Explaining the Prospective Association of Positive and Negative Parenting Behaviors and Child ADHD:
Pathways Through Child Executive Function and Reward Responsivity

Qi Zhang ¹ and James J. Li ^{2,3,4}

¹ Department of Educational Psychology, University of Wisconsin-Madison

² Department of Psychology, University of Wisconsin-Madison

³ Waisman Center, University of Wisconsin-Madison

⁴ Center for Demography for Health and Aging, University of Wisconsin-Madison

Acknowledgements

The authors express deep gratitude to all the families that participated in the study. The study was supported by grants from Wisconsin Alumni Research Foundation and the UW-Madison Center for Human Genomics and Precision Medicine. J.J.L was supported by a center grant to the Waisman Center from the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (P50HD105353).

Correspondence

James J. Li, Ph.D., Department of Psychology, University of Wisconsin-Madison, 1202 West Johnson Street, Madison, WI, 53706, USA, Email: james.li@wisc.edu, Tel: 1-608-265-1091

Author Biographies

Qi Zhang is a doctoral student in the Department of Educational Psychology at the University of Wisconsin-Madison. She received a bachelor's degree in social work from Beijing Institute of Technology a master's degree in applied psychology from Peking University Health Science Center.

James J. Li, Ph.D. is an Associate Professor in the Department of Psychology, a Waisman Center Investigator, and a faculty affiliate at the Center for Demography for Health and Aging at the University of Wisconsin-Madison. His research examines the interplay between biological and environmental factors underlying the development of childhood externalizing disorders.

Abstract

Objective: Parenting behavior is a well-established correlate of offspring ADHD. Yet, little is known about *how* parenting exerts its effects on offspring ADHD. We examined whether prospective associations between positive and negative parenting behaviors and child ADHD symptoms are mediated by deficits in child executive function (EF) and reward responsivity (RR). **Method:** A total of 135 children, with and without ADHD were assessed at mean ages 6 and 8. Children completed tasks on EF, and parents completed questionnaires about their parenting, and their children's RR and children's ADHD symptoms. **Results:** Negative parenting (but not positive parenting) was indirectly associated with offspring ADHD subtypes via the effects of Wave 1 EF and RR at Wave 2. **Conclusion:** Individual differences in EF and RR during the early childhood years may constitute a potential pathway by which negative parenting behaviors exerts its effects on subsequent offspring ADHD. Treatment implications are discussed.

Keywords: parenting, ADHD, executive function, reward responsivity, longitudinal

Attention-deficit/hyperactivity disorder (ADHD) is one of the most common childhood disorders (Willcutt, 2012). According to the *Diagnostic and Statistical Manual for Mental Disorders* (DSM), ADHD is characterized by developmentally inappropriate and impairing levels of inattention and/or hyperactivity/impulsivity (American Psychiatric Association, 2013). There are three subtypes for ADHD: predominantly hyperactive (ADHD-H), predominantly inattentive (ADHD-I), and a combined subtype (ADHD-C) that features impairing symptoms of both inattention and hyperactivity/impulsivity (Feczko et al., 2019). ADHD not only impairs children's ability to function across multiple domains, including at home, school, and social relationships, but also increases their risk for future occupational instability, criminality, and substance use problems (Langley et al., 2010; Owens et al., 2009).

Negative parenting behaviors are strong correlates as well as robust predictors of ADHD in children (Deault, 2010; Modesto-Lowe et al., 2008). For instance, parents of children with ADHD are often more harsh, critical, and less involved with their children compared to parents of typically-developing children (Sonuga-Barke & Halperin, 2010). Moreover, inconsistent parental discipline predicts child ADHD, even after accounting for parent ADHD and concurrent child behavioral disorders (i.e., oppositional defiant disorder and conduct disorder) (Ellis & Nigg, 2009). Choenni et al. (2019) found that lower levels of maternal sensitivity measured at child age three predicted more children's ADHD symptoms at age eight, even after accounting for cognitive functioning problems at age four. Collectively, evidence from both cross-sectional and prospective longitudinal studies converge on the strong role that parenting behaviors play in development of childhood ADHD. However, there has been comparatively less research on the underlying mechanisms by which negative parenting behaviors exert their impact on ADHD symptoms. Furthermore, relatively few studies have differentiated the effects of negative parenting behaviors (e.g., hostility, harshness) from positive parenting behaviors (e.g., praise, warmth, involvement). Prior work has shown that low negative parenting does not necessarily equate to high positive parenting, as these domains may have differing consequences on child development (Kaiser et al., 2011; Li, 2018). Understanding how variations in parenting behaviors influence child development

has critical importance in ADHD because it may shed light on explaining individual differences in response to evidence-based parent training interventions (Fabiano et al., 2015) .

One possible mechanism that may explain how parenting behaviors influence the development of childhood ADHD symptoms is through deficits in child executive functions (EF), which refer to the cognitive processes that are used to respond to novel or ambiguous situations and to achieve goals (Hughes et al., 2004). It is well established that children with ADHD typically exhibit some form of an EF deficit (Barkley, 2002; Martel et al., 2007). However, there is also evidence that negative parenting behaviors may also have an impact on EF development in early childhood, before an ADHD diagnosis (Blair et al., 2014; Pechtel & Pizzagalli, 2011). For instance, maternal insensitivity and hostility were associated with offspring EF as early as age three (Blair et al., 2011), while hostility was negatively correlated with EF abilities in another study at child age four (Hopkins et al., 2013). Lower levels of parental control were also prospectively associated with EF development two to three years later among preschool-aged children (Bindman et al., 2013). Collectively, these studies provide reason to suspect that offspring EF deficits may mediate associations between negative parenting behaviors and ADHD symptoms in later childhood. However, it is also important to note that the literature on parenting behaviors and offspring EF are inconsistent, especially when different domains of parenting behavior were assessed. Holochwost (2013) found that positive parenting, which was a composite of sensitivity, engagement, positive regard, animation, and stimulation of development, did not significantly predict general EF in preschoolers. Similarly, parental stimulation and disciplinary practices were found to be unrelated to children's general EF in early childhood (Blankson et al., 2011; Weber, 2011). Developmentally sensitive (e.g., longitudinal) research may help to address these discrepancies.

