ABSTRACT

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Parental psychopathology has consistently emerged as an important risk factor for negative developmental outcomes in children with ADHD; yet, this literature has focused almost exclusively on mothers. The few studies that do focus on fathers have identified significant associations between paternal psychopathology and child conduct problems (CP). This study sought to replicate these findings among families of children with ADHD and to extend prior research by considering paternal involvement as a potential moderator. Direct relationships between paternal psychopathology and child CP were not found. However, paternal involvement moderated the relationship between paternal ADHD and child CP. For involved fathers, these two variables were positively correlated, which suggests that children whose fathers are both involved and display psychopathology may be at higher risk for concurrent CP.

ASSOCIATIONS BETWEEN PATERNAL PSYCHOPATHOLOGY AND CONDUCT PROBLEMS IN CHILDREN WITH ADHD.

By

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Dedication

To my parents who have always encouraged me and to all of the fathers who put their all into parenting day in and day out.

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Table of Contents

List of Tables

List of Figures

Introduction

Theoretical Framework

Within the child psychopathology literature, there is no single overarching theory to explain the body of relevant findings. Belky's (1984) process model of parenting suggests that the etiology of parenting behavior is rooted in three basic sources: parental contributions (e.g., personality and psychopathology), child contributions (e.g., temperament and psychopathology), and contextual factors (e.g., parental marital relationship and socioeconomic status). Another theoretical model more specific to parent and child psychopathology is Patterson's (1982) reciprocal transactional model in which child externalizing behavior and parental psychopathology exert reciprocal influences on one another whereby parents of children with ADHD may suffer from negative psychosocial wellbeing in part as a result of negative child behavior, which may contribute to the use of maladaptive parenting practices, and exacerbate child behavior problems (Patterson, 1982). In general, these models from the developmental and clinical literatures, respectively, fit with a developmental psychopathology perspective of child behavior problems, which refers to the general framework for understanding both normal and maladaptive development (Mash & Dozois, 2003). With this theoretical background in mind, the most recent literature related to paternal psychopathology and conduct problems in children with ADHD is discussed.

ADHD in Children and Related Risks

Attention-deficit/hyperactivity disorder (ADHD) is among the most commonly diagnosed childhood psychological disorders, affecting between four and

six percent of school-age children (*DSM-IV-TR*; American Psychiatric Association, 2000; Nair, Ehimare, Beitman, Nair, & Lavin, 2006). Characterized by symptoms associated with hyperactivity, impulsivity, and inattention, children with ADHD experience significant functional impairment across various life areas (APA, 2000). For instance, they often have more conflict with parents and siblings, have trouble succeeding in the classroom, and face unique challenges in maintaining social relationships (Coie & Dodge, 1998; Fischer, 1990; Mikami & Pfiffner, 2008; Zentall, 2007). Additionally, these children tend to be at higher risk for serious maladaptive outcomes later in development, such as substance abuse, delinquency, and chronic criminality (Burke, Loeber, & Lahey, 2001).

Conduct Disorder (CD) has been shown to moderate the development of these more serious outcomes among children with ADHD (Elkins, McGue, & Iacono, 2007; Foley, Carlton, & Howell, 1996). Because CD is both highly comorbid with ADHD and associated with the most negative developmental outcomes, it has been the focus of much recent research. Indeed, community samples, CD has been shown to co-occur with ADHD at rates between 2.6 percent (Romano, Tremblay, Vitarro, Zoccolillo, Pagani, 2005) and 17.1 percent (Bird, Gould, & Staghezza-Jaramillo, 1994). Moreover, some clinical samples have found comorbidity rates as high as 52.4 percent (Jensen, Martin & Cantwell, 1997). CD is characterized by consistent behaviors in any of the following categories: aggression to people and animals, destruction of property, deceitfulness or theft, and serious violations of rules (APA, 2000). Oppositional Defiant Disorder (ODD) is also highly comorbid with ADHD, at rates between 36.5 percent (Yang, Wang, Qian, Biederman, & Faraone, 2004) and 65

percent in clinical samples (Biederman et al., 1996b). ODD is characterized by a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures (APA, 2000). Symptoms of CD and ODD are often collectively referred to as "conduct problems" (CP). Though some youth develop CP in later adolescence, those who develop such symptoms in childhood are more likely to have comorbid ADHD (Lahey et al., 1998). Additionally, children with early-onset CP are more likely to go on to exhibit severe antisocial behavior and psychopathic characteristics in adulthood (Henry, Caspi, Moffitt, & Silva, 1996; Lynam, 1996; Lynam, 1998). Thus, understanding the various environmental factors associated with the presence of CP is of utmost clinical and public health significance.

Parental characteristics have emerged as perhaps the most significant environmental factors associated with the development of child CP. Undeniably, parents are crucial in managing the impairments of their children with ADHD and in protecting against the development of more serious outcomes (Johnston & Mash, 2001). In fact, the most salient predictors of such outcomes in children with ADHD, including the development of CP, are the presence of parental psychopathology (specifically, maternal depression) and the degree of early positive parenting (Chronis et al., 2007).

Although similar parental factors have been shown to be predictive of negative outcomes among normative child samples, researchers have emphasized the importance of studying these relationships specifically in children with ADHD. As reviewed hererin, children with ADHD are more likely to have both mothers and fathers with psychopathology (Chronis, Lahey, Pelham, Kipp, Baumann, & Lee,

2003). They are also at significantly greater risk for CP (Jensen et al., 1997; Biederman et al., 1996b) and for developing such outcomes as substance use and criminality when comorbid CP are present (Elkins et al., 2007; Foley et al., 1996). Thus, children with ADHD have been identified as a high-risk population in need of continued study, with a focus on risk factors (e.g. parental factors) for those outcomes which have consistently emerged as societal concerns for public welfare.

Psychopathology in Parents: Associations with Child ADHD and Behavior Problems

Evidence from cross-sectional studies of both parents of children with ADHD and from studies of the offspring of adults with psychopathology has shown that the presence of parental psychopathology is significantly associated with the presence of child ADHD (Chronis et al., 2003; Cummings, Keller, & Davies, 2005; Fischer, 1990; Johnston & Mash, 2001). Furthermore, developmental studies have established that the presence of parental psychopathology influences the later development of CP among children in general (Loeber, Green, Lahey, Frick, & McBurnett, 2000) and among children with ADHD in particular (Chronis et al., 2007). In fact, among children with ADHD, this longitudinal relationship has been established *independent* of differences in socioeconomic status (Chronis et al., 2007; Nigg & Hinshaw, 1998) and maternal parenting behavior (Chronis et al., 2007; Frick, Lahey, Loeber, Stouthamer-Loeber, Christ, & Hanson, 1992). Indeed, various models of parenting have suggested that parents with a history of psychopathology are more likely to expose their children to maladaptive cognitions, behavior, and affect, which likely contribute to these negative outcomes (Jaffe, Belsky, Harrington, Caspi, & Moffitt, 2006).

Maternal psychopathology.

One of the most highly-prevalent and highly-researched disorders among mothers of children with ADHD is depression. Within samples of children with ADHD, approximately forty percent of mothers have suffered from a major depressive disorder at some point in their lives (Chronis et al., 2003). Cross-sectional research with a community sample also posits that even sub-clinical levels of depressive symptomatology in mothers may be associated with negative psychosocial functioning of their children as well as with higher likelihood of externalizing behavior problems within these children (Cummings et al., 2005).

Much like the research on maternal depression, the majority of previous research examining the relationship between psychopathology in parents and CP in children with ADHD has typically focused on the psychopathology of mothers and largely ignored fathers. This lack of knowledge about the relationship between paternal psychopathology and the developmental outcomes of children with ADHD leaves much to be learned in this area.

Paternal psychopathology.

There is a small literature that has linked the presence of psychopathology in fathers to negative psychosocial outcomes in their offspring, albeit among children without ADHD (Bronte-Tinkew, Moore, Matthews, & Carrano, 2007; Dietz, Jennings, Kelley, & Marshal, 2009; Kane & Garber, 2004; Kopp & Beauchaine, 2007; Mezulis, Hyde, & Clark, 2004). A meta-analysis reviewing 34 studies reported a significant cross-sectional relationship between paternal depression and the presence of both internalizing and externalizing psychopathology in their offspring,

with effect sizes of .24 and .19, respectively (Kane & Garber, 2004). For example, in a community sample of adolescents, lifetime paternal major depressive disorder (MDD) was significantly related to the presence of MDD in their 24-year-old offspring (Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005). Several studies have also established significant associations between child CP and paternal substance abuse (Frick et al., 1992), Antisocial Personality Disorder (ASPD; Frick et al., 1992), and alcohol dependence (although this final study was conducted with a boys-only sample; Loukas, Zucker, Fitzgerald, & Krull, 2003). In fact, some studies have found that children whose fathers have ASPD are more likely to display CP, regardless of maternal depression levels (Kopp & Beauchaine, 2007). The fact that these studies were conducted with community samples, however, limits our ability to generalize their findings to the population of children with ADHD. It is therefore crucial to examine the correlates of paternal psychopathology within this high-risk population.

