ABSTRACT

Title of Dissertation: PATERNAL ADHD, PARENTING, AND

CHILD CONDUCT PROBLEMS: POTENTIAL MECHANISMS.

Abigail D. Mintz, Doctor of Philosophy, 2012

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Parental factors, specifically psychopathology and parenting, robustly predict negative developmental outcomes among children with attention-deficit/hyperactivity disorder (ADHD). Indeed, emergent findings have linked maternal ADHD symptoms both with sub-optimal parenting and child conduct problems within families of children with ADHD. Despite considerable research supporting the important and unique contributions of fathers to their children's development, the role of fathers within families of children with ADHD has seldom been examined. In particular, little research has been conducted with regard to paternal ADHD symptoms and parenting, despite clear evidence for an association between maternal ADHD symptoms and maladaptive parenting. The current study examined psychopathology and parenting behavior among a sample of fathers (N=102) and their 5-12 year-old children with previously-diagnosed ADHD. Results indicated that paternal antisocial personality disorder (ASPD)

symptoms (rather than ADHD symptoms) were robustly associated with child conduct problems, and paternal negative parenting mediated this relationship.

Future research using prospective longitudinal designs should examine multiple forms of psychopathology and parenting behavior among fathers of children with ADHD in order to identify potential risk factors and associated mechanisms for the development of child conduct problems.

PATERNAL ADHD, PARENTING, AND CHILD CONDUCT PROBLEMS: POTENTIAL MECHANISMS.

By

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Dedication

To my parents for their unending support and encouragement. To my husband for riding alongside me on the long and winding road toward my degree. To my teachers in all aspects of my training: instructional, research, and clinical. Finally, to the tireless efforts of all parents, both mothers and fathers, who are raising a child with any type of special need.

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Introduction

Children with ADHD and Comorbid Conduct Problems

ADHD is present in 3-7% of school-age children in the United States (American Psychiatric Association, 2000). Characterized by symptoms of hyperactivity, impulsivity, and inattention, children with ADHD experience significant functional impairment across important life areas (APA, 2000). For instance, they often have more conflict with parents and siblings, have trouble succeeding in the classroom both academically and socially, and face unique challenges in maintaining peer relationships (Coie & Dodge, 1998; Fischer, 1990; Mikami & Pfiffner, 2008; Zentall, 2007). Additionally, these children are at substantially higher risk for maladaptive outcomes later in development, such as early initiation and abuse of substances, depression, suicidal behavior, and delinquency (Burke, Loeber, & Lahey, 2001; Chronis-Tuscano et al., 2010; Elkins et al., 2007).

Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) are both highly comorbid with ADHD. 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). In community samples, CD co-occurs 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 between ADHD and CD as high as 52.4 percent (Jensen, Martin & Cantwell, 1997). ODD is characterized by a recurrent pattern of

negativistic, defiant, disobedient, and hostile behavior toward authority figures (APA, 2000), which has been shown to co-occur with ADHD at rates between 36.5 percent (Yang, Wang, Qian, Biederman, & Faraone, 2004) and 65 percent in clinical samples (Biederman et al., 1996b). Symptoms of CD and ODD are often collectively referred to as "conduct problems" (CP).

Though ADHD alone is associated with negative developmental outcomes, the early comorbidity of ADHD and CP has been shown to be especially predictive of the most serious negative developmental outcomes, including serious substance abuse, persistent aggression (both verbal and physical), and chronic criminality (Burke, Loeber, & Lahey, 2001; Elkins, McGue, & Iacono, 2007; Foley, Carlton, & Howell, 1996; Harty, Miller, Newcorn, & Halperin, 2008; Lahey et al., 1988). Indeed, children with early-onset CP (who are more likely to have ADHD; Lahey, McBurnett, & Loeber, 2000) are also more likely to go on to exhibit severe antisocial behavior and psychopathic characteristics in adulthood (Henry, Caspi, Moffitt, & Silva, 1996; Lynam, 1996; Lynam, 1998). In line with a developmental psychopathology framework, identifying risk and protective factors that are associated with the presence of CP in these children is therefore critical. Moreover, identifying *modifiable* risk factors is a priority in the literature, as such research has the potential to inform the development of specific treatments aimed to prevent children from embarking on this lethal developmental trajectory.

It is important to note that recent studies have suggested that a genetic predisposition for the development of psychopathology in children is well-

established, and this is especially true with ADHD for which the heritability rate exceeds .75 (Burt, 2009; Faraone et al., 2005; Forero, Arboleda, Vasquez, & Arboleda, 2009; Thapar, Langley, Owen, & O'Donovan, 2007). Similar genetic relationships have been found between parental and child antisocial behavior (Ge et al., 1996). Importantly, though, a recent review of behavioral-genetic studies on child aggressive behavior has suggested that genetics account for approximately half (and in many studies even less) of the observed variance in child antisocial behavior (i.e., CP; Moffitt & Caspi, 2007). Thus, additional factors still seem to play a crucial role. Among children with ADHD, specifically, modifiable factors such as parenting have been found to interact with genetics to predict variance in the development of CP (Lahey et al., 2011). Indeed, focusing on modifiable risk factors (e.g., parenting) is supported both empirically and by relevant theories.

Theoretical Framework

The developmental psychopathology perspective provides an overarching framework for understanding both normal and maladaptive development (Mash & Dozois, 2003). A developmental psychopathology perspective acknowledges that, in order to understand maladaptive behavior, it is important to first understand normative behavior. Upon understanding normal behavior, this approach emphasizes the study of risk and protective factors that predict adaptive and maladaptive developmental trajectories. The developmental psychopathology perspective also encompasses several specific theories. Those most relevant to the relationship between parental characteristics and child behavior include Belsky's

(1984) process model of parenting and Patterson's (1982) reciprocal transactional model for child delinquent behavior.

Belsky (1984) specifically suggests that the etiology of parenting behavior is rooted in three basic sources: parental factors (e.g., personality and psychopathology), child factors (e.g., temperament and psychopathology), and contextual factors (e.g., parental marital relationship and socioeconomic status) (Belsky, 1984). The impact of these factors is especially salient among families of children with ADHD. For example, psychopathology in parents is more prevalent in these families (as compared to families of typically-developing children; Chronis et al., 2003), the divorce rate in these families is significantly higher (Barkley, Fischer, Edelbrock, & Smallish, 1991; Wymbs et al., 2008), and these children have a more difficult temperament and display challenging behavior. Thus, a comprehensive understanding of the multiple influences on parenting behavior within families of children with ADHD must consider the psychological health of the parent and child (including comorbid child psychopathology), and acknowledge relevant contextual factors (such as marital and socioeconomic status).

Patterson's (1982) coercion model describes a transactional relationship whereby child externalizing behavior, parenting, and parental psychopathology exert reciprocal influences on one another. Specifically, parents of children with ADHD may suffer from negative psychosocial well-being in part as a result of repeated exposure to negative child behavior, which may contribute to the use of maladaptive parenting practices and ultimately exacerbate child behavior

problems (Patterson, 1982). Furthermore, it is theorized that parents in these families often negatively reinforce their child's behavior by withdrawing demands when enforcing them becomes too stressful or seems fruitless due to repeated noncompliance. Parent behavior is also negatively reinforced because withdrawal of parent demands leads to a short-term reduction in child misbehavior. Alternatively, parents may utilize increased levels of hostility and coercive parenting behaviors when enforcing commands, and this too is negatively reinforced when the result is a short-term reduction in child misbehavior. Ultimately, the combination of both of these types of parenting behavior (i.e., avoiding conflict by withdrawing demands and being overly harsh) often result in patterns of inconsistent discipline, which is known to exacerbate child misbehavior (Loeber & Dishion, 1983; Pfiffner, McBurnett, Rathouz, & Judice, 2005). It stands to reason, therefore, that special attention must be paid to the bidirectionality of parent and child behavior, as well as to how parental psychopathology may contribute to both (Johnston, Mash, Miller, & Ninowski, 2012).

Within families of children with ADHD, recent research focus has turned to parental ADHD symptoms, given that ADHD is a highly heritable disorder (Faraone et al., 2005). ADHD in adulthood is characterized by the same core symptoms as in childhood (inattention and hyperactivity/impulsivity; Faraone et al., 2000b); yet, these symptoms typically manifest as more adult-relevant difficulties, including impaired parenting and parent-child interactions (Chronis-Tuscano et al., 2008; Murray & Johnston, 2006; Weiss et al., 2000). Specifically,

parents with ADHD often face issues related to impulsive decision-making, emotional lability, poor sustained attention, and poor persistence. It follows that adult ADHD symptoms may exacerbate the maladaptive parenting behaviors that are already more common among parents of children with ADHD (Chronis-Tuscano et al., 2008).

Taken together, the specific theories of Belsky (1984) and Patterson (1982), as well as a general developmental psychopathology approach, provide an important context for understanding findings linking parent and child factors within families of children with ADHD. With this theoretical context in mind, the most recent literature related to parental psychopathology (particularly ADHD), parenting behavior, and conduct problems in children with ADHD is discussed.

Maternal Characteristics and Child CP

Consistent with Belsky's (1984) theory on parenting behavior, recent findings have suggested that parental characteristics may be among the most significant risk and protective factors associated with the development of negative outcomes among children with ADHD (Chronis et al., 2007; Luthar & Zelazo, 2003). Undeniably, parents are crucial in managing the impairments of their children with ADHD (Pelham & Fabiano, 2008) and in protecting against the development of more serious long-term outcomes (Johnston & Mash, 2001). In fact, the most salient predictors of negative outcomes in children with ADHD, including the development of CP, are the presence of parental psychopathology and the degree of early positive parenting (Chronis et al., 2007). Psychopathology is present at higher rates among both mothers and fathers of children with ADHD

(Chronis et al., 2003; Cunningham, Benness, & Siegel, 1988; Faraone et al., 2005; Nigg & Hinshaw, 1998); yet, as with the majority of developmental psychopathology research, these risk factors have been most extensively studied with mothers.

Maternal psychopathology, parenting, and child CP. Cross-sectional research has linked maternal psychopathology and parenting to the presence of concurrent CP among children with ADHD. Mothers of children with ADHD have an elevated risk for psychopathology (including depression, anxiety, substance abuse, and alcohol abuse), as compared to controls (Chronis et al., 2003). Indeed, the highest rates of psychopathology have been observed among mothers of children with comorbid ADHD and CP (Chronis et al., 2003). In terms of maternal parenting, Kashdan et al. (2004) found that symptoms of ODD among children with ADHD were uniquely associated with maternal parenting characterized by less warmth and positive involvement, and more intrusive and negative discipline. It is important to note, however, that the cross-sectional design of these studies limits the conclusions that can be made with regard to direction of effect (i.e., parent to child or child to parent); rather, they simply reflect relationships between concurrent maternal psychopathology, parenting behavior, and child behavior.

Longitudinal research has also found maternal psychopathology to significantly predict both maternal parenting behavior and child CP. For example, Chronis and colleagues (2007) found that the presence of maternal depression is a risk factor for later CP among children with ADHD and that positive maternal

parenting is a protective factor against this outcome. These cross-sectional and longitudinal findings among families of children with ADHD suggest that symptoms of depression in mothers may be related to sub-optimal parenting and the presence of conduct problems in their children. Importantly, Patterson's theory (1982) suggests that this relationship may be bidirectional such that challenging child behavior may also contribute to the use of sub-optimal parenting and to parental psychosocial well-being.

Maternal ADHD and parenting. Because ADHD is highly heritable (Faraone et al., 2005), maternal ADHD has been a recent focus in the literature on families of children with ADHD. Mothers of children with ADHD are significantly more likely to have high levels of ADHD symptoms themselves as compared to typically-developing children, and are 24 times more likely to have a childhood diagnosis of ADHD compared to mothers of controls (Chronis et al., 2003). Importantly, ADHD in adults is characterized by many of the same symptoms as childhood ADHD, including inattention, impulsivity, and disorganization, and is highly comorbid with mood, anxiety, and substance use disorders (Kessler et al., 2006; Miller, Nigg, & Faraone, 2007). Moreover, adults with ADHD experience significant impairment, such as higher rates of marital problems and higher likelihood of unemployment (Barkley, Murphy, & Fischer, 2008; Kessler et al., 2006; Johnston et al., 2002; Mannuzza et al., 2011). Most relevant to this review, however, is the impairment that adults with ADHD experience in the parenting role (Barkley, 2011b; Chronis-Tuscano et al., 2008; Johnston et al., 2012; Murray & Johnston, 2006).

A handful of studies have examined the relationship between maternal ADHD and various aspects of parenting. For example, maternal self-reported inattention has been associated with lax parenting with their children with ADHD, and this continued to be the case even after mothers received parent training in behavior management techniques (Harvey, Danforth, McKee, Ulaszek, & Friedman, 2003). Similarly, using self-report measures, Chronis-Tuscano and colleagues (2008) found maternal ADHD symptoms to be associated with less positive parenting, more inconsistent discipline, and less involvement overall. Importantly, observational methods used in this same study showed that maternal ADHD symptoms were associated with more negative parenting and less positive parenting (Chronis-Tuscano et al., 2008). Similarly, in another study, mothers of children with ADHD who had ADHD diagnoses themselves reported using less consistent discipline as well as poorer parental monitoring as compared to mothers of children with ADHD who did not have the disorder (Murray & Johnston, 2006). Moreover, a laboratory-based problem-solving task indicated that mothers with ADHD were less efficient and utilized lower levels of planning to develop solutions in response to a simulated parenting problem (Murray & Johnston, 2006).

Notably, there have been some conflicting findings within this small body of literature linking maternal ADHD to maladaptive parenting. Psychogiou and colleagues (2007, 2008) have proposed a "similarity-fit hypothesis," whereby higher levels of maternal ADHD symptoms may promote more positive parenting when children also have high levels of ADHD symptoms. This hypothesis is

behavioral tempo and therefore have a greater understanding for each other's behavior. At best, however, only mixed support for this hypothesis has emerged in the research. Specifically, results from some of this group's most recent studies have suggested that high levels of adult ADHD symptoms in mothers of children with ADHD are associated with the use of *less* positive and affectionate parenting and *more* negative parenting (Psychogiou, Daley, Thompson, & Sonuga-Barke, 2007; Psychogiou, Daley, Thompson, & Sonuga-Barke, 2007; Psychogiou, Daley, Thompson, & Sonuga-Barke, 2008). Thus, the majority of available studies on maternal ADHD have found that mothers with ADHD (or elevated ADHD symptoms) display impaired parenting, consistent with core deficits related to inattention and hyperactivity/impulsivity.

Importantly, since over fifty-percent of adults with ADHD have children with the disorder as well (Adler & Cohen, 2004; Biederman, Faraone, Mick, & Spencer, 1995; Kessler et al., 2006; Minde et al., 2003), one might expect that parental ADHD would interfere with successful treatment delivery for children with ADHD. Indeed, two studies have shown that maternal ADHD symptoms were associated with diminished effects on child outcomes in an evidence-based treatment for their children with ADHD (Chronis-Tuscano et al., 2011; Sonuga-Barke, Daley, & Thompson, 2002). Moreover, the relationship between maternal ADHD symptoms and improvement in child behavior following parent training interventions is mediated by the degree of parenting improvement (i.e., ability to inhibit negative parenting response to child misbehavior; Chronis-Tuscano et al.,

2011). Thus, mothers with ADHD-related difficulties seem to display parenting impairments which are resistant to change following behavioral interventions.

In sum, emergent findings that link maternal psychopathology (and specifically ADHD) and maladaptive parenting within families of children with ADHD support Belsky's (1984) theory of parenting behavior. This research, however, has focused almost entirely on mothers, and our lack of knowledge about paternal psychopathology and parenting represents a major gap in the literature. To address this gap, research examining ADHD symptoms and parenting in *fathers* is needed in order to mirror existing research with mothers.

Paternal Characteristics and Child Psychosocial Outcomes: Are Fathers Important?

A developmental psychopathology framework suggests that, prior to examining paternal psychopathology, it is essential to first consider normative paternal involvement and parenting. Though it is generally well-accepted that fathers are important figures in the lives of their children, a brief review of the research on the *unique* role of fathers is presented.

Paternal involvement. Reviews of relevant research have suggested convincingly that fathers, like mothers, make a crucial and unique contribution to the psychosocial outcomes of their children (Lamb & Tamis-LeMonda, 2004, Lewis & Lamb, 2003). Developmental research has consistently 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 (Lamb & Lewis, 2004; Lewis & Lamb, 2003). The way that fathers engage in play with their children has been found to uniquely influence later child psychosocial adjustment (Lewis & Lamb, 2003).

Studies that focus on the level of paternal involvement have generally found significant associations with child behavior. Interestingly, one crosssectional, community-based study found that adolescent behavior problems are more significantly associated with paternal involvement than with maternal involvement (Day & Padilla-Walker, 2009). Longitudinal findings also support the influence of father involvement on child behavior. For example, one longitudinal study found that higher levels of father involvement with their 7year-old children predicted lower levels of police contact at age 16, yet this relationship was significant only for boys in this particular sample (Flouri & Buchanan, 2002). A more recent longitudinal study, however, found support for such a relationship among both boys and girls, such that higher levels of involvement by non-residential fathers predicted decreases in CP over time among adolescents who originally displayed high levels of CP (Coley & Medeiros, 2007). Within families of children with ADHD, having a biological father present in the home is related to lower levels of child CP, independent of socioeconomic status (SES; Pfiffner, McBurnett, & Rathouz, 2001). Overall, the literature suggests that paternal involvement is both uniquely and significantly linked to child behavior.

Is involvement enough? A review of studies of paternal involvement has suggested that it is *positive* paternal involvement in particular (rather than paternal involvement more generally) that results in positive psychosocial outcomes in children (Cabrera, Tamis-LeMonda, Bradley, Hofferth, & Lamb, 2000; Nettle, 2008). Consistent with this, a review by Parke (2000) suggests that the quality of paternal involvement is equally as important, if not more important, to examine as the quantity. For example, higher frequency of fathers' rough and tumble play was found to be associated with less aggression in preschoolers when fathers were able to set limits during playtime; otherwise, more frequent rough and tumble play was associated with higher levels of aggression in children (Flanders, Leo, Paquette, Pihl, & Seguin, 2009). Similarly, simply having a father present in the home may not be enough, as evidenced by findings by Coley and colleagues (2009) demonstrating that fathers with more knowledge about their adolescents' friends and activities had children who were less likely to engage in risky sexual behavior (Coley, Votruba-Drzal & Schindler, 2009).