Another possible mechanism that may explain how parenting behavior influences childhood ADHD is through disruptions to the child's reward sensitivity system (Becker et al., 2013). Children with ADHD have been shown to demonstrate aberrant sensitivities to rewards and reinforcement (Luman et al., 2005). For instance, children with ADHD tend to prefer smaller and immediate rewards over larger and delayed rewards (Sonuga-Barke et al., 2008). A recent meta-analysis found that there was an increase in

inhibitory control task performance in children with ADHD (relative to children without ADHD) when rewards are presented (Ma et al., 2016), suggesting that youths with ADHD may have a heightened sensitivity to reward. But to what extent does parenting behavior influence associations between child reward sensitivity system and their ADHD? Li (2018) found that only young children with high reward responsivity (RR) (as measured from a parent questionnaire) had more symptoms of ADHD, but only under conditions of low negative parenting and high positive parenting. Given that parenting behavior itself has been shown to independently predict children's RR across a multitude of methods (King et al., 2013; Kopala-Sibley et al., 2020), it is plausible that individual differences in children's RR may be an constituent in mechanistic models of ADHD.

Importantly, disruptions to RR may be independent from EF deficits, suggesting that mechanistic models of ADHD involving EF and RR may not necessarily be "one in the same". Sonuga-Barke (2002) proposed a dual pathway model in which there are two independent systems affecting ADHD: executive dysfunction grounded in dorsal frontostriatal dysregulation, and aberrant reward processing underpinned by disruptions in the ventral frontostriatum. Follow up research on this model suggest that deficits in these pathways may be ADHD-subtype specific. For example, Thorell (2007) found that EF deficits were more associated to symptoms of ADHD-I symptoms whereas delay aversion (i.e., a closely linked construct to RR) was more associated with ADHD-H symptoms among kindergarten children. Similarly, two studies showed children with the ADHD-H subtype exhibited fewer EF deficits by late childhood and adolescence (Chhabildas et al., 2001; Schmitz et al., 2002) relative to children with the ADHD-I subtype (Chhabildas et al., 2001). Overall, these lines of research suggest EF and RR deficits may be differentiated, where deficits in either (or both) these pathways may lead to subtype differences in the development of ADHD in children (Sonuga-Barke et al., 2010). Our study extends this work by examining whether deficits in either of these pathways (EF and RR) may explain the prospective association between parenting behavior and child ADHD.

The present study utilized a well-characterized sample of 4–6-year-old children, with and without ADHD, who have been followed at two time points (roughly two years apart) to test the hypothesis that

the association between positive and negative parenting behaviors (measured at Wave 1) and ADHD (measured at Wave 2) are mediated by child EF and RR (both measured at Waves 1 and 2). Because there may be differential associations between EF/RR deficits and the ADHD subtypes (Nigg et al., 2005), we separately tested sequential multiple mediation models for each of the three ADHD subtypes. Based on prior evidence from the dual pathway model, EF deficits are hypothesized to mediate associations between both negative and positive parenting behaviors and ADHD-I symptoms specifically, RR deficits are expected to mediate associations between both dimensions of parenting behaviors and ADHD-H symptoms, and both of EF and RR are expected to mediate the association between both dimensions of parenting behaviors and ADHD-C symptoms.

Method

Participants and Procedures

The current study is part of an ongoing longitudinal study on biological and environmental predictors of externalizing behaviors in 4–6-year-old children, with and without attention and behavioral problems, who are followed over time. The baseline (Wave 1) sample included 210 children (mean age = 6.02, *S.D.* = .43) and their parents (93% parents were mothers). Participants were recruited through multiple resources, including via “backpack mail” delivered via children in local pre-kindergarten and kindergarten classrooms, child development research registries, social media, and via brochures and flyers distributed throughout local communities. Participants were ineligible to participate if they were previously diagnosed with an intellectual disability or autism spectrum disorder, did not live with a biological parent at least half of the time, or were not fluent in English. Crucially, the study was not designed to be case-control, but rather, enriched with young child participants with elevated levels of ADHD and other externalizing problems. These children are being followed prospectively to study the environmental and biological factors that predict the remission, development, and persistence of ADHD over time. Thus, at Wave 1, 20.5% of child participants met full diagnostic criteria for any subtype of ADHD (4.8% ADHD-I, 9.0% ADHD-H, and 6.7% ADHD-C), and an additional 26.2% were “subthreshold” for ADHD (defined as having met 3 or more symptoms within either the ADHD-I or

ADHD-H clusters). 135 children returned for a follow-up approximately two years later (mean age = 8.35, *S.D.* = .64, 92% parents were mothers), among which 20.7% met diagnostic criteria of any subtype of ADHD (7.4% ADHD I, 5.9% ADHD-H, and 7.4% ADHD-C) and 23.0% were subthreshold for ADHD. The present study used data from participants who completed the first and the second Waves of data collection. Among the final analytic sample, 56.3% of the children were boys, and 85.2% were White, 1.5% Black/African American, 2.2% Asian, 2.2% Latino/Latina, and 8.1% Multiracial American. Moreover, 79.2% of the parents in the study reported having a bachelor's degree or higher. Parent-reported gross household income ranged from \$70,000 to \$400,000, with a median income of \$115,000. The study sample is demographically representative of the community (i.e., small urban city in the United States with a very large public university).

