Published in final edited form as:

J Clin Child Adolesc Psychol. 2015 ; 44(5): 787–799. doi:10.1080/15374416.2014.913250.

Parenting Behavior Mediates the Intergenerational Association of Parent and Child Offspring ADHD Symptoms

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Abstract

Although there are likely to be multiple mechanisms underlying parent attention-deficit/hyperactivity disorder (ADHD) symptoms as a key risk factor for offspring ADHD, potential explanatory factors have yet to be reliably identified. Given that parent ADHD symptoms independently predict parenting behavior and child ADHD symptoms, we tested whether individual differences in multiple dimensions of positive and negative parenting behavior (i.e., corporal punishment, inconsistent discipline, positive parenting behavior, observed negative talk, and observed praise) mediated the association between parental and offspring ADHD. We used a prospective design that featured predictors (i.e., parent ADHD symptoms) and mediators (i.e., parenting behavior) that temporally preceded the outcome (i.e., offspring ADHD symptoms). Using a well-characterized sample of 120 children with and without ADHD (ages 5–10 at Wave 1, 7–12 at Wave 2) and their biological parents, we examined multimethod (i.e., observed, self-report) measures of positive and negative parenting behavior as simultaneous mediators of the association of Wave 1 parent and Wave 2 offspring ADHD symptoms. Using a multiple mediation framework, consisting of rigorous bootstrapping procedures and controlling for parent depression, child's baseline ADHD and oppositional defiant disorder, and child's age, corporal punishment significantly and uniquely mediated the association of Wave 1 parent ADHD symptoms and Wave 2 offspring ADHD. We consider the role of parenting behavior in the intergenerational transmission of ADHD as well as implications of these findings for the intervention and prevention of childhood ADHD.

Attention-deficit/hyperactivity disorder (ADHD) is a childhood-onset disorder characterized by developmentally aberrant and impairing levels of inattention and/ or hyperactivity. ADHD affects approximately 8% of children in the United States and 8 to 12% of school-

aged children worldwide, persisting into adulthood for 50 to 80% of these children (Faraone, Biederman, & Mick, 2006; Faraone, Sergeant, Gillberg, & Biederman, 2003). Among key impairments, ADHD prospectively predicts substandard academic achievement, occupational instability, accidental injuries requiring medical attention, elevated rates of criminality, and substance use problems (Barkley, Fischer, Smallish, & Fletcher, 2006; Biederman et al., 2012; S. S. Lee, Lahey, Owens, & Hinshaw, 2008; Merrill, Lyon, Baker, & Gren, 2009; Molina et al., 2009; Owens, Hinshaw, Lee, & Lahey, 2009). Beyond individual-level consequences, ADHD-related mental health and educational services are also substantial (Pelham, Foster, & Robb, 2007). Given the clinical and public health significance of lifetime ADHD, identifying factors that contribute to its onset and development is a priority.

INTERGENERATIONAL CONTINUITY OF ADHD

There is replicated evidence that parent ADHD is a key risk factor for offspring ADHD as well as other dimensions of psychopathology (Chronis et al., 2003; Loeber, Hipwell, Battista, Semboer, & Stouthamer-Loeber, 2009). Compared to children without ADHD, children with ADHD were nearly 8 times more likely to have a parent previously diagnosed with ADHD (Mick, Biederman, Prince, Fischer, & Faraone, 2002). Parent ADHD similarly predicts higher rates of offspring externalizing and internalizing problems, as well as poor social functioning (Minde et al., 2003). The intergenerational continuity of ADHD (i.e., parental and offspring ADHD) reflects substantial shared genetic influences: In a meta-analysis of 22 studies, genetic factors accounted for 71% and 73% of the variance in inattention and hyperactivity symptoms, respectively (Nikolas & Burt, 2010). However, imperfect concordance rates of ADHD among monozygotic (MZ) twins indicate the importance of environment influences: Among 1,480 MZ and dizygotic twins, the cross-MZ twin correlations at age 8, 13, 16, and 19 ranged from $r = .53$ to $.70$ for parent-rated attention problems and from $r = .09$ to $.33$ for self-rated attention problems (Chang, Lichtenstein, Asherson, & Larsson, 2013). Furthermore, only 25 to 50% of parents with ADHD have a biological child with ADHD (Biederman, Faraone, & Monuteaux, 2002; Chronis et al., 2003; Wilens et al., 2005). The inconsistent concordance in rates of ADHD between parent and offspring highlights the need to identify environmental factors and processes that underlie the intergenerational continuity of ADHD.

However, beyond genetic influences, far less is known about additional explanatory processes underlying the association between parent and offspring ADHD. Comparatively, the literature with respect to the intergenerational continuity of other clinical phenotypes, including antisocial behavior, aggression, and depression is more well developed (Gershon et al., 2011; Hammen, Shih, & Brennan, 2004; Serbin & Karp, 2004). For example, the intergenerational transmission of parents' childhood conduct problems to their male offspring's current conduct problems was almost entirely *environmentally* mediated by current parental risk factors (e.g., harsh parenting, divorce), although common genetic factors fully accounted for the parents' transmission of conduct problems to their female offspring (D'Onofrio et al., 2007). Relatedly, family factors (e.g., parenting quality) and interpersonal stress are central to theories of the intergenerational transmission of depression (Hammen et al., 2004). Consistent with this formulation, in a sample of 299 mother-child

dyads, the association between maternal depression and offspring depression was mediated by maternal hostility, although this effect was attenuated after controlling for maternal antisocial behavior (Sellers, Harold, et al., 2013). Together, these studies highlight the importance of identifying environmental mediators contributing to the intergenerational transmission of ADHD.

PARENTING BEHAVIOR AS A MEDIATOR

Individual differences in positive and negative parenting behavior are plausible factors underlying the association of parent and offspring ADHD, given that parent ADHD is correlated with variation in parenting behavior, which is similarly associated with offspring ADHD (Banks, Ninowski, Mash, & Semple, 2008; Ellis & Nigg, 2009). Parents with elevated ADHD symptoms frequently use maladaptive discipline (e.g., harsh punishment, inconsistent discipline; Banks et al., 2008). Elevated maternal ADHD symptoms were also associated with less self-reported involvement and less observed positive parenting (e.g., labeled praise) in a parent–child interaction task, even after controlling for parent depression (Chronis-Tuscano et al., 2008). Parent ADHD symptoms were also positively associated with inconsistent discipline and nonsupportive responses to offspring negative emotions (Mokrova, O'Brien, Calkins, & Keane, 2010). Although the association between parenting behavior and offspring ADHD reflects reciprocal and transactional processes (Burke, Pardini, & Loeber, 2008; Psychogiou, Daley, Thompson, & Sonuga-Barke, 2007), frequent negative parenting and low positive parenting exacerbate ADHD (Li & Lee, 2012; Sonuga-Barke & Halperin, 2010) and independently predict the development of comorbid oppositional defiant disorder (ODD) and conduct disorder (CD; Chronis et al., 2007; Ellis & Nigg, 2009). In a cross-sectional study of 181 children with and without ADHD, maternal inconsistent discipline uniquely predicted offspring ADHD, controlling for parent ADHD as well as child ODD and CD (Ellis & Nigg, 2009). Notably, the parenting difficulties associated with adult ADHD may be further driven by the difficult temperament and behaviors (e.g., hyperactivity) associated with ADHD offspring, which may elicit negative parenting and further contribute to the intergenerational continuity of ADHD (Anastopoulos, Guevremont, Shelton, & DuPaul, 1992; Gau & Chang, 2013).

Parenting behavior contributes to the developmental course of childhood ADHD, including the development of comorbidity (Alizadeh, Applequist, & Coolidge, 2007); thus, parenting behavior may be one *modifiable* mechanism through which parent ADHD leads to offspring ADHD. Individual differences in parenting behavior are a potential pathway through which diverse risk factors (e.g., demographic, clinical) affect child outcome and facilitate efficacious child-focused interventions. For example, there is replicated evidence that individual differences in parenting behavior partially mediate the association of risk factors ranging from socioeconomic disadvantage (McLoyd, 1998), marital dissatisfaction, parental depression and alcohol problems, and parental stress with similarly diverse youth outcomes (e.g., substance problems, psychopathology, academic functioning; Latendresse et al., 2008). There is now emerging evidence that similar processes may underlie the association of parent and offspring ADHD. Controlling for pretreatment ADHD, maternal ADHD symptoms predicted posttreatment child behavior problems (i.e., ODD and CD symptoms) in an intervention study of 6- to 10-year-old children with ADHD; of importance, this

association was mediated by change in observed negative parenting (Chronis-Tuscano et al., 2011). Thus, the relation between maternal ADHD symptoms and attenuated child's response to parent training was partially explained by the differences in reductions of maternal negative parenting behavior. Although Chronis-Tuscano et al. (2011) did not directly examine the association between parent ADHD symptoms and change in child's ADHD symptoms, such findings underscore the potential role of parenting as a mechanism underlying the intergenerational transmission of risk and offspring psychopathology. Of importance, existing studies often ignore the incremental role of different parenting dimensions (e.g., discipline, involvement) on the association between parent and offspring ADHD. That is, simultaneously considering *multiple* facets of parenting behavior is necessary to discern both the collective and unique influences of specific parenting behaviors with respect to parent and offspring ADHD. This approach has important clinical implications given that the most crucial dimensions of parenting behavior underlying the transmission of risk for offspring can be targeted for intervention.

Furthermore, despite replicated evidence on the intergenerational continuity of ADHD and links between parent ADHD symptoms and parenting behavior, the *specificity* of these patterns are unclear given that parent ADHD frequently co-occurs with other mental health problems such as depression, which also predicts individual differences in parenting behavior (Kessler et al., 2006; Lovejoy, Graczyk, O'Hare, & Neuman, 2000). Similar to ADHD, parent depression robustly predicts negative parenting and offspring psychopathology (Gau & Chang, 2013; P. Lee et al., 2013; Mars et al., 2012). In one study, parents reporting elevated depression were four times more likely to have a preschool child with ADHD than nondepressed parents (Bauer, Gilbert, Carroll, & Downs, 2013). Despite the frequent co-occurrence of parent ADHD and depression, as well as evidence that comorbid parent psychopathology is particularly predictive of offspring psychopathology (Loeber et al., 2009; Sellers, Collinshaw, et al., 2013), there is relatively little knowledge about the intergenerational continuity of ADHD in the context of co-occurring parent depression. That is, few studies have directly explored the influence of parenting behavior on the association of parent and offspring ADHD symptoms above and beyond the potential confounding role of parent depression. In one study of 4- to 8-year-old boys, maternal inattention uniquely and positively predicted maternal inconsistent discipline and negatively predicted maternal involvement, even after controlling for parent depression and child characteristics (i.e., age and behavior problems; Chen & Johnston, 2007). Similarly, mothers who reported the most inattention engaged in the most negative parent-child interactions after a parent-training intervention, results that were weakened but still significant after controlling for parent depression and alcohol use (Harvey, Danforth, McKee, Ulaszek, & Friedman, 2003). These results substantiate the potentially independent association of maternal ADHD and maternal depression, beyond other potential child and contextual risk factors, in predictions of parenting behavior and offspring ADHD. Given that previous studies inconsistently considered the role of concurrent parent psychopathology in the intergenerational continuity of parent and offspring ADHD, improved traction on the role of parenting behavior in the context of parent and offspring ADHD requires careful attention to co-occurring parent depression.

PRESENT STUDY

Despite evidence that parent ADHD symptoms are associated with more negative parenting and less positive parenting, and that these differences prospectively predict child ADHD symptoms, few studies have formally evaluated whether individual differences in positive and negative parenting behavior mediate the intergenerational continuity of parent and offspring ADHD. Using a prospective design, which temporally separated the predictor and mediators relative to the outcome, we examined multimethod (i.e., observed, self-report) measures of positive and negative parenting behavior as simultaneous mediators of the association of Wave 1 parent ADHD symptoms and Wave 2 offspring ADHD symptoms. We hypothesized that parent ADHD symptoms would be positively associated with corporal punishment, inconsistent discipline, and observed negative talk and negatively associated with positive parenting behaviors and observed praise. We further predicted that corporal punishment, inconsistent discipline, and observed negative talk would positively predict ADHD symptoms in offspring, whereas positive parenting behaviors and observe praise would inversely predict child ADHD. Finally, we hypothesized that each dimension of parenting behavior would uniquely and significantly mediate the association between parent and offspring ADHD symptoms.

METHOD

Participants

Participants were 120 children (67% male) with ($n = 61$) or without ($n = 59$) ADHD and their biological parents (90% mothers). Children were 5 to 10 years old during their initial visit (i.e., Wave 1), and 7 to 12 years old when they completed their follow-up visit approximately two years later (i.e., Wave 2). Sixty-six percent of parents were Caucasian, 6% African-American, 14% Hispanic, 7% Asian, and 7% Mixed/Other. Families were recruited from a large metropolitan area in the western United States using presentations to self-help groups for ADHD, advertisements mailed to local elementary schools, pediatric offices, clinical service providers, and some referrals from mental health clinics. English fluency was required for parents and children. Families were excluded if their child had a Full Scale IQ less than 70 or a current/previous diagnosis of an autism spectrum, seizure, or neurological disorder that prevented full participation in the study.

Procedures

At baseline (i.e., Wave 1; ages 5–10), study eligibility for interested families was determined through a telephone screening based on the inclusion and exclusion criteria just listed. Eligible families ($n = 230$) were then mailed rating scales (95% returned completed or partially completed rating scales) and invited to our laboratory for in-person assessments. After obtaining parental consent and child assent, parents completed a structured diagnostic interview of child psychopathology and an interview about parenting; in a separate room, children's cognitive, academic, and social-emotional functioning were assessed. In addition, parents and children were videotaped during a parent–child interaction task. All interviewers were initially blind to the child's diagnostic status, although blindness was difficult to maintain following the completion of the Diagnostic Interview Schedule for Children,

Version IV, Parent Version (DISC-IV). Approximately 85% of children were assessed in our laboratory without psychotropic medication (e.g., stimulants). If a child was normally medicated, we asked that parents provide ratings based on the child's unmedicated behavior.

Approximately two years after the baseline evaluation, families were invited back to the laboratory for a follow-up assessment (i.e., Wave 2; ages 7–12). Procedures for the follow-up visit were highly parallel to those of Wave 1 (e.g., structured diagnostic interviews for child and parent psychopathology). Approximately 91% of the initial Wave 1 sample participated in some aspect of the Wave 2 follow-up assessment. Families who participated in the follow-up had a higher mean number of child ADHD symptoms than families who did not participate in Wave 2, $t(226) = -2.08, p = .04$, but there were no other significant demographic (i.e., child age and sex, parent race-ethnicity and sex) or clinical (i.e., parent ADHD and depression symptoms) differences between these two groups of families. In the final analyses of this study, 120 participants had complete data for all primary variables (see Table 1). The Institutional Review Board approved all study procedures.

Measures

Parent ADHD symptoms—Parents self-reported their ADHD symptom severity using the Adult ADHD Self Report Scale (ASRS; Kessler et al., 2005) at Wave 1. The ASRS is an 18-item self-report measure of adult ADHD. Each item is scored on 5-point scale (i.e., *never, rarely, sometimes, often, very often*). The ASRS has excellent test–retest reliability and internal consistency (Kessler et al., 2005; Kessler et al., 2007). In addition, the scale demonstrated convergent validity with clinician-rated measures of ADHD (Adler et al., 2006) as well as external validity with measures of substance abuse and candidate genotypes for ADHD (Reuter, Kirsch, & Hennig, 2006; van de Glind et al., 2013). Although formal clinical cutoffs have not been established for the 18-item ASRS, compared to population-based data, 49.4% of the parents in the current study were at or above the 90th percentile of ADHD symptoms (National Comorbidity Survey, 2005), which reflects the elevated ADHD symptomatology of parents in a case-control study. Given that dimensional ratings of ADHD demonstrate more predictive validity relative to disorder-based comparisons, we used the total score ($\alpha = .94$) to represent parent ADHD symptoms severity (Fergusson & Horwood, 1995).

Parental depression—The Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a 21-item self-report measure of current depression symptomatology. At Wave 1, parents rated the severity of their depression symptoms over the past 2 weeks on a 4-point Likert scale; items were summed to form a composite measure of depression (Cronbach's $\alpha = .84$). The Beck Depression Inventory has been extensively validated and possesses excellent psychometric properties (Beck, Steer, & Garbin, 1988; Yin & Fan, 2000).

Parenting behavior—The Alabama Parenting Questionnaire (APQ; Shelton, Frick, & Wootton, 1996) is a widely used self-report measure consisting of 42 items related to parenting behavior. At Wave 1, parents rated the frequency of each item on a 5-point Likert scale from 1 (*never*) to 5 (*always*). Several distinct APQ factors have been identified

(Chronis-Tuscano et al., 2008; Hawes & Dadds, 2006), including corporal punishment (e.g., “You spank your child with your hand when he/she has done something wrong”), inconsistent discipline (e.g., “You threaten to punish your child and then do not actually punish him/her”), as well as positive aspects of parenting such as involvement (e.g., “You help your child with his/her homework”) and positive reinforcement (“You praise your child if he/she behaves well”). We used the inconsistent discipline (Cronbach’s $\alpha = .64$) and corporal punishment (Cronbach’s $\alpha = .66$) factors for the current analyses. Due to their significant covariation ($r = .69, p < .001$), we created a composite positive parenting factor (Cronbach’s $\alpha = .67$) consisting of a sum of the involvement and positive reinforcement items.

In addition to self-reported parenting behavior, we used the Dyadic Parent Child Interaction Coding System (DPICS; Eyberg, Nelson, Duke, & Boggs, 2005) to obtain observed measures of parenting during a parent–child interaction task administered at Wave 1. The DPICS is a well-validated system of rating parent–child interaction in children with disruptive behavior disorders. Discrete parent and child behaviors were coded continuously, and then composite categories of parenting were created (Chronis-Tuscano et al., 2008; Eyberg et al., 2001; Li & Lee, 2013). Negative talk was coded when parents made hostile or critical comments to their child (e.g., “You’re so irritating sometimes,” “You’re doing that wrong”), negative commands (e.g., “Stop doing that!”), or sarcastic and condescending remarks (e.g., “You think you’re so clever, don’t you?”). Examples of praise included positive appraisals for a child’s behavior, attribute, or a product that the child created (e.g., “You’re a good builder,” “That’s a really pretty picture of a dog you drew”). All parent–child interactions were digitally recorded.

Research assistants completed intensive training on DPICS coding procedures until at least 70% agreement was attained. Coders participated in a full day of training followed by 2 months of practice where each coding category was discussed with reviews/quizzes. Weekly coding meetings were held to ensure reliability and resolve disagreements. To estimate reliability of each composite measure, 20% of the videos was randomly selected and coded by two separate raters. Normative data have been established for the DPICS coding system (Robinson & Eyberg, 1981), and DPICS composite categories have shown moderate to high interrater and test–retest reliability (Chronis-Tuscano et al., 2008). The intraclass correlations (ICC) for our categories were as follows: negativity (ICC = .75), praise (ICC = .88), and child noncompliance (ICC = .78). Moreover, the DPICS has discriminated between treatment and control families in intervention studies, correctly classifying 100% of control families and 85% of treatment families (Robinson & Eyberg, 1981), and has demonstrated predictive validity (Chronis et al., 2007).

Offspring ADHD and ODD—We used the ADHD and ODD modules of the Diagnostic Interview Schedule for Children, Version IV, Parent Version (DISC; Shaffer, Fisher, Lucas, Dulcan, & Schwab-Stone, 2000) to measure *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) offspring ADHD and ODD diagnosis at Wave 1, as well as ADHD symptoms at Wave 2. The DISC is a computer-assisted, fully structured diagnostic interview with the parent that assesses child psychopathology using symptom count, duration, age of onset, and impairment across settings. The

ADHD module of the DISC possesses good psychometric properties, including high internal consistency (Cronbach's $\alpha = .84$) and test-retest reliability ($r = .79$ after 1 year) in a large community sample (Shaffer et al., 2000). At Wave 1, children with at least six symptoms in the inattention or hyperactivity cluster present before the age of 7 and demonstrating impairment across settings were diagnosed with ADHD. Children with at least four symptoms of ODD present for the past 6 months and demonstrating clinical impairment were diagnosed with ODD during Wave 1. We analyzed the total number of the child's ADHD symptoms at Wave 2, controlling for Wave 1 ADHD and ODD diagnostic status.

Data Analyses

Multiple mediation was used to examine whether the association of Wave 1 parent ADHD symptoms, controlling for Wave 1 parent depression and offspring ADHD and ODD, and the number of Wave 2 child ADHD symptoms was collectively and individually mediated by the multimethod and multidimensional measures of parenting behavior from Wave 1 (i.e., self-reported positive parenting, corporal punishment, and inconsistent discipline as well as observed praise and observed negative talk). Unlike traditional mediation (Baron & Kenny, 1986), we implemented a particular type of multiple mediation based on bootstrapping, a powerful nonparametric resampling procedure that enables evaluation of multiple mediators *simultaneously* with adjustment for potential covariates (MacKinnon, Krull, & Lockwood, 2000; Preacher & Hayes, 2008). Notably, evaluation of causal mediation through bootstrapping is not contingent on a significant direct effect of the predictor on the outcome (MacKinnon et al., 2000; Preacher & Hayes, 2008; Zhao, Lynch, & Chen, 2010). In addition to evaluating the total mediation effect in the model, multiple mediation stringently controls for intercorrelation among each mediator, thus discerning the *unique* role of each individual mediator. In addition to these critical advantages, bootstrapping-based multiple mediation is also statistically more powerful than traditional approaches (i.e., Sobel test; Zhao et al., 2010).

Following recommended procedures (Preacher & Hayes, 2008), each multiple mediation model estimated the following parameters: (a) the total effect of Wave 1 parent ADHD symptoms on the number of Wave 2 child ADHD symptoms (i.e., excluding the mediators), (b) specific effect of Wave 1 parent ADHD symptoms on each Wave 1 parenting mediator, (c) specific effects of each Wave 1 mediator on Wave 2 child ADHD symptoms, and (d) the direct effect of Wave 1 parent ADHD symptoms with respect to Wave 2 child ADHD symptoms through each proposed mediator. Given the case-control design, all analyses controlled for offspring ADHD and ODD diagnostic status at Wave 1. Furthermore, given the high comorbidity between parent ADHD and depression symptoms, as well as the association between parent depression and child ADHD (Bauer et al., 2013), we controlled for parent depression to improve the specificity of the putative association of parent ADHD symptoms with child outcome. Parameter estimates and 95% bias-corrected and accelerated confidence intervals for total and specific indirect effects were generated based on 1,000 bootstrap resamples (Preacher & Hayes, 2008). Mediation analyses were conducted using SPSS 20.0 and the publicly available SPSS macro for multiple mediation (<http://afhayes.com/spss-sas-and-mplus-macros-and-code.html>).

RESULTS

Preliminary Analyses

Descriptive statistics of all study variables for offspring with and without ADHD are presented in Table 1; Table 2 shows the intercorrelations among demographic characteristics and other study variables (e.g., parenting behavior, parent ADHD symptoms and depression, offspring ADHD and ODD). Bivariate correlations revealed that child ADHD and ODD at Wave 1 were both correlated with each other, as well as with higher ADHD symptoms at Wave 2, higher parent ADHD symptoms and depression symptomatology, as well as more corporal punishment. As expected, parent depression was positively correlated with parent ADHD symptoms. Based on demographic correlations, several potential demographic variables were evaluated as covariates, including parent's sex and race-ethnicity, as well as the child's age and sex. In the final model, the only demographic covariate that was marginally associated with the dependent variable was the child's age. Thus, in addition to parental depression and offspring ADHD and ODD at Wave 1, child's age was included as a covariate in the final multiple mediation model.

Parenting Behavior Mediating Parent and Child ADHD

The fully saturated model including the independent variable (parent ADHD symptoms), covariates (child age, Wave 1 ADHD and ODD, and parent depression), and mediators (corporal punishment, inconsistent discipline, positive parenting behavior, observed praise, observed negative talk) accounted for 53.6% of the variance in offspring's Wave 2 ADHD symptoms ($F = 12.59$, $R^2 = .54$, $p < .01$). Child's Wave 1 ADHD ($B = 6.78$, $SE = .91$, $p < .01$), and age (marginally; $B = .59$, $SE = .35$, $p = .10$) had unique partial effects on Wave 2 ADHD. In contrast to predictions, the unique partial effect of parent depression and offspring ODD on Wave 2 ADHD was not significant after controlling for other covariates ($B = -.07$, $SE = .08$, $p = .37$ and $B = -.50$, $SE = .90$, $p = .58$, respectively).

Controlling for child age, Wave 1 ADHD and ODD, and parent depression (Figure 1), the total effect of Wave 1 parent ADHD symptoms on Wave 2 child ADHD symptoms was not significant ($B = .06$, $SE = .04$, $p = .12$). Parent ADHD symptoms were significantly associated with higher levels of corporal punishment ($B = .02$, $SE = .01$, $p = .04$) and marginally related to inconsistent discipline and positive parenting behavior ($B = .04$, $SE = .02$, $p = .08$ and $B = -.08$, $SE = .05$, $p = .09$, respectively), above and beyond the effect of parent depression and offspring ADHD and ODD at Wave 1. However, parent ADHD symptoms were unrelated to observed praise or observed negative talk ($B = .06$, $SE = .09$, $p = .49$ and $B = .01$, $SE = .07$, $p = .85$, respectively). Next, Wave 1 observed praise, observed negative talk, and corporal punishment (marginally) each *independently* predicted the number of the child's Wave 2 ADHD symptoms, controlling for parent ADHD symptoms and depression as well as child ADHD and ODD at Wave 1 ($B = .09$, $SE = .04$, $p = .02$; $B = .10$, $SE = .05$, $p = .05$; and $B = .58$, $SE = .34$, $p = .09$, respectively). However, Wave 1 positive parenting behavior and inconsistent discipline were each unrelated with the total number of Wave 2 child ADHD symptoms ($B = -.03$, $SE = .07$, $p = .66$ and $B = .06$, $SE = .17$, $p = .71$, respectively).

Controlling for all five parenting variables, the direct effect of Wave 1 parent ADHD symptoms with respect to Wave 2 child ADHD was not significant ($B = .03$, $SE = .04$, $p = .37$). Total and specific indirect effects of parent ADHD symptoms on child ADHD through the five parenting variables are presented in Table 3, including point estimates and 95% bias corrected and accelerated confidence intervals calculated using 1,000 bootstrap simulation samples. The total indirect effect (i.e., difference between the total effect and direct effect through all mediators) was nonsignificant, although specific indirect effects of parenting variables were observed. Specifically, controlling for child's age, ADHD and ODD at Wave 1, and parent depression, the association of parent ADHD symptoms and number offspring ADHD symptoms at Wave 2 was significantly and independently mediated by corporal punishment (Table 3).

DISCUSSION

Despite replicated evidence that parent ADHD symptoms predict more negative parenting and less positive parenting, which themselves are associated with offspring ADHD symptoms, few studies have tested whether the intergenerational continuity of parent and offspring ADHD is mediated by individual differences in positive and negative parenting behavior. Using a prospective study of a well-characterized sample of 120 children with and without ADHD, we evaluated multimethod parenting dimensions (i.e., corporal punishment, inconsistent discipline, positive parenting behavior, observed negative talk, observed praise) as simultaneous and independent mediators of the prospective association between Wave 1 (i.e., baseline) parent and Wave 2 (i.e., follow-up) offspring ADHD symptoms.

Bootstrapping procedures within a multiple mediation framework yielded several key findings: First, controlling for parent depression and baseline child ADHD and ODD diagnostic status, Wave 1 parent ADHD symptoms were associated with more corporal punishment and marginally related to inconsistent discipline and positive parenting behavior (but parent ADHD symptoms were unrelated to other parenting behaviors). Second, Wave 1 corporal punishment (marginally), observed praise, and observed negative talk each uniquely predicted the number of Wave 2 offspring ADHD symptoms, beyond offspring's baseline ADHD and ODD, parent ADHD and depression symptoms, and all other parenting dimensions. Finally, corporal punishment significantly and independently mediated the prospective association between parent and offspring ADHD, controlling for offspring baseline ADHD and ODD, parent depression, and the remaining parenting behaviors. These findings suggest that individual differences in parenting behavior, specifically corporal punishment, may represent an intermediate construct partly underlying the intergenerational continuity of parent and offspring ADHD.

These findings are consistent with previous research illustrating that parenting behavior partially mediates the intergenerational transmission of diverse problems ranging from antisocial behavior and aggression to depression and substance disorders (D'Onofrio et al., 2007; Hammen et al., 2004; Latendresse et al., 2008). These findings suggest that similar processes may underlie the temporally ordered association of parent and offspring ADHD symptoms reported herein. Notably, previous studies often ignored the potential confounding role of concurrent parent psychopathology, whereas the present findings suggest that multiple dimensions of parenting behavior significantly mediated Wave 1

parent and Wave 2 offspring ADHD symptoms, above and beyond parent depression as well as child characteristics at baseline (e.g., child age, ADHD, and ODD). Traditionally, the intergenerational continuity of ADHD is primarily attributed to genetic factors, which has perhaps indirectly hindered efforts to identify potential *environmental* mediators of this process. However, environmental factors, such as frequent parental use of corporal punishment, may also contribute to the development of ADHD (Ellis & Nigg, 2009), which further reflects the broader importance of parenting behavior to children's social-emotional, cognitive, and behavioral development (Amato & Fowler, 2002; Baumrind, 1991; Hoeve et al., 2009). The present findings suggest that a focus on parenting behavior among *parents* with elevated psychopathology may clarify how offspring behavior problems emerge.

In the current study, corporal punishment significantly and independently mediated the link between parent and offspring ADHD symptoms, even with rigorous statistical control of four different parenting dimensions, offspring's baseline ADHD and ODD, and parent depression. Efforts to examine the differential and incremental role of separate parenting dimensions (e.g., positive vs. negative as well as different parenting facets) in studies of parenting and child development are relatively infrequent, and even fewer studies examine these effects in the context of parent and offspring ADHD. A study that examined an array of parenting dimensions as simultaneous mediators of parent and offspring alcohol use revealed that parental monitoring and discipline were *unique* mediators of the intergenerational transmission of alcohol use, controlling for all other parenting behaviors measured (e.g., warmth, autonomy granting, shared activities; Latendresse et al., 2008). To improve the precision in understanding how specific parenting behaviors affect offspring psychopathology, the simultaneous evaluation of *multiple* facets of parenting behavior is a strength of the present study. The specificity of corporal punishment, beyond all other parenting dimensions, underlying the parent-offspring ADHD association may reflect several key processes. Parents with ADHD may have specific deficits in executive functioning (e.g., inhibitory control, working memory) and self-regulation (e.g., emotion regulation), which may increase the use of corporal punishment (Johnston, Mash, Miller, & Ninowski, 2012; McKay & Halperin, 2001). For example, lower maternal working memory (a key characteristic of individuals with ADHD) predicted increased reactive negativity to difficult offspring behaviors (i.e., opposition and distractibility) in an observed parent-child interaction task, implicating working memory as an important predictor of harsh reactive parenting (Deater-Deckard, Sewell, Petrill, & Thompson, 2010). Furthermore, beyond links to general harsh or negative parenting, inhibitory deficits associated with adult ADHD may be particularly limited to effortful inhibition of *motor* responses (vs. suppressing irrelevant information; Nigg, Butler, Huang-Pollock, & Henderson, 2002). These findings are consistent with the specificity observed in the present study between parent ADHD symptoms and use of physical punishment. Given that the present study did not measure parents' cognitive processes, future research that directly tests the role of cognitive functioning can help to clarify the mechanisms facilitating the association between parent ADHD symptoms and corporal punishment to help alleviate the intergenerational cycle of ADHD.

These preliminary findings suggest potential clinical implications with respect to childhood ADHD. First, given that corporal punishment independently mediated the prospective association between parent and offspring ADHD symptoms, improving parenting behavior may help to interrupt the intergenerational continuity of ADHD. Second, despite evidence that high levels of parent ADHD symptoms are associated with significantly less improvement in offspring symptoms following parent training (Sonuga-Barke, Daley, & Thompson, 2002), parent-training interventions typically do not actively integrate the role of parent ADHD symptoms. Our findings highlight the potential need for tailoring parent-training interventions for *parents* with elevated ADHD. For example, compared to parents without ADHD, parents with ADHD have more deficits in planning, working memory, and emotion regulation (Johnston et al., 2012). Although research on the effects of parent self-regulation on child outcomes is relatively rare, recent studies found that the prospective association of maternal self-regulation with offspring negative emotionality was partially mediated by the child's home environment (i.e., home chaos, spousal relationship; Bridgett, Burt, Laake, & Oddi, 2013). Thus, tailored interventions might require dedicated sessions to target parents' emotion regulation (e.g., anger and frustration), to increase prosocial behaviors (i.e., praise, attending), and/or provide more tools and structure to help parents organize their parenting behaviors.

Several study limitations should be considered in the context of the present findings. First, conceptualizations of causal mediation necessitate temporal ordering of the predictor, mediator, and outcome (i.e., three-occasion data; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). Although this study featured temporally ordered predictors (i.e., parent ADHD symptoms) and mediators (i.e., parenting behavior) relative to the outcome (i.e., offspring ADHD symptoms), precise inferences about the directionality of effects (and hence causal mediation) cannot be made. However, given that ADHD is a childhood-onset disorder, frequently characterized by persistent symptomatology into adulthood (Faraone et al., 2006), parent ADHD symptoms likely preceded parenting behavior for many participants. Second, 90% of the parents in this study were mothers. Given that there may be unique parenting effects and comorbid problems (e.g., antisocial behavior) from fathers that are important to children's socio-emotional functioning (Lewis & Lamb, 2003), future studies that include both parents will help to further understand the intergenerational transmission of ADHD in two-parent families. Third, shared method variance may have influenced the observed associations between self-reported parent ADHD symptoms and parenting behavior, and parent-reported offspring ADHD. To partially address this limitation, we employed multiple methods of assessing parenting, including coded observations during a structured parent-child interaction task. Fourth, we observed lower internal consistency estimates in the Alabama Parenting Questionnaire factors, which is relatively consistent with previous studies demonstrating lower estimates particularly for corporal punishment and inconsistent discipline (Dadds, Maujean, & Fraser, 2003; Ellis & Nigg, 2009; Shelton et al., 1996). However, previous studies from this sample, using the same APQ parenting factors, have demonstrated their predictive validity with relevant criteria, including offspring inattention, ODD, and CD symptoms, aggression, and rule-breaking behavior (Falk & Lee, 2012; Li & Lee, 2012; Tung & Lee, 2014; Tung, Li, & Lee, 2012). Fifth, because adult ADHD and offspring ADHD were measured using adult self-report versus parent structured interview,

respectively, parent and offspring ADHD may not necessarily reflect the same underlying construct. Furthermore, the reliance on self-reported adult ADHD may be susceptible to inconsistent self-reporting. Thus, longitudinal studies that can follow children with ADHD until parenthood are important to confirm these intergenerational effects. Sixth, because families were recruited from unspecified sources of either clinical or community settings, the generalizability of our findings to specific populations is unknown. However, our findings are consistent with studies of both clinic-referred and community-based samples (Chronis-Tuscano et al., 2011; Harvey et al., 2003; Hinshaw et al., 2000). Finally, given that this study did not focus on genetic mediators of ADHD, the observed mediation effects may partially reflect a potential gene–environment correlation between the parent’s corporal punishment and offspring ADHD symptoms. Given that twin studies may overestimate genetic effects due to environmental similarity of twins (Kendler, Neale, Kessler, Heath, & Eaves, 1993), we await future studies that can replicate our findings in a genetically informed adoption sample, thus enabling control of genetic risk for ADHD while testing parenting behavior as an environmental mediator of the intergenerational transmission of ADHD.

This study found preliminary evidence that corporal punishment mediated the prospective association between parent and offspring ADHD symptoms, which is consistent with research demonstrating that parent ADHD influences the quality of parenting behavior. However, whereas previous studies often ignored the differential role of separate parenting dimensions, this study simultaneously evaluated multiple facets of parenting behavior and demonstrated that corporal punishment specifically mediated the association of parent and offspring ADHD symptoms. Furthermore, these mediation effects were evident even after controlling for baseline child ADHD and ODD, child’s age, and parent depression. The specificity of our findings carries potential implications for parent-training interventions for parent–child dyads with ADHD. Although replication studies are needed, these initial findings suggest that interventions for ADHD, particularly those that feature parent training, may benefit from tailoring treatments for parents with ADHD. We conclude by emphasizing that multiple risk factors must be considered in the context of ADHD development, and future research must further identify the underlying mechanisms through which the intergenerational continuity of ADHD occurs.

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FIGURE 1. Multiple mediation of parent and offspring attention-deficit/hyperactivity disorder (ADHD) symptoms by parenting behavior, controlling for child's age, Wave 1 ADHD and oppositional defiant disorder (ODD), and parent depression (unstandardized beta coefficients and standard errors). Note. $^{*}p < .05$. $^{\dagger}p < .10$.

TABLE 1

Descriptive Statistics for Children With and Without ADHD at Wave 1

	<i>ADHD^a</i>	<i>Non-ADHD^b</i>	<i>Test Statistic</i>	<i>P</i>
Child's Age (Wave 1)	7.59 (1.11)	7.95 (1.07)	$t = -1.19$	.08
Child's Sex (% Male)	70.5%	64.4%	$\chi^2 = .51$	.56
Child ODD (% Diagnosed at Wave 1)	54.1%	13.6%	$\chi^2 = 21.91$	<.01
Child's ADHD Symptoms (Wave 2)	11.10 (4.67)	3.39 (3.75)	$t = 9.98$	<.01
Parent's Sex (% Mothers)	89.8%	91.5%	$\chi^2 = 10$	.75
Parent Race-Ethnicity (% Caucasian)	63.8%	68.4%	$\chi^2 = -28$	.60
Parent ADHD Symptoms	28.05 (14.02)	20.81 (8.40)	$t = 3.44$	<.01
Parental Depression Symptomatology	7.51 (6.16)	4.85 (3.82)	$t = 2.86$	<.01
Positive Parenting Behaviors	43.67 (6.66)	42.08 (4.83)	$t = 1.50$	.14
Corporal Punishment	4.33 (1.26)	3.69 (1.15)	$t = 2.87$	<.01
Inconsistent Discipline	9.64 (2.39)	9.02 (2.26)	$t = 1.47$	.15
Observed Praise	13.64 (11.30)	11.73 (10.18)	$t = 0.91$	.33
Observed Negative Talk	10.61 (9.11)	6.88 (6.73)	$t = 2.55$	.01

Note. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.

^a
 $n = 61$.

^b
 $n = 59$.

TABLE 2

Correlations Among All Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Child's Age	—													
2. Child's Sex	-.08	—												
3. Child ADHD (Wave 1)	-.16	.07	—											
4. Child ODD (Wave 1)	-.05	.05	.43**	—										
5. Child ADHD (Wave 2)	-.24**	.04	.68**	.24**	—									
6. Parent's Sex	.11	-.04	-.03	-.02	-.02	—								
7. Parent Race-Ethnicity	.01	<.01	-.05	.09	-.05	-.05	—							
8. Parent ADHD Symptoms	-.03	-.04	.30**	.23*	.28**	.11	.02	—						
9. Parental Depression	.06	-.01	.25**	.24**	.14	.15	.05	.43**	—					
10. Positive Parenting Behaviors	-.14	.20*	.14	-.06	.11	-.60**	.01	-.16	-.14	—				
11. Corporal Punishment	-.03	-.01	.26**	.26**	.28**	.03	-.28**	.27**	.14	-.02	—			
12. Inconsistent Discipline	.06	-.12	.13	.13	.11	.22*	.18	.24**	.18*	-.21*	.24**	—		
13. Observed Praise	-.14	.12	.09	-.11	.21*	.02	.18	.06	-.01	.21*	-.19*	-.13	—	
14. Observed Negative Talk	-.09	.01	.23*	.04	.29**	<.01	-.16	.11	.13	.12	.14	-.05	.01	—

Note. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.

* $p < .05$.

** $p < .01$.

TABLE 3

Mediation by Parenting Behavior on Parent and Child ADHD Symptoms

	Point Est.	SE	95% BCa Bootstrap CI	
			Lower	Upper
Positive Parenting Behavior	<.01	.01	-.0073	.0293
Corporal Punishment	.01	.01	.0004	.0530
Inconsistent Discipline	<.01	.01	-.0075	.0284
Observed Praise	.01	.01	-.0070	.0303
Observed Negative Talk	<.01	.01	-.0094	.0190
Total	.02	.02	-.0033	.0685

Note. Significant mediation effect bolded. Model controlled for child's age, Wave 1 attention-deficit/hyperactivity disorder (ADHD), and oppositional defiant disorder (ODD) diagnostic status, as well as parental depression. Point est. = point estimate of the indirect effect; BCa bootstrap CI = bias corrected and accelerated confidence intervals.