Paternal psychopathology and children with ADHD.

As mentioned previously, despite the copious amount of literature on the relationship between various maternal disorders and the behavioral correlates of their children with ADHD, our knowledge of the relationship between paternal psychopathology and children with ADHD is quite limited. For example, Lahey et al. (1998) found that the presence of paternal antisocial behavior increases the likelihood of later CP in their children with ADHD. Nigg and Hinshaw (1998) found greater levels of Generalized Anxiety Disorder (GAD) in fathers of children with comorbid ADHD and CP, as compared with children without either diagnosis. Additionally, higher rates of covert antisocial behavior in children with ADHD were associated

with paternal history of substance abuse (Nigg & Hinshaw, 1998). Like many studies, however, girls were not included in the sample, and more research is needed to replicate this relationship with samples of both boys and girls with ADHD. Additionally, these studies did not examine other problems common among fathers within this population, such as paternal ADHD, depression, and alcohol abuse.

Combined maternal and paternal psychopathology.

Since several studies have shown that maternal and paternal psychopathology are each associated with negative child outcomes, questions regarding the possible additive effects of maternal and paternal psychopathology on child behavioral problems has become the focus of much recent research. Research conducted with normative child samples has also examined the concurrent and longitudinal associations between maternal and paternal psychopathology and child psychopathology. For instance, children who have two depressed parents are at higher risk for developing both internalizing and externalizing disorders than children who have only one depressed parent (Brennan, Hammen, Katz, & LeBroque, 2002; Mezulis et al., 2004). Dietz et al. (2009) found similar results in a clinical sample where maternal history of depression predicted later externalizing behavior problems in toddlers only when fathers also had psychopathology. Related research conducted with community samples has also shown that the presence of a healthy parent can effectively buffer the negative effects of psychopathology in one parent, namely the development of internalizing and externalizing disorders in their children (Connell & Goodman, 2002). These findings have not been replicated, however, in samples of children with ADHD, who are at higher risk for parental psychopathology.

Proposed Mediators of the Parental Psychopathology — Child CP Relationship

Much like the presence of a healthy parent has emerged as an important protective factor in the relationship between parental psychopathology and child psychosocial outcomes, several other variables have also been proposed to mediate this relationship. Genetic factors (e.g., Forero, Arboleda, Vasquez, & Arboleda, 2009; Thapar, Langley, Owen, & O'Donovan, 2007) and parenting (e.g., Coley & Medeiros, 2007; Elgar, McGrath, & Waschbusch, 2004) are two of the most highly-researched of these potential mediators.

Genetics.

Recent studies have suggested that a genetic predisposition for the development of psychopathology in children is well-established (Burt, 2009 for antisocial behavior; Hetterna et al., 2001 for anxiety disorders; Levinson, 2006 for depression; Thapar et al., 2007 for ADHD); yet, there remains much support for the unique contribution of a child's environment. Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi (2005), for instance, found that child antisocial behavior was significantly associated *only* with maternal depression that occurred after, and not before, the child was born. Similarly, maternal depression among either biological or adoptive mothers was found to be associated with disruptive behavior problems in their children (Tully, Iacono, & McGue, 2008). Environmental influences (especially, maternal parenting) also seem to significantly interact with genetics to predict child disruptive behavior (Lee et al., 2008). Such studies argue for the continued need to understand environmental correlates of conduct problems among children with ADHD.

Parenting.

Within the domain of environmental influences on child behavior problems, several researchers have examined the extent to which parenting, in particular, mediates the relationship between parental psychopathology and child outcomes. For instance, lax and inconsistent disciplining strategies among depressed mothers mediate the development of CP and other such negative behavioral outcomes in their offspring (Cunningham, Benness, & Siegel, 1988; Elgar et al., 2004; Patterson, 1982). Similarly, among mothers of children with ADHD, highly anxious mothers are more likely to display lower levels of warmth and positive involvement (Kashdan et al., 2004). Thus, it is crucial to consider aspects of parenting, such as involvement, when researching the relationship between parental psychopathology and child developmental outcomes.

Father Involvement: A Unique Contribution

A review of recent research has suggested convincingly that fathers make a very crucial and unique contribution to the psychosocial outcomes of their children (Lamb & Tamis-LeMonda, 2004). Underscoring the importance of studying fathers separately from mothers, the majority of recent cross-sectional and developmental research has uncovered differences in how mothers and fathers relate to their children (Lamb & Lewis, 2004; McBride, Dyer, Liu, Brown, & Hong, 2009). While mothers tend to focus on caretaking, fathers more often engage in play and recreation, although both parents tend to be involved in academic activities (Lewis & Lamb, 2003; Lamb & Lewis, 2004). Thus, mothers and fathers may play different roles in

shaping the developmental outcomes of their children (Chang, Schwartz, Dodge, & McBride-Chang, 2003; Deater-Deckard & Dodge, 1997).

Father involvement and child outcomes.

Recently, the specific relationship between father involvement and child outcomes has become the focus of much research (Lamb & Lewis, 2004; Lamb & Tamis-LeMonda, 2004; Lewis & Lamb, 2003). For example, higher levels of father involvement with their 7-year-old children has been found to predict lower levels of police contact at age 16 (Flouri & Buchanan, 2002). A review of studies of paternal involvement has further suggested that positive paternal involvement in particular results in positive psychosocial outcomes in children (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000). Consistent with this conclusion, a recent study of an adolescent, low-income community sample, found that higher levels of involvement by non-residential fathers were predictive of lower levels of offspring CP (Coley & Medeiros, 2007). Furthermore, a decrease in CP over time was observed among those adolescents who originally displayed high levels of CP when fathers were highly involved (Coley & Medeiros, 2007). Indeed, positive father involvement is predictive of positive outcomes even among samples of Chinese adolescent boys (Shek, 2005).

High levels of positive father involvement have also been found to attenuate the effects of maternal depressive symptoms on the development of child behavior problems (Chang, Halpern, & Kaufman, 2007). Although this study did not examine psychopathology in fathers, it suggests that father involvement is a potential buffer in the relationship between maternal psychopathology and child CP. Furthermore, father

involvement should be examined specifically within families of children with ADHD, as these families are at considerably greater risk for both child CP and parental psychopathology.

It should be noted that, within certain communities (e.g. African-American households), researchers have found no significant differences in psychosocial adjustment between children whose fathers are present and living in the home and those whose fathers are completely absent (Black, Dubowitz, & Starr, 1999). It has been suggested, however, that the mere report of fathers' presence or absence in the home may be insufficient for measuring this construct, and future researchers should utilize more detailed measures of father involvement.

Maladaptive father involvement.

Father involvement has also been studied with a focus on maladaptive paternal parenting and child outcomes. In one study, negative paternal parenting was found to be significantly associated with the presence of CP in clinical and comparison samples of adolescent boys from two-parent homes (DeKylen, Speltz, & Greenberg, 1998). Rogers, Wiener, Marton, & Tannock (2009) examined father involvement in families of children with ADHD and found that these fathers reported using more coercive parenting as compared to fathers of typically developing children. Indeed, several studies have also found that fathers with high levels of ADHD symptoms tended to report more lax and over reactive parenting with their children with ADHD than fathers without ADHD symptoms (Arnold, O'Leary, & Edwards, 1997; Harvey, Danforth, McKee, Ulaszek, & Friedman, 2003). Taken together, these findings suggest both that paternal involvement can be harmful for

children if fathers use negative parenting and that fathers with psychopathology tend to display more negative parenting. Therefore, one can surmise that high levels of psychopathology among involved fathers may be associated with negative child outcomes, in contrast to the positive effects of paternal involvement that have been noted in non-clinical samples. However, to our knowledge, no studies have examined associations between paternal psychopathology and involvement in relation to child behavioral outcomes (e.g., CD and ODD symptoms).

Limitations of Previous Research

Significant strides have been made in understanding the ways in which fathers contribute to their offspring's development. Much research remains to be done, however, to address the dearth of knowledge that still exists, particularly as relevant to fathers of children with ADHD. In particular, several limitations have been noted in previous research.