Families who were eligible were then mailed a packet with questionnaires to complete before the laboratory visit. If the child was prescribed a stimulant medication for their ADHD, parents were asked to bring their children in on a medication wash of 24 hours prior to their visit. On the day of the laboratory visit, child participants were asked to complete a battery of age-appropriate EF tasks during both data collection waves. While the children were being assessed, a second research assistant administered a fully-structured clinical interview *Diagnostic Interview Schedule for Children, Version IV* (DISC-IV) (Shaffer et al., 2000) to the parents.

Measures

Alabama Parenting Questionnaire (APQ) (Shelton et al., 1996). The APQ was assessed at Waves 1 and 2. It is comprised of 42 items by which parents self-reported on the frequency of parenting behaviors on a 5-point Likert scale (i.e., 1 = *never* to 5 = *always*). There are five subscales, including parents' level of involvement (e.g., "you ask your child about his/her homework;" 10 items), frequency of using positive reinforcement strategies (e.g., "you let your child know when he/she is doing a good job with something;" 6 items), parents' self-reported frequency of using corporal punishment (e.g., "you slap your child when he/she has done something wrong;" 10 items), having poor monitoring (e.g., "your child is out with friends you don't know;" 10 items), and being inconsistent in discipline (e.g., "the punishment

you give your child depends on your mood;" 6 items) with their child. Based on previous factor analytic studies (Kaiser et al., 2011; Li & Lee, 2012), the positive parenting dimension of the APQ includes parents' level of involvement and frequency of using positive reinforcement strategies with their child. The negative parenting dimension of the APQ includes the parents' self-reported frequency of the use of corporal punishment, poor monitoring, and inconsistent disciplining of their child. Furthermore, higher scores on the positive parenting dimension represent more positive parental practices whereas higher scores on the negative parenting dimension represent less suitable parental practices. The APQ scales demonstrate a moderate internal consistency (Cronbach's alpha ranged from .58 to .70) (Shelton et al., 1996) and a convergent validity with observed parent-child interaction measures (Chronis-Tuscano et al., 2011). The Wave 1 APQ scores were used in this study. 76% of the measure were completed by mother only, 4.8% of the data were completed by father only, and 18.6% were completed by both parents. When both parents completed the APQ, the mean score of two informants was used. Cronbach's alpha values were .82 and .65 for the positive parenting (16 items) and negative parenting (26 items) dimensions, respectively.

EF Touch (Willoughby et al., 2010). EF Touch was assessed to children at Wave 1 and is a computerized-cognition battery of tasks that were specifically designed for preschool-aged children. A trained research assistant read the script displayed in one monitor while the child responded to stimuli using the other monitor. While the complete battery has seven EF and two non-EF tasks (one warm-up/orientation and one simple reaction time), we used data from seven tasks: *Spatial Conflict Arrows* (36-item spatial conflict task measuring inhibitory control and cognitive flexibility), *Silly Sounds Stroop* (17-item Stroop-like task measuring inhibitory control), *Animal Go/No-Go* (40-item go/no-go task measuring inhibitory control), *Working Memory Span* (18-item span task measuring working memory), *Pick the Picture* (32-item task measuring working memory), *Farmer* (36-item task measuring visual spatial working memory), and *Something's the Same* (30-item task measuring attention shifting and flexible thinking. Additional details can be found in supplementary materials. The EF Touch has modest test-retest reliability ($r = .60$) and strong criterion validity, as scores on the EF-touch were robustly associated

with ADHD symptoms and intelligence in 1,292 children (Willoughby et al., 2017). As recommended previously, scores for the six EF tasks were combined into an EF composite score (Willoughby et al., 2018). For the current study, all tasks were scored as the percentage of correct responding. The average score was standardized to yield a composite *z*-score to represent global levels of EF, with higher scores indicating higher levels of EF.

NIH Toolbox Cognition Battery (NIH-TCB) (Bauer & Zelazo, 2013). Given that children in the study “aged out” of the EF Touch by their one year follow up, we used the NIH-TCB to measure comparable EF domains in children at Wave 2. The NIH-TCB is a developmentally-sensitive battery of computerized assessments used to measure cognitive, emotional, sensory, and motor health from ages 3 to 89. Our study used the age 3-11 cognition battery. The NIH-TCB was normed using a U.S. sample of 1,020 typically developing children and adolescents aged 3 to 20 years (Akshoomoff et al., 2014). The battery consisted of seven tasks, including *Dimensional Change Card Sort Test* (a measure of cognitive flexibility), *Flanker Inhibitory Control and Attention Test* (a measure of inhibition and visual attention), *Picture Sequence Memory Test* (a measure of episodic memory), *List Sorting Working Memory Test* (a measure of working memory), *Pattern Comparison Processing Speed Test* (a measure of the speed of processing), *Oral Reading Recognition Test* (a measure of reading decoding skills), and *Picture Vocabulary Test* (a measure of receptive vocabulary). Additional details of the battery are described in supplementary materials. Age-corrected cognitive function composite scores were used in the current study, for which mean and standard deviation are 100 and 15, respectively. NIH-TCB have demonstrated adequate convergent and discriminant validity (Weintraub et al., 2013). The composite score also has shown acceptable internal consistency (Cronbach’s $\alpha = .77$) and excellent test-retest reliability ($r = .90$) (Heaton et al., 2014).