Father involvement has also been studied with a focus on *maladaptive* paternal parenting. In one study, fathers of preschool boys with an ODD diagnosis were more likely than comparison fathers to report using harsh and ineffective parenting practices, interacting angrily with their sons, physically threatening them, and generally experiencing less positive involvement (DeKlyen, Speltz, & Greenberg, 1998). Interestingly, within this same sample, fathers of boys with ODD were more likely than comparison fathers to report symptoms of depression and anxiety. Furthermore, fathers' use of harsh discipline mediated the

relationship between paternal psychiatric symptoms and child ODD status (DeKlyen, Biernbaum, Speltz, & Greenberg, 1998). Fathers of children with ADHD have also reported using more coercive and punitive parenting compared to controls (Rogers, Wiener, Marton, & Tannock, 2009). Finally, one cross-sectional study found that paternal inconsistent discipline and low involvement were uniquely associated with child inattention symptoms above and beyond both paternal ADHD and child comorbid conduct problems (Ellis & Nigg, 2009). Though these studies suggest that fathers of children with ADHD and CP may engage in relatively more negative parenting behavior, the relationship between paternal psychopathology, parenting, and child behavior remains to be clarified.

Paternal psychopathology and child CP. A small body of literature has found several types of paternal psychopathology to be associated with child CP. Meta-analytic findings have suggested that, for girls, the presence of paternal psychopathology may be even more strongly related to child externalizing behavior problems than is maternal psychopathology (Connell & Goodman, 2002). Paternal depressive symptoms, in particular, are associated with child externalizing problems in both community (Cummings, Keller, & Davies, 2005) and clinical (Dave, Sherr, Senior, & Nazareth, 2008) samples of preschoolers, although there are some studies that have failed to find this relationship among adolescents (Tully, Iacono, & McGue, 2008). Specifically among children with ADHD, significant positive associations have been found between paternal depression and child CP (Pfiffner et al., 2005). Few studies have examined other internalizing disorders among fathers of children with ADHD; however, Nigg and

Hinshaw (1998) found that fathers of children with ADHD and comorbid CP were more likely to have Generalized Anxiety Disorder (as compared to both children with ADHD only and to controls).

Among fathers, externalizing disorders such as substance abuse, alcohol abuse, and Antisocial Personality Disorder (ASPD) have been more extensively studied than internalizing forms of psychopathology, and this is especially true in relation to child CP. For instance, several studies have linked paternal alcohol abuse (Foley et al., 2001; Loukas, Zucker, Fitzgerald, & Krull, 2003) and illicit substance abuse (Frick et al., 1992) with the presence of CP in boys. Similar patterns have emerged in samples of children diagnosed with ADHD, such that child CP are associated with paternal alcohol abuse (Chronis et al., 2003) and illicit substance abuse (Nigg & Hinshaw, 1998).

Paternal ASPD symptoms have also been positively associated with CP in boys in both longitudinal (Foley et al., 2001) and cross-sectional (Frick et al., 1992; Lahey et al., 1998) research. Moreover, this has been the case even when maternal depression, one of the most robust predictors of child CP, is controlled (Kopp & Beauchaine, 2007). Among children diagnosed with ADHD, paternal ASPD and child CP are also significantly positively associated (Lahey et al., 1988; Pfiffner et al., 2005). Theoretically, this makes sense considering that ASPD is considered to be a developmental progression of CD (APA, 2000), and the antisocial behavior underlying both of these disorders is both partially heritable (Ge et al., 1996) and more common among individuals with ADHD (Bird et al., 1994; Jensen et al., 1997; Romano et al., 2005).

As mentioned previously, fathers of children with ADHD are more likely to have ADHD themselves (Chronis et al., 2003; Faraone et al., 2005); yet, paternal ADHD has received considerably less research attention relative to other paternal psychiatric disorders, including ASPD and depression. Moreover, to our knowledge, no studies have examined current ADHD among fathers in relation to child CP, though one study has considered this relationship with regard to paternal childhood ADHD (Chronis et al., 2003). Considering the higher rates of both paternal ADHD and child CP within families of children with ADHD, this represents a major gap in the literature.

Paternal psychopathology and parenting. As with maternal psychopathology, few studies have examined paternal psychopathology in relation to both paternal parenting and child behavior. The link between paternal psychopathology and parenting has been far more extensively studied in relation to paternal depression than other paternal psychiatric disorders. In a recent meta-analysis, Wilson and Durbin (2010) found that paternal depression is moderately associated with lower rates of positive parenting and higher rates of negative parenting. Fathers who are depressed also are less engaged with their children and experience more parenting stress (Bronte-Tinkew, Moore, Matthews, & Carrano, 2007).

With regard to paternal externalizing psychopathology, the degree of antisocial behavior in fathers was found to moderate the relationship between the amount of time fathers lived with their children and child CP, such that paternal involvement was negatively associated with child CP, but only when fathers had

low levels of antisocial behavior themselves (Jaffe, Belsky, Harrington, Caspi, & Moffitt, 2006). Despite these provocative findings, this small literature is clearly still in its infancy and little research has examined paternal ADHD in relation to parenting.

Paternal ADHD and parenting. Though ADHD in fathers has received considerably less research attention than has maternal ADHD, a handful of studies have found that fathers with high levels of ADHD symptoms reported more lax and over reactive parenting toward their children with ADHD than fathers with lower levels of ADHD symptoms (Arnold, O'Leary, & Edwards, 1997; Harvey et al., 2003). Similarly, Psychogiou and colleagues (2007) have reported that paternal ADHD symptoms moderated the relationship between child ADHD symptoms and negative paternal parenting such that negative parenting was more strongly related to child ADHD symptoms among fathers with high levels of ADHD symptoms themselves. Moreover, negative paternal parenting was associated with child CP (Psychogiou et al., 2007). Importantly, however, Psychogiou and colleagues did not use a sample of children diagnosed with ADHD. Similar to the findings of Jaffe and colleagues (2006) described above, our own recent study with children diagnosed with ADHD found that paternal ADHD symptoms and child CP were significantly positively related, but only when fathers were highly involved in parenting (Mintz & Chronis-Tuscano, under review).

Taken together, these findings suggest both that fathers with psychopathology tend to display maladaptive parenting and that paternal

involvement may be related to child CP, particularly if fathers display maladaptive parenting. Therefore, one can surmise that high levels of psychopathology among involved fathers may be associated with negative child outcomes. This may especially be the case for fathers of children with ADHD who have ADHD symptoms themselves. That is, the core deficits associated with adult ADHD (e.g., inattention, disorganization, impulsivity, lack of followthrough) may be especially impairing when parenting children with ADHD, who require high levels of structure and consistency. Though there is emerging evidence that paternal involvement may be detrimental when fathers have high levels of ADHD symptoms (Arnold, O'Leary, & Edwards, 1997; Harvey et al., 2003; Mintz & Chronis-Tuscano, under review), much remains to be learned about which specific parenting behaviors may explain this association. Understanding which specific paternal parenting behaviors are associated with CP among children with ADHD has the potential to inform focused parenting interventions for this population.

Summary of existing literature. Taken together, existing theory and empirical findings suggest strongly that parenting and parental psychopathology are related to child CP. Though the majority of research examining these factors has focused on mothers, an emerging body of literature suggests that the unique role of fathers should be neither disputed nor ignored. Considering that fathers of children with ADHD are more likely to have psychopathology themselves (Chronis et al., 2003; Cunningham et al., 1988) and to report maladaptive parenting practices (e.g., coercive parenting; Rogers et al., 2009), it follows that

associations between paternal psychopathology and parenting should be examined in these families. Because of the high heritability of ADHD within families (Faraone et al., 2005) and emerging associations between maternal ADHD and parenting (Chronis-Tuscano et al., 2008; Harvey et al., 2003), research should focus specifically on understanding relations between paternal ADHD and parenting. Furthermore, considering that maternal psychopathology and parenting predict the development of CP in children with ADHD, examining these characteristics in fathers should be a priority within the literature.

Challenges in Conducting Research with Fathers

Rates of father involvement in research. Though it may seem that the clinical literature on fathers is alarmingly small, interest in this important topic has generally increased quite a bit in recent years, despite the many challenges that researchers face in studying fathers. Still, a recent review of child psychological psychopathology studies examining parental contributions to child psychological maladjustment found that only 28% of studies collected information from both mothers and fathers and analyzed these data separately (Cassano, Adrian, Veits, & Zeman, 2006). Low rates of including fathers in research is highly problematic considering the unique role that fathers play across cultures (Cabrera et al., 2000). Moreover, even within studies that include fathers, the samples are typically biased toward healthy fathers, as those who participate in research tend to have the lowest levels of antisocial behavior (Pfiffner et al, 2001). Considering the increased rates of antisocial behavior (and other types of psychopathology) among fathers of children with ADHD, such research is clearly not capturing large

portions of the father population, and these fathers may actually be the most interesting targets of research.

Challenges in studying fathers. Traditionally, fathers have been recruited for research through mothers. This has proved challenging, as many mothers function as "gatekeepers" whereby they prefer not to allow researchers to contact fathers for many reasons, including to protect that father's time (Allen & Hawkins, 1999; Fagan & Barnett, 2003). Because of this, many researchers have opted to sample fathers directly; yet, this technique tends to result in select samples of high-functioning fathers (Gibson-Davis, Edin, & McLanahan, 2005; Jarret, Roy, & Burton, 2002). When efforts have been made in face-to-face data-collection to obtain more representative samples (of adults in general), doing so has required enormous resources (e.g., 300 paid interview staff, \$50 payments to each of nearly 10,000 participants, etc.; Kessler et al., 2004).

Researchers choosing to make face-to-face (and even telephone) contact with fathers have encountered several challenges (Mitchell et al., 2007). Even when successfully contacted, fathers often decline research participation, citing a host of reasons (e.g., lack of time, wanting to protect their privacy, and not viewing the research as important). Stigma associated with men seeking help for mental illness or even everyday stress also impacts fathers' decisions regarding participation in clinical research (Addis & Mahalik, 2003). Fathers from low-income or immigrant families, specifically, are often reluctant to provide identifying information due to their legal status or stigma related to research participation in some ethnic minority communities (Mitchell et el., 2007). Within

the child clinical literature, a handful of studies have found that fathers view their children with ADHD as both less-symptomatic and less-impaired than mothers do, which may also impact their interest in participating in related research (Langberg et al., 2010; Tallmadge & Barkley, 1983). Importantly, monetary incentives typically do not increase rates of father participation in research (Brick, Hagedorn, Montaquilla, Roth, & Chapman, 2006); in fact, studies that provide as much as \$200 to participating fathers may be considered coercive (Parke, 2004). Indeed, it has been suggested that we know very little about the appropriateness and effectiveness of compensating fathers financially for research participation (Mitchell et al., 2007).

It is clear that studying fathers is challenging, especially within a clinical population. Researchers currently struggle with balancing many factors: ensuring the feasibility of a study design (e.g., manpower and economic resources; Kessler et al., 2004), obtaining a representative sample (Gibson-Davis et al., 2005), making participation attractive and reasonable (Brick et al., 2006), and keeping procedures ethical (Parke, 2004). Given these challenges, studies on fathers should prioritize the need to obtain a representative sample while being creative about how to best reach fathers. Researchers should also take care to make participation feasible and appealing for fathers, while recognizing that monetary incentives alone will not likely accomplish these goals.

Recommendations by prior researchers. As a result of the challenges reviewed above, several recommendations have been proposed related to future research with fathers: (1) approach fathers directly about participation in research

(i.e., avoid having mothers serve as gate-keepers), (2) emphasize to fathers the importance of their role in their own child's life and in contributing to learning more about fathers in general, (3) obtain as much direct paternal-report information as possible, (4) include non-custodial fathers, (5) make participation practical and confidential, (6) use rating scales that are normed for and relevant to fathers (Duhig, Phares, & Birkeland, 2002; Fabiano, 2007; Mitchell et al., 2007; Phares, Lopez, Fields, Kamboukos, & Duhig, 2005).

Limitations of Prior Research

Due to the many challenges of studying fathers described above, much of the currently available research on fathers suffers from both methodological and theoretical limitations. First, even among the studies that do address paternal psychopathology and parenting in relation to child behavior, few have collected this information directly through paternal report. Relying on maternal report of paternal psychopathology is certainly not ideal, as mothers tend to report fewer paternal symptoms than fathers do about themselves (Caspi et al., 2001). Second, most samples of fathers in prior literature have been limited in terms of custodial status, race/ethnicity, and SES. Related to this issue is that few studies have truly tapped into the population of fathers of children with ADHD, since fathers who participate in research are typically healthier than we know to be the case for fathers of children with ADHD (Pfiffner et al., 2001).

Present Study

Though the extensive lack of knowledge about fathers (relative to mothers) has begun to be addressed in recent research, there are clearly many

questions left unanswered that relate specifically to the relationship between paternal ADHD, paternal parenting, and child CP. A developmental psychopathology approach and the relevant theories of Belsky (1984) and Patterson (1982) suggest that parenting behavior may be rooted in a complex system of child psychopathology, parental psychopathology, and contextual factors. Thus, among families of children with ADHD, fathers with ADHD symptoms themselves may have children with higher levels of CP. Moreover, existing relevant theory and research findings suggest that parenting may be the specific *mechanism* explaining this association between paternal and child psychopathology. Yet, to our knowledge, no single study has examined this possible mechanism among families of children with ADHD--a gap which was addressed in the current study.

This project sought to examine a more representative sample of fathers and their children with ADHD with a specific focus on the relationships among paternal ADHD, paternal parenting, and child CP. Parenting and maternal psychopathology have emerged as robust predictors of CP among children with ADHD and, despite clear evidence that fathers make a critical contribution to their children's psychosocial wellbeing, we have yet to understand if and how *paternal* psychopathology and parenting are related to child CP in this population. Moreover, since the presence of CP is associated with the most serious developmental outcomes among children with ADHD, identifying specific modifiable risk factors associated with CP is of utmost clinical and public health importance. Finally, given high rates of comorbid mood, anxiety, and substance

use problems among adults with ADHD (Kessler et al., 2006), clarifying potential associations between paternal ADHD symptoms, paternal parenting, and child CP in the context of comorbid symptoms is crucial.

The present study used internet data collection procedures with the goal of recruiting a larger and more representative sample of fathers of children with ADHD than previous studies. This method was also designed specifically to reduce previously-identified barriers to direct father participation in clinical research (e.g., high burden, lack of anonymity; Mitchell et al., 2007). Several prior studies have found that samples collected through the internet are considerably more diverse with respect to socioeconomic status, geographic region, gender, and ethnicity (Gosling, Vazire, Srivastava, & John, 2004; Murray & Fisher, 2002). Moreover, findings using this form of data collection are not significantly different from those obtained with traditional methods (e.g., mailings or university-based) (Gosling et al., 2004; Murray & Fisher, 2002). Thus, this innovation in data collection was expected to result in a larger and more diverse sample (in terms of demographic and clinical characteristics) than has been obtained in prior research on fathers.

Aims

Primary Aims

Primary Aim 1a. To obtain and describe a sample of fathers and their previously-diagnosed children with ADHD.

Hypothesis 1a. It was hypothesized that a sizeable number of fathers of children with ADHD would demonstrate clinically-significant levels of ADHD themselves as well as levels of other forms of psychopathology (i.e., depression, anxiety, and antisocial personality disorder) that was equal to or greater than population base-rates (Lenzenweger, Lane, Loranger, & Kessler, 2007; Kessler, Chiu, Demler, & Walters, 2005; Kessler et al., 2006). Additionally, it was hypothesized that previously diagnosed children with ADHD would evidence clinically-significant levels of impairment in the home and school environments, as well as high rates of comorbid Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) symptoms (i.e., CP).

Primary Aim 1b. To examine correlations among parenting, demographic and clinical characteristics of fathers and their children with ADHD.

Hypothesis 1b. Based on previous literature (Arnold et al., 1997; Harvey et al., 2003; Mintz & Chronis-Tuscano, under review), it was hypothesized that paternal ADHD symptoms would be negatively associated with paternal positive parenting, positively associated with paternal negative parenting, and positively associated with child CP. No specific hypotheses were put forth regarding correlations among variables beyond the primary study variables of interest.

Primary Aim 2a. To examine positive parenting as a mediator in the relationship between paternal ADHD levels and child CP, controlling for significant demographic variables.

Hypothesis 2a. It was hypothesized that paternal ADHD symptoms would be positively associated with child CP and negatively associated with paternal positive parenting. Moreover, it was hypothesized that the relationship between paternal ADHD symptoms and child CP would no longer be significant when paternal positive parenting was included in the equation. That is, paternal positive parenting was expected to significantly mediate the relationship between paternal ADHD levels and child CP, even when controlling for demographic variables.

Primary Aim 2b. To examine positive parenting as a mediator in the relationship between paternal ADHD levels and child CP, controlling for *both* significant demographic variables and additional forms of paternal psychopathology.

Hypothesis 2b. It was hypothesized that paternal ADHD symptoms would remain positively associated with child CP and negatively associated with paternal positive parenting. Moreover, it was hypothesized that the relationship between paternal ADHD symptoms and child CP would no longer be significant when paternal positive parenting was included in the equation. That is, paternal positive parenting was expected to significantly mediate the relationship between paternal ADHD levels and child CP, even when controlling for demographic variables and additional forms of paternal psychopathology.

Primary Aim 2c. To examine whether any differences in results were identified when child gender was considered as a moderator (i.e., moderated mediation).

Hypothesis 2c. Based on previous literature (Connell & Goodman, 2002; Flouri & Buchanan, 2002), it was hypothesized that results might differ when child gender was included in the model; however, no more specific hypotheses were set forth due to the lack of research including child gender as a variable in studies of families of children with ADHD (Johnston et al., 2012).

Primary Aim 3a. To examine negative parenting as a mediator in the relationship between paternal ADHD levels and child CP, controlling for significant demographic variables.

Hypothesis 3a. It was hypothesized that paternal ADHD symptoms would be positively associated with child CP and positively associated with paternal negative parenting. Moreover, it was hypothesized that the relationship between paternal ADHD symptoms and child CP would no longer be significant when paternal negative parenting was included in the equation. That is, paternal negative parenting was expected to significantly mediate the relationship between paternal ADHD levels and child CP, even when controlling for demographic variables.

Primary Aim 3b. To examine negative parenting as a mediator in the relationship between paternal ADHD levels and child CP, controlling for *both* significant demographic variables and additional forms of paternal psychopathology.

Hypothesis 3b. It was hypothesized that paternal ADHD symptoms would remain positively associated with child CP and positively associated with paternal negative parenting. Moreover, it was hypothesized that the relationship between paternal ADHD symptoms and child CP would no longer be significant when paternal negative parenting was included in the equation. That is, paternal negative parenting was also expected to significantly mediate the relationship between paternal ADHD levels and child CP, even when controlling for demographic variables and other forms of paternal psychopathology.

Primary Aim 3c. To examine whether any differences in results were identified when child gender was considered as a moderator (i.e., moderated mediation).

Hypothesis 3c. As with Aim 2c, it was hypothesized that results might differ when child gender was included in the model; however, no more specific hypotheses were put forth.

Method

Participants

Participants included 102 fathers and their 5-12 year-old children who had been previously diagnosed with ADHD. Fathers were recruited through a variety of methods, including print and email advertisements sent to schools and physicians' offices, advertisements on websites frequented by parents of children with ADHD (e.g., CHADD), announcements sent to parenting listserves, and through established ADHD clinical and research programs throughout the United States. A special effort was made to advertise through newsletters, websites, and listserves primarily serving fathers as parents (in an effort to reduce the impact of mothers functioning at gatekeepers to participation). For inclusion in the study, children were required to: (1) have a previous diagnosis of ADHD as reported by fathers, (2) live with at least one biological parent, and (3) be between the ages of 5 and 12. Fathers in this study were also required to be the biological parent of the target child, to increase variability in paternal ADHD symptoms. Children and fathers taking ADHD medications were included in the study, but medication status was examined as a covariate in the analyses where appropriate. Demographic characteristics for fathers and children included in the study are presented in Tables 1 and 2, respectively. The sample is also discussed in more detail as part of the Aim 1a results.

Procedures

Fathers who saw the study advertised were provided with a web-based link allowing them to participate through Qualtrics (www.qualtrics.com). Each

father first completed an online informed consent form. Following consent, he completed online measures assessing demographic information regarding himself and his child, his parenting behaviors, and his own psychopathology (see Appendix A). Once a father completed these measures, he was provided with the option of using his email address to be entered into a raffle for a 1 in 50 chance to win a gift card for Amazon.com. Throughout the data collection period, two fathers were awarded with these gift certificates. Fathers were not required to provide identifying information about themselves or their child (i.e., email) unless they wished to be entered into the raffle or contacted for future studies conducted by the Maryland ADHD Program.

Measures

Child psychopathology measures. The Disruptive Behavior Disorder (DBD) symptom checklist (Pelham et al., 1992), which includes all DSM-IV symptoms of ADHD, ODD, and CD, was completed by each father as a measure of both child ADHD and CP. ADHD symptoms endorsed by fathers as occurring "pretty much" or "very much" in either the school or home setting were considered clinically significant and were included in the ADHD symptom count. Internal consistency for the DBD was excellent to good on the ADHD ($\alpha = 0.87$), ODD ($\alpha = 0.80$) and CD ($\alpha = 0.92$) scales.

To examine impairment across settings, fathers completed two items from the Children's Impairment Rating Scale (CIRS; Fabiano et al., 2006): (1) "How do your child's problems affect his or her academic progress in school?" and (2)

"How do your child's problems affect your family in general?" 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 as well as predictive validity (Fabiano et al., 2006). A score of 3 or above on one of these 7-point scales indicates clinical impairment (Fabiano et al., 2006). Internal consistency for these two items from the CIRS was good (α = 0.82). Notably, both the DBD and CIRS have been used in studies of fathers of children with ADHD (Fabiano et al., 2009).

Paternal parenting measures. Fathers completed four subscales of the Alabama Parenting Questionnaire (APQ; Shelton et al., 1996) to assess certain parenting practices. This version of the APQ is a 39-item measure on which fathers were asked to indicate the frequency with which they implement the following parenting practices: Involvement (e.g., "You have a friendly talk with your child," "You attend PTA meetings, parent/teacher conferences, or other meetings at your child's school"), Positive Parenting (e.g., "You let your child know when he/she is doing a good job with something," "You praise your child if he/she behaves well"), Inconsistent Discipline (e.g., "You threaten to punish your child and then do not actually punish them," "The punishment you give your child depends on your mood), and Poor Monitoring/Supervision (e.g., "Your child is out with friends that you don't know," "Your child is at home without adult supervision"). Internal consistency for this version of the APQ was good to acceptable on the Involvement ($\alpha = 0.80$), Positive Parenting ($\alpha = 0.78$), Inconsistent Discipline ($\alpha = 0.83$), and Poor Monitoring/Supervision ($\alpha = 0.76$)

scales. Importantly, the original sample for testing the reliability of this measure included fathers (Shelton et al., 1996). Of note, the 3-item Corporal Punishment subscale from the original 42-item questionnaire was not included due to the fact that participants were anonymous and endorsement of some of these items might have constituted an ethical responsibility to follow up with families on the part of the researchers as mandated reporters of child maltreatment.

As an additional measure of paternal parenting, the Laxness and Overreactivity subscales of the Parenting Scale (PS; Arnold et al., 1993) were completed by fathers. The PS is comprised of Likert-style items with prompts and associated specific anchors. For example, the Laxness subscale includes prompts (e.g., "When my child is out of my sight...") and anchors (e.g., "I often don't know what my child is doing" or "I always have a good idea of what my child is doing") assessing lax parenting. Similarly, the Overreactivity subscale includes prompts (e.g., "If my child misbehaves and then acts sorry...") and anchors (e.g., "I handle the problem like I usually would" or "I let it go that time") assessing overreactive parenting. This questionnaire reliably measures parenting strategies, and internal consistency was good for the Laxness ($\alpha = 0.82$) and Overreactivity $(\alpha = 0.80)$ subscales. The PS has also been used in prior studies with fathers (Arnold et al., 1997; Harvey et al., 2003). Of note, one item from the Overreactivity subscale was removed for this study that assessed parenting behavior related to spanking. This was done for the same reasons described above with regard to reconciling ethical responsibilities in the context of the anonymous nature of internet data collection.

The APQ and PS have been used in previous studies examining paternal ADHD symptoms and parenting (Arnold et al., 1997; Harvey et al., 2003). These particular subscales from the APQ and PS were selected based on existing evidence that parents of children with ADHD (either with or without ADHD symptoms themselves) experience difficulty with these specific parenting behaviors (Arnold et al., 1997; Chronis-Tuscano et al., 2008; Harvey et al., 2003; Murray and Johnston, 2006; Rogers et al., 2009). For instance, as reviewed previously, the impulsivity and inattention associated with adult ADHD symptom presentation likely impacts the consistency with which parents discipline and monitor their children, respectively. This may result in lax or overreactive parenting, as well as lower rates of positive parenting behavior—all of which have been associated with child CP.

Paternal parenting variables were measured via direct-report only from fathers, rather than also obtaining collateral maternal reports. Collection of measures from other informants could interfere with our intention to keep father participation confidential and minimize participant burden (Mitchell et al., 2007). Furthermore, collecting self-reports of parenting behavior is well-accepted within the literature (e.g., Arnold, O'Leary, Wolff, & Acker, 1993; Shelton, Frick, & Wooton, 1996).

Paternal psychopathology. Fathers completed the Conners' Adult ADHD Rating Scales – Short Version (CAARS-S:S, Conners, Erhardt, & Sparrow, 2003) as a dimensional measure of their current ADHD symptoms. The CAARS is a reliable and valid measure of ADHD symptoms for use with adults that assesses

the core features of ADHD as seen in children and adolescents, while adding content unique to the adult expression of ADHD (Conners et al., 1999; Erhardt et al., 1999). The original sample testing the reliability of and establishing norms for this measure included adult males (Erhardt et al., 1999). The short version of the original scale consists of 26 items that are rated using a 4-point Likert scale. This version consists of four subscales (inattention/memory, hyperactivity, impulsivity, and self-concept) and a 12-item overall ADHD index score (Conners, Erhardt, & Sparrow, 1998) that are converted to T-scores, which allows for comparison to a normative population. A T-score equal to or higher than 65 is considered clinically-significant. Internal consistency for the CAARS-S:S in this study was good to acceptable on the Inattention/Memory ($\alpha = 0.83$), Hyperactivity/ Restlessness ($\alpha = 0.82$), Impulsivity/Emotionality ($\alpha = 0.77$), Self-Concept ($\alpha = 0.86$), and Overall ADHD Index ($\alpha = 0.86$) scales. The overall index score was used to measure paternal ADHD levels in the primary analyses.

As with the paternal parenting variables, paternal psychopathology was measured through direct father-report. The practice of collecting collateral report for adult psychopathology, specifically ADHD, is rooted in concerns about the accuracy with which adults are able to report on their own ADHD symptoms (McGough & Barkley, 2004). However, there is ample evidence suggesting that adults likely report on their own ADHD symptoms with relative accuracy (Conners et al., 1999; Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999), and the inter-rater reliability between self- and investigator-report of ADHD in adults is .45-.65 (Adler et al., 2008). Therefore, though collecting both self- and

collateral-report of paternal ADHD symptoms (and other forms of psychopathology) is preferable, supplementing with collateral report does not appear to be critical. Moreover, as discussed above, a primary goal of this study was to facilitate confidential and anonymous father participation.

Fathers also completed a self-report broadband measure of their own psychopathology. This information was used to clarify that any findings with regard to associations between paternal ADHD and either parenting or child CP were not better accounted for by the presence of other forms of paternal psychopathology. The Adult Self-Report Form (ASR; Achenbach & Rescorla, 2003) contains broadband scales measuring mental health problems experienced by adults within the last six months. The scales that were utilized in this study were DSM-oriented scales measuring depressive problems, anxiety problems, and antisocial personality problems. Though the measure allows for both dimensional and categorical diagnostic impressions, dimensional measures were used to maximize variability. Additionally, raw score data (rather than T-score data) were utilized for analyses, as recommended by the developers in order to take account of the full range of variation in these scales (Achenbach & Rescorla, 2003). Tscores were utilized for descriptive purposes, as scores 70 or higher are considered in the clinical-range and scores 65-69 are considered to be borderlineclinical. The ASR has been found to have good test-retest reliability (mean r for all DSM-oriented scales is .83) as well as good content and criterion-related validity (Achenbach & Rescorla, 2003). Internal consistency for the ASR in this

study was good to acceptable on the Depressive Problems ($\alpha = 0.84$), Anxiety Problems ($\alpha = 0.71$), and Antisocial Personality Problems ($\alpha = 0.87$) scales.

Fathers also completed one-item scales that assessed alcohol and substance use problems. Specifically, fathers rated the frequency with which they were either drunk or used illegal substances in the past six months as either "not at all," "sometimes," or "often."

Demographic information. Fathers were also asked to complete a brief demographic form about their child and themselves. Child demographic information included: age, race/ethnicity, gender, previous diagnosis of ADHD (yes/no), and ADHD medication status (yes/no). Paternal demographic information included: age, race/ethnicity, educational level, marital status (in relation to the child's biological mother), residential status (yes/no), frequency of contact with his child (if not residential), ADHD medication status (yes/no), and annual family income.

Marital/relationship satisfaction. Fathers also completed a one-item scale assessing their marital/relationship satisfaction if they were married to or cohabitating with their child's mother. This item was a 7-point Likert scale ranging from "very dissatisfied" to "very satisfied", which was adapted from the

¹ These items were initially intended to be given as part of the ASR, which asks respondents to estimate the number of instances over the past six months when they have been drunk/used illegal substances. However, an error in designing the online form resulted in fathers' responses being coded by frequency label (i.e., 0=not at all, 1=sometimes, 2=often), similar to the other ASR

items.

Dyadic Adjustment Scale (DAS; Spanier, 1976). This item is correlated .76 with the full DAS (Goodwin, 1992).

Results

Missing Data

Two hundred fathers began the qualtrics survey and 140 completed the majority of items regarding paternal and child demographic information (see Figure 1 for disposition of participants). Fathers who completed all online measures (i.e., DBD, CAARS-S:S, ASR, APQ, and PS) were considered to have complete data. Missing data due to missing items from questionnaires were handled in two ways. First, all data from measures assessing child psychopathology, paternal psychopathology, and paternal parenting were prorated, such that participants who completed at least a predetermined minimum number of items within each subscale (≥80%) were given a prorated score based on the number of items completed. Second, participants for whom prorated scores could not be computed based on an abundance of missing items were dropped from the analyses (N=2).

Because bootstrapping analyses cannot be performed with missing data codes (Hayes, 2012), hot deck imputation (Andridge & Little, 2010; Myers, 2011) was implemented for missing demographic variables that were included as covariates in mediation analyses. This was necessary only for marital satisfaction. Because fathers who were not married or cohabitating with their child's mother were not asked to complete a measure of marital/relationship satisfaction, dropping these fathers from the analyses completely would have limited the sample substantially in terms of both size and representativeness. Hot deck imputation replaces missing values with the value of a similar "donor" participant

in the dataset that matches the "donee" on researcher-specified parameters. Parameter variables should be: (1) likely to be related to the variable with missing data, (2) missing little to no data themselves, and (3) not of substantial theoretical interest to the research question at hand. The number of parameters that can be specified is commensurate with sample size; thus, one parameter variable was specified for this imputation (i.e., paternal education level). Hot deck imputation holds several advantages over other methods used for missing data (e.g., list-wise deletion, mean substitutions), including: (1) its utility for data that are Missing Not at Random (MNAR), (2) the conservation of statistical power by avoiding the need to drop cases, and (3) the likelihood that imputed values are more realistic given that they are based on values elsewhere in the sample (Roth, 1994).

Preliminary Analyses

All data were downloaded from the Qualtrics website, and the database was cleaned and verified by the principal investigator using SPSS Statistics GradPack 19.0.0 (www.spss.com). Prior to conducting planned analyses, all variables were examined for distributional properties and outliers using methods discussed by Tabachnick & Fidell (1996, 2001, 2007). Prior to the main analyses, preliminary analyses were conducted to examine if composite factors could be created for paternal parenting, in order to minimize the number of analyses conducted for Aims 2 and 3.

Comparison of completers to non-completers. Preliminary analyses were also conducted to determine if the final sample for this study (N=102) differed significantly on a variety of demographic characteristics from those with

insufficient data or who discontinued early (and for whom demographic data were available; N=38). Independent samples t-tests were conducted for the following continuous variables: family income, paternal education level, child age, paternal age, and paternal marital satisfaction. Chi-square analyses were conducted for the following dichotomous variables: child gender, paternal residential status, paternal marital status (to child's mother), paternal ADHD medication status, child ADHD medication status, paternal minority status, and child minority status. Fathers in the final sample were significantly older (M=41.90, SE=0.74) than fathers who began but did not complete the survey (M=37.74, SE=1.65) (t(88)=2.51, p < .05). Fathers in the final sample also had a significantly higher mean family income level (M = \$156,230.10, SE = \$14,628.11) than those who began but did not complete the survey (M=\$71,159.74, SE=\$10,039.91) (t(115)=-2.53, p <.05). No significant differences were found for paternal education level (t(129)= .52, p > .05), child age (t(131)=-1.48, p > .05), or paternal marital satisfaction (t(42)=1.30, p > .05). The groups also differed on percentage of children who were racial/ethnic minorities ($\chi^2(1, N=130)=10.50, p < .05$), such that the odds of children being Caucasian were 4.35 times higher if their fathers were included in the final sample. The groups also differed on percentage of fathers who were racial/ethnic minorities ($\chi^2(1, N=130)=11.72, p < .05$), such that the odds of fathers being Caucasian were 5.00 times higher if included in the final sample. The groups also differed on percentage of children taking ADHD medication $(\chi^2(1, N=131)=17.02, p < .001)$, such that the odds of children taking medication was 5.85 times higher if included in the final sample. No differences between

groups emerged regarding child gender ($\chi^2(1, N=135)=3.81, p > .05$), paternal residential status ($\chi^2(1, N=131)=.96, p > .05$), paternal marital status ($\chi^2(1, N=131)=0.01, p > .05$), and paternal ADHD medication status ($\chi^2(2, N=130)=2.50, p > .05$).

Variable examination. Normality was assessed for all variables via visual inspection of the distribution graphs and assessment of skewness and kurtosis values (Field, 2004; Hair, et al., 2006; Tabachnick & Fidell, 2001, 2007). First, all variables were examined for outliers using both visual inspection of Boxplots and statistically by converting the variables to standardized z-scores and examining those with z-scores greater than 3.29 (Tabachnick & Fidell, 2001, 2007). Though it appeared as though outliers were present in the data for several of the variables, it was determined that these outliers were part of the expected variability within a clinical population and were of central interest to the aims of this study. Therefore, no outliers were removed from subsequent analyses.

Z-scores were then computed for skewness and kurtosis using the standard error term, for all variables, and z-scores equal to or less than 3.29 was the criterion used, as recommended for small samples (Field, 2004; Hair, et al., 2006). Though several variables indicated high skew/kurtosis, it was determined that no variables would be transformed for these analyses. This is based on specific recommendations for bootstrapping analyses, which use resampling techniques and do not assume distribution normality (Hayes & Preacher, 2010), as well as statistician recommendations against transforming study variables (with the exception of the dependent variable; Pedhazur, 1997; M. Wang, personal

communication, March 26, 2012) Beyond this, however, the high skew (z=8.561) and kurtosis (z=9.637) of the Antisocial Personality Disorder (ASPD) symptoms variable were of particular concern in the current sample. A visual examination of this variable revealed that dichotomizing this variable was not appropriate given its distribution (Appendix B). Moreover, despite its positive skew, considerable variability appeared to be present in the variable. Specifically, scores on this variable ranged from T≤55 (i.e., completely asymptomatic; n=52) to T≥65 (i.e., borderline clinical; n=12). Therefore, the ASPD variable was left in its original form for the study analyses. Distribution statistics for all variables, including mean, standard deviation, range, skewness and kurtosis are presented in Appendix C.

Data reduction. Preliminary analyses were conducted to examine if data reduction was possible for the following constructs: Negative Parenting and Positive Parenting. For both of these constructs, multiple measures were used to assess the construct. Relatedness of measures within each construct was assessed using Pearson-product moment correlations between different measures of a construct. Specifically, for Positive Parenting, correlations examined the relation between the following subscales: Involvement (APQ) and Positive Parenting (APQ). Results examining subscales indicated for the Positive Parenting composite suggested a high degree of relatedness. Specifically, APQ Involvement was significantly positively related to APQ Positive Parenting, r=.658 p<.01. As a result, z-scores for these two subscales were averaged to create a composite

Positive Parenting score in which higher scores are reflective of higher levels of positive parenting behaviors.

For Negative Parenting, correlations examined the relation between the following subscales: Inconsistent Discipline (APQ), Poor Monitoring/Supervision (APQ), Laxness (PS), and Overreactivity (PS). Results examining subscales indicated for the Negative Parenting composite suggested medium to high degrees of relatedness (ranging from r=.284 to r=.583, all p<.01). As a result, z-scores for these four subscales were averaged to create a composite Negative Parenting score in which higher scores are reflective of higher levels of negative parenting behaviors.

Lastly, reliability analyses examined alpha levels within the newly-created composites. These analyses yielded good internal consistency (Positive Parenting: α =0.86, Negative Parenting: α =0.89). Therefore, the composites of Positive Parenting (PP) and Negative Parenting (NP) were used for analyses to examine Aims 2 and 3.

Preliminary regression analyses. Prior to running the primary analyses, we conducted regression analyses to determine the extent to which the various demographic variables were associated with paternal positive parenting (PP), paternal negative parenting (NP), and child CP, necessitating their inclusion as control variables in the primary analyses. For each predicted variable (i.e., paternal PP, paternal NP, and child CP), demographic variables were examined in two separate regression analyses: (1) child-related demographics, which included

age, race/ethnicity (Caucasian/Non-Caucasian), gender (male/female), and ADHD medication status (yes/no), and (2) paternal demographics, which included age, race/ethnicity (Caucasian/Non-Caucasian), educational level, total annual family income, ADHD medication status (yes/no), marital/cohabitating status (yes/no), marital satisfaction, and residential status (yes/no). All predictor variables associated with paternal PP, paternal NP, or child CP at a significance level of p < .05 in preliminary analyses were considered as covariates in the appropriate primary analyses.

Results indicated that significant demographic variables associated with child CP included marital status, residential status, marital satisfaction and paternal ADHD medication status. It was determined that paternal ADHD medication status was likely a proxy for the independent variable (paternal ADHD symptoms) based on theoretical reasons as well as the significant correlation between these two variables (r=.334, p<.01), and therefore this variable was not included as a covariate. Similarly, because paternal marital status and paternal residential status were significantly correlated (r=.389, p<.01), it was determined that each variable was likely a proxy for the other; thus, of the two, only paternal marital status was included in the model as a covariate. Therefore, paternal marital status and paternal marital satisfaction were included as covariates in all subsequent primary analyses (i.e., Aims 2 and 3). The only significant demographic variable that predicted paternal PP was child ADHD medication status. This variable was therefore included as a covariate in primary analyses with paternal PP as the proposed mediator (i.e., Aim 2). The only

significant demographic variable predicting paternal NP was paternal ADHD medication status. As explained previously, paternal ADHD medication status was not included as a covariate in the primary analyses. Therefore, no additional covariates were included in primary analyses with paternal NP as the proposed mediator (i.e., Aim 3).

Regression analyses were also conducted to determine the extent to which the various forms of paternal psychopathology (other than ADHD) were associated with paternal PP, paternal NP, and child CP. For each predicted variable (i.e., paternal PP, paternal NP, and child CP), all of the following psychopathology variables were examined together: depressive symptoms, anxiety symptoms, antisocial personality disorder (ASPD) symptoms, alcohol use, and substance use. All predictor variables associated with paternal PP, paternal NP, or child CP at a significance level of p < .05 in preliminary analyses were considered as additional covariates in Aims 2b and 3b in order to determine if results differed when additional forms of psychopathology were accounted for. Results indicated that only paternal ASPD symptoms were significantly related to both child CP and paternal NP. No variables significantly predicted paternal PP. Therefore, ASPD symptoms were included as an additional covariate in analyses examining Aims 2b and 3b.

Primary Analyses

Primary Aim 1a: To describe a sample of fathers and their previously-diagnosed children with ADHD. To address the current limitations in research on fathers of children with ADHD, our first aim for this project was to

obtain and describe a sample of this population (N=102). Despite efforts to obtain a demographically diverse sample in terms of race/ethnicity, educational level, and marital- and residential-status, fathers in this sample were primarily Caucasian (89%), highly-educated (68% with bachelors degree or higher), married to or cohabitating with their child's mother (83%), and living with the target child (97%). Sixty-six percent of fathers met the clinical cut-off on the CAARS ADHD Index (i.e., T-score \geq 65); 13% were taking medication for ADHD. Fathers also displayed significant levels of other types of psychopathology. Specifically, rates of clinically-significant depressive, anxiety, and ASPD symptoms were 10%, 4%, and 4%, respectively. Furthermore, there were high rates of borderline-clinical levels of depressive (22%), anxiety (8%), and ASPD (8%) symptoms. Fathers also engaged in a fair amount of alcohol and substance use. Specifically, 10% of fathers reported that they "sometimes" used substances in the last 6 months and another 1% reported they used "often." Regarding alcohol use, 20% of fathers reported having "sometimes" been drunk in the last 6 months and another 6% reporting being drunk "often." Please see Table 1 for a full description of the fathers in this sample.

Children in this sample were also primarily Caucasian (87%), and the gender-breakdown was consistent with that which is typically found among children with ADHD (76% male). The majority of these children were taking medication for ADHD (76%). Although these children were all reported by their fathers to have a previous diagnosis of ADHD, when strict DSM-IV criteria for ADHD diagnosis was applied to this sample (i.e., 6 or more clinically-significant

symptoms of inattention and/or hyperactivity/impulsivity as well as evidence of cross-situational impairment), only 66% of the children met the symptom and impairment criteria threshold based on father report alone.² Of note, rates of clinically-significant impairment across both home (88%) and school (93%) were high. Please see Table 2 for a full description of the children in this sample.

Primary Aim 1b. To examine correlations among parenting, demographic and clinical characteristics of fathers and their children with ADHD. Correlation analyses were conducted to examine the relationships between the independent, dependent, mediator and demographic variables of interest. Pearson product-moment correlations were used to examine the relationships between continuous variables while Kendall's tau was used to examine the relationships between dichotomous variables or a dichotomous and a continuous variable. The resulting correlation matrix is presented in Table 3.

Results indicated that paternal ADHD levels were positively related to paternal NP (r=.260, p<.01). However, paternal ADHD levels were not significantly related to either paternal PP or child CP. Child CP and paternal NP were positively related (r=.477, p<.01), but child CP were not related to paternal PP. Given these findings, exploratory analyses were conducted examining the two subscales comprising the paternal PP composite (APQ Positive Parenting and APQ Involvement) separately as related to paternal ADHD levels and child CP;

² Subsequent analyses were therefore repeated with the smaller sample of fathers and their children (N=67) whose diagnoses of ADHD were confirmed via the process described above. No differences in any of the findings emerged.

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results confirmed that neither of these subscales was significantly associated with either paternal ADHD levels or child CP.

In terms of demographic and paternal psychopathology variables, paternal ADHD levels were positively related to paternal alcohol abuse (r=.235, p<.05), substance abuse (r=.285, p<.01), depressive symptoms (r=.652, p<.01), anxiety symptoms (r=.467, p<.01), and ASPD symptoms (r=.518, p<.01), such that fathers with higher levels of ADHD also had higher levels of other forms of psychopathology. Child CP were also negatively related to paternal marital/relationship satisfaction (r=-.325, p<.01) and positively related to paternal depressive symptoms (r=. 364, p<.01), and ASPD symptoms (r=.528, p<.01), such that children with higher levels of CP had fathers with lower levels of marital/relationship satisfaction and higher levels of depressive and ASPD symptoms.

PP was negatively related to paternal substance abuse (r=-.223, p<.05) and depressive symptoms (r=-.197, p<.05), such that fathers who reported using higher levels of PP had lower levels of substance abuse and depressive symptoms. Finally, NP was negatively related to paternal education level (r=-.237, p<.05), marital/relationship satisfaction (r=-.292, p<.01), and total household income (r=-.226, p<.05) and positively related to paternal ADHD medication status $(\tau=.252, p<.01)$, substance abuse (r=.322, p<.01), alcohol abuse (r=.302, p<.01), depressive symptoms (r=.447, p<.01), anxiety symptoms (r=.318, p<.01), and ASPD symptoms (r=.540, p<.01), such that fathers who displayed higher levels of NP had lower levels of education, lower marital/relationship satisfaction, lower

annual income, were more likely to be medicated for ADHD, and had higher levels of various forms of psychopathology.

Mediation analyses. As defined by Baron and Kenny (1986), a mediator is a variable which may explain why certain effects occur. Mediation has been traditionally assessed using the causal steps approach (Baron & Kenny, 1986), which requires that each pathway of the mediation model (e.g., Figures 2-5) meet statistical significance. The causal steps approach, however, has been highly criticized in recent years with the advancement of statistical strategies and software tools (for a review, see Hayes, 2012b). First, the traditional approach to mediation is one of the lowest in power (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002), as the requirement of three separate statistically significant results to support mediation increases the likelihood that a false null hypothesis will not be rejected. Second, causal steps analysis logically infers the presence of an intervening effect of an independent variable (IV) on a dependent variable (DV) through a mediator by demonstrating that the IV affects the mediator and the mediator, in turn, affects the DV. This approach, however, fails to directly measure or quantify the size of the intervening effect (Hayes, 2009). Third, the causal steps approach requires that a total effect of an IV on a DV must exist in order for mediation to be present, so no further analysis is required to explain the process by which non-significant total effects occur. There are many cases, however, in which an IV may impact a DV indirectly through a mediator when no total effect is found (Hayes, 2009, 2012b). For example, Hayes (2012b) offers the scenario in which an IV is linked to a DV indirectly through only two

mechanisms working in equal but opposite directions (i.e., one mediating effect is positive and the other is negative). Though the IV clearly impacts the DV indirectly through two mediating variables, the total effect will sum to zero. Hence, the causal approach, by requiring the presence of a significant total effect prior to further analysis, may fail to investigate significant underlying indirect effects.

Unlike traditional statistical approaches to mediation analysis, mediation was assessed in the present study by directly examining the indirect effect of the IV (i.e., paternal ADHD symptoms) on the DV (i.e., child CP) through the mediator (i.e., paternal positive or negative parenting). Although some authors have drawn a distinction between "mediation" and "indirect effects," the present study follows other statisticians in using the terms interchangeably (e.g., Hayes, 2009; MacKinnon, Lockwood, & Williams, 2004; Rucker, Preacher, Tormala, & Petty, 2011). Hence, results of the various causal steps pathways are presented in the present study only to aid in interpretation of mediation findings. Support for mediation in the present study rested primarily on findings of significant indirect effects.

Bootstrapping analysis is the method currently recommended for testing of indirect effects, especially in small samples (Hayes, 2009; MacKinnon et al., 2004; Preacher & Hayes, 2004; Williams & MacKinnon, 2008). Unlike other tests of indirect effects (e.g., the Sobel test), bootstrapping makes no assumptions about the shape of the sampling distribution or the normality of the individual variables (Hayes, 2009). It treats the study sample as a smaller representation of

the larger population. It iteratively resamples from the data pool, selecting data from an individual participant at random, placing that participant's data back into the pool, and randomly selecting again from the overall pool. Once the resample is constructed, the indirect effect is estimated. This process is completed k times, with statisticians recommending that k equal at least 5000 (Hayes, 2009). The values of the indirect effect are then sorted from smallest to largest, and endpoints are adjusted to yield a bias-corrected confidence interval to estimate the size of the indirect effect. The effect is considered statistically significant at p < .05 if the 95% confidence interval range does not include zero.

Primary Aim 2: To examine paternal PP as a mediator in the relationship between paternal ADHD levels and child CP, controlling for significant demographic variables (Aim 2a) and for additional paternal psychopathology (Aim 2b). The proposed mediation model is outlined in Figure 2. In the figure, the direct effect of paternal ADHD symptoms on child CP is noted in path c; while the indirect effect of paternal ADHD symptoms is the product (i.e., ab) of paternal ADHD symptoms on paternal PP (path a) and of paternal PP on child CP after controlling for paternal ADHD symptoms (path b). The path denoted c' represents the coefficient of paternal ADHD symptoms on child CP after the addition of paternal PP. In the model, paternal marital status, paternal marital satisfaction, and child ADHD medication status were included as covariates.

Primary Aim 2a. The test of this path is presented in Table 4. Paternal ADHD symptoms did not exert a significant effect on either child CP (B=.069,

p=.093) or paternal PP (B=-.005, p=.607). Additionally, paternal PP did not exert a significant effect on child CP when the effects of paternal ADHD symptoms were partialled out (B=-.344, p=.440). Further, the relationship between paternal ADHD symptoms and child CP were not significant after accounting for paternal PP (B=.067, p=.106). Finally, bootstrapping analyses confirmed that <u>paternal PP</u> did *not* significantly mediate the relationship between paternal ADHD symptoms and child CP, M_{β} =.0015, S.E.=.0051, 95% CI=-.0043 to .0190.

Primary Aim 2b. These analyses were repeated including paternal ASPD symptoms as an additional covariate to examine whether results changed when other forms of paternal psychopathology were included in the model. The test of this path is presented in Table 5.

Paternal ADHD symptoms did not exert a significant effect on either child CP (B=-.038, p=.383) or paternal PP (B=.004, p=.739). Additionally, paternal PP did not exert a significant effect on child CP when the effects of paternal ADHD symptoms were partialled out (B=-.078, p=.848). Further, the relationship between paternal ADHD symptoms and child CP were not significant after accounting for paternal PP (B=-.038, p=.386). Finally, bootstrapping analyses confirmed that paternal PP did *not* significantly mediate the relationship between paternal ADHD symptoms and child CP, M_{β} =-.0003, S.E.=.0043, 95% CI=-.0117 to .0072.

Primary Aim 2c. Based on previous findings that paternal psychopathology may differentially impact either the parenting process for or the psychosocial adjustment of male and female children (Connell & Goodman,

2002; Flouri & Buchanan, 2002), analyses were conducted considering gender as a possible moderator. Results were consistent with those reported above when these moderated mediation models were examined (i.e., gender was not a moderator). A more comprehensive report of these results is presented in Appendix D.

Primary Aim 3: To examine paternal NP as a mediator in the relationship between paternal ADHD levels and child CP, controlling for significant demographic variables (Aim 3a) and for additional paternal psychopathology (Aim 3b). The proposed mediation model is outlined in Figure 3. In the figure, the direct effect of paternal ADHD symptoms on child CP is noted in path c; while the indirect effect of paternal ADHD symptoms is the product (i.e., ab) of paternal ADHD symptoms on paternal NP (path a) and of paternal NP on child CP after controlling for paternal ADHD symptoms (path b). The path denoted c' represents the coefficient of paternal ADHD symptoms on child CP after the addition of paternal NP. In the model, paternal marital status and paternal marital satisfaction were included as covariates.

Primary Aim 3a. The test of this path is presented in Table 6. Paternal ADHD symptoms did not exert a significant effect on child CP (B=.069, p=.093), but it exerted a significant effect on paternal NP (B=.020, p=.008). Additionally, paternal NP exerted a significant effect on child CP when the effects of paternal ADHD symptoms were partialled out (B=2.339, p=.000). Further, the relationship between paternal ADHD symptoms and child CP was not significant after accounting for paternal NP (B=.026, p=.506). Finally, bootstrapping analyses

indicated that paternal NP mediated the relationship between paternal ADHD symptoms and child CP, M_{β} =.0434, S.E.=.0241, 95% CI=.0083 to .1057.

Primary Aim 3b. These analyses were repeated including paternal ASPD symptoms as an additional covariate to examine whether results changed when other forms of paternal psychopathology were included in the model. The test of this path is presented in Table 7.

Paternal ADHD symptoms did not exert a significant effect on child CP (B=-.038, p=.383) or paternal NP (B=-.002, p=.785). Additionally, paternal NP exerted a significant effect on child CP when the effects of paternal ADHD symptoms were partialled out (B=1.528, p=.008). Further, the relationship between paternal ADHD symptoms and child CP was not significant after accounting for paternal NP (B=-.036, p=.393). Finally, bootstrapping analyses confirmed that paternal NP did *not* significantly mediate the relationship between paternal ADHD symptoms and child CP when paternal ASPD symptoms were included in the model, M_{β} =-.0020, S.E.=.0137, 95% CI=-.0330 to .0226. In other words, paternal NP no longer significantly mediated the relationship between paternal ADHD symptoms and child CP when paternal ASPD symptoms were accounted for.

Primary Aim 3c. As described above (Aim 2c), analyses were conducted considering gender as a possible moderator. Results were consistent with those reported above when these moderated mediation models were examined (i.e., gender was not a moderator). A more comprehensive report of these results is presented in Appendix D.

Exploratory Analyses

Exploratory Analysis 1: Examining the role of paternal ASPD symptoms. Based on the results of the primary analyses above, exploratory analyses were conducted to better understand the role of paternal ASPD symptoms in the relationship between paternal ADHD symptoms, parenting, and child CP. Specifically, because the inclusion of paternal ASPD symptoms impacted whether or not paternal NP was found to significantly mediate the relationship between paternal ADHD symptoms and child CP, the same model was examined with paternal ASPD symptoms as the independent variable and controlling for paternal ADHD symptoms.

Exploratory Analysis 1a: To examine paternal PP as a mediator in the relationship between paternal ASPD symptoms and child CP in children. The proposed mediation model for these analyses is outlined in Figure 4. In this figure, the direct effect of paternal ASPD symptoms on child CP is noted in path c; while the indirect effect of paternal ASPD symptoms is the product (i.e., ab) of paternal ASPD symptoms on paternal PP (path a) and of paternal PP on child CP after controlling for paternal ASPD symptoms (path b). The path denoted c' represents the coefficient of paternal ASPD symptoms on child CP after the addition of paternal PP. In the model, paternal marital status, paternal marital satisfaction and child ADHD medication status were included as demographic covariates. Paternal ADHD symptoms were included as an additional covariate in order to further control for the effect of other forms of paternal psychopathology (Table 8).

Paternal ASPD symptoms exerted a significant effect on child CP (B=.419, p=.000) but not on paternal PP (B=-.030, p=.134). Additionally, paternal PP did not exert a significant effect on child CP when the effects of paternal ASPD symptoms were partialled out (B=-.078, p=.848). Further, the relationship between paternal ASPD symptoms and child CP were significant after accounting for paternal PP (B=.416, p=.000). Finally, bootstrapping analyses confirmed that paternal PP did *not* significantly mediate the relationship between paternal ASPD symptoms and child CP, M β =.0027, S.E.=.0153, 95% CI=-.0190 to .0361.

Exploratory Analysis 1b: To examine paternal NP as a mediator in the relationship between paternal ASPD symptoms and child CP. The proposed mediation model for these analyses is outlined in Figure 5. In this figure, the direct effect of paternal ASPD symptoms on child CP is noted in path c; while the indirect effect of paternal ASPD symptoms is the product (i.e., ab) of paternal ASPD symptoms on paternal NP (path a) and of paternal NP on child CP after controlling for paternal ASPD symptoms (path b). The path denoted c' represents the coefficient of paternal ASPD symptoms on child CP after the addition of paternal NP. In the model, paternal marital status and paternal marital satisfaction were included as demographic covariates. Paternal ADHD symptoms were included as an additional covariate in order to further control for the effect of other forms of paternal psychopathology (Table 9).

Paternal ASPD symptoms exerted a significant effect on both child CP (B=.419, p=.000) and paternal NP (B=.080, p=.000). Additionally, paternal NP exerted a significant effect on child CP when the effects of paternal ASPD

symptoms were partialled out (B=1.528, p=.008). Further, the relationship between paternal ASPD symptoms and child CP remained significant after accounting for paternal NP (B=.300, p=.002). Finally, bootstrapping analyses indicated that <u>paternal NP mediated the relationship between paternal ASPD symptoms and child CP</u>, M β =.1188, S.E.=.0527, 95% CI=.0353 to .2448. Overall, this model accounted for 36% of the variance in child CP.

Exploratory Analysis 1c: To examine whether any differences in results were identified when child gender was considered as a moderator (i.e., moderated mediation). Results were consistent with those reported above when these moderated mediation models were examined (i.e., gender was not a moderator). A more comprehensive report of these results is presented in Appendix E.

Exploratory Analysis 2: Examining interactions between paternal ADHD and ASPD symptoms. Based on the results above, which indicated that paternal ASPD symptoms were a stronger predictor than paternal ADHD symptoms in the proposed model, further exploratory analyses were examined to determine potential interactive effects of paternal ADHD and ASPD symptoms. Specifically, it was hypothesized that paternal ASPD and ADHD symptoms might interact such that higher levels of both disorders would be associated with the lowest levels of paternal PP, the highest levels of paternal NP, and the highest levels of child CP. A series of linear regression analyses were therefore conducted in order to examine these potential interactive effects.

Exploratory Analysis 2a: To examine the interaction between paternal

ADHD and ASPD symptoms as related to child CP. On the first step of these analyses, demographic variables significantly related to child CP (i.e., paternal marital satisfaction and paternal marital status) were entered. On the second step, paternal ADHD and ASPD symptoms were entered simultaneously. In the third step, we examined the extent to which the interaction between paternal ADHD and ASPD symptoms contributed to the prediction of child CP above and beyond the first two steps. The following interaction variable was created by computing the product of paternal ADHD and ASPD symptoms: ASPD x ADHD. Results indicated that there was *not* a significant interactive effect of paternal ADHD and ASPD symptoms on child CP; however, paternal ASPD symptoms were independently associated with child CP when paternal ADHD symptoms were considered in the model, whereas paternal ADHD symptoms were not (Table 10).

Exploratory Analysis 2b: To examine the interaction between paternal ADHD and ASPD symptoms as related to paternal PP. On the first step of these analyses, demographic variables significantly related to paternal PP (i.e., child ADHD medication status) were entered. On the second step, paternal ADHD and ASPD symptoms were entered simultaneously. In the third step, we examined the extent to which the interaction between paternal ADHD and ASPD symptoms contributed to the prediction of paternal PP above and beyond the first two steps. Results indicated that there was no significant interactive effect on paternal PP. Additionally, neither paternal ADHD symptoms nor paternal ASPD symptoms were independently associated with paternal PP (Table 11).

Exploratory Analysis 2c: To examine the interaction between paternal

ADHD and ASPD symptoms as related to paternal NP. On the first step of these analyses, paternal ADHD and ASPD symptoms were entered simultaneously. In the second step, we examined the extent to which the interaction between paternal ADHD and ASPD symptoms contributed to the prediction of paternal NP above and beyond the first step. Results indicated that there was not a significant interactive effect of paternal ADHD and ASPD on paternal NP; however, paternal ASPD symptoms were independently associated with paternal NP when paternal ADHD symptoms were considered in the model, while paternal ADHD symptoms were not (Table 12).

Discussion

The present study is the first to examine paternal parenting as a potential mediator in the relationship between paternal ADHD symptoms and child CP in children with previously-diagnosed ADHD. Importantly, this was also the first study to assess other forms of paternal psychopathology which may account for these relationships. Indeed, paternal negative parenting (which was comprised of the Alabama Parenting Questionnaire's Poor Monitoring and Supervision and Inconsistent Discipline subscales and the Parenting Scale's Laxness and Overreactivity subscales) emerged as a potential mediator in the relationship between paternal ADHD symptoms and child conduct problems; however, these findings were largely driven by paternal ASPD symptoms. These results are described and discussed in turn.

Consistent with prior findings suggesting links between paternal ADHD symptoms and negative paternal parenting (Arnold et al., 1997; Harvey et al., 2003) and between negative paternal parenting and child CP (Psychogiou et al., 2007), paternal ADHD symptoms were significantly associated with negative parenting, and negative parenting was also significantly associated with child CP. Paternal negative parenting significantly mediated the relationship between paternal ADHD symptoms and child CP, such that a significant amount of variance in child CP was better accounted for by paternal negative parenting than by paternal ADHD symptoms. These results are relatively consistent with longitudinal findings suggesting that *maternal* negative parenting may be the mechanism by which effects of a behavioral intervention for children with ADHD

were attenuated when mothers had higher levels of ADHD symptoms (Chronis-Tuscano et al., 2011).

When paternal ASPD symptoms were included in the models above, however, all of the previously-discussed associations were rendered nonsignificant. Exploratory analyses subsequently clarified that paternal ASPD symptoms (and not paternal ADHD symptoms) were associated with child CP, and this association was mediated by paternal negative parenting. Moreover, the uniquely powerful nature of paternal ASPD symptoms in the model was further confirmed by the lack of interactive effects between paternal ADHD and ASPD symptoms in predicting both paternal parenting and child CP. Given our hypotheses that paternal ADHD would be associated with various forms of negative parenting (e.g., inconsistency, overreactivity, laxness, poor monitoring), these findings were somewhat surprising. It was also surprising that there were no significant, unique associations between paternal ADHD and child CP. This suggests that the form of paternal psychopathology which may be of greatest concern clinically is ASPD, and negative parenting may be a primary mechanism by which paternal ASPD confers risk for CP upon children with ADHD.

The current results fit with prior findings demonstrating that paternal ASPD is associated with child CP (e.g., Kopp & Beauchaine, 2007; Lahey et al., 1988; Pfiffner et al., 2005) and that paternal ASPD is associated with negative parenting (e.g., Psychogiou et al., 2007). At the same time, these findings stand in contrast to the small literature examining paternal ADHD symptoms and parenting, which has consistently found significant associations between these

variables (Arnold et al., 1997; Harvey et al., 2003; Psychogiou et al., 2007). With the exception of Psychogiou and colleagues (2007), these previous studies linking paternal ADHD and maladaptive parenting did *not* measure paternal ASPD symptoms; thus, their findings and related conclusions may be called into question in light of the current study. Indeed, a recent review highlights the importance of considering other forms of psychopathology in research on parental ADHD (Johnston et al., 2012), and these findings support that recommendation.

Paternal negative parenting and ASPD symptoms accounted for a significant amount of the variance in child CP (i.e., 36%) in the current study. Given the documented intergenerational heritability of antisocial behavior (Ge et al., 1996), genetics likely account for a large portion of unexplained variance. Behavioral-genetic studies, however, suggest that genetics account for at most half of the observed variance in child CP, and therefore additional unmeasured factors likely also contribute to child CP in the current study (Moffitt & Caspi, 2007). Maternal parenting, specifically, has been identified as a moderator in the relationship between genetic risk factors and comorbid ADHD and CP in children (Lahey et al., 2011). Thus, in the context of available behavioral-genetic studies, the current findings highlight the need for future studies to examine additional forms of paternal psychopathology, the role of relevant maternal factors, and the role of genetics as related to CP among children with ADHD.

Paternal positive parenting (comprised of the Alabama Parenting

Questionnaire's Involvement and Positive Parenting subscales) was not a

significant mediator in the relationship between paternal ADHD symptoms (or

ASPD symptoms) and child CP. Consistent with this, the majority of previous literature among families of children with ADHD has not found paternal positive parenting to be significantly associated with paternal psychopathology (Arnold et al., 1997; Harvey et al., 2003; Psychogiou et al., 2007) or child CP (Kashdan et al., 2004; Pfiffner et al., 2005; Psychogiou et al., 2007). This stands in contrast to our hypotheses supported by cross-sectional research showing that lower levels of self-reported and observed maternal positive parenting were associated with the presence of child CP (Johnston, Murray, Hinshaw, Pelham, & Hoza, 2002; Kashdan et al., 2004). Moreover, longitudinal findings have identified early maternal positive parenting as a robust protective factor against child CP among families of children with ADHD (Chronis et al., 2007). Thus, perhaps child CP are associated with positive parenting only in mothers, but with negative parenting among both parents. To further clarify these relationships, future studies should examine psychopathology and positive and negative parenting in both parents simultaneously, ideally using a longitudinal design in order to more accurately examine these factors as predictors of the development of later child CP.

The results of the current study should be considered in light of its limitations. First, the use of anonymous participation via the internet was specifically chosen with the hope of obtaining a larger sample of fathers that was diverse with regard to race/ethnicity, education level, marital status, and residential status. Indeed, several previous studies examining internet data-collection have suggested that this method promotes the collection of a sample that is more diverse with respect to socioeconomic status and ethnicity (Gosling et

al., 2004; Murray & Fisher, 2002). On one hand, our sample evidenced rates of paternal psychopathology that were remarkably higher than rates found in epidemiological studies. Most notably, 66% of fathers in this study were above the clinical cutoff for ADHD, compared with 4.4% in epidemiological studies (Kessler et al., 2006). Rates of clinically significant depressive (33%) and ASPD (12%) symptoms in the current sample were also elevated compared to epidemiological rates of 8.2% and 1%, respectively (Lenzenweger et al., 2007; Kessler et al., 2005).

Also, children in the current sample were representative of the general population with regard to clinical characteristics. Specifically, they displayed elevated rates of meeting symptom threshold for ODD (32%) and CD (8%) as well as clinically-significant levels of father-reported home (88%) and school (93%) impairment, all of which are consistent with previous literature (Biederman et al., 1996b; Bird et al., 1994; Fabiano et al., 2006; Jensen et al., 1997; Romano et al., 2005; Yang et al., 2004). Of note, unlike the epidemiological studies described above, the current study did not utilize diagnostic interviews with either fathers or children. Still, it is unlikely that this methodological difference entirely accounts for the elevated rates of psychopathology in the current study. Moreover, the significant variability in both paternal and child psychopathology in this sample further underscores the fact that associations between paternal ADHD symptoms and child CP would have been observed had they been present. Indeed, associations among variables including child CP, paternal psychopathology, paternal marital status and marital/relationship satisfaction, and paternal education level were as expected and consistent with existing literature (Amato & Rogers, 1997; Barkley, Anastopoulos, Guevremont, & Fletcher, 1992; Emery, 1999, Lindahl, 1998; Rogge & Bradbury, 1999; Schermerhorn, Cummings, De-Carlo, & Davies, 2007; Wymbs et al., 2007; Wymbs, et al., 2008).

On the other hand, the current sample consisted primarily of fathers who were Caucasian, highly-educated, married, and living with their child. It is widely established that children are diagnosed with ADHD across ethnic groups (in fact, some studies have suggested that rates of ADHD diagnosis are higher among African-American children; Reid et al., 2000). Similarly, although rates of treatment with ADHD medication in children may be higher among families of higher education- and income-level, there is mixed evidence with regard to comparative rates of diagnosis among low-income families (e.g., Bussing, Zima, Gary & Garvan, 2003; Froehlich et al., 2007). Finally, despite evidence of elevated divorce rates among parents of children with ADHD (as compared to typically-developing children; Wymbs et al., 2008), only 12% of fathers in the current sample were separated or divorced from their child's mother. Similarly, 74% of fathers in the current sample reported that they were at least somewhat satisfied with their marriage/relationship despite evidence that marital dissatisfaction is higher among families of children with ADHD (Barkley et al., 1991). Thus, the current sample is clearly a select one and is likely not entirely representative of the general population of families of children with ADHD, which limits the generalizability of the current findings.

Future studies should continue to develop study methodology with the equally important goals of maximizing sample diversity and reducing barriers for father participation. One potential strategy may be targeted recruitment via established resource centers for minority and low-income families across the country. Ideally, hard-copy study packets would be mailed to these institutions with pre-paid return envelopes to (a) facilitate anonymous participation for those without access to or familiarity with the internet and (b) reduce the financial and time burden on participants. Translating study measures into additional languages (e.g., Spanish) may also increase the diversity of future study samples. Indeed, participation data from the current study suggested that some ethnic and socioeconomic diversity was lost as the survey progressed, highlighting the need to continue to reduce participation time.

A second major limitation of the current study concerns its cross-sectional nature. Ideally, mediation is examined using a longitudinal research design. In the current study, all constructs were measured at a single time-point. Thus, it cannot be assumed that paternal psychopathology and negative parenting *pre-dated* child CP nor that these paternal factors contributed to the *development* of child CP. A longitudinal design was beyond the scope of the current study whose more modest aims and design were based on (1) a lack of sizeable samples of direct-report information from fathers of children with ADHD in the literature and (2) the well-established challenges associated with recruiting these fathers to participate in research (Mitchell et al., 2007). However, the next step in this line of research will be for future studies to replicate these models within a prospective longitudinal

design to establish risk factors (and potential related mechanisms) for the later development of CP in children with ADHD.

Third, due to the fact that the current study was based solely on fatherreport rating scales, we were unable to confirm the ADHD diagnoses of their children. In line with DSM-IV criteria, a diagnosis of ADHD includes 6 or more symptoms of inattention and/or hyperactivity/impulsivity (ideally via diagnostic interview and both parent- and teacher-report) as well as evidence of clinically significant cross-situational impairment (typically home and school; Fabiano et al., 2006). Within the confines of the current study design, we attempted to confirm father-reported diagnoses of ADHD in their children via father-report rating scales of child ADHD symptoms and cross-situational impairment; however, this method still falls short of the gold-standard diagnostic procedures (Pelham, Fabiano, & Massetti, 2005). Indeed, although these children were all reported by their fathers to have a previous diagnosis of ADHD, when these stricter criteria were applied to the sample, only 66% of the children met diagnostic criteria based on father report alone (though the primary findings did not differ when only the subsample meeting full diagnostic criteria was examined). This is consistent with evidence that fathers view their children with ADHD as less symptomatic than mothers do (Langberg et al., 2009; Tallmadge & Barkley, 1983). It is likely that mother and teacher ratings would have added to symptom counts and impairment ratings as well had they been collected; however, this was deliberately avoided to increase paternal anonymity and comfort with research participation. Thus, the current findings may be limited in

terms of their generalizability to the population of children with more strictly-diagnosed ADHD and future larger-scale studies should ideally include a diagnostic interview as well as both maternal- and teacher-report of child ADHD symptoms and related impairments.

A fourth limitation concerns the way in which child psychopathology, paternal psychopathology, and paternal parenting were measured. As mentioned previously, the current study was designed to collect information directly from fathers in an efficient and anonymous manner which would not require them to come into the laboratory. This design consideration was based on the lack of direct paternal-report in previous studies on fathers of children with ADHD and recommendations by prior researchers (e.g., Mitchell et al., 2007). Relying on maternal report of paternal psychopathology (as previous studies have) is certainly not ideal, as mothers tend to report fewer paternal symptoms than fathers do about themselves (Caspi et al., 2001). Collecting information only from fathers, however, is also problematic for several reasons. For example, having fathers report on their own parenting and psychopathology as well as their child's behavior raises the issue of shared method variance. Important information can also be gained through corroborative maternal report of fathers' parenting and psychopathology (especially ADHD symptoms; McGough & Barkley, 2004). Additionally, parenting is ideally measured using a multi-method approach, including both self-report and observational methods (Johnston et al., 2012). Therefore, future studies should strive to utilize multi-informant, multi-method approaches to measuring paternal parenting, paternal psychopathology and child

psychopathology. Additionally, the current study did not collect specific data on the quantity of direct contact that fathers in this study had with their children. Although fathers in the current sample primarily lived with their children, it is unclear how much they were directly involved in everyday caretaking and parenting tasks. This lack of quantitative information somewhat limits the conclusions that can be drawn from the current study's findings. For example, it is possible that paternal positive parenting was not related to either paternal ADHD symptoms of child CP in the sample overall but may have been had we considered the amount of time fathers spent with their children as a potential moderator. Indeed, frequency of contact has emerged in a previous study as a moderator in the relationship between paternal ADHD symptoms and child CP (Mintz & Chronis-Tuscano, under review). Thus, future studies examining paternal parenting should therefore take care to quantify the direct contact that fathers have with their children.

A fifth and related limitation concerns the lack of data collected regarding maternal parenting and psychopathology. Both a developmental psychopathology approach (Mash & Dozois, 2003) and a great deal of empirical evidence (Connell & Goodman, 2002; Dierker, Merikangas, & Szatmari, 1999; Goodman, Brogan, Lynch, & Fielding, 1993) suggest that children may be at greater risk for psychopathology when both parents exhibit mental health problems relative to children with only one affected parent (Brennan, Hammen, Katz, & Le Broque, 2002; Conger, Ge, Elder, Lorenz, & Simons, 1994), and conversely, that having a healthy parent may serve as a protective factor (Goodman et al., 1993).

Considering both maternal and paternal psychopathology in models of risk for child CP is of even greater importance in the context of assortative mating, such that mental health problems tend to co-occur in mothers and fathers (particularly paternal antisocial behavior with maternal depression; Krueger, Moffitt, Caspi, Bleske, & Silva 1998; Maes et al., 1998; Marmorstein et al., 2004). Similarly, previous findings suggest that psychopathology is present at higher rates and associated with parenting deficits among both mothers and fathers of children with ADHD (Arnold et al., 1997; Chronis et al., 2003; Chronis-Tuscano et al., 2008; Harvey et al., 2003; Murray & Johnston, 2006; Rogers et al., 2009). Finally, in contrast to the current findings regarding paternal ASPD, negative parenting, and child CP, Pfiffner and colleagues (2005) found a moderated effect such that maternal dysfunctional parenting was significantly positively associated with child CP, but only when fathers had *lower* levels of ASPD symptoms. When fathers had higher levels of ASPD symptoms, maternal parenting was not associated with child CP. Therefore, it follows that future studies should examine more comprehensive models of maternal and paternal psychopathology and parenting as related to child CP to clarify the relationships among these variables.