First, prior research on paternal psychopathology and child developmental outcomes has largely failed to include non-residential fathers (Cabrera et al., 2000). Considering the changing family structure and the elevated divorce rates in ADHD families (Barkley, Fischer, Edelbrock, & Smallish, 1991; Wymbs, Pelham, Molina, Gnagy, Wilson, & Greenhouse, 2008), examining only residential fathers of children with ADHD ignores important aspects of the child's environment. Furthermore, those studies that have included non-residential fathers (e.g. Arnold et al., 1997; Loukas et al., 2003; Nigg et al., 1998) have simply studied the presence or absence of fathers rather than the quality or level of their involvement, and several studies have established that studying variations of paternal involvement is meaningful (Chang et

al., 2007; Coley & Medeiros, 2007; Flouri & Buchanan, 2002). Thus, the relationship between father involvement and child psychosocial adjustment in the context of paternal psychopathology remains largely unknown.

Second, many samples within the ADHD literature have consisted exclusively of boys. The prevalence of ADHD is three times higher in boys than girls, and boys and girls with ADHD present with different patterns of impairment, comorbidities, different rates of school suspension, and are treated at different rates (Arnold, 1996; Bauermeister et al., 2007; Gaub & Carlson, 1997). Thus, findings from boys-only samples cannot be applied to girls with ADHD (APA, 2000; Nolan, Volpe, Gadow, & Sprafkin, 1999).

Third, most studies on father involvement and paternal psychopathology reviewed herein have not focused specifically on families of children with ADHD (e.g. Bronte-Tinkew et al., 2007; Connell & Goodman, 2002; Dietz et al., 2009; Flouri and Buchanan, 2002; Kane & Garber, 2004; Kopp et al., 2007; Loukas et al., 2003; Mezulis et al. 2004; Wilens et al., 2005), and therefore our ability to generalize their findings to this high-risk population is limited. In light of the many unique differences between the populations of children with and without ADHD, discussed previously, addressing this gap in the ADHD research is of utmost importance.

Lastly, most studies have also failed to gather information geared toward examining interactions between maternal and paternal psychopathology in relation to child adjustment. This is particularly important in families of children with ADHD, for whom rates of psychopathology is higher in both mothers and fathers (Chronis et al., 2007).

In sum, the limitations that have been discussed herein point to a need for research regarding psychology and involvement among fathers of children with ADHD. Future research should take care to ensure the following: (1) recruitment of a sample of families of children with ADHD, (2) recruitment of a sample of fathers with varying levels of involvement (including non-residential fathers), (3) inclusion of both boys and girls, and (4) consideration of both father involvement and maternal psychopathology as possible moderators of the paternal psychopathology - child CP relationship.

Present Study

Significance.

Though gaps in our knowledge of fathers have begun to be addressed, many questions remain unanswered that relate specifically to paternal psychosocial characteristics within families of children with ADHD. The proposed project sought to explore the extent to which psychopathology among fathers of children with ADHD was associated with concurrent CP in their offspring, and to consider the extent to which father involvement and maternal psychopathology each interacted with paternal psychopathology. Identifying correlates of CP within samples of children with ADHD is of critical importance given that this comorbidity is associated with the most serious developmental outcomes within this high-risk population.

This study was designed to address the limitations of previous research by utilizing a multi-method approach to the assessment of paternal psychopathology,

including both self-report and interview data. Moreover, this study aimed to include both boys and girls with ADHD, and a sample that is demographically diverse. Additionally, due to our recruitment procedures, many of these children come from families with at least mildly depressed mothers which is both common in families of children with ADHD and poses significant risks for negative child outcomes. We were therefore able to examine the interaction of maternal and paternal psychopathology and its association with child CP.

Aims and Hypotheses.

(1) The first aim of this study was to examine the relationship between paternal psychopathology and parent- and teacher-reported CP in their children. It was hypothesized that continuous levels of paternal psychopathology (e.g. Depression, ADHD, Alcohol Dependence, Non-Alcohol Substance Dependence, and ASPD) would be significantly and positively associated with concurrent child CP. This hypothesis was based on research findings that children with ADHD are more likely to develop CP when their fathers display antisocial behavior problems (Lahey et al., 1998) as well as other studies on unselected populations (Loukas et al., 2003; Kane & Garber, 2004). As reviewed previously, Lahey et al. (1998) did not examine level of father involvement in relation to paternal psychopathology. Since fathers with psychopathology have been shown to display more negative parenting (Arnold et al., 1997; Harvey et al., 2003), and negative paternal parenting has been found to be significantly associated with child behavior problems (DeKylen et al., 1998), father involvement was explored in our second aim.

(2) The second aim of this study was to examine whether paternal involvement moderated the relationship between paternal psychopathology and child CP. It was hypothesized that paternal involvement would significantly moderate the relationship between continuous measures of psychopathology and child CP such that children at highest risk for child CP would be those who fathers have higher levels of both involvement and psychopathology. This hypothesis was based on indirect evidence from several studies. First, Kim-Cohen et al. (2005) reported that maternal depression was associated with child antisocial behavior only when the depression occurred in the child's lifetime. This suggests that parental psychopathology may only be associated with child problem behavior when the child is exposed to the parent with psychopathology (i.e., this relationship is not purely the result of genetics). Also, studies have found that fathers with high levels of ADHD symptoms display more negative parenting, suggesting that these fathers' involvement with their children may be less optimal when he has psychopathology (Arnold et al., 1997; Harvey et al., 2003). Child outcomes were not examined in these studies on fathers; however, their findings serve as a basis for hypotheses proposing that the negative effects of paternal psychopathology on child CP may be reduced when the child has less contact with the father. Thus we hypothesized that paternal involvement would significantly interact with paternal psychopathology and child CP to predict variance in child CP. It was expected that low levels of both paternal involvement and paternal psychopathology would be associated with the lowest levels of child CP, while high levels of both paternal involvement paternal psychopathology would be associated with the highest levels of child CP.

(3) The third aim of this study was to examine whether the interaction between maternal and paternal psychopathology was related to child CP. It was hypothesized that the interaction of continuous measures of maternal and paternal psychopathology would be significantly associated with child CP. This hypothesis was based on findings that children with two depressed parents have the greatest likelihood for developing externalizing behaviors problems (Brennan et al., 2002; Conger, Ge, Elder, Lorenz, & Simons, 1994) and that the presence of a healthy parent may buffer the negative effects of having one parent with psychopathology (Connell & Goodman, 2002). Following these hypotheses, it was expected that the absence of psychopathology in both parents would be associated with the lowest levels of child CP, while the presence of psychopathology in both parents would be associated with the highest levels of child CP.

Method

Participants

Forty families for this study were originally recruited through fliers sent to local schools and pediatricians as well as advertisements in the local newspaper as part of three research studies examining the effects of an Integrated Treatment for Depressed Mothers of Children with ADHD (NIMH R34 MH073567-01; N=113), the Associations Between Maternal ADHD Symptoms and Parenting Behavior (NIMH R03 MH070666-1; N=70), and a Behavioral Homework Intervention for Middle School Students with ADHD (N=12).

To recruit for the current study, 195 mothers who were participants of these larger studies and who had previously agreed to be contacted for future research were

asked for permission to contact the biological father of the child who had participated in these studies. All mothers received at least three letters, three follow-up emails, and three follow-up phone calls. If permission to contact fathers was granted (N=49), fathers also received at least three letters, three follow-up emails, and three phone calls (see Figure 1 for recruitment procedures). These recruitment procedures resulted in a sample of 40 fathers from all three original studies. It should be noted that, because including non-residential fathers was a priority in this study, effort was often expended above and beyond the follow-up procedures described herein (i.e., additional phone calls, letters, and emails). Fathers were paid twenty-five dollars for completing the self-report questionnaires and twenty-five dollars for completing the telephone interview. This study was funded by a University of Maryland General Research Board (GRB) Summer Research Award.

Families included in this sample had children who: (1) met full DSM-IV criteria for ADHD according to parent and teacher reports and structured diagnostic interviews; (2) had an estimated IQ above 70, as assessed by the block design and vocabulary subtests of the Wechsler Intelligence Scale for Children, 3rd Ed. (Wechsler, 1991); and (3) were between the ages of 6 and 12. Families of children who met criteria for any pervasive developmental disorder were excluded. Additionally, mothers from the study examining an Integrated Treatment for Depressed Mothers of Children with ADHD were required to have elevated depressive symptoms (as determined by a score ≥ 10 on the Beck Depression Inventory). Because research has indicated a strong genetic basis for ADHD (Thapar et al., 2007), only biological fathers were eligible to participate in this study, in an

effort to control for the possible differential contributions of biological and non-biological fathers to the presence of CP in their children with ADHD (Swanson & Catsellanos, 2002). However, because children with ADHD tend to be more likely to have parents who are divorced or unmarried (Barkley et al., 1990; Wymbs et al., 2008), this study made a considerable attempt to recruit non-residential biological fathers in addition to residential fathers (reviewed above). Additional characteristics of the final sample (*N*=40) are included in Tables 1 and 2.

Procedure

Eligibility for the larger studies was determined through a screening phone call to the Maryland ADHD Program, upon which participants underwent a telephone screen and were sent a packet of parent and teacher measures to complete. Participants then completed a full diagnostic assessment to assess psychopathology in the child. For the current study, fathers completed a brief set of questionnaires as well as a brief telephone interview (described below) to further assess for the presence of current and past psychopathology. Questionnaires were sent by mail and returned by mail. Neither mothers nor fathers were required to travel to the University of Maryland for this part of the study. All assessments and interviews (including telephone interviews) were conducted by students in the doctoral program in clinical psychology at the University of Maryland under the supervision of Andrea Chronis-Tuscano, Ph.D.

Study Materials

Child Diagnostic Measures.

A diagnosis of ADHD was made according to DSM-IV criteria. The Disruptive Behavior Disorder (DBD) symptom checklist (Pelham, Gnagy, Greenslade, & Milich, 1992), which includes all DSM-IV symptoms of ADHD, ODD, and CD, was completed by the child's mother as well as the child's teacher. Symptoms endorsed as occurring "pretty much" or "very much" in either the school or home setting were considered clinically significant and were included in the symptom count. The DBD has demonstrated internal consistency of .96, .96, and .81 for the ADHD, ODD, and CD subscales, respectively.

In addition to the DBD, mothers were interviewed using the Schedule for Affective Disorders for School-Aged Children- Present and Lifetime Version (K-SADS-PL; Kaufman, Birmaher, Brent, Rao & Ryan, 1997). The K-SADS-PL is a semi-structured clinical interview assessing DSM-IV child/adolescent symptomatology for ADHD, ODD, CD, Separation Anxiety Disorder, Post-Traumatic Stress Disorder, Major Depression, Panic Disorder, Obsessive Compulsive Disorder, and Generalized Anxiety Disorder, which provides information about current and lifetime clinical diagnoses. The K-SADS-PL demonstrates strong psychometric properties of reliability (Ambrosini, 2000) and validity (Kaufman et al., 1997). On the K-SADS-PL, each symptom was rated on a three-point scale, ranging from 1 (not present) to 3 (threshold: definitely present), and symptoms were counted as clinically significant on this measure if the clinician rated it as a three. A symptom of ADHD, ODD, or CD was counted if it was rated as clinically significant on either the parent or teacher DBD or the KSADS.

The Children's Impairment Rating Scale (CIRS; Fabiano et al., 2006) was also completed by both the child's mother and teacher, as evidence for impairment in multiple settings (as required by DSM-IV for an ADHD diagnosis). The CIRS contains ratings of impairment on a 7-point scale across multiple domains, and it demonstrates strong psychometric properties of stability and cross-informant reliability (.54 to .76 and .64, respectively) as well as predictive validity (.38).

Maternal Psychopathology Screening.

All mothers (with the exception of the two mothers in the Homework Intervention study) completed the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) to assess depressive symptomatology. The BDI-II is a psychometrically sound, 21-item self-report instrument. Psychometric data indicate coefficient alphas of .92 for an outpatient sample and .93 for a college sample, demonstrating high reliability (Beck et al., 1996). For analyses examining the interaction of maternal psychopathology with paternal psychopathology, the presence of maternal psychopathology was examined as a continuous variable.

Paternal Psychopathology.

Fathers completed the Short Michigan Alcohol Screening Test (MAST-S; Seltzer, Vinokur, & Van Rooijen, 1975) to assess alcohol-related problems. The MAST-S is a 13-item self-report measure of alcohol consumption. The sensitivity of the 13-item MAST-S (about 0.70) in reference to a formal psychiatric interview is somewhat lower than the 25-item full MAST (0.83) (Rounsaville, Weissman, & Wilber, 1983), but this difference reflects only a ‘‘modest’’ loss of sensitivity and

specificity (Schuckit, Irwin, Howard, & Smith,1988). Internal consistency of the items within the MAST-S exceed 0.80, and the measure has been shown to be relatively valid as well (Seltzer et al., 1975). They also completed the Beck Depression Inventory-II (BDI-II; Beck et al., 1996) to assess depressive symptomatology.

Fathers were also administered the Mini International Neuropsychiatric Interview (M.I.N.I Plus; Sheehan et al., 1998) by telephone to further assess for the presence and extent of current and past psychopathology, specifically symptoms of ASPD, ADHD, alcohol dependence, and non-alcohol substance dependence. The M.I.N.I. demonstrates strong psychometric properties of reliability and validity (Lecrubier et al., 1997).

Paternal Involvement.

Fathers completed the Alabama Parenting Questionnaire (APQ; Shelton, Frick, & Wooton, 1996), a 42-item measure on which parents are asked to indicate the frequency with which they implement the following parenting practices: Corporal Punishment, Inconsistent Discipline, Poor Monitoring/Supervision, Involvement, and Positive Parenting. Internal consistency for all scales is moderate to high (Shelton et al., 1996), and test-retest reliability across a 3-year interval averages .65 (McMahon, Munson, & Spieker, 1997). For the purposes of this study, only the Involvement subscale was analyzed.

As an additional measure of father involvement, fathers completed the engagement/ activities section of the Early Head Start Father Interview (Cabrera, Ryan, Shannon, Brooks-Gunn, Vogel, & Raikes, 2004). The engagement/activities

section consists of items that ask fathers to report on how often they have done specific activities with the target child in the past month, such as "Attended a teacher conference at his/her school?," "Took him/her to visit relatives?," and "Played outside in the yard, a park, or a playground with him/her?" All four subscales of the original measure have demonstrated good internal consistency, with alphas ranging from .77 to .86 (Cabrera et al., 2004). In collaboration with its developer, this measure was modified for the present study to include only those items that are relevant to children between the ages of six through twelve, and additional items were added to address activities that are specific to children with ADHD (e.g., "Attended a parent-teacher conference at his/her school"). This modified Father Involvement Questionnaire also demonstrated good internal consistency, with an alpha of .93. Additionally, the measure was highly correlated with the APQ Involvement scale within our sample (r = .62).

Finally, fathers completed a brief demographic questionnaire to assess their level of satisfaction with their amount of involvement and hours they spend at work per week.

Results

Preliminary Analyses

A first set of preliminary analyses was conducted to determine if the final sample for this study (N=40) differed significantly on a variety of demographic and clinical characteristics from those who chose not to participate (N=155). Independent samples t-tests were conducted for the following continuous variables: family income level, maternal education level, maternal depressive symptomatology (according to

BDI-II), and child CD/ODD symptoms. Chi-square analyses were conducted for the following dichotomous variables: paternal residential status and paternal history of psychological diagnosis/treatment (per maternal report on the initial application form). There was a significant difference between the two groups, such that those who participated had a higher mean family income than those who did not participate ($t(202) = -2.19$, $p < .05$). No significant effects were found for maternal education level ($t(202) = 1.65$, $p > .05$), maternal depressive symptomatology ($t(202) = .26$, $p > .05$), or child CD/ODD symptoms ($t(202) = 1.76$, $p > .05$). In terms of the percentage of non-residential fathers in each sample, the group that participated had a much lower percentage than those who did not participate ($\chi^2(1, N = 202) = 10.26$, $p < .01$). No differences between groups emerged regarding the percentage of fathers who had ever been diagnosed with or treated for a psychological disorder ($\chi^2(1, N = 202) = 0.00$, $p > .05$).

The primary outcome variable selected for this study was the total number of child symptoms of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD), as reported by parents and/or teachers using the "or rule". This decision to use the sum of child ODD and CD symptoms was based on a lack of variability of CD symptoms within this sample ($M = 0.95$, $SD = 1.03$, $Range = 4.0$). The combination of ODD and CD symptoms ($M = 4.52$, $SD = 2.89$, $Range = 11.0$) provided adequate variability to examine the proposed hypotheses. Precedent for combining ODD and CD symptoms to represent a larger category of "disruptive behavior problems" or "conduct problems (CP)" can be found in several studies (e.g. Christiansen et al., 2008; D'Onofrio, Goodnight, Van Hulle, Waldman, Rodgers, & Rathouz, 2009

Ingoldsby, Kohl, McMahon, Lengua, & The Conduct Problems Prevention Group, 2006; Hipwell, Pardini, Loeber, Sembower, Keenan, & Stouthamer-Loeber, 2006). These two disorders are also both categorized by the *Diagnostic and Statistical Manual of Mental Disorders* as "Disruptive Behavior Disorders" (APA, 2000), and they load onto a single factor in dimensional measures of child psychopathology, such as the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2001).

A second set of preliminary analyses was conducted to determine which child demographic variables were associated with child CP to then control in subsequent analyses. As reviewed herein, existing literature has shown that boys are more likely to develop CD than girls (Arnold, 1996; Bauermeister et al., 2007), a higher prevalence rate of CD in samples which include minority races than in samples of only Caucasian children (Lahey & Waldman, 2003), and that older children are more likely to display CP than younger children (Rutter, 2003). Child gender was coded into two dichotomous (dummy) variables (male; female), and child race was similarly coded (White; Non-White). Then, child race, child age, and child gender were entered into a preliminary regression analysis predicting CP. None of these child-related demographic variables were significantly related to child CP at the $p < .05$ level; thus, none were entered into subsequent regression analyses as control variables (see Table 3).

Finally, a third set of preliminary analyses was also conducted to determine which parental demographic variables were associated with child CP and should therefore be controlled in the primary analyses. Parental demographic variables that have been shown to be associated with the development of child CP include family

income (Chronis et al., 2007) and maternal education level (Chronis et al., 2007). Maternal education level, total family income, and paternal residential status were entered into a second preliminary regression analysis predicting CP. None of these demographic variables were significantly related to child CP at the $p < .05$ level; thus, none were entered into subsequent regression analyses as control variables (see Table 3). Descriptive statistics for all demographic and predictor variables are presented in Tables 1 and 2.

Aim1: Paternal Psychopathology

For the first aim, correlational analyses were conducted between paternal psychopathology and child CP. To examine this aim regarding the relationship between various types of paternal psychopathology and concurrent child CP, correlations were examined between child CP and continuous levels of the following types of paternal psychopathology: depression, alcohol problems, past and current alcohol dependence, past non-alcohol substance dependence, ASPD, and ADHD. Symptoms of these disorders were examined continuously in order to maximize variability among the fathers within our sample. Contrary to the proposed hypothesis, results suggested that there were no significant relationships between any of these continuous measures of paternal psychopathology and child CP. Results for these analyses are presented in Table 4.[1]

[1] When paternal psychopathology was examined diagnostically (presence; absence) for each disorder, no significant associations were found. Therefore, continuous levels of paternal psychopathology were used in all subsequent analyses.

Aim 2: Paternal Involvement and Psychopathology

To examine the extent to which father involvement moderated the relationship between paternal psychopathology and child CP, we conducted linear regression analyses in two steps. In the first step, father involvement and paternal psychopathology were entered continuously to examine how much variance in the outcome variable was explained by each of these variables while considering the other. In the second step, we examined the extent to which the interaction between paternal psychopathology and involvement contributed to the prediction of child CP above and beyond the first step. For this second step, the following interaction variables were created by computing the product of the Father Involvement Questionnaire (INV) and each continuous measure of paternal psychopathology: INV x depression, INV x alcohol problems, INV x alcohol dependence (past and current), INV x non-alcohol substance dependence (past), INV x ASPD, and INV x ADHD (childhood and adult).

For this aim, separate regression analyses were initially conducted using two measures of paternal involvement: the Father Involvement Questionnaire (INV) and the well-established involvement subscale of the Alabama Parenting Questionnaire (APQ-Inv). These two measures are highly correlated ($r = .61$, $p < .01$), and similar trends were found such that each interacted with paternal psychopathology to predict child CP (see Tables 5 and 6). We therefore report only on results using the Father Involvement Questionnaire. This measure was preferred over the APQ involvement scale because it provides a greater number of items which described the frequency

and nature of paternal involvement with their children, as well as items that are more relevant to parenting a child with ADHD.

Results indicated that neither paternal psychopathology nor involvement were significantly associated with child CP when both were considered in these models. The INV x ADHD (childhood) interaction term, however, did contribute significantly to the overall relationship with child ODD/CD symptoms, accounting for 11.9% of the variance in child CP (see Table 5). Furthermore, the relationship between the INV x ADHD (adulthood) interaction term and child CP was significant, accounting for 20.1% of the variance in child CP (see Table 5).[2] No other interaction terms contributed significantly to these models at the level of $p < .05$; however, several other interactions terms showed a trend toward significance ($p < .10$). While these trends should not be interpreted given their non-significance, they have been reported in Table 5 for use in generating new hypotheses for future larger studies.

To first probe the interaction between paternal ADHD and involvement, we examined correlations within dichotomous groups of fathers with high and low levels of involvement based on a median split. Among fathers with lower levels of involvement, paternal ADHD symptomatology was negatively correlated with child CP (childhood ADHD: r = -.269, N = 21, p > .05; adulthood ADHD: r = -.217, N = 21, p > .05). Among fathers with higher levels of involvement, however, paternal ADHD symptomatology was positively correlated with child CP (childhood ADHD: r = .579, N = 16, p <.05; adulthood ADHD, r = .775, N = 16, p < .01).

Considering our small sample size and the fact that median splits reduced our

[2] It should be noted that, when fathers who see their children less than once per week (n = 3) were removed from these analyses, the same patterns of significant relationships emerged.

power even further, we also conducted simple slopes tests to probe the region of significance for the interaction between paternal ADHD and involvement in predicting child CP. At levels of paternal ADHD of 8 symptoms or more, paternal involvement is significantly and positively related to child CP (childhood ADHD: $\beta = 0.528$, $F(1,32) = 4.641$, $p < .05$; adulthood ADHD: $\beta = 0.442$, $F(1,32) = 5.533$, $p < .05$). A negative relationship between paternal involvement and child CP was only found when there were zero paternal ADHD symptoms (childhood ADHD: $\beta = -0.550$, $F(1,32) = 4.202$, $p < .05$; adulthood ADHD: $\beta = -0.501$, $F(1,32) = 5.228$, $p < .05$).

Aim 3: Interaction of Maternal and Paternal Psychopathology

To examine the extent to which maternal psychopathology interacted with paternal psychopathology to predict variance in child CP, we conducted linear regression analyses in two steps. In the first step, maternal psychopathology and paternal psychopathology, both entered continuously, were examined in terms of how much variance in child CP was explained by each of these variables while considering the other. In the second step, we examined the extent to which the interaction between maternal and paternal psychopathology contributed above and beyond the first step. For this second step, the following interaction variables were created by computing the product of Maternal BDI-II (MOM BDI-II) and each continuous measure of paternal psychopathology: MOM BDI-II x depression, Maternal BDI-II x alcohol problems, MOM BDI-II x alcohol dependence (past and current), MOM BDI-II x non-alcohol substance dependence (past), MOM BDI-II x ASPD, and MOM BDI-II x ADHD (childhood and adult). Results indicated that

neither paternal psychopathology nor maternal psychopathology were significantly associated with child CP when both were considered in these models. Furthermore, no interaction terms contributed significantly to these models at the level of $p < .05$. Results for these analyses are presented in Table 7.

Discussion

This study adds to the small but growing literature on the associations between paternal psychopathology and child conduct problems (CP) within families of children with ADHD. In the context of a developmental psychopathology perspective, we have extended this literature by considering level of paternal involvement to determine whether paternal psychopathology is most strongly related to child outcomes when the father is highly involved in childrearing. Second, we also examined the interaction between maternal and paternal psychopathology as associated with child CP, which has not, to our knowledge, been examined in families of children with ADHD. Third, whereas the majority of prior studies have focused on the psychosocial outcomes of boys with ADHD, the results of this study are based on a clinical sample consisting of both boys and girls.

Contrary to our hypothesis, paternal psychopathology was not significantly associated with concurrent child CP (see Table 4). Theoretical models of child psychopathology, especially that of Belsky (1984) and Patterson (1982), suggest that children and parents each reciprocally contribute to the psychological wellbeing of the other such that the likelihood of mental illness in both counterparts is positively related. Consistent with these models, previous literature examining paternal psychopathology (including ASPD, alcohol dependence, non-alcohol substance

dependence, depression, and anxiety disorder) has found significant associations between these predictors and child CP, albeit within samples that are not specific to children with ADHD (Bronte-Tinkew et al., 2007; Dietz et al., 2009; Frick et al., 1992; Kane & Garber, 2004; Kopp & Beauchaine, 2007; Loukas et al., 2003; Mezulis et al., 2004). Even within ADHD-specific samples, however, significant associations have been found between paternal antisocial behavior and child CP (Lahey et al., 1998). Thus, our findings are inconsistent with much of the previous literature and well-accepted theory; however, this may be attributable to the size and composition of our sample. Indeed, prior studies that have found significant associations between paternal psychopathology and child CP have had considerably larger sample sizes and more variability within paternal psychopathology than our own study (Bronte-Tinkew et al., 2007; Dietz et al., 2009; Frick et al., 1992; Kopp & Beauchaine, 2007; Loukas et al., 2003; Mezulis et al., 2004). This may be because many of these studies did not collect data on paternal psychopathology directly from fathers; rather, they asked mothers to report on fathers (Lahey et al., 1998). Indeed, studies have suggested that fathers who agree to participate in research may be healthier (Pfiffner, McBurnett, & Rathouz, 2001). Indeed, within our own sample of fathers reporting on themselves, considerable variability emerged within paternal ADHD symptoms, but far less so within other disorders (see Table 2). Thus, future studies should aim to recruit larger, more representative samples of families of children with ADHD in order to elucidate the associations between paternal psychopathology and child CP in this population and allow for better examination of moderators of this relationship.

Interestingly, even within this small sample, paternal involvement significantly moderated the relationship between paternal ADHD symptoms and child CP. Among fathers with higher levels of involvement, paternal ADHD symptoms (both childhood and current) was positively associated with child CP; however, among fathers with lower involvement, paternal ADHD symptoms was negatively associated with child CP (although these latter associations did not reach statistical significance). Paternal involvement did not interact significantly with any of the other forms of psychopathology that were examined (i.e., paternal depressive symptomatology, alcohol problems, alcohol dependence, non-alcoholic substance dependence, and ASPD) (see Table 5).[3] Our results with regard to paternal ADHD are inconsistent with developmental studies of paternal involvement that have generally found higher levels of paternal involvement to be associated with *better* psychosocial outcomes in children (e.g., Flouri et al., 2002; Cabrera et al., 2000; Shek, 2005; Coley & Medeiros, 2007). Our findings *are* consistent, however, with one study that found the interaction of paternal ADHD symptoms and father involvement to be associated with negative paternal parenting, though they did not consider child behavioral correlates of this interaction (Arnold et al., 1997). Furthermore, these findings fit with the theoretical models proposed by both Belksy (1984) and Patterson (1982), whereby parenting (e.g., parental involvement) is multiply determined by child factors (e.g., child ADHD), parental factors (e.g. parental ADHD), and contextual factors, and negative parenting may exacerbate already-existing child behavior problems. Our study is among the first to consider both paternal psychopathology and paternal

[3] Trends toward significance, however, suggest that similar patterns might be detected in larger samples for all of these types of paternal psychopathology, with the exception of paternal alcohol problems, past alcohol dependence, and ASPD.

involvement in relation to child CP, and its results suggest that the psychological health of the father may determine whether his involvement in childrearing has a positive or detrimental influence on child CP.

Maternal psychopathology did not significantly interact with paternal psychopathology to relate to child CP (see Table 7), contrary to our hypothesis. Our results were inconsistent with prior studies, which have typically found that children who have two parents with significant psychopathology (e.g., depression) are at greater risk for both internalizing and externalizing problems, as compared to children with only one affected parent (Brennan et al., 2002; Dierker, Merikangas, & Szatmari, 1999; Foley et al., 2001; Goodman, Brogan, Lynch, & Fielding, 1993; Reich, Earls, Frankel, & Shayka, 1993). Notably, however, these studies all examined parents with psychiatric diagnoses, whereas the current study examined parental psychopathology continuously within a sample of parents with relatively little psychopathology (see Tables 1 and 2). Our findings do fit with one study, which found the interaction between maternal and paternal psychopathology to be significantly associated with child internalizing problems, but not externalizing problems (Mezulis et al., 2004). Future studies could clarify relationships between the interaction of maternal and paternal psychopathology and child CP and broaden this research-base to examine additional forms of maternal psychopathology beyond depression.

The results of this study should be considered in light of its limitations. First, this study was limited by its small sample size and (perhaps therefore) limited variability within the paternal disorders that were examined. When considering

paternal involvement as a moderator, we only found significant relationships between paternal ADHD (in childhood and adulthood) and child CP, but not with other paternal disorders. Indeed, as compared to the 37% of fathers our sample that had four or more ADHD symptoms, fewer had similar symptom-levels within other disorders. Additionally, there were only five non-residential fathers recruited for our sample (12.5%), as compared to that of the larger sample from which we recruited (38%). This rate is much lower than would be expected within the population of families of children with ADHD, in which the divorce rate is estimated to be three times that of parents of children without the disorder (Barkley et al., 1991; Wymbs et al., 2008). As with much prior research, we encountered great difficulty in recruiting non-residential fathers, as mothers often refused to give permission to contact them in order to preserve these fathers' limited free time for visitation (Mitchell, See, Tarkow, Cabrera, McFadden, & Shannon, 2007). The families in our sample also had significantly higher mean income-levels than that of the larger sample from which this study recruited. Although these paternal characteristics (i.e., high-income, residential, and relatively healthy) are typical of fathers who are willing to participate in research (Pfiffner et al., 2001), these sample characteristics limit the generalizability of our findings to the broader population of families of children with ADHD. This high-risk population typically has lower family income levels and a higher incidence of non-residential fathers (as compared to families of healthy children). Future studies should therefore recruit samples that are more representative of the ADHD population, which may be accomplished by recruiting through

community centers and other establishments that are not necessarily for the purpose of treatment-seeking.

A second limitation is the cross-sectional nature of this study. Our results can not be interpreted from a developmental psychopathology standpoint because all constructs were measured at a single time-point; thus, we can not assume that paternal psychopathology and involvement pre-dated child conduct problems and therefore contributed to the development of these problems. Still, among involved fathers, paternal reports of their own childhood ADHD symptoms were positively associated with mother-reported child CP (although reports of paternal childhood symptoms were retrospective). Possible predictors and correlates of child psychopathology should, in future studies, be studied longitudinally so that we can more clearly establish whether these are indeed risk factors for the later development of CP in children with ADHD.

Finally, the way in which information on fathers was collected was limited in our study. We required mothers' permission to contact fathers, and prior research has found that mothers often function as "gatekeepers" to protect fathers' time. This is the case for both residential and non-residential fathers who are less involved in parenting, where mothers (and other family members) often screen phone calls and refuse to pass along messages regarding research participation (Mitchell, See, Tarkow, Cabrera, McFadden, & Shannon, 2007). These phone calls were also made by a female investigator; thus, future studies should attempt to obtain the resources necessary to make face-to-face visits and to involve male research assistants in contacting fathers, as these procedures have been found to be considerably more

effective for recruiting fathers (regardless of income-level and residential-status) (Mitchell et al., 2007). Additionally, our reliance on father-report for information on paternal involvement without corroborating maternal report may have limited the accuracy of this information for use in analyses, and future studies should collect paternal psychopathology and involvement information from a collateral informant to maximize the accuracy of such information. This method has been shown to be especially important for adults who have high levels of ADHD symptoms due to concerns about individuals' awareness of current symptoms and parenting behavior and ability to recall past symptoms (McGough & Barkley, 2004).

Despite these limitations, this study uniquely contributes to previous literature by examining both paternal psychopathology and involvement (and each considering the other) as correlates of child CP, and by collecting direct reports from fathers (which is rarely obtained in this literature; Pfiffner et al., 2001). The merits of collecting self-report information from fathers has been debated. Indeed, it has been found that fathers of children with ADHD who willingly participate in research have lower levels of psychopathology (Singh, 2003). Supplementing father-report with mother-report information, however, still provides a conservative estimate of paternal psychopathology, as mothers tend to under-report the psychopathology of their children's fathers (Caspi, Taylor, Smart, Jackson, Tagami, & Moffit, 2001). Thus, collecting father-report information remains a priority within research on paternal psychopathology.

Additionally, while a moderately-sized literature on the interaction between maternal and paternal psychopathology currently exists, few studies have considered

paternal externalizing disorders and even fewer have done so within an ADHD-specific sample. Additionally, this study is one of the first to examine the interaction of paternal psychopathology and involvement as associated with child CP. Indeed, the findings of this study suggest that paternal involvement moderates the relationship between paternal psychopathology and concurrent child CP, such that the degree to which paternal involvement is helpful or harmful may depend on the father's psychological health. Thus, future research should examine paternal psychopathology and paternal involvement in tandem in order to paint a more complete picture of the impact of fathers on child adjustment.

These findings also emphasize the importance of continued examination of environmental factors, especially parenting, as risk factors for child psychopathology. Though much recent research has pointed to a genetic etiology of ADHD (e.g., Thapar et al., 2007), it seems that environmental exposure to paternal ADHD may be related to the development of conduct problems among children with ADHD. Thus, such environmental factors should continue to be a primary research focus.

Although this study requires replication with a larger and more representative sample, these findings suggest that clinicians may wish to consider assessing for paternal psychopathology and involvement as part of routine child assessment. Moreover, when fathers are highly involved in childrearing, it may be particularly important to engage them in treatment to enhance the likelihood that will positively contribute to the psychosocial wellbeing of their children with ADHD (see Fabiano, 2007 for a review of father participation in behavioral parent training). In addition, clinicians might also consider integrating treatment for paternal psychopathology,

especially ADHD, as part of routine behavioral parent training for their children with ADHD.

Tables

Table 1

Sample Demographic and Diagnostic Characteristics (N = 40)

	N (%)	M (SD)
Child demographic variables		
Age		8.275 (1.935)
Sex		
Male	28 (70.0%)	
Female	12 (30.0%)	
Ethnic characteristics		
White	24 (60.0%)	
African American	6 (15.0%)	
Hispanic or Latino	1 (2.5%)	
Biracial	8 (20.0%)	
Other/refused	1 (2.5%)	
DSM-IV diagnoses		
ODD diagnosis	21 (52.5%)	
CD Diagnosis	2 (5.0%)	
ODD Symptoms		3.575 (2.218)
CD Symptoms		0.950 (1.037)
CD + ODD Symptoms		4.525 (2.891)
Maternal Demographic Variables		
Age		40.45 (6.164)
Total Family Income		$125,756.82 ($65,398.86)
Highest Level of Education (years post middle school)		7.525 (3.080)
BDI-II Score		17.263 (11.351

Note. DSM-IV = Diagnostic and Statistical Manual, Fourth Edition; ODD = Oppositional Defiant Disorder; CD = Conduct Disorder; BDI-II = Beck Depression Inventory

Table 2

Paternal Demographic and Diagnostic Characteristics (N = 40)

	N (%)	M (SD)
Age		42.50 (7.307)
Residential status		
Residential father	35 (87.5%)	
Non-residential father	5 (12.5%)	
BDI-II score (depressive symptomatology)		5.359 (8.028)
MAST-S score (alcohol problems)		3.325 (1.542)
Major Depressive Disorder		
Current diagnosis	2 (5.0%)	
Past diagnosis	4 (10.0%)	
Alcohol Dependence		
Current diagnosis	2 (5.0%)	
Total current symptoms		0.865 (1.546)
Past diagnosis	12 (30.0%)	
Total past symptoms		2.695 (2.054)
Non-Alcohol Substance Dependence		
Past diagnosis	5 (12.5%)	
Total past symptoms		0.919 (1.656)
Antisocial Personality Disorder (ASPD)		
Lifetime diagnosis	1 (2.5%)	
Total lifetime symptoms		1.162 (1.463)
Attention-Deficit/Hyperactivity Disorder (ADHD)		
Childhood diagnosis	9 (22.5%)	
Total childhood symptoms		3.703 (2.905)
Adulthood diagnosis	5 (12.5%)	
Total adulthood symptoms		3.676 (3.232)
Total externalizing symptoms (current)		4.541 (4.004)
Presence of psychiatric diagnosis (lifetime)	19 (47.5%)	

Table 3

Correlations Between Demographics and Child Conduct Problems

	Child CP (*r* value)
Child demographics	
Age	-.269
Sex – Male	.140
Sex – Female	.140
Race – Caucasian	-.089
Race - African American	.083
Race – Hispanic or Latino	.079
Race – Biracial	.002
Maternal demographics	
Age	-.087
Total family income	.005
Highest education level (post middle school)	.170
Paternal demographics	
Age	-.014
Residential status – lives with child	-.036
Residential status – does not live with child	.036

Note. CP = Conduct Problems; No correlations were significant at the level $p < .05$.

Table 4

Aim #1: Correlations between Paternal Psychopathology Symptoms and Child CP

	Child CP (*r* value)
Depressive Symptomatology (BDI-II)	-.093
Alcohol Problems (MAST-S)	-.091
Alcohol Dependence (Current)	-.123
Alcohol Dependence (Past)	-.014
Non-Alcohol Substance Use Dependence (Past)	-.030
Antisocial Personality Disorder (Lifetime)	-.120
ADHD (Childhood)	.041
ADHD (Adulthood)	.181

Note. CP = Conduct Problems; No correlations were significant at the level $p < .05$.

Table 5

Aim 2: Paternal Psychopathology and Paternal Involvement predicting child CP (Father Involvement Questionnaire)

Depressive symptomatology	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 35	.158	.009	.009		
BDI-II					.064	-.096
INV total					.025	-.003
Step 2	1, 34	1.234	.098	.089+		
BDI-II x INV					.002	.664+
Alcohol problems	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 36	.173	.010	.010		
MAST-S					.315	-.098
INV total					.023	-.003
Step 2	1, 35	.560	.046	.036		
MAST-S x INV					.013	1.013
Alcohol Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.241	.014	.014		
Current Sx					.393	-.132
INV total					.027	.035
Step 2	1, 32	1.126	.095	.081		
Current Sx x INV					.014	.741
	df	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 32	.022	.001	.001		
Past Sx					.268	-.037
INV total					.026	.003
Step 2	1, 31	1.192	.103	.102+		
Past Sx x INV					.008	.972+
Non-Alcohol Substance Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.059	.004	.004		
Past Sx					.331	-.055
INV total					.025	.014
Step 2	1, 32	1.324	.110	.107+		
Past Sx x INV					.010	.816+
ASPD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.278	.017	.017		
Lifetime Sx					.361	-.131
INV total					.025	-.009
Step 2	1, 32	1.164	.098	.082		
Lifetime Sx x INV					.012	.897
ADHD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.022	.001	.001		
Childhood Sx					.185	.027
INV total					.025	.030
Step 2	1, 32	2.581	.195	.194**		
Childhood Sx x INV					.007	1.554**
	df	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2,33	.503	.030	.030		
Adulthood Sx					.164	.171
INV total					.024	.046
Step 2	1,32	3.935	.269	.240**		
Adulthood Sx x INV					.006	1.744**

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

Note. CP = conduct problems; INV = Paternal Involvement Questionnaire; BDI-II = Beck Depression Inventory; MAST-S = Michigan Alcohol Screening Test – Short Form; Sx = Symptoms; Dx = Diagnosis; ASPD = Antisocial Personality Disorder Symptoms; ADHD = Attention-Deficit/Hyperactivity Disorder Symptoms

Table 6

Aim #2: Paternal Psychopathology and Paternal Involvement predicting child CP (Alabama Parenting Questionnaire – Involvement Scale)

Depressive symptomatology	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 35	.175	.010	.010		
BDI-II					.067	-.111
APQInv					.110	-.048
Step 2	1, 34	1.175	.094	.084+		
BDI-II x APQInv					.008	1.254+
Alcohol problems	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 36	.280	.015	.015		
MAST-S					.323	-.122
APQInv					.098	-.033
Step 2	1, 35	1.487	.113	.098+		
MAST-S x APQInv					.063	2.522+
Alcohol Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.247	.015	.015		
Current Sx					.369	-.128
APQInv					.107	-.038
Step 2	1, 32	.369	.033	.019		
Current Sx x APQInv					.055	.662
	df	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 32	.039	.002	.002		
Past Sx					.305	-.055
APQInv					.116	-.022
Step 2	1, 31	1.068	.094	.091+		
Past Sx x APQInv					.035	1.342+
Non-Alcohol Substance Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.063	.004	.004		
Past Sx					.349	-.066
APQInv					.110	-.022
Step 2	1, 32	1.366	.113	.110+		
Past Sx x APQInv					.044	1.443+
ASPD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.436	.026	.026		
Lifetime Sx					.380	-.167
APQInv					.105	-.043
Step 2	1, 32	.768	.067	.041		
Lifetime Sx x APQInv					.052	1.002
ADHD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.034	.002	.002		
Childhood Sx					.178	.045
APQInv					.104	.009
Step 2	1, 32	1.117	.095	.093+		
Childhood Sx x APQInv					.029	1.809+
	df	***F***	R^2	$R^2\Delta$	**SE**	**β**
Step 1	2, 33	.593	.035	.035		
Adulthood Sx					.157	.187
APQInv					.102	.024
Step 2	1, 32	1.612	.131	.097		
Adulthood Sx x APQInv					.024	1.788

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

Note. CP = conduct problems; APQInv = Involvement Scale of Alabama Parenting Questionnaire; BDI-II = Beck Depression Inventory; MAST-S = Michigan Alcohol Screening Test – Short Form; Sx = Symptoms; Dx = Diagnosis; ASPD = Antisocial Personality Disorder Symptoms ;ADHD = Attention-Deficit/Hyperactivity Disorder Symptoms

Table 7

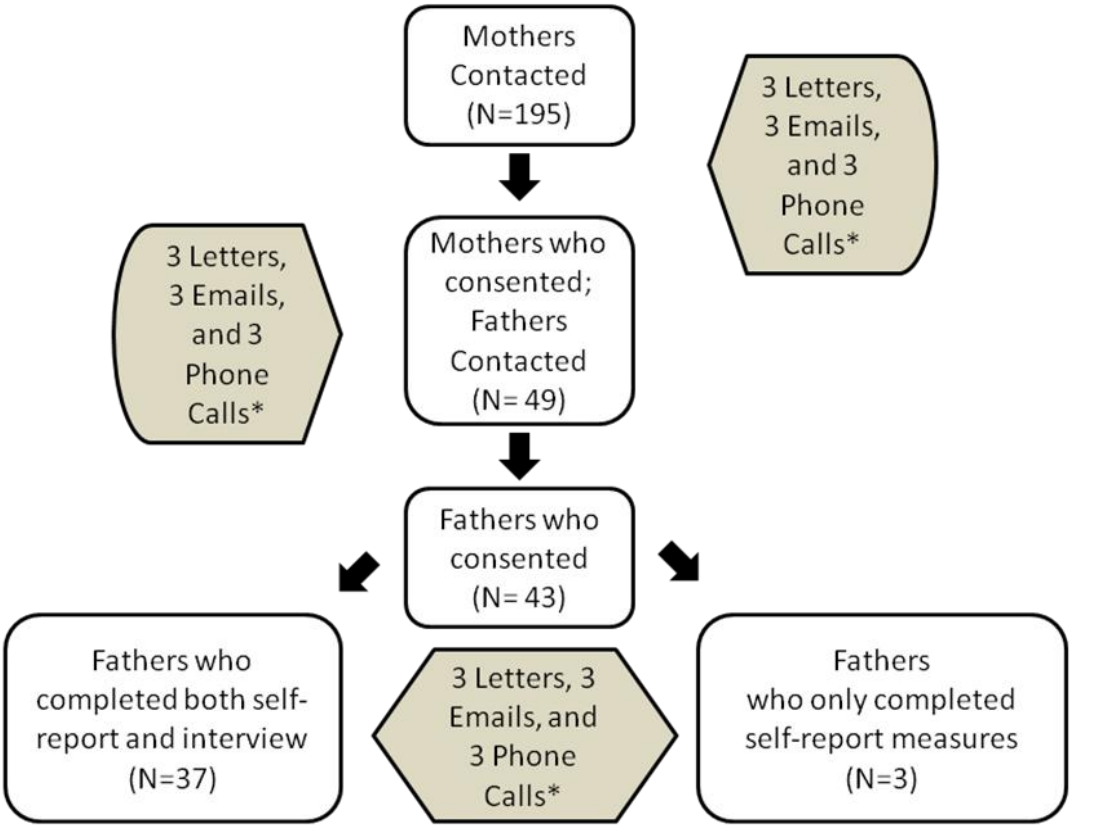
Aim #3: Paternal Psychopathology and Maternal Depression predicting child CP

Depressive symptomatology	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 34	.512	.029	.029		
Father BDI-II					.067	-.197
Mother BDI-II					.048	.109
Step 2	1, 33	.484	.042	.013		
Father BDI-II x mother BDI-II					.004	-.313
Alcohol problems	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 35	.265	.015	.015		
Father MAST-S					.309	-.122
Mother BDI-II					.043	.055
Step 2	1, 34	.188	.016	.001		
Father MAST-S x mother BDI-II					.034	-.143
Alcohol Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 32	.253	.016	.016		
Father current Sx					.339	-.124
Mother BDI-II					.044	.024
Step 2	1, 31	.534	.049	.034		
Father current Sx x mother BDI-II					.019	-.350
	df	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 31	.111	.007	.007		
Father past Sx					.269	-.090
Mother BDI-II					.048	.027
Step 2	1, 30	.690	.065	.057		
Father Past Sx x mother BDI-II					.017	-.665
Non-Alcohol Substance Dependence	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 32	.138	.009	.009		
Father Past Sx					.345	
Mother BDI-II					.050	
Step 2	1, 31	.428	.040	.031		
Father Past Sx x mother BDI-II					.019	-.443
ASPD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 32	.526	.032	.032		
Father lifetime Sx					.350	-.185
Mother BDI-II					.045	.062
Step 2	1, 31	.604	.055	.023		
Father lifetime Sx x mother BDI-II					.024	-.337
ADHD	***df***	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 32	.011	.001	.001		
Father childhood Sx					.176	.023
Mother BDI-II					.045	.008
Step 2	1, 31	.217	.021	.020		
Father childhood Sx x mother BDI-II					.013	-.351
	df	***F***	R^2	$R^2\Delta$	**SE**	**B**
Step 1	2, 32	.300	.018	.018		
Father adulthood Sx					.166	.145
Mother BDI-II					.047	-.039
Step 2	1, 31	.273	.026	.007		
Father adulthood Sx x mother BDI-II					.011	-.216

Note. CP = conduct problems; BDI-II = Beck Depression Inventory; MAST-S = Michigan Alcohol Screening Test – Short Form; Sx = Symptoms; Dx = Diagnosis; ASPD = Antisocial Personality Disorder Symptoms; ADHD = Attention-Deficit/Hyperactivity Disorder Symptoms

Figures

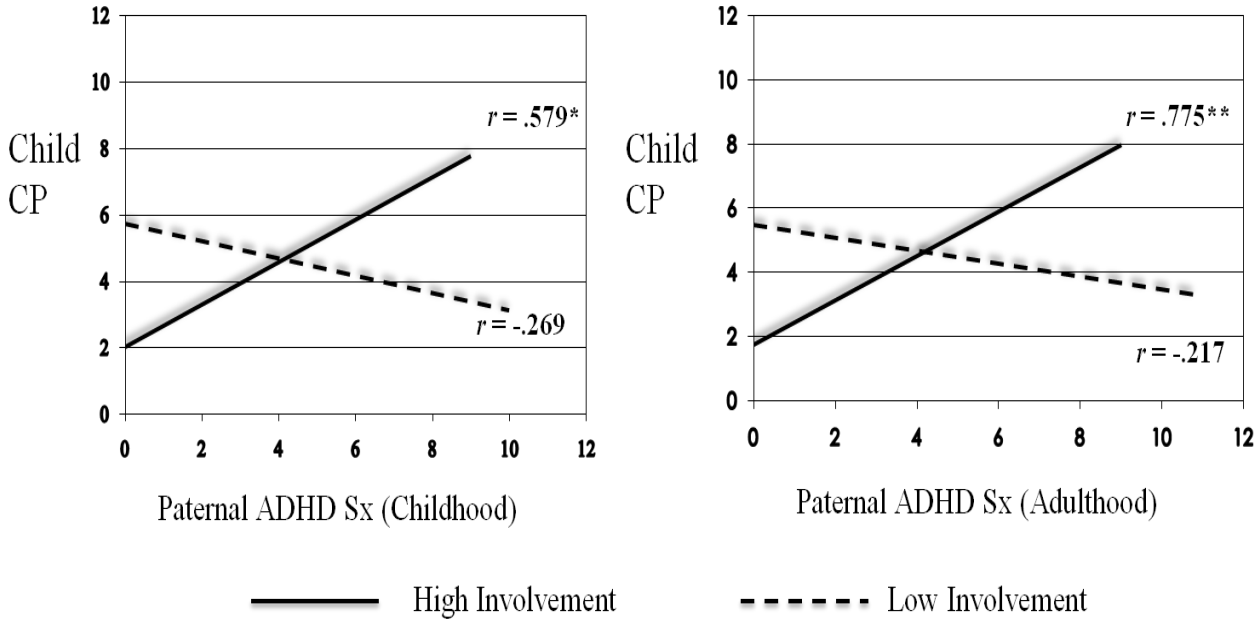
Figure 1
Recruitment Procedures



*Note that these steps represent only the minimum effort for each final participant. In cases where the father was non-residential or some response was received, there were more letters, emails, and phone calls than are indicated in this figure.

Figure 2

Results of Aim 2: Paternal Psychopathology and Paternal Involvement predicting child CP (Father Involvement Questionnaire)



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