Sensitivity to Punishment/Reward Responsivity Questionnaire for Children (SPSRQ-C) (Colder & O’Connor, 2004). SPSRQ-C was assessed at Waves 1 and 2 to parents and is a 33-item measure of a child’s sensitivity to punishment and rewards, where items were rated on a 5-point Likert scale (1 = strongly disagree and 5 = strongly agree). This measure includes two composite scales: Sensitivity to

Punishment (15 items) and Sensitivity to Reward (18 items). Sensitivity to Reward consisted of three subscales: reward responsivity (RR), drive, and impulsivity/fun-seeking. Due to the overlap between the drive (i.e., “your child likes displaying their physical abilities even though it may involve danger”) and impulsivity/fun-seeking (i.e., “your child often has trouble resisting the temptation of doing forbidden things”) subscales and ADHD symptoms, the present study used only the RR subscale. The SPSRQ-C has demonstrated good external and convergent validity (Luman et al., 2012). For the current study, RR demonstrated high internal consistency at both time points (Cronbach’s $\alpha = .85$ and $.88$, respectively).

Vanderbilt Assessment Scale (NICHQ, 2002). The Vanderbilt Assessment Scale was assessed to parents at Wave 2 and consists of DSM-5 screening criteria for ADHD, oppositional defiant disorder (ODD), conduct disorder (CD), and a brief assessment for anxiety and depressive disorders. Parents were asked to rate on the frequency of these symptoms in their children over the past six months on a 4-point Likert scale, where 0 = rarely and 3 = very often. A score of 2 or 3 is coded as a positive endorsement of the symptom and a score of 0 or 1 indicates no presence of the symptom. Present inattention symptoms (9 items) were summed to derive an inattentive subtype (ADHD-I) symptom score, and present hyperactive-impulsive symptom (9 items) were summed to create a hyperactive-impulsive subtype (ADHD-H) symptom score. Present ADHD symptoms (18 items) were summed to create a total ADHD (ADHD-C) symptom score. 47% of the scores were completed by mother only ($n = 63$), and 53% were completed by both parents ($n = 72$). In cases where both parents completed the measure, the average scores of both parent reports were used. The Vanderbilt Assessment Scale has demonstrated a high level of internal consistency and concurrent validity (Wolraich et al., 2003). ADHD-I, ADHD-H, and ADHD-C symptom scores were used in the current study, all of which showed excellent internal consistency (Cronbach’s $\alpha = .92$, $.91$, and $.94$, respectively).

Diagnostic Interview Schedule for Children, Version IV (DISC-IV) (Shaffer et al., 2000). ADHD symptoms were also assessed via DISC-IV to parents at Wave 2. The DISC-IV is a structured, computer-assisted clinical interview conducted with parents based on DSM-IV criteria for a range of disorders (including ADHD). The DISC-IV requires the interviewer to ask questions and enter answers directly on

the computer, and based on previous answers, a computer algorithm determines the questions and ultimately, the diagnosis for the child. The ADHD module has demonstrated a good test-retest reliability ($r = .79$) and internal consistency (Cronbach's $\alpha = .84$) in a large community sample (Shaffer et al., 2000). Instead of characterizing ADHD categorically, the current study used a dimensional conceptualization of ADHD by summing the symptom count for child ADHD in all analytic models where higher scores represent higher levels of ADHD symptoms. The ADHD-I, ADHD-H, and ADHD-C symptoms scores measured were used in the current study.

Data Analysis

Analyses were performed in *R 4.0.3*. Serial multiple mediator models using structural equation modeling (SEM) were tested to investigate longitudinal indirect and direct effects of predictors (i.e., positive and negative parenting) on the outcome (i.e., ADHD symptoms), via the effects of EF and RR measured between the predictor (Wave 1) and the outcome (Wave 2). The effect of the predictor on the outcome is considered a “stage sequential” effect as predictor, outcome, and mediator are not required to be correlated according to updated tests of mediation (Preacher & Hayes, 2008). ADHD was a latent construct as measured via two observed variables (i.e., DISC-IV and Vanderbilt Assessment Scale).

A total of six models were tested using SEM. General model 1 examined the direct and indirect effects of *positive parenting* behaviors on child ADHD symptoms through the effects of EF and RR at Wave 1 and Wave 2, while controlling for child sex, gross household income, and children's age at second visit. Because we analyzed the subtypes of ADHD separately, there were three sub-models under this general model, which examined the direct and indirect effects of positive parenting on latent dimensions ADHD-I, ADHD-H, and ADHD-C symptoms, respectively. General model 2 examined the direct and indirect effects of *negative parenting behaviors* on children's ADHD through the effects of EF and RR at Wave 1 and Wave 2, while controlling for child sex, gross household income, and children's age at second visit. Once again, we tested three sub-models reflecting the three latent ADHD subtypes as separate outcomes.

Results

Descriptives

Table 1 presents means, standard deviations, and bivariate correlations of all study variables. All of three subtypes of ADHD measured by the DISC-IV were positively correlated with negative parenting ($r = .20, .18$, and $.19$ for ADHD-I, ADHD-H, and ADHD-C, respectively; $p < .05$), although negative parenting was not correlated with ADHD symptoms measured by Vanderbilt Assessment Scale. Positive parenting was not correlated with ADHD using either DISC-IV or Vanderbilt Assessment Scale. Child ADHD-I and ADHD-C symptoms measured by both DISC-IV and the Vanderbilt Assessment Scale were negatively correlated with child Wave 1 EF (r 's = $-.42 - -.47$, $p < .001$) and Wave 2 EF (r 's = $-.21 - -.27$, $p < .05$), whereas ADHD-H symptoms measured by two measures were only significantly correlated with Wave 1 EF (r 's = $-.30 - -.41$, $p < .001$). All of three subtypes of ADHD symptoms measured by two measures were also positively correlated with Wave 1 and Wave 2 RR (r 's = $.18 - .45$, $p < .05$).

Model 1: Positive Parenting on Child ADHD Symptoms via EF and RR

First, we examined the direct and indirect effects of positive parenting on a latent dimension of ADHD symptoms through the effects of EF and RR at Wave 1 and Wave 2. Model fit indices are presented in Table 2. Overall, the models examining latent ADHD-I (CFI = .983, TLI = .954, RMSEA = .052, SRMR = .048) and latent ADHD-C (CFI = .981, TLI = .950, RMSEA = .055, SRMR = .048) as outcome variables fit the data well, whereas the model with latent ADHD-H showed acceptable fit (CFI = .957, TLI = .886, RMSEA = .078, SRMR = .051).

Table 3 provides the standardized indirect effects of positive parenting on child ADHD symptoms through EF and RR and confidence intervals. Figure 1a provides the results of each regression path in the model using ADHD-I as the outcome. Although positive parenting was not associated with latent ADHD-I with or without accounting for the effects of EF and RR, latent ADHD-I were associated with Wave 1 EF ($\beta = -.443$, 95% CI $[-.608, -.277]$, $p < .001$) and Wave 2 RR ($\beta = .302$, 95% CI $[.124, .479]$, $p < .01$). There was no evidence of indirect effects of positive parenting on latent ADHD-I through the effects of EF and

RR, but Wave 1 EF was significantly associated with Wave 2 RR ($\beta = -.236$, 95% CI $[-.371, -.101]$, $p < .01$).

Next, we examined the direct and indirect effects of the positive parenting on latent ADHD-H symptoms through the effects of EF and RR at Wave 1 and Wave 2 (Table 3). Results from this model were in line with the model with latent ADHD-I (Figure 1b). Positive parenting was not significantly associated with latent ADHD-H, whereas Wave 1 EF and Wave 2 RR were associated with latent ADHD-H ($\beta = -.517$, 95% CI $[-.694, -.340]$, $p < .001$, and $\beta = .435$, 95% CI $[.246, .624]$, $p < .001$, respectively). Moreover, Wave 1 EF was associated with Wave 2 RR ($\beta = -.236$, 95% CI $[-.371, -.101]$, $p < .01$). There was no evidence of indirect effects of positive parenting on latent ADHD-H through the effects of EF and RR.

We then examined the direct and indirect effects of the positive parenting on child ADHD-C symptoms through the effects of EF and RR at Wave 1 and Wave 2 (Table 3 and Figure 1c). Once again, positive parenting was not associated with latent ADHD-C, whereas Wave 1 EF and Wave 2 RR were associated with latent ADHD-C ($\beta = -.489$, 95% CI $[-.647, -.331]$, $p < .001$, and $\beta = .413$, 95% CI $[.243, .582]$, $p < .001$, respectively). In line with previous models, Wave 1 EF was associated with Wave 2 RR ($\beta = -.236$, 95% CI $[-.371, -.101]$, $p < .01$). There was no evidence of indirect effects of positive parenting on latent ADHD-C symptoms through the effects of EF and RR.

Model 2: Negative Parenting on Child ADHD Symptoms via EF and RR

The direct and indirect effects of negative parenting on child ADHD symptoms through the effects of EF and RR at Wave 1 and Wave 2 were examined. As shown in Table 2, the model with latent ADHD-I as the outcome fit the data well (CFI = .969, TLI = .917, RMSEA = .070, SRMR = .052), whereas the ADHD-H model (CFI = .925, TLI = .802, RMSEA = .106, SRMR = .057) and ADHD-C models (CFI = .960, TLI = .895, RMSEA = .082, SRMR = .053) both exhibited acceptable data fit.

First, we examined the direct and indirect effects of the negative parenting on child ADHD-I symptoms through the effects of EF and RR at Wave 1 and Wave 2 (Table 3 and Figure 2a). With and without accounting for the effects of EF and RR, negative parenting was not significantly associated with

latent ADHD-I. However, negative parenting indirectly influenced latent ADHD-I via the effects of Wave 1 EF (95% CI [.036, .228]). Latent ADHD-I was significantly associated with Wave 1 EF and Wave 2 RR ($\beta = -.486$, 95% CI [-.653, -.318], $p < .001$, and $\beta = .276$, 95% CI [.095, .457], $p < .001$, respectively). In addition, Wave 2 RR was significantly associated with Wave 1 EF and negative parenting ($\beta = -.269$, 95% CI [-.404, -.134], $p < .001$, and $\beta = -.149$, 95% CI [-.281, -.017], $p < .05$, respectively); no significant indirect effect of Wave 2 RR was found.

Next, we examined the direct and indirect effects of the negative parenting on child ADHD-H symptoms through the effects of EF and RR at Wave 1 and Wave 2 (Table 3 and Figure 2b). Without accounting for the effects of EF and RR, negative parenting was significantly associated with latent ADHD-H ($\beta = .199$, 95% CI [.017, .382], $p < .05$). After accounting for the effects of EF and RR, the association between the negative parenting and latent ADHD-H was not significant. The indirect effect of negative parenting on latent ADHD-H through the effects of Wave 1 EF was significant (95% CI [.036, .229]). Moreover, the indirect effect of negative parenting on latent ADHD-H through Wave 1 EF and Wave 2 RR was also significant (95% CI [.003, .061]). There was no evidence of an indirect effect of the negative parenting on latent ADHD-H through the effects of Wave 2 RR.

Finally, we examined the direct and indirect effects of the negative parenting on child ADHD-C symptoms through the effects of EF and RR at Wave 1 and Wave 2 (Table 3 and Figure 2c). With and without accounting for the effects of EF and RR, the association between the negative parenting and latent ADHD-C was not significant. The indirect effect of negative parenting on latent ADHD-C through the effects of Wave 1 EF was significant (95% CI [.039, .231]), and the indirect effect through the effects of Wave 2 RR was significant (95% CI [-.121, -.001]). Furthermore, the indirect effect of negative parenting on latent ADHD-C through Wave 1 EF and Wave 2 RR was also significant (95% CI [.003, .057]).

Discussion

Variations in parenting behaviors are extremely robust and well-established correlates of offspring ADHD. Yet, little is known about *how* parenting, and particularly negative parenting, exerts its

effects on offspring ADHD. This study tested the primary hypothesis that individual differences in child EF and RR may constitute as potential mechanisms underlying prospective associations between variations in parenting behaviors and the development of offspring ADHD symptoms. We tested our hypotheses using a sequential multiple mediation model in SEM from a well-characterized sample of children, with and without ADHD, followed longitudinally (two years apart). No significant indirect effects of EF and RR between positive parenting behaviors and ADHD symptoms were found. However, we found that negative parenting and ADHD-I symptoms were partially mediated via the effects of children's EF at Wave 1, and the association between negative parenting and ADHD-H and ADHD-C symptoms were partially mediated via both EF at Wave 1 and RR at Wave 2. Overall, our findings provide preliminary evidence for the importance of considering individual differences in children's EF and RR as possible mechanisms by which parenting exerts its effects on offspring ADHD.

First, positive parenting was not significantly associated with either children's EF, RR or their ADHD symptoms. While prior studies have also shown mixed results with respect to the links between positive parenting and preschooler EF (Bindman et al., 2013; Holochwost, 2013), we were surprised that positive parenting was not prospectively associated with ADHD symptoms (either directly or indirectly via EF/RR). We surmise that the quality of the parental involvement also needs to be considered with the quantity of parental involvement (Rogers et al., 2009). Additionally, it is also possible that the APQ may not have been sensitive to other (important) forms of positive parenting, which may be more related to ADHD and EF/RR in children. For instance, studies have reported on the association of autonomy support (i.e., scaffolding) on the development of child EF (Bernier et al., 2010), as well as the effects of parental emotional sensitivity and its associations with child ADHD symptoms (Choenni et al., 2019). These constructs were not assayed on the APQ. More research is needed to consider other dimensions of positive parenting behaviors and their contributions to child EF and RR development.

In contrast to our findings with positive parenting, we found that negative parenting was negatively associated with child EF at Wave 1, which in turn contributed to child ADHD at Wave 2. These associations are broadly congruent with findings from previous EF studies (Blair et al., 2011;

Hopkins et al., 2013) that have shown how negative parenting (such as harsh punishment and hostility) negatively impacts children's development of their EF skills, perhaps directly via altered brain development (Moriguchi, 2014). However, EF not only partially mediated the association between negative parenting and ADHD-I/ADHD-C symptoms as we had hypothesized, but also partially mediated the association between negative parenting and ADHD-H symptoms as well. EF deficits are predictive of a broad number of negative outcomes, and likely beyond just a single subtype of ADHD (Chhabildas et al., 2001; Schmitz et al., 2002). With respect to the indirect pathway of negative parenting on ADHD symptoms via RR, high levels of negative parenting may be especially associated with ADHD in children with already heightened levels of RR (Becker et al., 2013; Li, 2018), as these children may exhibit greater sensitivity to environmental stress and adversity. Our findings suggest that negative parental behaviors in early childhood may affect the development of the offspring's EF and RR abilities, which in turn may explain the development of certain subtypes of ADHD. However, we caution that individual differences in offspring EF and RR are also influenced by other crucial factors, such as genetics (Li & Roberts, 2017). It is also important to consider that EF and RR are constituents of a much larger constellation of factors that mediate the complex association between parental behavior and offspring ADHD, including parent-child relationships (Lifford et al., 2008) and socioeconomic disadvantage (Russell et al., 2016).

Our findings should be interpreted in light of some limitations. First, each of our primary measures were based on the parents' report. Our findings could be affected by social desirability bias, whereby (for example) parents might over report on their positive parenting while potentially under reporting on their negative parenting practices. Integration of multiple measures, such as observational or ecological momentary assessments, may help to address informant biases in our measures of parenting behavior. Given the on-going nature of this study, we plan to probe additional measures of parenting in follow up studies. Second, although standardized lab-based cognitive tasks were used to assess children's EF at both Waves, the task at Wave 1 (EF touch) was different from the task at W2 (NIH toolbox). While both tasks demonstrated good evidence of validity, replication of the findings using the same measure of EF over time is warranted. Third, the sample was predominantly white, which is representative of the

surrounding community but may lack generalizability to other racial-ethnic groups. Fourth, although the current study used a prospective longitudinal design with which to conduct serial mediation models, our sample sizes were still relatively small (after accounting for attrition by Wave 2) and limited to two time points. Until more data is collected at later time points, we refrained from drawing strong causal inferences regarding parenting effects on child EF, RR, and ADHD.

Despite these limitations, the results may have important clinical implications, particularly with respect to assessments and treatments based on ADHD subtypes. For example, our findings suggest that child RR and EF may be important to track in treatments for children with ADHD, especially as RR were shown to directly and indirectly mediate the link between negative parenting practices and the emergence of ADHD-H and ADHD-C symptoms. Additionally, behaviorally-based treatments such as parent management training could consider how individual differences in child EF and RR may temper or augment the beneficial effects of these treatments. In sum, our findings provide insights into *how* negative parenting behaviors eventuate into ADHD symptoms as a function of childhood EF and RR deficits. Such advances in our understanding may help to explain why some children with ADHD are more responsive to parenting-based interventions than others, and eventually lead to more tailored interventions for these children.

References

- Akshoomoff, N., Newman, E., Thompson, W. K., McCabe, C., Bloss, C. S., Chang, L., ... Frazier, J. A. (2014). The NIH Toolbox Cognition Battery: results from a large normative developmental sample (PING). *Neuropsychology*, 28(1), 1.
- American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders. *BMC Med*, 17, 133–137.
- Barkley, R. A. (2002). International consensus statement on ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41(12), 1389.
- Bauer, P. J., & Zelazo, P. D. (2013). Ix. Nih toolbox cognition battery (cb): summary, conclusions, and implications for cognitive development. *Monographs of the Society for Research in Child Development*, 78(4), 133–146.
- Becker, S. P., Fite, P. J., Garner, A. A., Greening, L., Stoppelbein, L., & Luebke, A. M. (2013). Reward and punishment sensitivity are differentially associated with ADHD and sluggish cognitive tempo symptoms in children. *Journal of Research in Personality*, 47(6), 719–727.
- Bernier, A., Carlson, S. M., & Whipple, N. (2010). From External Regulation to Self-Regulation: Early Parenting Precursors of Young Children's Executive Functioning. *Child Development*, 81(1), 326–339.
- Bindman, S. W., Hindman, A. H., Bowles, R. P., & Morrison, F. J. (2013). The contributions of parental management language to executive function in preschool children. *Early Childhood Research Quarterly*, 28(3), 529–539.
- Blair, C., Granger, D. A., Willoughby, M., Mills-Koonce, R., Cox, M., Greenberg, M. T., ... Investigators, F. L. P. (2011). Salivary cortisol mediates effects of poverty and parenting on executive functions in early childhood. *Child Development*, 82(6), 1970–1984.
- Blair, C., Raver, C. C., & Berry, D. J. (2014). Two approaches to estimating the effect of parenting on the development of executive function in early childhood. *Developmental Psychology*, 50(2), 554–565.
<https://doi.org/10.1037/a0033647>

- Blankson, A. N., O'Brien, M., Leerkes, E. M., Marcovitch, S., & Calkins, S. D. (2011). Shyness and vocabulary: The roles of executive functioning and home environmental stimulation. *Merrill-Palmer Quarterly (Wayne State University. Press)*, 57(2), 105.
- Chhabildas, N., Pennington, B. F., & Willcutt, E. G. (2001). A comparison of the neuropsychological profiles of the DSM-IV subtypes of ADHD. *Journal of Abnormal Child Psychology*, 29(6), 529–540.
- Choenni, V., Lambregtse-van den Berg, M. P., Verhulst, F. C., Tiemeier, H., & Kok, R. (2019). The Longitudinal Relation between Observed Maternal Parenting in the Preschool Period and the Occurrence of Child ADHD Symptoms in Middle Childhood. *Journal of Abnormal Child Psychology*, 47(5), 755–764. <https://doi.org/10.1007/s10802-018-0492-9>
- Chronis-Tuscano, A., O'Brien, K. A., Johnston, C., Jones, H. A., Clarke, T. L., Raggi, V. L., ... Seymour, K. E. (2011). The relation between maternal ADHD symptoms & improvement in child behavior following brief behavioral parent training is mediated by change in negative parenting. *Journal of Abnormal Child Psychology*, 39(7), 1047–1057.
- Colder, C. R., & O'Connor, R. M. (2004). Gray's reinforcement sensitivity model and child psychopathology: Laboratory and questionnaire assessment of the BAS and BIS. *Journal of Abnormal Child Psychology*, 32(4), 435–451.
- Crockett, L. J., Raffaelli, M., & Shen, Y. (2006). Linking self-regulation and risk proneness to risky sexual behavior: Pathways through peer pressure and early substance use. *Journal of Research on Adolescence*, 16(4), 503–525.
- Deault, L. C. (2010). A systematic review of parenting in relation to the development of comorbidities and functional impairments in children with Attention-Deficit/ Hyperactivity Disorder (ADHD). *Child Psychiatry and Human Development*, 41(2), 168–192. <https://doi.org/10.1007/s10578-009-0159-4>
- Ellis, B., & Nigg, J. (2009). Parenting practices and attention-deficit/hyperactivity disorder: New findings suggest partial specificity of effects. *Journal of the American Academy of Child & Adolescent*

Psychiatry, 48(2), 146–154.

Fabiano, G. A., Schatz, N. K., Aloe, A. M., Chacko, A., & Chronis-Tuscano, A. (2015). A systematic review of meta-analyses of psychosocial treatment for attention-deficit/hyperactivity disorder.

Clinical Child and Family Psychology Review, 18(1), 77–97.

Feczko, E., Miranda-Dominguez, O., Marr, M., Graham, A. M., Nigg, J. T., & Fair, D. A. (2019). The heterogeneity problem: Approaches to identify psychiatric subtypes. *Trends in Cognitive Sciences*, 23(7), 584–601.

Holochwost, S. J. (2013). Cumulative Risk, Parenting Behaviors, and the Development of Executive Functions in Early Childhood: A Moderating Role for Physiological Self-Regulation? The University of North Carolina at Chapel Hill.

Hopkins, J., Lavigne, J. V., Gouze, K. R., LeBailly, S. A., & Bryant, F. B. (2013). Multi-domain models of risk factors for depression and anxiety symptoms in preschoolers: Evidence for common and specific factors. *Journal of Abnormal Child Psychology*, 41(5), 705–722.

Hughes, C., Graham, A., & Grayson, A. (2004). Executive function in childhood: Development and disorder. *Cognitive and Language Development in Children*, 205–230.

Kaiser, N. M., McBurnett, K., & Pfiffner, L. J. (2011). Child ADHD severity and positive and negative parenting as predictors of child social functioning: Evaluation of three theoretical models. *Journal of Attention Disorders*, 15(3), 193–203.

King, K. M., Lengua, L. J., & Monahan, K. C. (2013). Individual differences in the development of self-regulation during pre-adolescence: Connections to context and adjustment. *Journal of Abnormal Child Psychology*, 41(1), 57–69.

Kopala-Sibley, D. C., Cyr, M., Finsaas, M. C., Orawe, J., Huang, A., Tottenham, N., & Klein, D. N. (2020). Early Childhood Parenting Predicts Late Childhood Brain Functional Connectivity During Emotion Perception and Reward Processing. *Child Development*, 91(1), 110–128.
<https://doi.org/10.1111/cdev.13126>

Li, J. J. (2018). Children's Reward and Punishment Sensitivity Moderates the Association of Negative

- and Positive Parenting Behaviors in Child ADHD Symptoms. *Journal of Abnormal Child Psychology*, 46(8), 1585–1598.
- Li, J. J., & Lee, S. S. (2012). Association of positive and negative parenting behavior with childhood ADHD: Interactions with offspring monoamine oxidase A (MAO-A) genotype. *Journal of Abnormal Child Psychology*, 40(2), 165–175.
- Li, J. J., & Roberts, D. K. (2017). Genetic influences on executive functions across the life span. In *Executive Function* (pp. 106-123). Routledge.
- Lifford, K. J., Harold, G. T., & Thapar, A. (2008). Parent–child relationships and ADHD symptoms: a longitudinal analysis. *Journal of abnormal child psychology*, 36(2), 285-296.
- Luman, M., Oosterlaan, J., & Sergeant, J. A. (2005). The impact of reinforcement contingencies on AD/HD: a review and theoretical appraisal. *Clinical Psychology Review*, 25(2), 183–213.
- Luman, M., Van Meel, C. S., Oosterlaan, J., & Geurts, H. M. (2012). Reward and punishment sensitivity in children with ADHD: validating the sensitivity to punishment and sensitivity to reward questionnaire for children (SPSRQ-C). *Journal of Abnormal Child Psychology*, 40(1), 145–157.
- Ma, I., van Duijvenvoorde, A., & Scheres, A. (2016). The interaction between reinforcement and inhibitory control in ADHD: A review and research guidelines. *Clinical Psychology Review*, 44, 94–111.
- Martel, M., Nikolas, M., & Nigg, J. T. (2007). Executive function in adolescents with ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, 46(11), 1437–1444.
- Modesto-Lowe, V., Danforth, J. S., & Brooks, D. (2008). ADHD: Does parenting style matter? *Clinical Pediatrics*, 47(9), 865–872. <https://doi.org/10.1177/0009922808319963>
- Moriguchi, Y. (2014). The early development of executive function and its relation to social interaction: a brief review. *Frontiers in Psychology*, 5, 388.
- Nigg, J. T., Willcutt, E. G., Doyle, A. E., & Sonuga-Barke, E. J. S. (2005). Causal heterogeneity in attention-deficit/hyperactivity disorder: do we need neuropsychologically impaired subtypes? *Biological Psychiatry*, 57(11), 1224–1230.

- Owens, E. B., Hinshaw, S. P., Lee, S. S., & Lahey, B. B. (2009). Few girls with childhood attention-deficit/hyperactivity disorder show positive adjustment during adolescence. *Journal of Clinical Child & Adolescent Psychology, 38*(1), 132–143.
- Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology, 214*(1), 55–70.
- Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879–891.
- Rogers, M. A., Wiener, J., Marton, I., & Tannock, R. (2009). Supportive and controlling parental involvement as predictors of children's academic achievement: Relations to children's ADHD symptoms and parenting stress. *School Mental Health, 1*(2), 89–102.
- Russell, A. E., Ford, T., Williams, R., & Russell, G. (2016). The association between socioeconomic disadvantage and attention deficit/hyperactivity disorder (ADHD): a systematic review. *Child Psychiatry & Human Development, 47*(3), 440–458.
- Schmitz, M., Cadore, L., Paczko, M., Kipper, L., Chaves, M., Rohde, L. A., ... Knijnik, M. (2002). Neuropsychological performance in DSM-IV ADHD subtypes: An exploratory study with untreated adolescents. *The Canadian Journal of Psychiatry, 47*(9), 863–869.
- Shaffer, D., Fisher, P., Lucas, C. P., Dulcan, M. K., & Schwab-Stone, M. E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(1), 28–38.
- Shelton, K. K., Frick, P. J., & Wootton, J. (1996). Assessment of parenting practices in families of elementary school-age children. *Journal of Clinical Child Psychology, 25*(3), 317–329.
- Sonuga-Barke, E., Bitsakou, P., & Thompson, M. (2010). Beyond the Dual Pathway Model: Evidence for the Dissociation of Timing, Inhibitory, and Delay-Related Impairments in Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 49*(4), 345–355. <https://doi.org/10.1016/j.jaac.2009.12.018>

- Sonuga-Barke, E. J. S. (2002). Psychological heterogeneity in AD/HD—a dual pathway model of behaviour and cognition. *Behavioural Brain Research*, 130(1–2), 29–36.
- Sonuga-Barke, E. J. S., Sergeant, J. A., Nigg, J., & Willcutt, E. (2008). Executive Dysfunction and Delay Aversion in Attention Deficit Hyperactivity Disorder: Nosologic and Diagnostic Implications. *Child and Adolescent Psychiatric Clinics of North America*, 17(2), 367–384.
<https://doi.org/10.1016/j.chc.2007.11.008>
- Sonuga-Barke, E. J. S., & Halperin, J. M. (2010). Developmental phenotypes and causal pathways in attention deficit/hyperactivity disorder: potential targets for early intervention? *Journal of Child Psychology and Psychiatry*, 51(4), 368–389.
- Thorell, L. B. (2007). Do delay aversion and executive function deficits make distinct contributions to the functional impact of ADHD symptoms? A study of early academic skill deficits. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 48(11), 1061–1070.
<https://doi.org/10.1111/j.1469-7610.2007.01777.x>
- Weber, R. C. (2011). *How hot or cool is it to speak two languages: Executive function advantages in bilingual children* [Doctoral dissertation, Texas A&M University]. ProQuest dissertations and Theses Global.
- Weintraub, S., Bauer, P. J., Zelazo, P. D., Wallner-Allen, K., