This suggests that family risk has impairing effects that are ADHD-specific, rather than simply indicating global impairment. Though it is important to note that impairment is a requirement for the ADHD diagnosis, these results support research that found an association between ADHD and high levels of impairment (Gaub & Carlson, 1997; Gershon, 2002; Strine et al., 2006), and extends the literature to include syndrome-specific effects. Family risk,

while generally impairing, has particular effects on child behavior. Those effects have been outlined herein, and are further supported by this finding.

Clinical Implications

Family risk has been shown to pinpoint key cognitive factors in the development of a subsequent expression of ADHD symptoms. Children's perceptions of interparental conflict appear to be keenly involved in this relationship, therefore helping children develop appropriate coping mechanisms and methods of interpretation of conflict events may prevent maladaptive behavior. In particular, it would be important to determine whether early intervention could weaken the noticeably stronger relationship observed between family risk and ADHD behavior in children. The unique effects of self-blame and other possible interpretations of interparental conflict may be areas to begin such an intervention.

The present study highlighted a mechanism whereby the family can affect ADHD behavioral outcomes. In particular, parent training programs have become recognized as important components of empirically supported treatment protocols for ADHD. Such programs help parents understand the nature of the ADHD syndrome and teach behavior management skills to facilitate a more productive experience for the child (DuPaul, Guevremont, & Barkley, 1994). The measurement of parent training outcomes ranges from assessing changes in parent stress, child behavior, or family functioning (Anastopoulos & Farley, 2003).

Another important treatment consideration is the lack of empirical support for *cognitively* based treatment of ADHD, which involves strategies to help alter maladaptive thinking patterns that affect behavioral functioning. Findings of the present study may

help explain the phenomenon of ineffective cognitive treatments for young children, as their level of development may render these strategies inaccessible or require access via alternate pathways. Adolescents may fare better, as they are developmentally more equipped to tolerate cognitive skills training. However, there is a dearth of research on CBT treatment efficacy in teens (Chronis, Jones, & Raggi, 2006). One study found support for parent-teen training (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001), consistent with family and neurocognitive pathways investigated in the present study. That is, the family can be used as another tool to help remediate cognitive difficulty in children with ADHD.

Finally, ethnic identity appeared to be a protective factor in relation to family risk in the ethnic minority group such that individuals with greater ethnic identity had less family risk. Recognition of these culture-specific effects can be useful in facilitating treatment retention and compliance.

Limitations

The cross-sectional design of the present study limited the ability to look at temporal effects (i.e. whether the interparental conflict was recent), which would be available if a longitudinal design was used. Also, as mentioned, genetic effects may have been implicit in family risk factors, and there may be a feedback loop of ADHD effects with respect to family risk. Also, sample size and statistical power were generally adequate-to-ample in this investigation, however they may have been lower (though still statistically viable) in ethnicity analyses involving relatively smaller groups (ethnic minority group $N = 103$ (26%), although representative of the local population). In

addition, within-group specificity was limited as several cultures were included in the ethnic minority group, which may affect generalizability (Tyson, 2004).

Conclusion

Overall, the findings support the relationship between family risk factors and ADHD via disruption of neurocognitive functioning. The present study replicated previous investigations of factors related to family risk. Further, it validated a latent family risk variable, which was used to examine cognitive mechanisms involved in its effect on ADHD. Analyses were conducted in a large diverse sample that included boys and girls across a broad age range. The data further suggest that the relation among these factors depends upon age and race. The present study extended existing research and contributed new findings that will inform theory and clinical practice regarding key family risk factors associated with ADHD, a significantly impairing condition in children and adolescents.

APPENDICES

APPENDIX A

Main Tables and Figures

Table 1a. Description of Sample

	Control	ADHD	*p*	Full Sample
N (%)	184 (47%)	210 (53%)	--	394
Number (%) Male	81 (42%)	139 (63%)	<.001	220 (56%)
Number (%) Caucasian	132 (45%)	159 (55%)	.370	291 (74%)
Age group (%) Adolescents	125 (55%)	103 (45%)	<.001	228 (58%)
(%) Children under 13	59 (36%)	107 (64%)		166 (42%)
Age in years	13.9 (2.9)	12.7 (3.1)	<.001	13.3 (3.1)
Oppositional Defiant Disorder Sx	.87 (1.6)	2.5 (2.5)	<.001	1.7 (2.3)
Conduct Disorder Sx	.10 (.33)	.40 (.79)	<.001	.25 (.64)
Parent 1 Current ADHD Sx	1.3 (2.7)	2.5 (3.9)	.001	1.9 (3.5)
Parent 2 Current ADHD Sx	.98 (2.4)	1.6 (2.8)	.109	1.3 (2.6)
Conners' ADHD Total Score	4.2 (5.3)	16.5 (8.9)	<.001	10.8 (9.7)
# Current DSM Inattentive Sx	1.0 (1.7)	7.6 (1.4)	<.001	4.5 (3.6)
# Current DSM Hyp/Imp Sx	.63 (1.3)	4.6 (3.2)	<.001	2.7 (3.2)
CPIC Conflict Quality	2.0 (.63)	2.2 (.57)	.002	2.1 (.61)
CPIC Marital Unhappiness	1.3 (.40)	1.4 (.44)	.010	1.3 (.43)
CPIC Self Blame	1.2 (.28)	1.4 (.38)	<.001	1.3 (.36)
CPIC Perceived Threat	1.4 (.50)	1.7 (.59)	<.001	1.5 (.57)
Parent 1 Depression Score	.59 (.72)	.79 (.82)	.020	.69 (.78)
SSRT	217.8 (76.9)	262.6 (119.3)	<.001	241.4 (103.9)
RT Variability	132.2 (35.0)	152.9 (40.7)	<.001	143.2 (39.5)
FSIQ	109.3 (14.5)	102.9 (13.1)	<.001	105.8 (14.1)
MEIM	2.7 (.53)	2.8 (.57)	.872	2.7 (.55)
GAF	80.9 (9.5)	69.3 (8.9)	<.001	74.8 (10.9)

Notes to Table 1a: P-value reflects the two group comparison (control vs ADHD) and is based on an independent samples T-test for continuous variables or a chi-square test for dichotomous variables. N's vary slightly for some measures due to missing data for some disorders. "Sx" refers to symptoms. DSM = Diagnostic and Statistical Manual of Mental Disorders, fourth edition, text revision (DSM-IV-TR). CPIC = Children's Perception of Interparental Conflict Scale. SSRT = Logan Stop Task Stop Signal Reaction Time measured in milliseconds, RT Variability = Logan Stop Task Response Time Variability. FSIQ = full scale IQ, MEIM = Multigroup Ethnic Identity Measure, and GAF = DSM Global Assessment of Functioning score.

Table 1b. Description of Household Demographics Across Groups

	Full Sample	Child Group	Adolescent Group	*p*	Caucasian Group	Minority Group	*p*
Annual Household Income	66k (35k)	60k (32k)	70k (36k)	.005	68k (34k)	59k (37k)	.027
Income ≤ $25,000 Per Year	39 (10%)	23 (14%)	16 (7%)	.047	21 (7%)	18 (18%)	.005
Parent Education (%) High School	105 (27%)	61 (37%)	44 (19%)	.056	67 (23%)	38 (37%)	.040
(%) Some College	117 (30%)	58 (35%)	59 (26%)		85 (29%)	32 (31%)	
(%) Bachelor's	71 (18%)	28 (17%)	43 (19%)		62 (21%)	9 (9%)	
(%) Master's	31 (8%)	14 (8%)	17 (8%)		21 (7%)	10 (10%)	
(%) Doctorate	1 (.3%)	0 (0%)	1 (.4%)		1 (.3%)	0 (0%)	
Child Lives With Both Parents	279 (71%)	121 (73%)	158 (70%)	.216	221 (76%)	58 (56%)	.001
One Parent	101 (26%)	42 (25%)	59 (26%)		59 (20%)	42 (41%)	
Other Relative	6 (2%)	0 (0%)	6 (3%)		5 (2%)	1 (1%)	

Notes to Table 1b: P-value reflects the two group comparison (control vs ADHD) and is based on an independent samples T-test for continuous variables. Percentages may not equal 100 in cases when data were not available. For Annual Household Income, values were rounded to the nearest thousand, where k = thousands of US dollars. The child group includes participants under age 13, whereas the adolescent group includes children ages 13 – 18 years of age. The Caucasian group includes participants who self-identified as such, whereas the ethnic minority group includes children of African American, Latino, other, biracial, or mixed origin.

Table 2. Regression Model: Teacher-rated ADHD Symptoms Regressed Onto Predictors of Risk (Hypothesis 1a)

		R^2	*B*	*SE B*	β
Step 1	Developmental History	.004	.119	.123	.067
Step 2	Developmental History	.023	.101	.124	.056
	Parent 1 Current ADHD Sx		.105	.082	.089
	Parent 2 Current ADHD Sx		.096	.073	.091
Step 3	Developmental History	.198	.088	.116	.049
	Parent 1 Current ADHD Sx		.000	.092	.000
	Parent 2 Current ADHD Sx		.057	.070	.055
	FES Values		-.121	.082	-.134
	FES Expressiveness		.074	.099	.071
	CPIC Conflict Quality		.045	.085	.045
	CPIC Marital Unhappiness		.032	.087	.031
	CPIC Self Blame		.149	.073	.143*
	CPIC Perceived Threat		.126	.077	.121
	Maternal Depression		.018	.075	.019
	Income		-.085	.057	-.101
	ODD Sx		.203	.076	.206**
	CD Sx		.062	.089	.053

Notes to Table 2: "Sx" refers to symptoms. FES = Family Environment Scale, CPIC = Children's Perception of Interparental Conflict Scale, ODD = Oppositional Defiant Disorder, CD = Conduct Disorder. The dependent variable used in this analysis was teacher-reported total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$, ** $p < .01$.

Figure 1. CFA Model of Family Risk (Hypothesis 2a)

Notes to Figure 1: *Mplus* StdYX values were reported herein. These values are standardized based on the variances of the latent and observed variables to show the amount of change in Y per standard deviation unit of X. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. CPIC = Children's Perception of Interparental Conflict Scale. * $p <$ .05. χ^2 (8, N = 394) = 4.97, $p > .05$, CFI = 1.00, RMSEA = 0.00, and SRMR = 0.02.

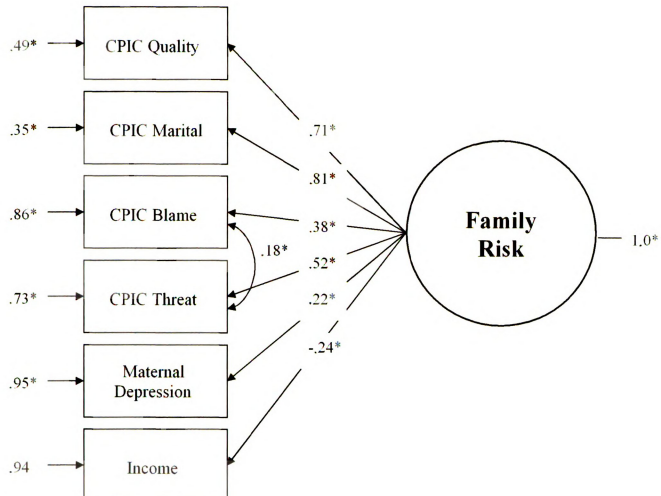

Figure 1. CFA Model of Family Risk (Hypothesis 2a)

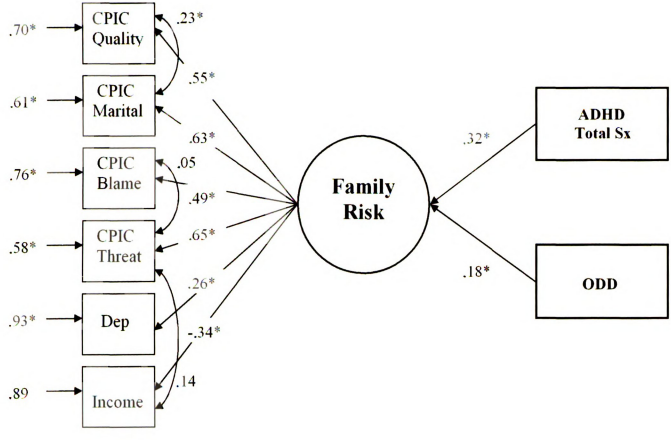

Figure 2. SEM of Family Risk Regressed onto ADHD Total Symptoms and ODD (Hypothesis 2b)

Notes to Figure 2: *Mplus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. CPIC = Children's Perception of Interparental Conflict Scale, Dep = Maternal Depression, Sx = symptoms, ODD = Oppositional Defiant Disorder. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. $\chi^2(16, N = 394) = 21.31, p > .05$, CFI = .98, RMSEA = .03, and SRMR = .03.

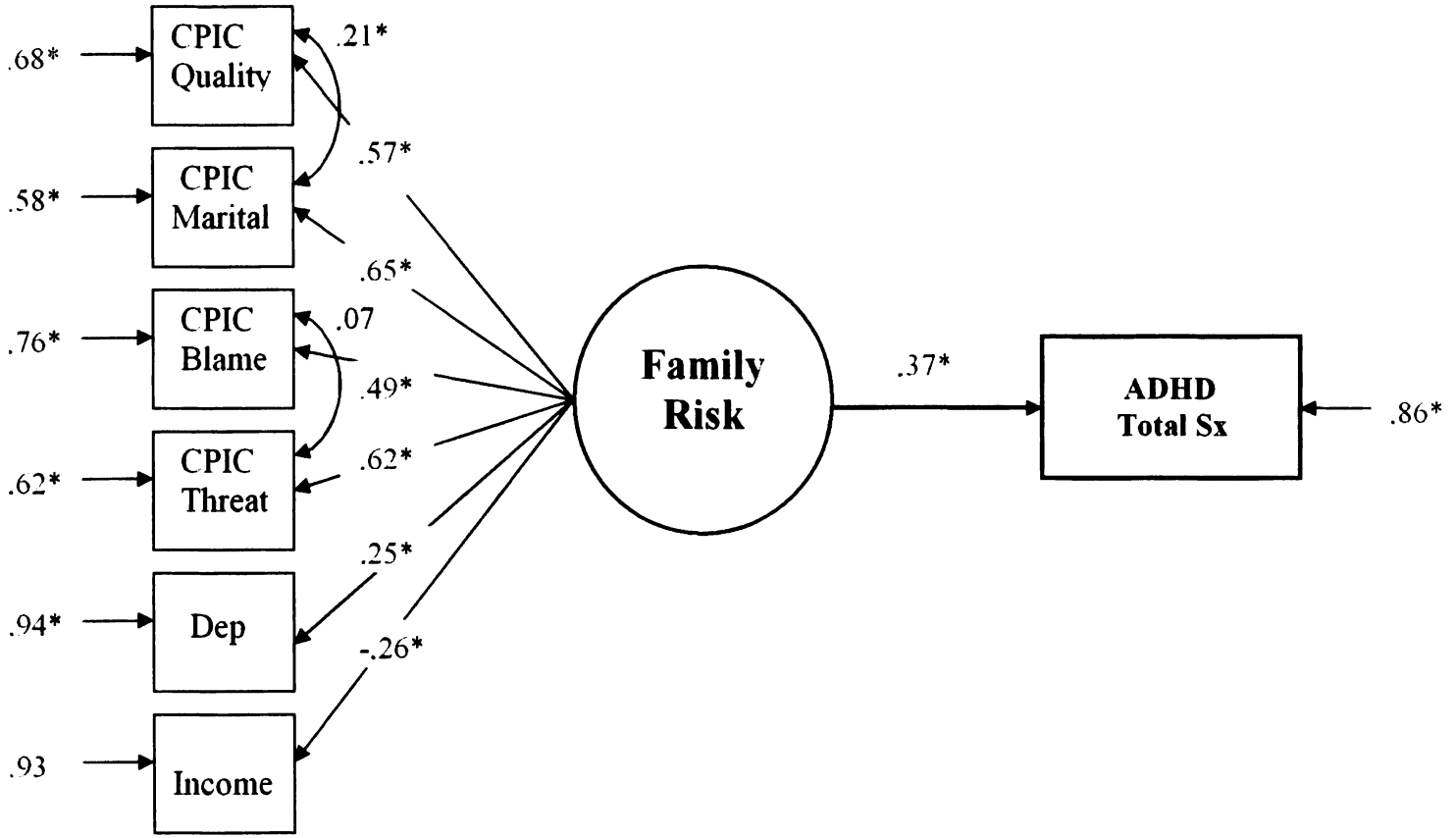

Figure 3. SEM of ADHD Total Symptoms Regressed onto Family Risk (Hypothesis 2b)

Notes to Figure 3: M*plus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. CPIC = Children's Perception of Interparental Conflict Scale, Dep = Maternal Depression, Sx = symptoms. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. In the current figure, age is not covaried, but results were essentially the same when it was covaried. χ^2 (12, N = 394) = 20.47, $p > .05$, CFI = .97, RMSEA = .04, and SRMR = .03.

Figure 4. SEM of CTRS Items Regressed onto Family Risk (Hypothesis 2c)

Notes to Figure 4: *Mplus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. CPIC = Children's Perception of Interparental Conflict Scale, Dep = Maternal Depression, Inattn = ADHD Inattention, Hyper = ADHD Hyperactivity/Impulsivity, CTRS = Conners' Teacher Rating Scale. * $p < .05$. χ^2 (123, N = 394) = 147.80, $p > .05$, CFI = .99, RMSEA = .02, and SRMR = .04.

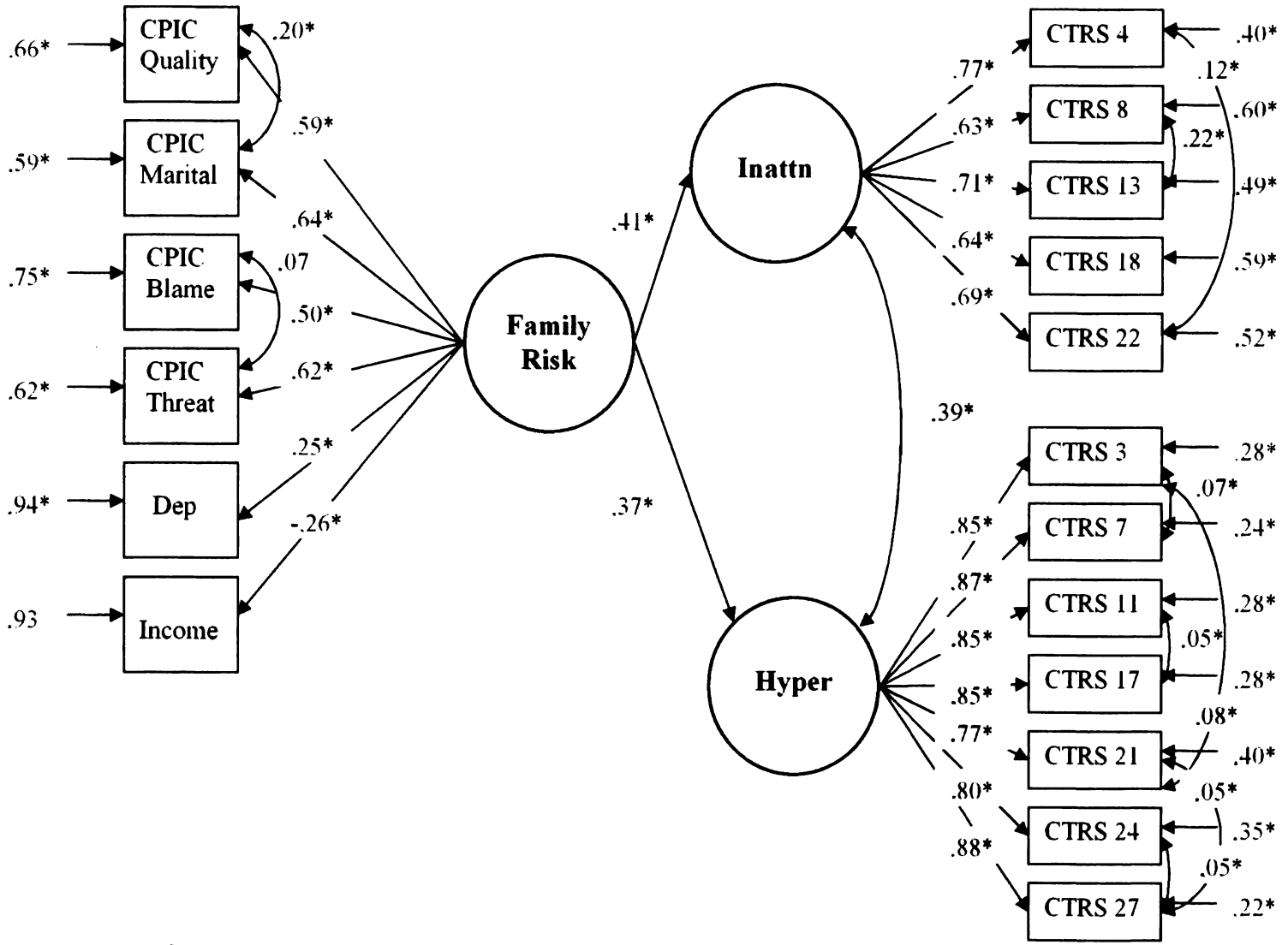

Figure 4. SEM of CTRS Items Regressed onto Family Risk (Hypothesis 2c)

Figure 5. Mediation Model using Clustering, without Bootstrap Method (Hypothesis 3a)

Notes to Figure 5: *Mplus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, Sx = symptoms, FSIQ = Full Scale Intelligence Quotient. The dependent variable was teacher-reported total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. In the current figure, age is not covaried, but results were essentially the same when it was covaried. χ^2 (23, $N = 394$) = 27.45, $p > .05$, CFI = .98, RMSEA = .02, SRMR = .02, and BIC = 9079.18.

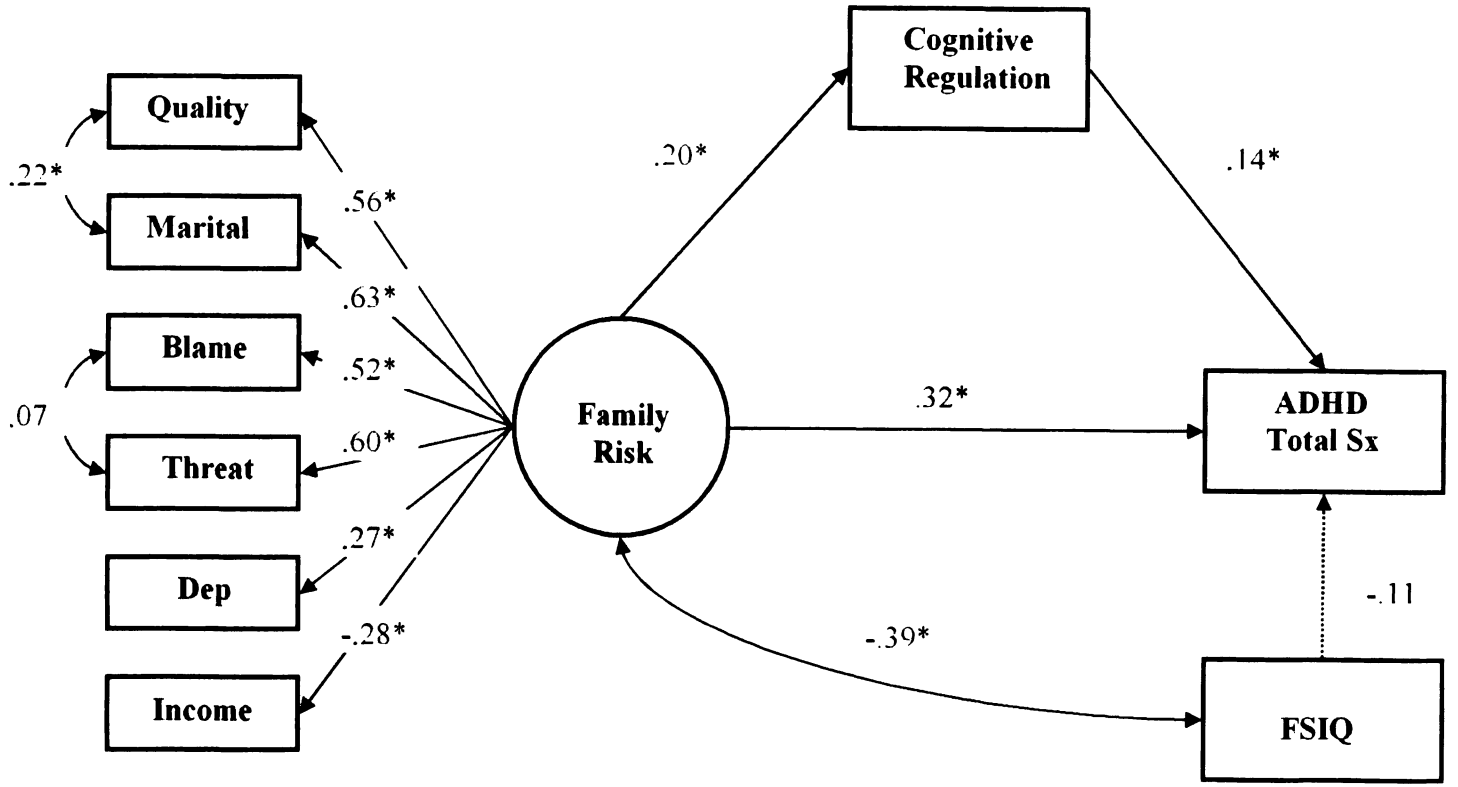

Figure 5. Mediation Model using Clustering, without Bootstrap Method (Hypothesis 3a)

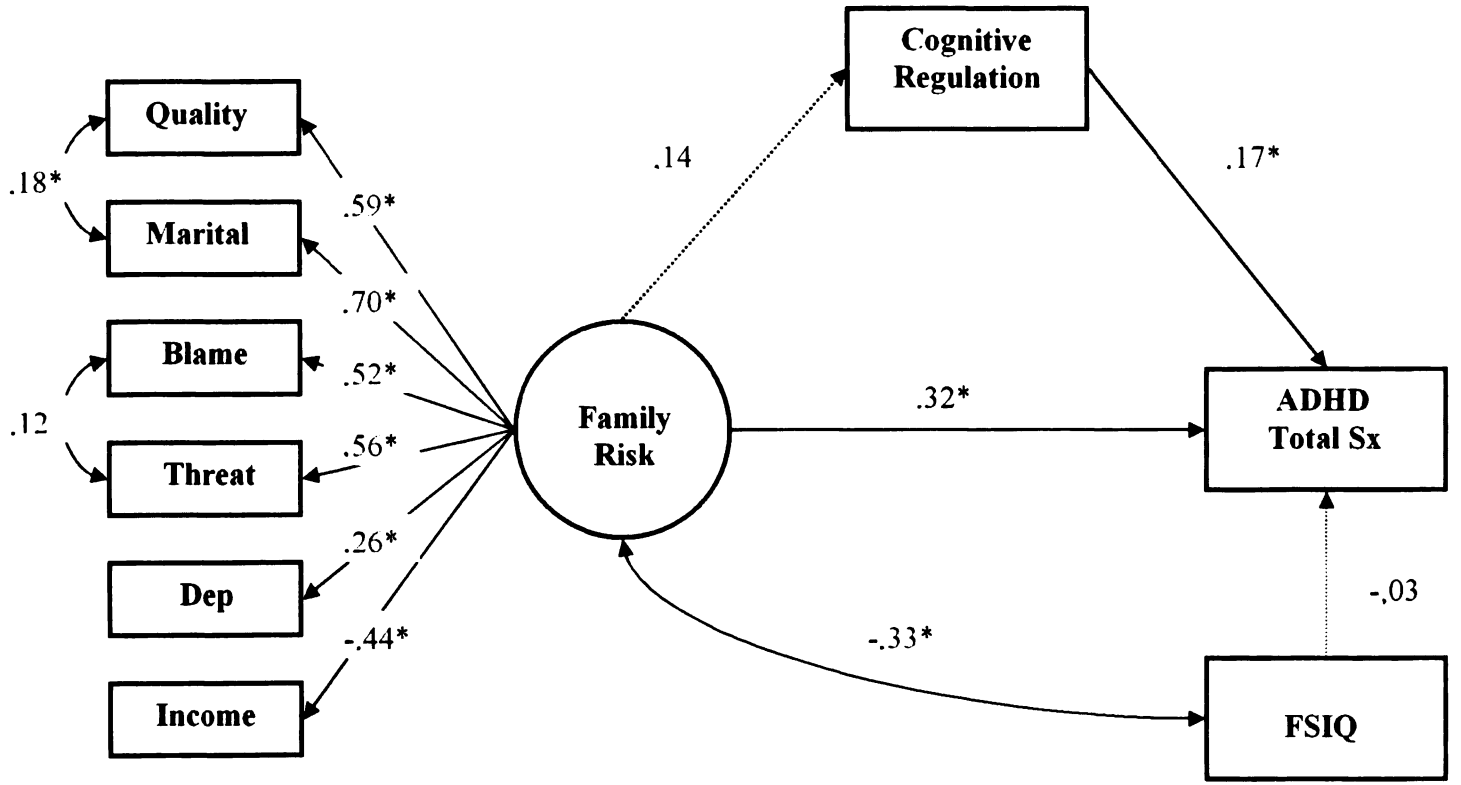

Figure 6. Mediation Model using Clustering, and Multigroup Age Analyses – Child Group (Hypothesis 3b)

Notes to Figure 6: M*plus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, Sx = symptoms, FSIQ = Full Scale Intelligence Quotient. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. χ^2 (56, N = 394) = 121.36, $p < .001$, CFI = .84, RMSEA = .08, SRMR = .07, and BIC = 8956.92.

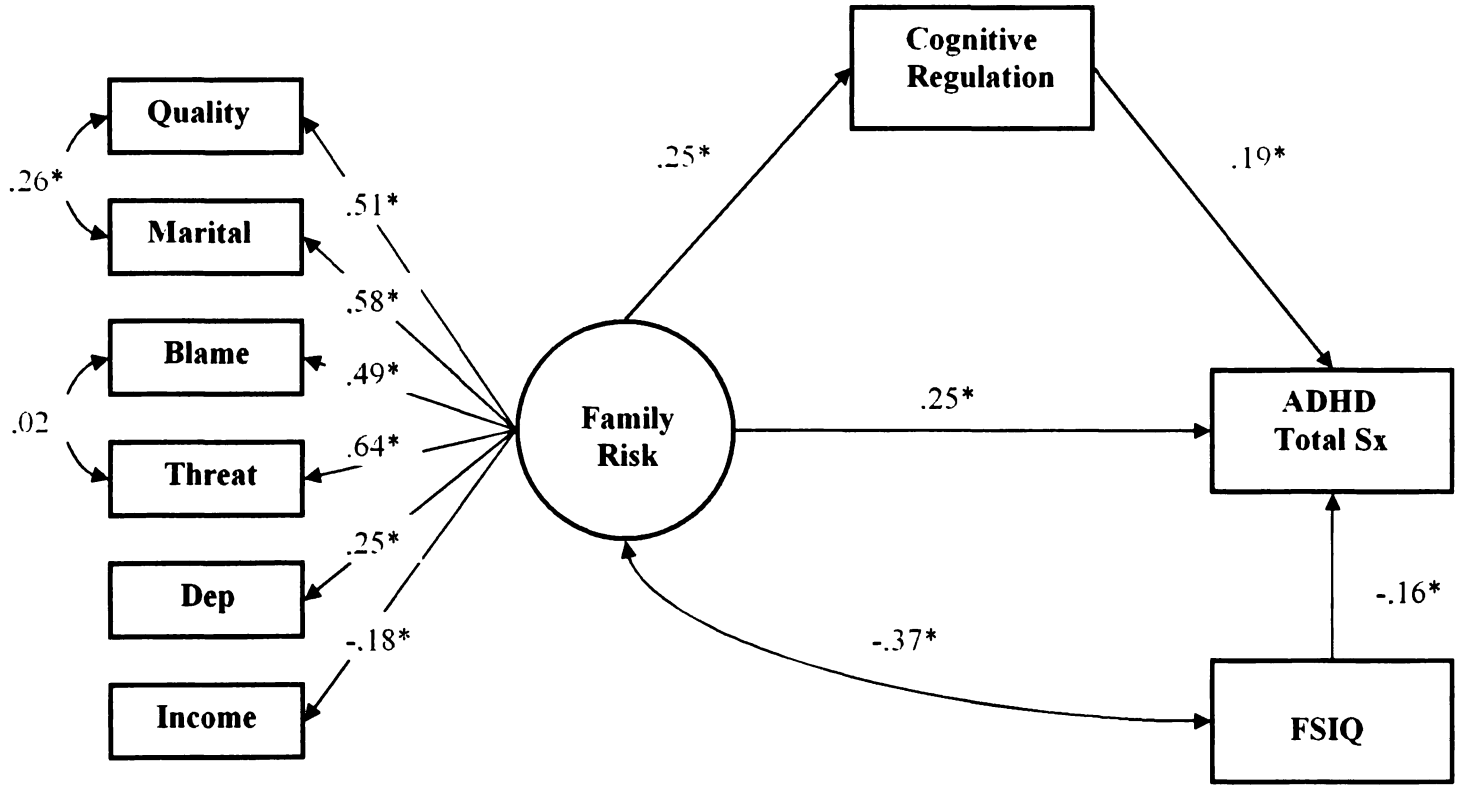

Figure 7. Mediation Model using Clustering, and Multigroup Age Analyses – Adolescent Group (Hypothesis 3b)

Notes to Figure 7: *Mplus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, Sx = symptoms, FSIQ = Full Scale Intelligence Quotient. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. $\chi^2(56, N = 394) = 121.36$, $p < .001$, CFI = .84, RMSEA = .08, SRMR = .07, and BIC = 8956.92.

Figure 8. Mediation Model using Clustering, and Multigroup Analyses – Caucasian Group (Hypothesis 3c)

Notes to Figure 8: M*plus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, Sx = symptoms, FSIQ = Full Scale Intelligence Quotient, MEIM = Multigroup Ethnic Identity Measure. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. χ^2 (68, N = 394) = 123.33, $p < .001$, CFI = .87, RMSEA = .06, SRMR = .07, BIC = 9947.08.

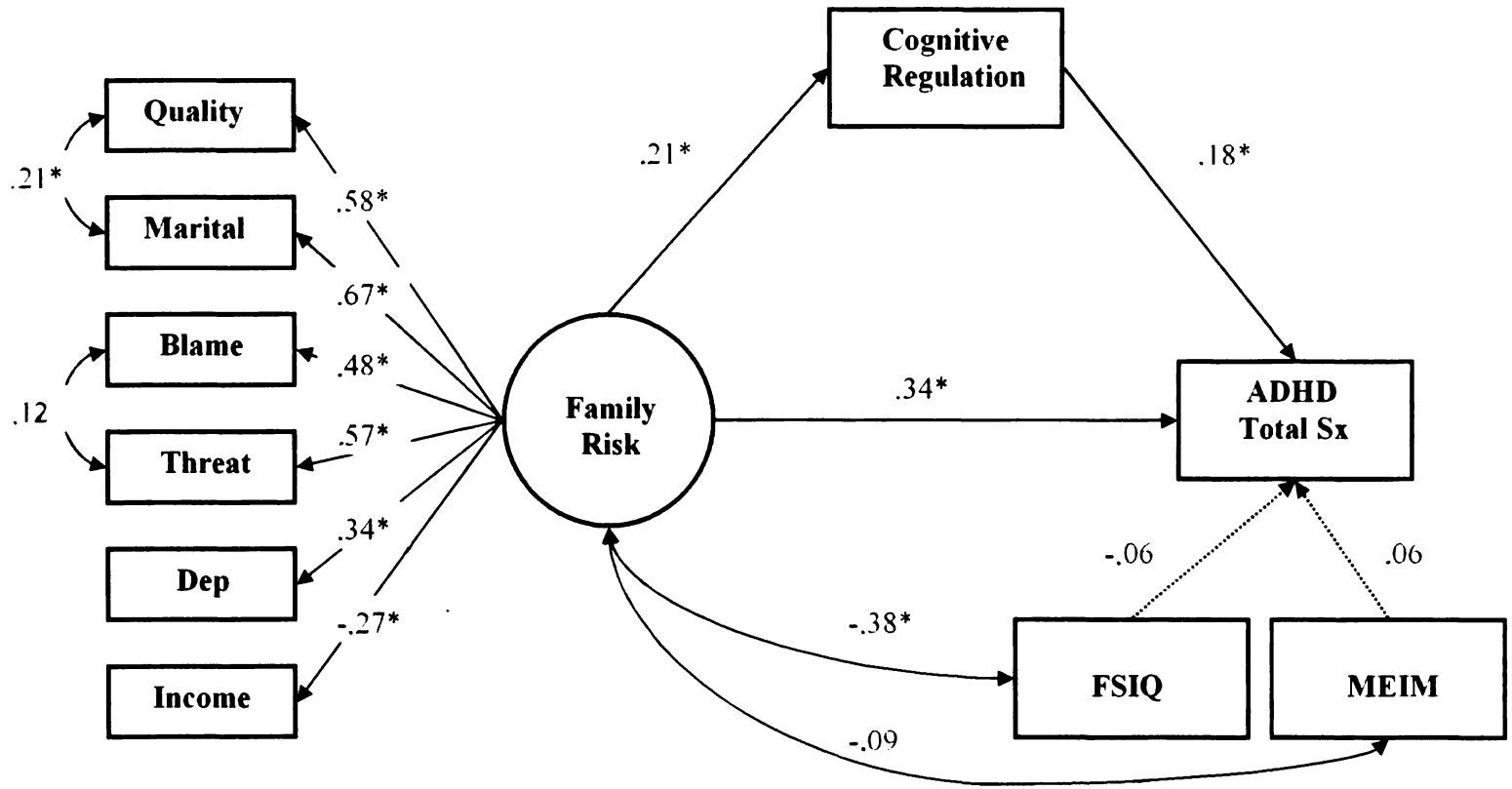

Figure 8. Mediation Model using Clustering, and Multigroup Analyses – Caucasian Group (Hypothesis 3c)

Figure 9. Mediation Model using Clustering, and Multigroup Analyses – Ethnic Minority Group (Hypothesis 3c)

Notes to Figure 9: M*plus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, Sx = symptoms, FSIQ = Full Scale Intelligence Quotient, MEIM = Multigroup Ethnic Identity Measure. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. χ^2 (68, N = 394) = 123.33, $p < .001$, CFI = .87, RMSEA = .06, SRMR = .07, BIC = 9947.08.

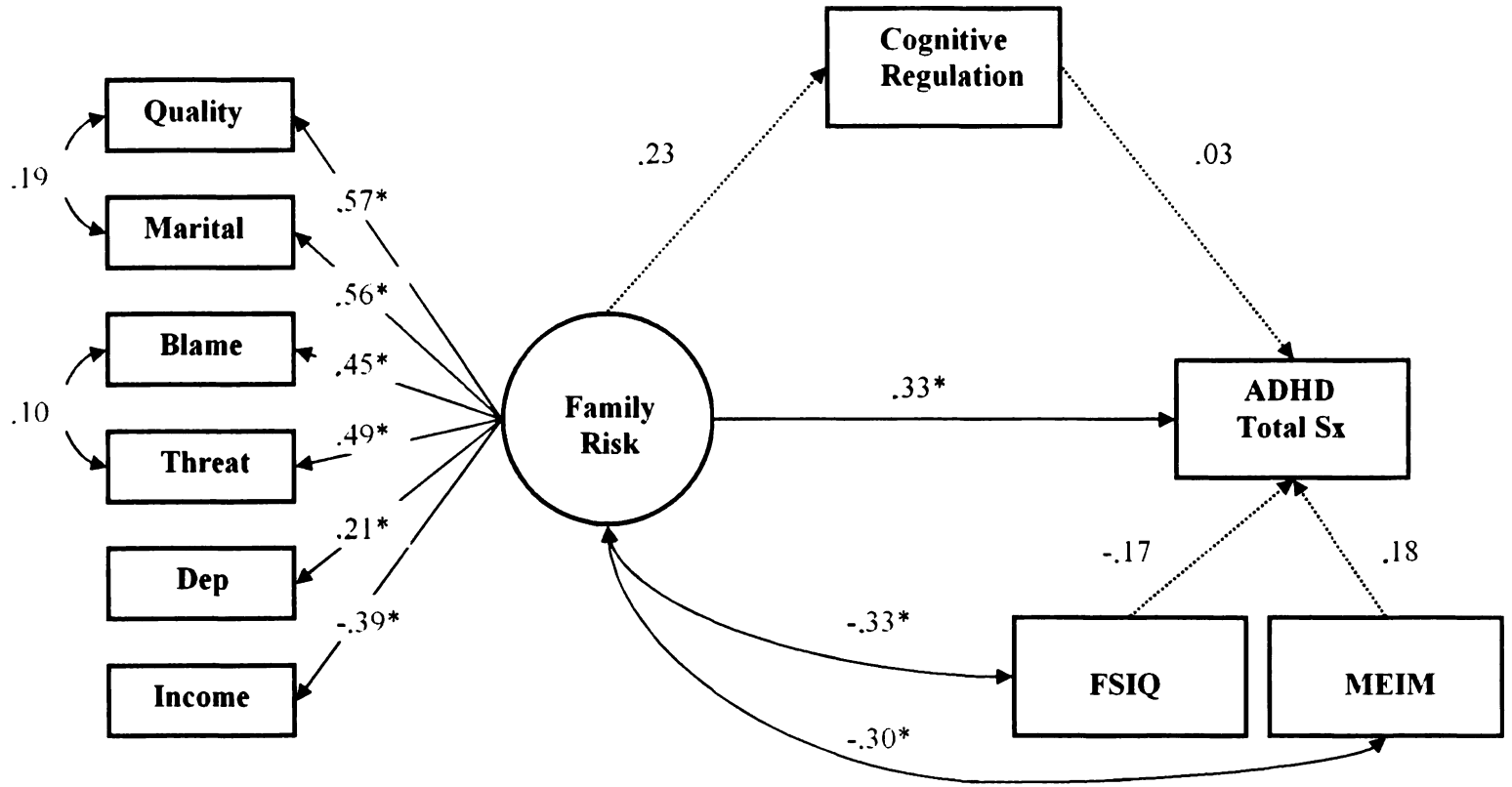

Figure 9. Mediation Model using Clustering, and Multigroup Analyses – Ethnic Minority Group (Hypothesis 3c)

Figure 10. ADHD Mediation of Family Risk and Adaptive Functioning (Hypothesis 4)

Notes to Figure 10: *Mplus* StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, ADHD Total Sx = teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. The dependent variable was a composite adaptive functioning score comprised by a mean of the clinician-reported Global Assessment of Functioning score and parent-reported total competency score on the Child Behavior Checklist. * $p < .05$. Non-significant paths are further indicated by a dashed line. The correlations on the far left side of the model are correlations among the Family Risk indicators, not among their residuals. In the current figure, age is not covaried, but results were essentially the same when it was covaried. $\chi^2 (17, N = 394) = 29.72, p < .05$, CFI = .97, RMSEA = .04, and SRMR = .04.

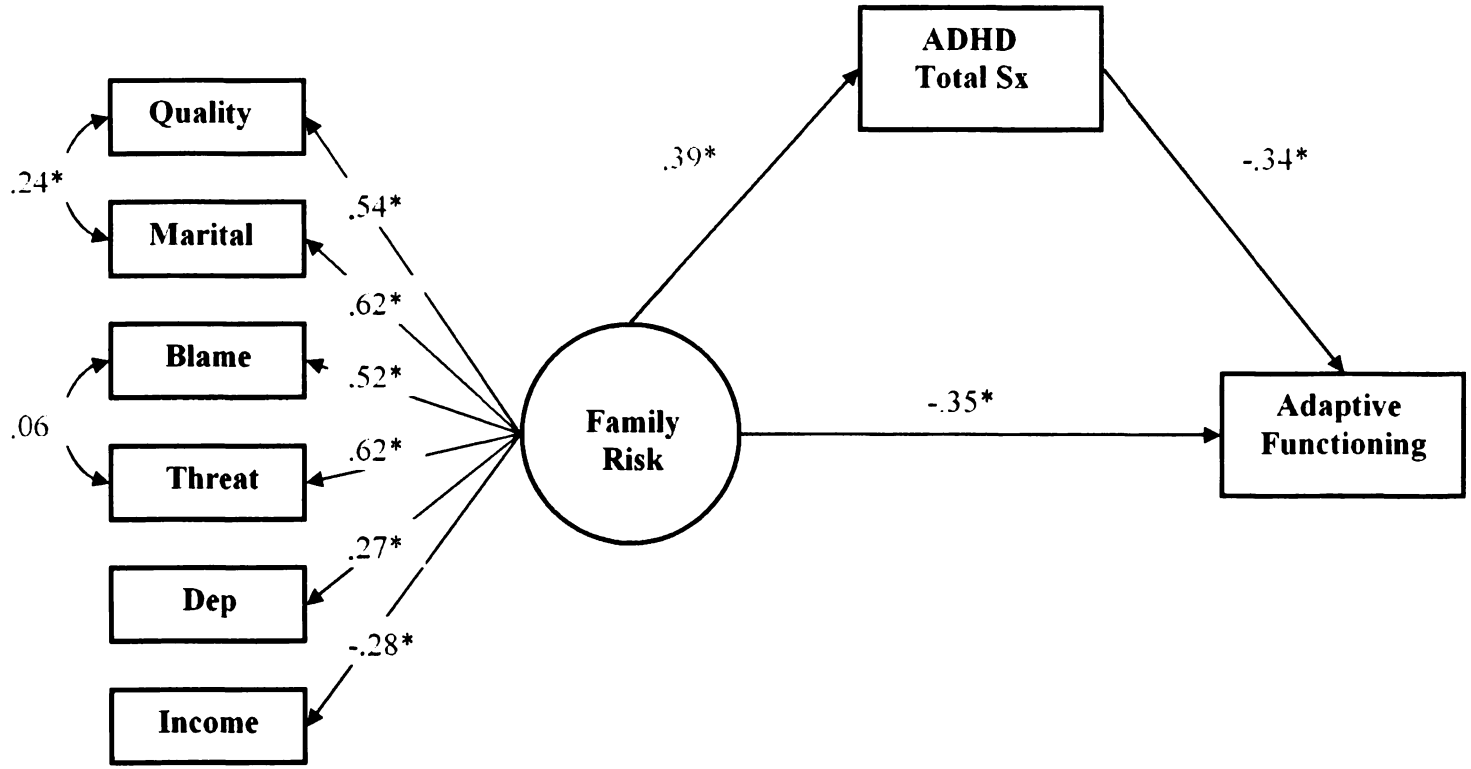

Figure 10. ADHD Mediation of Family Risk and Adaptive Functioning (Hypothesis 4)

APPENDIX B

Family Environment Scale Analyses

Table B1. Family Environment Scale EFA 1-factor Solution, 10 Subscales

FES Subscale	Factor 1
Cohesion	.605
Expressiveness	.367
Conflict	.120
Independence	.583
Achievement	.391
Intellectual-Cultural	.542
Active-Recreational	.572
Moral-Religious	.507
Organizational	.677
Control	.554

Notes to Table B1: M*plus* Analysis using FIML imputation, Maximum Likelihood extraction with Promax rotation; χ^2 (35, N = 374) = 200.41, $p < .001$, RMSEA = .11, RMR = .08.

APPENDIX B (cont'd)

Table B2. Family Environment Scale EFA 2-factor Solution, 10 Subscales

FES Subscale	Factor 1	Factor 2
Cohesion	.635	
Expressiveness	.527	
Conflict		.347
Independence	.463	
Achievement		.268
Intellectual-Cultural	.751	
Active-Recreational	.583	
Moral-Religious	.355	
Organizational	.447	
Control		.967

Notes to Table B2: M*plus* Analysis using FIML imputation, Maximum Likelihood extraction with Promax rotation; 2 factors, $r = .480$; $\chi^2 (26, N = 374) = 88.20, p < .001$, RMSEA = .08, RMR = .05.

APPENDIX B (cont'd)

Table B3. Family Environment Scale EFA 4-factor Solution, 10 Subscales

FES Subscale	Factor 1	Factor 2	Factor 3	Factor 4
Cohesion				.655
Expressiveness				.312
Conflict			-1.693	
Independence	.363			
Achievement	.267			
Intellectual-Cultural		.988		
Active-Recreational				.456
Moral-Religious	.295			
Organizational				.613
Control	.892			

Notes to Table B3: M*plus* Analysis using FIML imputation, Maximum Likelihood extraction with Promax rotation; 4 factors, $r < .68$; $\chi^2 (11, N = 374) = 13.02, p = .29$, RMSEA = .02, RMR = .02.

APPENDIX B (cont'd)

Table B4. Family Environment Scale EFA 2-factor Solution, 6 Subscales using FIML Imputation

FES Subscale	Factor 1	Factor 2
Control	.860	
Organizational	.580	
Independence	.415	
Moral-Religious	.425	
Intellectual-Cultural		.807
Expressiveness		.541

Notes to Table B4: *Mplus* Analysis using FIML imputation, Maximum Likelihood extraction with Promax rotation; 2 factors, $r = .506$; $\chi^2 (4, N = 374) = 6.02, p < .19$, RMSEA = .04, RMR = .02.

Table B5. Family Environment Scale EFA 2-factor Solution, 6 Subscales using Raw Data

FES Subscale	Factor 1	Factor 2
Control	.756	
Organizational	.641	
Independence	.577	
Moral-Religious	.500	
Intellectual-Cultural		.777
Expressiveness		.500

Notes to Table B5: SPSS Analysis using Raw Data, Maximum Likelihood extraction with Promax rotation; 2 factors accounted for 60.25% of the variance, $r = .447$, $\alpha = .71$; $\chi^2 (4, N = 374) = 5.94, p < .20$.

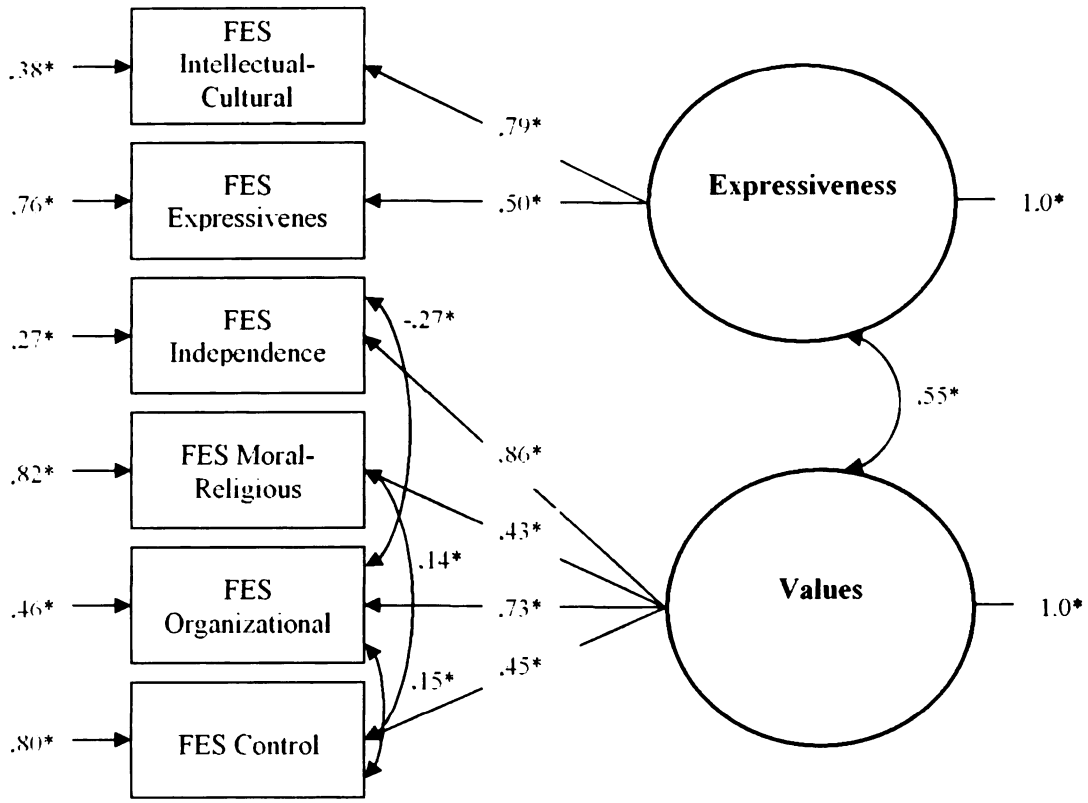

Figure B1. CFA of FES 2-Factor Solution with 6 Subscales

Notes to Figure B1: *Mplus* StdYX values were reported herein. These values are standardized based on the variances of the latent and observed variables to show the amount of change in Y per standard deviation unit of X. FES Intellectual-Cultural and FES Independence were fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. FES = Family Environment Scale. * $p < .05$. χ^2 (5, N = 374) = 10.84, $p > .05$, CFI = .99, RMSEA = .06, RMR = .03.

APPENDIX C

CPIC Factor Structure

Table C1. CPIC Factor Structure Validated by Nigg et al. (under review)

CPIC Item #	Conflict Quality	Marital Unhappiness	Self Blame	Perceived Threat
1	.465			
12	.765			
26	.747			
30	.787			
35	.809			
2		.587		
11		.733		
14		.612		
29		.711		
37		.456		
42		.404		
46		.766		
3			.622	
8			.347	
16			.722	
19			.850	
25			.591	
28			.719	
36			.688	
40			.452	
47			.350	
6				.663
13				.535
15				.767
23				.900
44				.625

Notes to Table C1: Maximum likelihood extraction with Promax rotation. 4 factors accounted for 52.59% of the variance, $\alpha = .89$. The confirmatory analysis conducted by Nigg et al. (under review) resulted in the following fit indices for the 4-factor solution outlined above: $\chi^2(293, N = 1190) = 1389, p > .05$, CFI = .935, RMSEA = .056.

APPENDIX D

Alternative Regression Analyses for Hypothesis 1a

Table D1. Teacher-rated ADHD Symptoms Regressed Onto Predictors of Risk, With ODD & CD in Step 2

		R^2	*B*	*SE B*	β
Step 1	Developmental History	.004	.119	.123	.067
Step 2	Developmental History	.113	.078	.118	.044
	ODD Sx		.227	.077	.231**
	CD Sx		.135	.089	.116
	Parent 1 Current ADHD Sx		.039	.079	.033
	Parent 2 Current ADHD Sx		.070	.070	.067
Step 3	Developmental History	.198	.088	.116	.049
	ODD Sx		.203	.076	.206**
	CD Sx		.062	.089	.053
	Parent 1 Current ADHD Sx		.000	.092	.000
	Parent 2 Current ADHD Sx		.057	.070	.055
	FES Values		-.121	.082	-.134
	FES Expressiveness		.074	.099	.071
	CPIC Conflict Quality		.045	.085	.045
	CPIC Marital Unhappiness		.032	.087	.031
	CPIC Self Blame		.149	.073	.143*
	CPIC Threat		.126	.077	.121
	Maternal Depression		.018	.075	.019
	Income		-.085	.057	-.101

Notes to Table D1: Sx = symptoms, ODD = Oppositional Defiant Disorder, CD = Conduct Disorder, CPIC = Children's Perception of Interparental Conflict Scale. * $p < .05$, ** $p < .01$. Step 3 brought a significant gain in r^2 ($\Delta r^2 = .085$, $p = .010$).

APPENDIX D (cont'd)

Table D2. Teacher-rated ADHD Symptoms Regressed Onto Predictors of Risk, Without ODD & CD

		R^2	*B*	*SE B*	β
Step 1	Developmental History	.005	.127	.122	.071
Step 2	Developmental History	.024	.108	.123	.060
	Parent 1 Current ADHD Sx		.112	.081	.095
	Parent 2 Current ADHD Sx		.093	.072	.089
Step 3	Developmental History	.147	.111	.118	.062
	Parent 1 Current ADHD Sx		.035	.093	.030
	Parent 2 Current ADHD Sx		.078	.071	.074
	FES Values		-.103	.082	-.114
	FES Expressiveness		.047	.099	.045
	CPIC Conflict Quality		.021	.086	.021
	CPIC Marital Unhappiness		.051	.088	.051
	CPIC Self Blame		.162	.073	.156*
	CPIC Threat		.158	.077	.152*
	Maternal Depression		.043	.076	.045
	Income		-.112	.058	-.134

Notes to Table D2: Sx = symptoms, ODD = Oppositional Defiant Disorder, CD = Conduct Disorder, CPIC = Children's Perception of Interparental Conflict Scale. * $p < .05$. Step 3 brought a significant gain in r^2 ($\Delta r^2 = .123$, $p < .001$).

APPENDIX E

Bootstrapping Method (Hypothesis 3a)

A path analysis was conducted in Mplus using bootstrapped standard errors for the indirect effects. The model tested whether the relationship between family risk and ADHD was partially mediated by cognitive regulation. Bootstrapped standard errors were computed using 1000 draws. Bootstrapping is a new method (Shrout & Bolger, 2002), and at this time statistical software does not allow for clustering of nested data while executing the bootstrap option, therefore clustering was not used in this model.

The model, shown below, provided a good overall fit: χ^2 (25, N = 394) = 34.18, p > .05, CFI = .97, RMSEA = .04, and SRMR = .05. The indirect effect for this model (StdYX = .028) was significant at p > .05. Thus, family risk predicted poorer regulation on cognitive tasks, and the relationship between family risk and ADHD was partially mediated by cognitive regulation.

The Mplus bootstrap option was used to prevent Type 2 error; however, this is of less concern in these data given the significant effect. Therefore, the cluster feature was used in place of the bootstrap option due to the necessity of clustering the nested family data.

Figure E1. Mediation using Bootstrap Method, without Clustering

Notes to Figure E1: Mplus StdYX values were reported herein. CPIC Quality was fixed at 1; however this is not apparent in the figure, as the StdYX value has been provided. Dep = Maternal Depression, FSIQ = Full Scale Intelligence Quotient. The dependent variable was teacher-rated total ADHD symptoms on the Conners' Teacher Rating Scale. * $p < .05$.

Appendix E (cont'd)

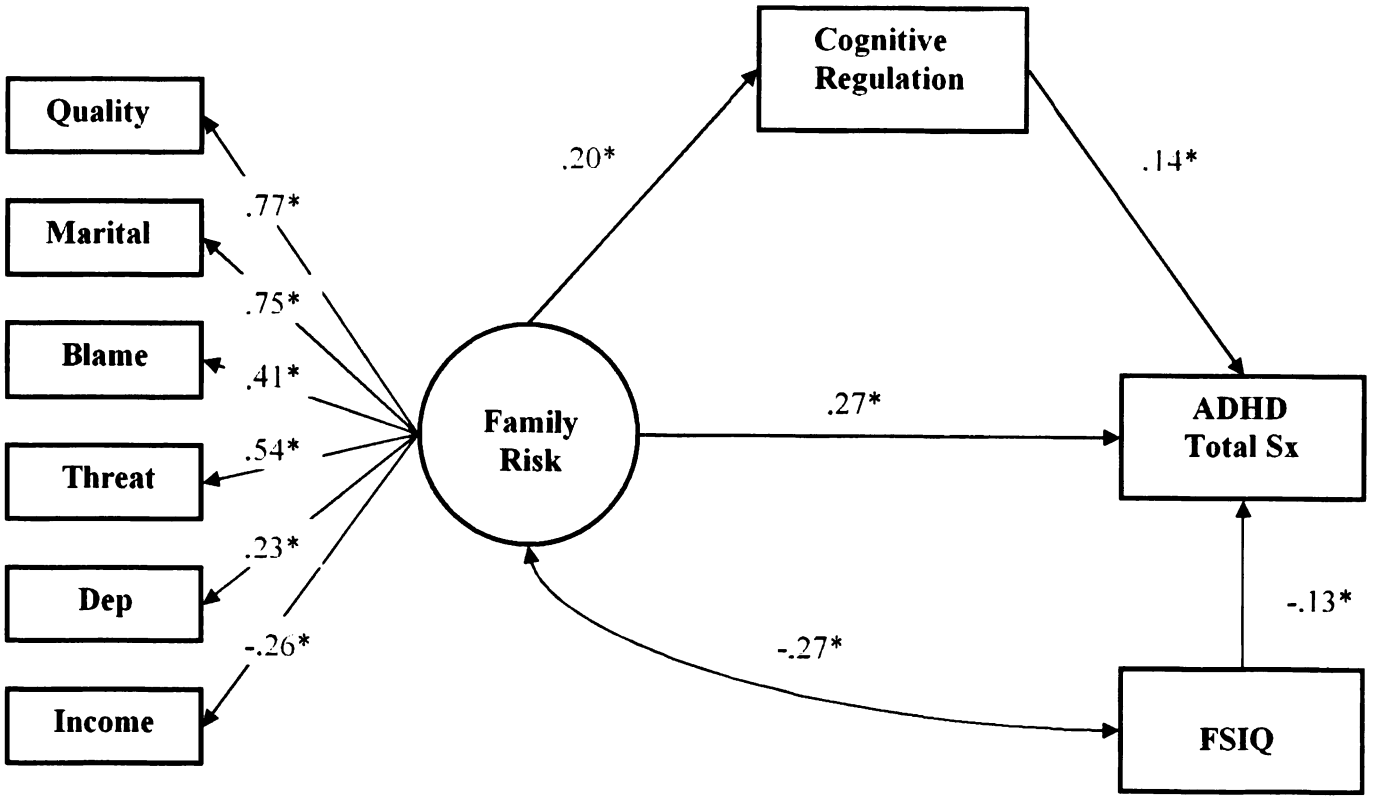

Figure E1. Mediation using Bootstrap Method, without Clustering

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