A final limitation concerns the number of consecutive analyses conducted given the relatively small sample size. Traditionally, the error rate (i.e., significance level) is adjusted commensurate with the number of analyses conducted. In the case of bootstrapping, the confidence interval of the analysis may be adjusted from the typical 95% to 99%. Of note, when the primary finding of this study (i.e., that paternal negative parenting mediated the relationship

between paternal ASPD symptoms and child CP; Exploratory Aim 1b) was reexamined with a confidence interval at the 99% level, the findings did not change. Regardless, future studies should take care to minimize the number of primary and exploratory analyses that are conducted in studies of relatively small sample sizes in order to minimize threats to conclusive validity.

Despite these limitations, the current study is the first to our knowledge to examine paternal psychopathology, paternal parenting, and child CP in a single model among families of children with ADHD, and its findings provide important information for future research and clinical endeavors. Specifically, the current results suggest that negative parenting may be a powerful potential mechanism by which paternal psychopathology (specifically, paternal ASPD symptoms) is associated with the presence of child CP among children with ADHD. As noted previously, the identification of risk factors for the development of child CP is crucial in preventing these children from embarking on a deleterious pathway. Thus, the current cross-sectional findings pave the way for these factors to be studied longitudinally in future research with this population.

Another primary strength of the current study is the measurement of other forms of paternal psychopathology in addition to paternal ADHD symptoms. As highlighted in a recent review by Johnston and colleagues (2012), a major limitation to the available body of literature on parents with ADHD is the lack of attention paid to other forms of parental psychopathology. The current study examined paternal depressive, anxiety, and ASPD symptoms as well as frequency of alcohol and substance use, and this crucial aspect of its methodology allowed

initial findings attributed to paternal ADHD symptoms to be clarified in the context of paternal ASPD symptoms. Otherwise, the current finding of negative parenting as a potential mechanism by which paternal psychopathology is associated with child CP might have been mistakenly attributed to paternal ADHD symptoms. Instead, a consistent and particularly robust finding emerged such that the association between paternal ASPD symptoms and child CP was mediated by paternal negative parenting, even when paternal ADHD symptoms were controlled, and there was no interactive effect of paternal ADHD and ASPD symptoms. Therefore, it is clear that the assessment of commonly co-occurring forms of paternal psychopathology should become a standard for future research examining paternal factors as related to child CP (Johnston et al., 2012). The current study may call into question previously-found associations between paternal ADHD symptoms, paternal parenting, and child CP, given that paternal ASPD symptoms have rarely been assessed in these studies (e.g., Arnold et al., 1997; Harvey et al., 2003).

Finally, this study also addresses another primary limitation of this body of literature (Johnston et al., 2012) by exploring the role of child gender as related to paternal psychopathology, paternal parenting, and child CP. Specifically, the primary results of this study did not change when child gender was examined. This is inconsistent with previous preliminary findings suggesting that paternal psychopathology and parenting may differentially impact the psychosocial adjustment of male and female children (Connell & Goodman, 2002; Flouri & Buchanan, 2002). However, as noted by Johnston and colleagues (2012), there is

very little research in general on the role of child gender in findings regarding families of children with ADHD and thus no previously-established pattern with which to specifically compare the current findings.

Clinical Implications

Several important clinical implications emerge from the preliminary findings of this study. Most importantly, this study highlights the powerful role that paternal negative parenting plays in the relationship between paternal psychopathology and concurrent child CP among families of children with ADHD. Additionally, in contrast to conclusions drawn from previous studies that paternal ADHD symptoms are associated with negative parenting (e.g., Arnold et al., 1997; Harvey et al., 2003), the current study suggests that perhaps paternal ASPD symptoms play a more central role in the presence of negative parenting and therefore child CP. Thus, these preliminary findings suggest that clinicians should carefully evaluate baseline paternal psychopathology (in particular ASPD symptoms) and parenting behavior as part of routine pre-intervention child ADHD assessment.

Additionally, it is clear that engaging fathers of children with ADHD in treatment focused on reducing negative parenting behavior is essential to improving their children's psychosocial well-being (Fabiano, 2007). Indeed, a recent waitlist-controlled trial of a behavioral parent training designed specifically for groups of fathers of children with ADHD was found to be efficacious (Fabiano et al., 2012). Specifically, the 8-week intervention that combined lecture-based skills-learning (e.g., watching videos of parenting errors) and in-

vivo practice of these skills (i.e., with their children on the soccer field) resulted in reduced rates of observed paternal negative talk and increased rates of observed praise as well as reduced father-report intensity of behavior problems (Fabiano et al., 2012). The results of the current study support a continued intervention focus on decreasing negative parenting behaviors for fathers of children with ADHD.

Figures

Figure 1. Disposition of participants

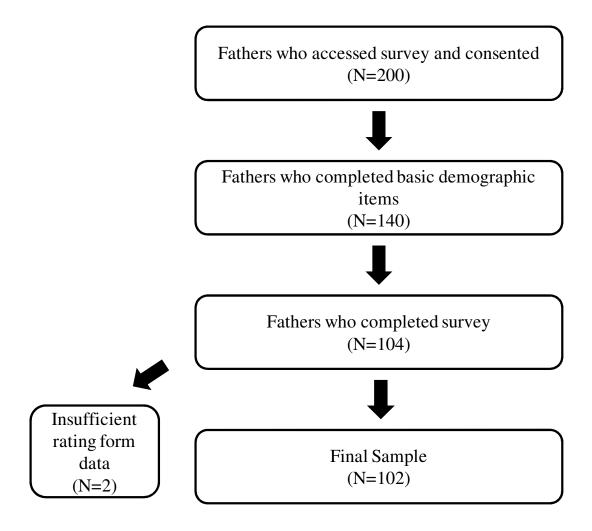
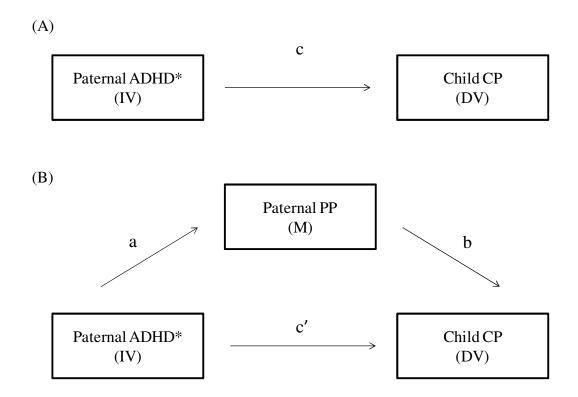


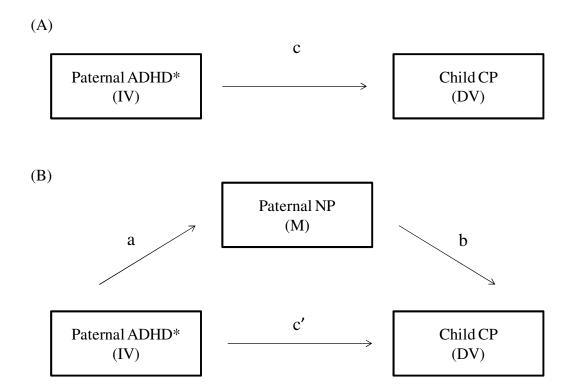
Figure 2. Model of direct effects (A) and simple mediation (B): Aims 2a and 2b



Note. ADHD=attention-deficit/hyperactivity disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, M=mediator, PP=positive parenting.

^{*} Refers to symptoms measured continuously

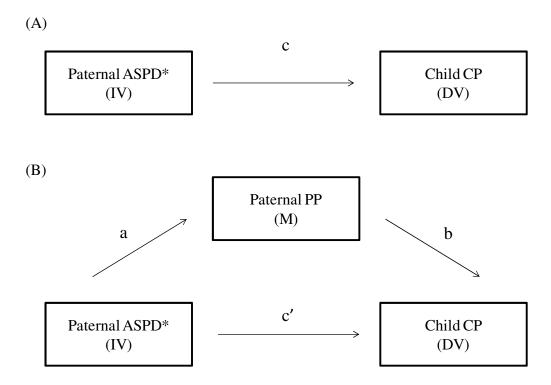
Figure 3. Model of direct effects (A) and simple mediation (B): Aims 3a and 3b



Note. ADHD=attention-deficit/hyperactivity disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, M=mediator, NP=negative parenting.

^{*} Refers to symptoms measured continuously

Figure 4. Model of direct effects (A) and simple mediation (B): Exploratory Analysis 1a

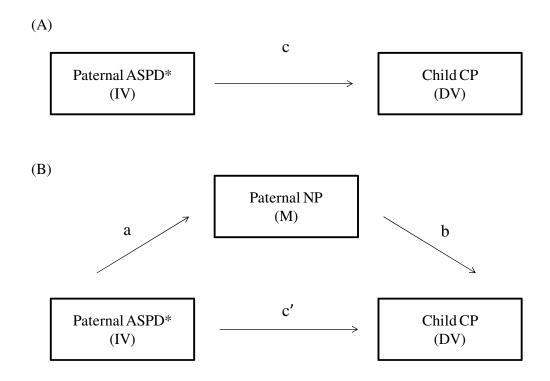


Note. ASPD=antisocial personality disorder, CP=conduct problems,

DV=dependent variable, IV=independent variable, M=mediator, PP=positive parenting.

^{*} Refers to symptoms measured continuously

Figure 5. Model of direct effects (A) and simple mediation (B): Exploratory Analysis 1b



Note. ASPD=antisocial personality disorder, CP=conduct problems,

DV=dependent variable, IV=independent variable, M=mediator, NP=negative parenting.

^{*} Refers to symptoms measured continuously

Tables

Table 1. Aim 1: Paternal Sample Demographic and Diagnostic Characteristics (N = 102)

Paternal Demographic Variables	44.05.15.55
Age	41.90 (6.20)
Race/Ethnicity	Range = $29 - 57$
Caucasian	90 (89.1)
African-American	2 (2.0)
Hispanic/Latino	3 (3.0)
Asian	2 (2.0)
Biracial	1 (1.0)
Other	3 (3.0)
Education Level (Highest Completed)	3 (3.0)
No High School Degree	1 (1.0)
High School Degree	5 (5.0)
Some College	26 (25.7)
College Degree	33 (32.7)
Master's Degree (Or Equivalent)	21 (20.8)
Doctoral Degree (Or Equivalent)	15 (14.9)
ADHD Medication Status (Current)	10 (10 5)
Yes	13 (12.7)
No	88 (86.3)
Marital Status	
Currently Married To And Cohabitating With Child's Mother	84 (82.4)
Separated From Child's Mother	4 (3.9)
Divorced From Child's Mother	8 (7.8)
Never Married To And Cohabitating With Child's Mother	1 (1.0)
Never Married To And Not Cohabitating with Child's Mother	4 (3.9)
Total Household Income	\$156,230.10 (\$144,810.95
	Median = $$135,000.00$
	Range = \$10,000 -
Residential Status	\$1,000,000
Live With Child	00 (07.1)
	99 (97.1)
Do Not Live with Child	3 (2.9)
Paternal Psychopathology Variables	
ADHD Symptomatology (CAARS-S:S)	22.00 (6.45) TF 70
Overall ADHD Index Score	23.00 (6.45); T=70
Inattention/Memory	9.93 (3.36); T=66
Hyperactive/Restless	10.65 (3.61); T=69
Impulsive/Emotional	9.53 (2.86); T=69
Self-Concept	9.74 (3.43); T=64
Substance Abuse	
Not At All	91 (89.2)
Sometimes	10 (9.8)
Often	1 (1.0)
Alcohol Abuse	
Not at all	76 (74.5)

Sometimes	20 (19.6)
Often	6 (5.9)
DSM Depressive Problems (ASR)	5.38 (4.17); T=58
DSM Anxiety Problems (ASR)	4.66 (2.63); T=56
DSM Antisocial Personality Problems (ASR)	5.48 (5.25); T=58
Paternal Parenting Variables	
Involvement (APQ)	35.92 (5.76)
Positive Parenting (APQ)	23.28 (3.63)
Poor Monitoring/Supervision (APQ)	15.20 (5.32)
Inconsistent Discipline (APQ)	14.90 (3.77)
Laxness (PS)	2.74 (0.93)
Overreactivity (PS)	3.41 (1.06)
Paternal Marital Satisfaction	5.70 (1.63)
Very Dissatisfied	1 (1.1)
Dissatisfied	7 (7.8)
Somewhat Dissatisfied	4 (4.4)
Neutral	4 (4.4)
Somewhat Satisfied	12 (13.3)
Satisfied	22 (24.4)
Very Satisfied	40 (44.4)

Note. Results presented as M (SD) or as percent (n). ADHD=attention-deficit/hyperactivity

disorder, APQ=Alabama Parenting Questionnaire, ASR=Adult Self Report, PS=O'Leary

Parenting Scale, CAARS-S:S=Conners Adult ADHD Rating Scale-Self Report: Short Version,

DSM=Diagnostic and Statistical Manual.

Table 2. Aim 1: Child Sample Demographic and Diagnostic Characteristics (N = 102)

Child Demographic Variables	
Age	8.96 (2.18)
Gender	Range = $5-12$
Male	77 (76.2)
Female	24 (23.8)
Race/Ethnicity	24 (23.6)
Caucasian	88 (87.1)
African-American	1 (1.0)
Hispanic/Latino	2 (2.0)
Asian	1 (1.0)
Biracial	7 (6.9)
Other	2 (2.0)
ADHD Medication Status (Current)	2 (2.0)
Yes	77 (75.5)
No	25 (24.5)
Child Psychopathology Variables (all rated by fathers)	20 (2.10)
ADHD Inattentive Symptoms (DBD)	5.92 (2.56)
ADHD Hyperactive/Impulsive Symptoms (DBD)	5.16 (2.68)
ODD Symptoms (DBD)	3.14 (2.47)
CD Symptoms (DBD)	1.15 (2.70)
ODD/CD Symptoms (DBD)	4.29 (4.34)
ADHD Diagnosis (DBD & CIRS)	67 (65.7)
ADHD – Predominantly Inattentive Type	17 (16.7)
ADHD - Predominantly Hyperactive/Impulsive Type	8 (7.8)
ADHD – Combined Type	42 (41.2)
ODD Diagnosis (DBD)	33 (32.4)
CD Diagnosis (DBD)	8 (7.8)
Child Impairment Variables	
Home Impairment (CIRS)	4.62 (1.62)
Clinically-Significant	90 (88.2)
Subthreshold	12 (11.8)
School Impairment (CIRS)	4.92 (1.66)
Clinically-Significant	95 (93.1)
Subthreshold	7 (6.9)

Note. Results presented as M (SD) or as percent (n). ADHD=attention-deficit/hyperactivity

disorder, CD=Conduct Disorder, CIRS=Children's Impairment Rating Scale, DBD=Disruptive Behavior Disorders Scale, ODD=Oppositional Defiant Disorder.

Table 3. Correlation Matrix of Independent, Dependent, Mediator and Demographic Variables (N = 102)

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
1. Child Age	-																				
2. Child Gender	.013	-																			
3. Child Minority Status	043	.061	-																		
4. Child ADHD Medication Status	.295**	016	328**	-																	
5. Child ODD/CD Symptoms (DBD)	.924	085	015	.010	-																
6. Father Age	.469**	001	086	.123	206	-															
7. Father Minority Status	129	.102	.909**	315**	.027	127	-														
8. Father Education Level	.121	.016	078	.056	180	.248*	105	-													
9. Father ADHD Medication Status	.112	024	153	.226*	.060	203*	134	027	-												
10. Father Marital Status	113	122	.094	133	139	.041	.072	.185*	124	-											
11. Father Marital Satisfaction	040	.080	080	064	325**	.175	070	008	122	.259**	-										
12. Father Residential Status	.085	.098	107	.036	016	.014	126	038	.069	.389**	.185	-									
13. Total Household Income	.129	.068	031	.056	156	.299*	077	.291**	073	.098	.117	.023	-								
14. Father ADHD Score (CAARS)	.075	113	014	.147	.193	110	034	092	.334**	069	072	077	069	-							
15. Father Substance Abuse	123	023	040	.052	.152	225	010	177	.410**	022	091	.060	136	.285**	-						
16. Father Alcohol Abuse	100	107	.053	.011	.117	285*	.017	159	.085	107	205	114	157	.235*	.350**	-					
17. Father Depressive Symptoms (ASR)	.047	185*	022	.095	.364**	237*	063	137	.290**	117	239*	.058	205*	.652**	.312**	.318**	-				
18. Father Anxiety Symptoms (ASR)	088	107	.098	.078	.132	070	.102	026	.136	030	258*	031	121	.467**	.193	.275**	.668**	-			
19. Father ASPD Symptoms (ASR)	076	075	.053	.071	.528**	398**	.019	166	.305**	178*	321**	094	246*	.518**	.472**	.357**	.628**	.381**	-		
20. Father Positive Parenting	.006	.125	.009	138	115	008	026	.194	159	.033	.133	062	.049	079	223*	135	197*	133	177	-	
21. Father Negative Parenting	.098	152	.091	.011	.477**	030	.072	237*	.252**	003	292**	.088	226*	.260**	.322**	.302**	.447**	.318**	.540**	442**	-

Note. Results reported as Pearson product-moment correlations or Kendall's tau as appropriate. ADHD=attention-deficit/hyperactivity disorder, APQ=Alabama Parenting Questionnaire, ASPD=Antisocial Personality Disorder, ASR=Adult Self Report, CAARS-S:S=Conners Adult ADHD Rating Scale-Self Report: Short Version, CD=Conduct Disorder, CIRS=Children's Impairment Rating Scale, DBD=Disruptive Behavior Disorders Scale, DSM=Diagnostic and Statistical Manual, PS=O'Leary Parenting Scale, ODD=Oppositional Defiant Disorder.

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

Table 4. Aim 2a: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	p
Effect of ADHD* on paternal PP (path a)	004	.009	464	.644
Effect of paternal PP on child CP controlling for ADHD* (path b)	356	.453	785	.434
Total effect of ADHD* on child CP (path c)	.069	.041	1.672	.098
Direct effect of ADHD* on child CP controlling for paternal PP (path c')	.068	.042	1.630	.107
Indirect effect of ADHD* on child CP through paternal PP (path a x b)	.002	.005	CI:00	04,
			.019; p	>.05
Partial effects of control variables on DV				
Marital status	-2.271	1.103	-	.042
			2.058	
Marital satisfaction	722	.236	-2.74	.007
Child ADHD medication status	004	.957	004	.997

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

CP=conduct problems, DV=dependent variable, PP=positive parenting.

Table 5. Aim 2b: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	p
Effect of ADHD* on paternal PP (path a)	.003	.011	.173	.863
Effect of paternal PP on child CP controlling for ADHD* (path b)	093	.415	224	.823
Total effect of ADHD* on child CP (path c)	037	.044	850	.398
Direct effect of ADHD* on child CP controlling for paternal PP (path c')	037	.044	839	.404
Indirect effect of ADHD* on child CP through paternal PP (path a x b)	000	.004	CI:01	12,
			.007; p	>.05
Partial effects of control variables on DV				
Marital status	-1.174	1.024	-	.254
			1.147	
Marital satisfaction	385	.248	-	.124
			1.552	
Child ADHD medication status	147	.866	170	.866
ASPD*	.419	.088	4.757	.000

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, PP=positive parenting.

^{*} Refers to paternal symptoms measured continuously

^{*} Refers to paternal symptoms measured continuously

Table 6. Aim 3a: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	p
Effect of ADHD* on paternal NP (path a)	.019	.007	2.577	.012
Effect of paternal NP on child CP controlling for ADHD* (path b)	2.338	.523	4.473	.000
Total effect of ADHD* on child CP (path c)	.069	.041	1.697	.093
Direct effect of ADHD* on child CP controlling for paternal NP (path c')		.039	.668	.506
Indirect effect of ADHD* on child CP through paternal NP (path a x b)		.024	CI: .008	3, .106;
			p<.05	
Partial effects of control variable on DV				
Marital status	-2.271	1.09	-2.083	.040
Marital satisfaction	721	.262	-2.758	.007

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

CP=conduct problems, DV=dependent variable, NP=negative parenting.

Table 7. Aim 3b: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	p
Effect of ADHD* on paternal NP (path a)	001	.008	172	.864
Effect of paternal NP on child CP controlling for ADHD* (path b)	1.528	.563	2.714	.008
Total effect of ADHD* on child CP (path c)	038	.043	877	.383
Direct effect of ADHD* on child CP controlling for paternal NP (path c')	036	.042	858	.393
Indirect effect of ADHD* on child CP through paternal NP (path a x b)		.014	CI:03	3,
			.023; p>	.05
Partial effects of control variable on DV				
Marital status	-1.156	1.013	-1.141	.257
Marital satisfaction	385	.247	-1.561	.122
ASPD*	.419	.088	4.778	.000

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, NP=negative parenting.

^{*} Refers to paternal symptoms measured continuously

^{*} Refers to paternal symptoms measured continuously

Table 8. Exploratory Aim 1a: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	р
Effect of ASPD* on paternal PP (path a)	029	.022	-1.345	.182
Effect of paternal PP on child CP controlling for ASPD* (path b)	093	.415	224	.823
Total effect of ASPD* on child CP (path c)	.419	.088	4.757	.000
Direct effect of ASPD* on child CP controlling for paternal PP (path c')	.416	.089	4.660	.000
Indirect effect of ASPD* on child CP through paternal PP (path a x b)	.003	.013	CI:019	9,
			.036; p>	.05
Partial effects of control variables on DV				
Marital status	-1.174	1.024	-1.147	.254
Marital satisfaction	385	.248	-1.552	.124
Child ADHD medication status	147	.866	170	.866
ADHD*	037	.044	850	.398

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, PP=positive parenting.

Table 9. Exploratory Aim 1b: Total, direct, indirect, and partial effects of the mediation model

Effects	В	S.E.	t	p
Effect of ASPD* on paternal NP (path a)	.078	.015	5.080	.000
Effect of paternal NP on child CP controlling for ASPD* (path b)	1.528	.563	2.714	.008
Total effect of ASPD* on child CP (path c)	.419	.088	4.778	.000
Direct effect of ASPD* on child CP controlling for paternal NP (path c')	.300	.096	3.140	.002
Indirect effect of ASPD* on child CP through paternal NP (path a x b)	.119	.053	CI: .035	, .245;
			p<.05	
Partial effects of control variable on DV				
Marital status	-1.156	1.013	-1.141	.257
Marital satisfaction	385	.247	-1.561	.122
ADHD*	038	.043	877	.383

Note. Standardized coefficients not reported (Hayes, 2012); ADHD=attention deficit/hyperactivity disorder,

ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, NP=negative parenting.

^{*} Refers to paternal symptoms measured continuously

^{*} Refers to paternal symptoms measured continuously

Table 10. Exploratory Aim 2a: Paternal ADHD and ASPD symptoms predicting child CP

	df	F	R^2	$R^2\Delta$	SE	В
Step 1	2, 99	7.299	.129	.129*		
Marital status					1.096	210*
Marital satisfaction					.264	266*
Step 2	4, 97	11.136	.315	.186*		
ADHD					.043	087
ASPD					.088	.506**
Step 3	5, 96	8.817	.315	.000		
ADHD x ASPD					.009	004

Note. ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder,

CP=conduct problems.

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

Table 11. Exploratory Aim 2b: Paternal ADHD and ASPD symptoms predicting paternal PP

	df	F	R^2	$R^2\Delta$	SE	ß
Step 1	1, 100	3.656	.035	.035		
Child ADHD medication status					.209	188
Step 2	3, 98	2.077	.060	.025		
ADHD					.011	.037
ASPD					.020	173
Step 3	4, 97	1.966	.075	.015		
ADHD x ASPD					.002	-1.168

Note. ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder,

PP=positive parenting.

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

Table 12. Exploratory Aim 2c: Paternal ADHD and ASPD symptoms predicting paternal NP

	df	$\boldsymbol{\mathit{F}}$	R^2	$R^2\Delta$	SE	ß
Step 1	2, 99	20.425	.292	.292**		
ADHD					.008	027
ASPD					.014	.554**
Step 2	3, 98	13.493	.292	.000		
ADHD x ASPD					.001	139

Note. ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder,

NP=negative parenting.

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

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Appendix A. Study Measures

i. Parent DBD Rating Scale

Check the column that best describes your child.	Not at All	Just a Little	Pretty Much	Very Much
1. Often interrupts or intrudes on others (e.g., butts into conversations or games)				
2. Has run away from home overnight at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)				
3. Often argues with adults				
 4. Often lies to obtain goods or favors or to avoid obligations (i.e., "cons" others) 5. Often initiates physical fights with other members of his or her household 				
6. Has been physically cruel to people				
7. Often talks excessively				
8. Has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery)				
9. Is often easily distracted by extraneous stimuli				
10. Often engages in physically dangerous activities without considering possible consequences (not for the purpose of thrill-seeking), e.g., runs into street without looking				
11. Often truant from school, beginning before age 13 yrs				
12. Often fidgets with hands or feet or squirms in seat				
13. Is often spiteful or vindictive				
14. Often swears or uses obscene language				
15. Often blames others for his/her mistakes or misbehavior				
16. Has deliberately destroyed others' property (other than by fire setting)				
17. Often actively defies or refuses to comply with adults' requests or rules				
18. Often does not seem to listen when spoken to directly				
19. Often blurts out answers before questions have been completed				
20. Often initiates physical fights with others who do not live in his or her household (e.g., peers at school or in the neighborhood)				

21. Often shifts from one uncompleted activity to another		
22. Often has difficulty playing or engaging in leisure activities quietly23. Often fails to give close attention to details or makes		
careless mistakes in schoolwork, work, or other activities		
24. Is often angry and resentful		
25. Often leaves seat in classroom or in other situation in which remaining seated is expected		
26. Is often touchy or easily annoyed by others		
27. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)		
28. Often loses temper		
29. Often has difficulty sustaining attention in tasks or play activities		
30. Often has difficulty awaiting turn		
31. Has forced someone into sexual activity		
32. Often bullies, threatens, or intimidates others		
33. Is often "on the go" or often acts as if "driven by a motor"		
34. Often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)		
35. Often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)		
36. Has been physically cruel to animals		
37. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)		
38. Often stays out at night despite parental prohibitions, beginning before age 13 years		
39. Often deliberately annoys people		
40. Has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery)		
41. Has deliberately engaged in fire setting with the intention of causing serious damage		
42. Often has difficulty organizing tasks and activities		
43. Has broken into someone else's house, building or car		
44. Is often forgetful in daily activities		

45. Has used a weapon that can cause serious physical harm		
to others (e.g., a bat, brick, broken bottle, knife, gun)		

ii. Children's Impairment Rating Scale (CIRS)

Instructions: Please complete the following ratings related to (1) his or her academic progress at school and (2) your family in general. For the ratings, please choose a number along the lines at the points that you believe reflect the impact of the child's problems on this area and whether he or she needs treatment or special services for the problems.

(1) Ho	ow your child	d's problems af	fect his or her	academic pro	gress at scho	ol?	
1	2	3	4	5	6	7	
No Pro	blem				Extreme	Problem	
	tely does not r cial services	need treatment				y needs treatn l services	nent
(2) Ho	ow your child	d's problems af	fect your fami	ly in general?	•		
1	2	3	4	5	6	7	
No Pro	oblem				Extreme	Problem	
Defini	tely does not r	need treatment			Definitel	y needs treatn	nent
or spec	cial services				or specia	l services	

iii. Alabama Parenting Questionnaire (APQ)

The following are a number of statements about your family. Please rate each item as to how often it TYPICALLY occurs in your home.

	I at	A1 4	l g 4.	Of	A 1
	Never	Almost Never	Sometimes	Often	Always
1		Nevel			
1. You have a friendly talk with					
your child.					
2. You let your child know when					
he/she is doing a good job with					
something.					
3. You threaten to punish your					
child and then do not actually					
punish them.					
4. You volunteer to help with					
special activities that your child is					
involved in (such as sports,					
boy/girl scouts, church youth					
groups)					
5. You reward or give something					
extra to your child for obeying					
you or behaving well.					
6. Your child fails to leave a note					
or to let you know where he/she is					
going.					
7. You play games or do other fun					
things with your child.					
8. Your child talks you out of					
being punished after he/she has					
done something wrong.					
9. You ask your child about					
his/her day in school.					
10. Your child stays out in the					
evening past the time he/she is					
supposed to be home.					
11. You help your child with his/her homework.					
12. You feel that getting your child to obey you is more trouble					
than it's worth.					
13. You compliment your child					
when he/she does something well.					
14. You ask your child what					
his/her plans are for the coming					
day.					
15. You drive your child to a					
special activity.					
special activity.					

16. You praise your child if he/she			
behaves well.			
17. Your child is out with friends			
that you don't know.			
18. You hug or kiss your child			
when he/she has done something			
well.			
19. Your child goes out without a			
set time to be home.			
20. You talk to your child about			
his/her friends.			
21. Your child is out after dark			
without an adult with him/her.			
22. You let your child out of a			
punishment early (like lift			
restrictions earlier than you			
originally said)			
23. Your child helps plan family			
activities			
24. You get so busy that you			
forget where your child is and			
what he/she is doing			
25. Your child is not punished			
when he/she has done something			
wrong			
26. You attend PTA meetings,			
parent/teacher conferences, or			
other meetings at your child's			
school			
27. You tell your child that you			
like it when he/she helps out			
around the house.			
28. You don't check that your			
child comes home at the time			
she/he was supposed to.			
29. You don't tell your child where you are going.			
30. Your child comes home from			
school more than an hour past the			
time you expect him/her.			
31. The punishment you give your			
child depends on your mood.			
32. Your child is at home without			
adult supervision.			
34. You ignore your child when			
he/she is misbehaving			
36. You take away privileges or			
money from your child as a			
punishment.			

37. You send your child to his/her room as a punishment.			
39. You yell or scream at your child when he/she has done something wrong.			
40. You calmly explain to your child why his/her behavior was wrong when he/she misbehaves.			
41. You use time out (make him/her sit or stand in a corner) as a punishment			
42. You give your child extra chores as a punishment			

iv. Parenting Scale

For each item, fill in the circle that best describes your style of parenting during the past two months.

1. When my child misbehaves...

I do something right **0---0---0---0** I do something about it away.

2. When I'm upset or under stress...

3. When my child pesters me...

I can ignore the pestering **0---0---0---0** I can't ignore the pestering

4. When my child misbehaves...

I usually get into a long argument with my child 0---0---0---0 I don't get into an argument

5. I am the kind of parent that...

Sets limits on what my 0---0---0---0 Lets my child do child is allowed to do whatever he/she wants

6. When my child misbehaves...

I raise my voice and yell **0---0---0---0** I speak to my child calmly

7. When I want my child to stop doing something...

I firmly tell my child to 0---0---0---0 I coax or beg my child to stop

8. When my child is out of my sight...

I often don't know what **0---0---0---0** I always have a good idea my child is doing of what my child is doing

9. After there's been a problem with my child...

I often hold a grudge 0---0---0---0 Things get back to normal quickly

10. When we're not at home...

I handle my child the way 0---0---0---0 I let my child get away I do at home with a lot more

11. When my child does something I don't like...

I do something about it **0---0---0---0** I often let it go every time it happens

12. When there is a problem with my child...

Things build up and I do 0---0---0---0 Things don't get out of things I don't mean to hand

13. When my child doesn't do what I ask...

I often let it go or end up **0---0---0---0** I take some other action doing it myself

14. When I give a fair threat or warning...

I often don't carry it out 0---0---0---0 I always do what I said

15. If saying "No" doesn't work...

I take some other kind of 0---0---0---0 I offer my child something nice so he/she will behave

16. When my child misbehaves...

I handle it without getting upset

O---0---0---0---0

I get so frustrated or angry that my child can see I'm upset

17. If my child misbehaves and then acts sorry...

I handle the problem like I **0---0---0---0** I let it go that time usually would

18. When my child misbehaves...

I rarely use bad language 0---0---0---0 I almost always use bad or curse language

19. When I say my child can't do something...

I let my child do it **0---0---0---0** I stick to what I said anyway

20. When I have to handle a problem...

I tell my child I'm sorry **0---0---0---0** I don't say I'm sorry about it

21. When my child does something I don't like, I insult my child, say mean things, or call my child names...

Never or rarely **0---0---0** Most of the time

22. If my child gets upset when I say "No"...

I back down and give in **0---0---0---0** I stick to what I said to my child

v. CAARS Self-Report: Short Version (CAARS-S:L)

Instructions: Listed below are items concerning behaviors or problems sometimes experienced by adults. Read each item carefully and decide how much or how frequently each item describes you recently. Indicate your response for each item by checking the box that corresponds to your choice.

	Not at All, Never	Just a Little, Once in a while	Pretty Much, Often	Very Much, Very Frequently
1. I interrupt others when talking.				
2. I am always on the go, as if driven by a motor.				
3. I'm disorganized.				
4. It's hard for me to stay in one place very long.				
5. It's hard for me to keep track of several things at once.				
6. I'm bored easily.				
7. I have a short fuse/hot temper.				
8. I still throw tantrums.				
9. I avoid new challenges because I lack faith in my abilities.				
10. I seek out fast paces, exciting activities.				
11. I feel restless inside even if I am sitting still.				
12. Things I hear or see distract me from what I'm doing.				
13. Many things set me off easily.				
14. I am un underachiever.				
15. I get down on myself.				
16. I act okay on the outside, but inside I'm unsure of myself.				

17. I can't get things done unless there is an absolute deadline.		
18. I have trouble getting started on a task.		
19. I intrude on others' activities.		
20. My moods are unpredictable.		
21. I'm absent-minded in daily activities.		
22. Sometimes my attention narrows so much that I'm oblivious to everything else; other times, it's so broad that everything distracts me.		
23. I tend to squirm or fidget.		
24. I can't keep my mind on something unless it's really interesting.		
25. I wish I had greater confidence in my abilities.		
26. My past failures make it hard for me to believe in myself.		

$vi.\ Adult\ Self-Report\ Scale\ (ASR)$

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Below is a list of items that describe people. For each item, please choose 0, 1, or 2 to describe yourself over the past 6 months. Please answer all items as well as you can, even if some do not seem to apply to you.

you can, even if some do not seem			T .
	0 = Not	1 = Somewhat or	2 = Very True
	True	Sometimes True	or Often True
1. I am too forgetful			
2. I make good use of my			
opportunities			
opportunities			
3. I argue a lot			
4. I work up to my ability			
5. I blame others for my problems			
6. I use drugs (other than alcohol and			
nicotine) for nonmedical purposes			
F 22 F 22 70			
7. I brag			
,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,			
8. I have trouble concentrating or			
paying attention for long			
paying attention for long			
9. I can't get my mind off certain			
•			
thoughts			
10. I have trouble sitting still			
10. I have trouble sitting still			
11 I am too donondont on others			
11. I am too dependent on others			
12. I feel lonely			
12. I feel foliety			
12 I feel confused on in a fee			
13. I feel confused or in a fog			
14. I cry a lot			
14.1 Cry a 10t			
15. I am pretty honest			
13. 1 am pretty nonest			
16. I am mean to others			
10. 1 am mean to others			
17 I daydysam a let			
17. I daydream a lot			
10. I tury to got a lot of attention			
19. I try to get a lot of attention.			
20 I damage and day			
20. I damage or destroy my things			

21. I damage or destroy things		
belonging to others		
22. I worry about the future		
23. I break rules at work or elsewhere		
24. I don't eat as well as I should		
25. I don't get along with other people		
26. I don't feel guilty after doing something I shouldn't		
27. I am jealous of others		
28. I get along badly with my family		
29. I am afraid of certain animals, situations, or places		
30. My relations with the opposite sex are poor		
31. I am afraid I might think or do something bad		
32. I feel that I have to be perfect		
33. I feel that no one loves me		
34. I feel that others are out to get me		
35. I feel worthless or inferior		
36. I accidentally get hurt a lot, accident-prone		
37. I get in many fights		
38. My relations with neighbors are poor		
39. I hang around people who get in trouble		
40. I hear sounds or voices that other people think aren't there		
41. I am impulsive or act without thinking		
42. I would rather be alone than with		

others		
43. I lie or cheat		
44. I feel overwhelmed by my responsibilities		
45. I am nervous or tense		
46. Parts of my body twitch or make nervous movements		
47. I lack self-confidence		
48. I am not liked by others		
49. I can do certain things better than other people		
50. I am too fearful or anxious		
51. I feel dizzy or lightheaded		
52. I feel too guilty		
53. I have trouble planning for the future		
54. I feel tired without good reason		
55. My moods swing between elation and depression		
56. I experience physical problems without known medical cause		
a. Aches or pains (not stomach or headaches)		
b. Headaches		
c. Nausea, feel sick		
d. Problems with eyes (not if corrected by glasses)		
e. Rashes or other skin problems		
f. Stomachaches		
g. Vomiting, throwing up		
h. Heart pounding or racing		

i. Numbness or tingling in body parts		
1. Ivulibriess of thighing in body parts		
57. I physically attack people		
58. I pick my skin or other parts of my body		
59. I fail to finish things I should do		
60. There is very little that I enjoy		
61. My work performance is poor		
62. I am poorly coordinated or clumsy		
63. I would rather be with older people than with people of my own age		
64. I have trouble setting priorities		
65. I refuse to talk		
66. I repeat certain acts over and over		
67. I have trouble making or keeping friends		
68. I scream or yell a lot		
69. I am secretive or keep things to myself		
70. I see things that other people think aren't there		
71. I am self-conscious or easily embarrassed		
72. I worry about my family		
73. I meet my responsibilities		
74. I show off or clown		
75. I am too shy or timid		
76. My behavior is irresponsible		
77. I sleep more than most other people during day and/or night		
78. I have trouble making decisions		

79. I have a speech problem	
73. I have a speech problem	
80. I stand up for my rights	
81. My behavior is very changeable	
82. I steal	
83. I am easily bored	
84. I do things that other people think are strange	
85. I have thoughts that other people would think are strange	
86. I am stubborn, sullen, or irritable	
87. My moods or feelings change suddenly	
88. I enjoy being with people	
89. I rush into things without considering the risks	
90. I drink too much alcohol or get drunk	
92. I do things that may cause me trouble with the law	
93. I talk too much	
94. I tease others a lot	
95. I have a hot temper	
96. I think about sex too much	
97. I threaten to hurt people	
98. I like to help others	
99. I dislike staying in one place for very long	
100. I have trouble sleeping	
101. I stay away from my even job when I'm not sick and not on vacation	
102. I don't have much energy	

103. I am unhappy, sad, or depressed		
104. I am louder than others		
105. People think I am disorganized		
106. I try to be fair to others		
107. I feel that I can't succeed		
108. I tend to lose things		
109. I like to try new things		
110. I wish I were of the opposite sex		
111. I keep from getting involved with others		
112. I worry a lot		
113. I worry about my relations with the opposite sex		
114. I fail to pay my debts or meet other financial responsibilities		
115. I feel restless or fidgety		
116. I get upset too easily		
117. I have trouble managing money or credit cards		
118. I am too impatient		
119. I am not good at details		
120. I drive too fast		
121. I tend to be late for appointments		
122. I have trouble keeping a job		
123. I am a happy person		

vii. Frequency of Alcohol and Substance Use

In the past six months, I have been drunk:

0 = Not at all 1 = Sometimes 2 = Often

In the past six months, I have used drugs for nonmedical purposes (including marijuana, cocaine, and other drugs except alcohol and nicotine):

0 = Not at all 1 = Sometimes 2 = Often

viii. Initial Application Form (Abbreviated)

1. Date	_
2. How did you learn about this study?	?
Information about your child	
3. Child's age	
4. Child's gender 1. Male	2. Female
5. Child's race/ethnicity (please circle	one)
1. Caucasian	5. Asian
2. African-American	6. Bi-racial (specify)
3. Hispanic or Latino	7. Other (specify)
4. Native American	
6. Has your child been previously diag	gnosed with ADHD or ADD?
1. Yes 2. No	
7. Is your child currently taking medic	ation for treatment of ADD or ADHD?
1. Yes	
2. No	
Information about yourself (Father)	
18. Father's age	
19. Father's race/ethnicity (please circ	le one)
1. Caucasian	5. Asian
2. African-American	6. Bi-racial (specify)
3. Hispanic or Latino	7. Other (specify)
4. Native American	

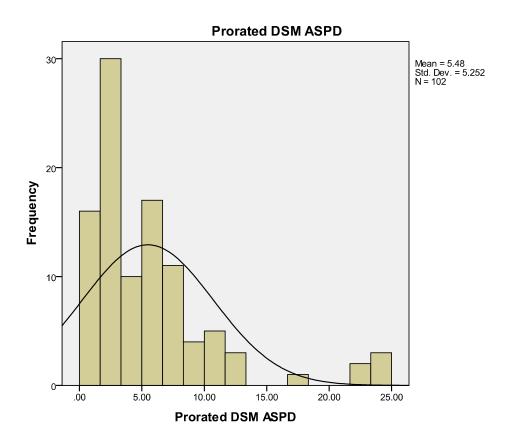
	20. What is the last grade you have complete	d?	
	1. Ninth grade	6.	Two years college
	2. Tenth Grade	7.	Three years college
	3. Eleventh Grade	8.	College Degree
	4. Twelfth Grade	9.	Master's Degree or equivalent
	5. One year college	10). Doctoral Degree or equivalen
21. W	hat is your marital status (in relation to this ch	ild's moth	er)?
	1. Never married to and not cohabitating with	n my child	l's mother
	2. Married to and cohabitating with my child	's mother	
	3. Separated from my child's mother		
	4. Divorced from my child's mother		
	5. Never married to and cohabitating with my	y child's n	nother
22. Do	you live with the above named child?	1. Yes	2. No
23. If 1	no, how many days per week do you see your	child?	
24. W	hat is your yearly family income?		

ix. Marital/Relationship Satisfaction

If you are married to or cohabitating with your child's mother, please rate your overall relationship satisfaction:

- 1 = Very Dissatisfied
- 2 = Dissatisfied
- 3 = Somewhat Dissatisfied
- 4 = Neutral
- 5 = Somewhat Satisfied
- 6 = Satisfied
- 7 = Very Satisfied

Appendix B: Histogram of ASPD Variable



Note. ASPD=antisocial personality disorder; DSM=Diagnostic and Statistical Manual.

Appendix C. Distribution Statistics for all Variables

Variable	N	M (SD)	Range	Skew (SE)	Skew z-score	Kurtosis (SE)	Kurtosis z-score
Alcohol Abuse	102	0.31 (.58)	0 - 2	1.703 (.239)	7.126	1.898 (.474)	4.004
ASR Anxiety Symptoms	102	4.66 (2.63)	0 - 12	.331 (.239)	1.385	186 (.474)	392
ASR ASPD Symptoms	102	5.48 (5.25)	0 - 24.21	2.046 (.239)	8.561	4.568 (.474)	9.637
ASR DSM Depressive Symptoms	102	5.38 (4.17)	0 - 15	.638 (.239)	2.669	646 (.474)	-1.363
CAARS ADHD Index (T-Score)	102	23.00 (6.45)	13 - 40	.647 (.239)	2.707	300 (.474)	633
DBD Conduct Problems	102	4.29 (.43)	0 - 23	2.192 (.239)	8.908	6.654 (.474)	14.038
Negative Parenting Composite (Z-Score)	102	00 (.76)	-1.85 - 1.86	.332 (.239)	1.389	140 (.474)	295
Positive Parenting Composite (Z-Score)	102	00 (.92)	-3.31 - 2.02	771 (.239)	-3.226	1.543 (.474)	3.255
Substance Abuse	102	.12 (.35)	0 - 2	.037 (.239)	4.184	9.512 (.474)	20.068

Note. ADHD=attention-deficit/hyperactivity disorder, ASR=Adult Self-Report, ASPD=antisocial personality disorder, CAARS=Conners Adult ADHD
Rating Scale, DBD=Disruptive Behavior Disorders scale, DSM=Diagnostic and Statistical Manual.

Appendix D. Aims 2c and 3c (Moderated Mediation)³

Given previous findings that paternal psychopathology may differentially impact either the parenting process for or the psychosocial adjustment of male and female children (Connell and Goodman, 2002; Flouri & Buchanan, 2002), analyses were conducted to clarify the impact of gender as a moderator on the proposed mediation relationships between paternal ADHD symptoms and child CP. Specifically, bootstrapping analyses were again completed to assess for the presence of moderated mediation by which the moderator variable (i.e., child gender) affects path a (Figures 6 and 7). The following interaction variable was created by computing the product of child gender and paternal ADHD symptoms: gender x ADHD.

Analyses first examined this model with paternal positive parenting as the mediator (Aim 2c). Bootstrapping analyses confirmed that gender did not significantly moderate the proposed mediation relationship, M_{β} = .005, S.E. = .015, 95% CI = -.0021 to .046 (Table 13). Next, analyses examined this model with paternal negative parenting as the mediator (Aim 3c). Bootstrapping analyses confirmed that gender did not significantly moderate the proposed mediation relationship, M_{β} = .027, S.E. = .022, 95% CI = -.003 to .088 (Table 14). Thus, the results described above (Aims 2b and 3b) were the same for both boys and girls.

 $^{\rm 3}$ Please see Appendix E for relevant tables and figures.

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Appendix E. Exploratory Analysis 1c (Moderated Mediation)⁴

Given previous findings that paternal psychopathology may differentially impact either the parenting process for or the psychosocial adjustment of male and female children (Connell and Goodman, 2002; Flouri & Buchanan, 2002), analyses were conducted to clarify the impact of gender as a moderator on the proposed mediation relationships between paternal ASPD symptoms and child CP. Specifically, bootstrapping analyses were again completed to assess for the presence of moderated mediation by which the moderator variable (i.e., child gender) affects path a (Figure 8). The following interaction variable was created by computing the product of child gender and paternal ASPD symptoms: gender x ASPD.

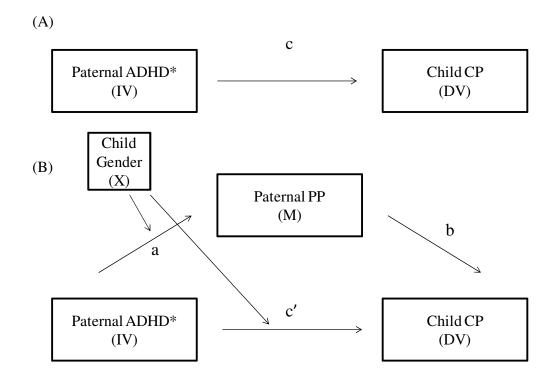
Analyses first examined this model with paternal positive parenting as the mediator. Bootstrapping analyses confirmed that gender did not significantly moderate the proposed mediation relationship, M_{β} = .044, S.E. = .040, 95% CI = -.009 to .166 (Table 15). Next, analyses examined this model with paternal negative parenting as the mediator. Bootstrapping analyses confirmed that gender did not significantly moderate the proposed mediation relationship, M_{β} = .017, S.E. = .047, 95% CI = -.072 to .110 (Table 16). Thus, the results described above (Exploratory Analyses 1a and 1b) were the same for both boys and girls.

⁴ Please see Appendix E for relevant tables and figures.

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Appendix F. Figures and Tables for Results Discussed in Appendices C-F

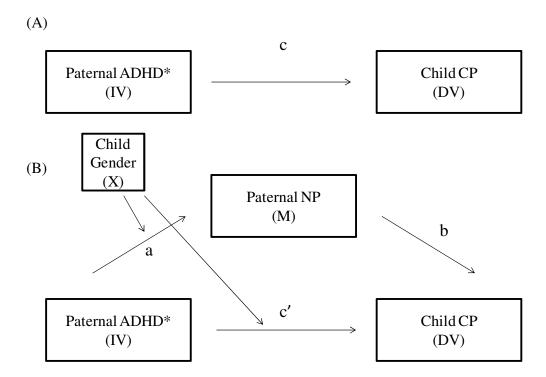
Figure 6. Model of direct effects (A) and moderated mediation (B): Aim 2c



Note. ADHD=attention-deficit/hyperactivity disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, M=mediator, PP=positive parenting, X=moderator.

^{*} Refers to symptoms measured continuously

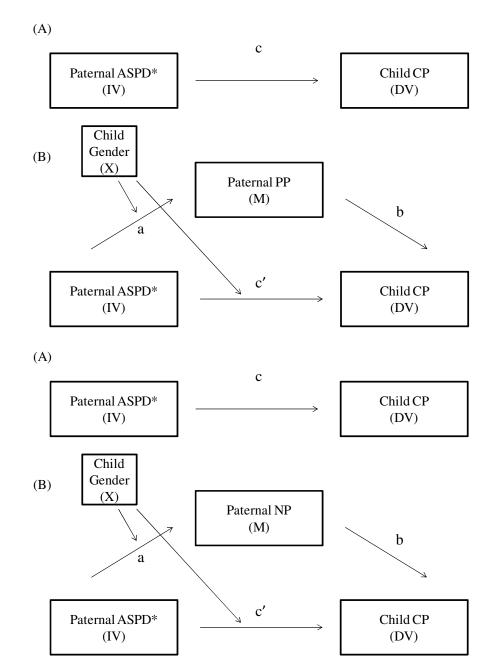
Figure 7. Model of direct effects (A) and moderated mediation (B): Aim 3c



Note. ADHD=attention-deficit/hyperactivity disorder, CP=conduct problems, DV=dependent variable, IV=independent variable, M=mediator, NP=negative parenting, X=moderator.

^{*} Refers to symptoms measured continuously

Figure 8. Models of direct effects (A) and moderated mediation (B): Exploratory analysis 1c



Note. ASPD=antisocial personality disorder, CP=conduct problems,
DV=dependent variable, IV=independent variable, M=mediator, PP=positive
parenting, NP=negative parenting, X=moderator.

^{*} Refers to symptoms measured continuously

Table 13. Aim 2c: Total, direct, indirect, and partial effects of the moderated mediation model

Effects	В	S.E.	t	p
Effect of ADHD* x gender on paternal PP (path a)	041	.021	-1.939	.055
Effect of paternal PP on child CP controlling for ADHD* (path b)	125	.430	291	.772
Total effect of ADHD* on child CP (path c)	037	.044	850	.398
Males	048	.049	968	.336
Females	.046	.093	.498	.625
Direct effect of ADHD* on child CP controlling	.097	.120	.808	.421
for paternal PP (path c')				
Males	016	.049	333	.740
Females	130	.082	-1.584	.117
Indirect effect of ADHD* on child CP	.005	.015	CI:002	21,
through paternal PP (path a x b)			.046; p>	.05
Males	002	.007	CI:020	0, .007;
			p>.05	
Females	.003	.011	CI:01	1, .039;
			p>.05	
Partial effects of control variables on DV				
Marital status	-1.270	1.043	-1.218	.227
Marital satisfaction	349	.251	-1.391	.168
Child ADHD medication status	020	.902	022	.983
ASPD*	.418	.090	4.652	.000

Note. Standardized coefficients not reported (Hayes, 2012); Analyses with child gender conducted with

N=101 (1 subject dropped due to missing data); ADHD=attention deficit/hyperactivity disorder,

ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, PP=positive parenting.

^{*} Refers to paternal symptoms measured continuously

Table 14. Aim 3c: Total, direct, indirect, and partial effects of the moderated mediation model

Effects	В	S.E.	t	р	
Effect of ADHD* x gender on paternal NP (path a)	.017	.015	1.146	.255	
Effect of paternal NP on child CP	1.571	.573	2.743	.007	
controlling for ADHD* (path b)					
Total effect of ADHD* on child CP (path c)	038	.043	877	.383	
Males	048	.049	990	.325	
Females	.030	.081	.370	.715	
Direct effect of ADHD* on child CP	.127	.113	1.127	.263	
controlling for paternal NP (path c')					
Males	008	.047	162	.872	
Females	143	.077	-1.854	.067	
Indirect effect of ADHD* on child CP	.027	.022	.022 CI:003, .088;		
through paternal NP (path a x b)	p>.05				
Males	011	.018 CI:056, .017;		5, .017;	
			p>.05		
Females	.017	.015	CI:00	5, .058;	
			p>.05		
Partial effects of control variables on DV					
Marital status	-1.505	.996	-1.511	.134	
Marital satisfaction	264	.242	-1.090	.279	
ASPD*	.300	.096	3.123	.002	

Note. Standardized coefficients not reported (Hayes, 2012); Analyses with child gender conducted with N=101 (1 subject dropped due to missing data); ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, NP=positive parenting.

^{*} Refers to paternal symptoms measured continuously

Table 15. Exploratory Aim 1c: Total, direct, indirect, and partial effects of the moderated mediation model

Effects	В	S.E.	t	р	
Effect of ASPD* x gender on paternal PP (path a)	115	.043	-2.664	.009	
Effect of paternal PP on child CP	383	.417	918	.361	
controlling for ASPD* (path b)					
Total effect of ASPD* on child CP (path c)	.381	.076	5.040	.000	
Males	.500	.085	5.920	.000	
Females	136	.149	915	.372	
Direct effect of ASPD* on child CP	1.101	1.259	1.703	.092	
controlling for paternal PP (path c')					
Males	.510	.090	5.661	.000	
Females	082	1.176	466	.642	
Indirect effect of ASPD* on child CP	.044	.040	CI:009	CI:009, .166;	
through paternal PP (path a x b)			p>.05		
Males	.004	.010	CI:007	', .039;	
			p>.05		
Females	.049 .043 CI:010, 0174;), 0174;		
			p>.05		
Partial effects of control variables on DV					
Marital status	-1.641	.999	-1.644	.104	
Marital satisfaction	323	.240	-1.347	.181	
Child ADHD medication status	394	.864	456	.650	
ADHD*	031	.042	734	.465	

Note. Standardized coefficients not reported (Hayes, 2012); Analyses with child gender

conducted with N=101 (1 subject dropped due to missing data); ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, PP=positive parenting.

^{*} Refers to paternal symptoms measured continuously

Table 16. Exploratory Aim 1c: Total, direct, indirect, and partial effects of the moderated mediation model

Effects	В	S.E.	t	р
Effect of ASPD* x gender on paternal NP (path a)	.011	.032	.348	.728
Effect of paternal NP on child CP	1.530	.545	2.809	.006
controlling for ASPD* (path b)				
Total effect of ASPD* on child CP (path c)	.379	.075	5.064	.000
Males	.495	.083	5.943	.000
Females	135	.145	928	.364
Direct effect of ASPD* on child CP	.958	.214	4.476	.000
controlling for paternal NP (path c')				
Males	.397	.096	4.147	.000
Females	165	.168	985	.327
Indirect effect of ASPD* on child CP	.017	.047	CI:072	2, .110;
through paternal NP (path a x b)			p>.05	
Males	.116	.051	CI: .038	, .238;
			p<.05	
Females	.133	.064	CI: .037	, .309;
			p<.05	
Partial effects of control variables on DV				
Marital status	-1.825	.956	-1.909	.059
Marital satisfaction	248	.232	-1.071	.287
ADHD*	031	.040	768	.445

Note. Standardized coefficients not reported (Hayes, 2012); Analyses with child gender

conducted with N=101 (1 subject dropped due to missing data); ADHD=attention deficit/hyperactivity disorder, ASPD=antisocial personality disorder, CP=conduct problems, DV=dependent variable, NP=negative parenting.

^{*} Refers to paternal symptoms measured continuously

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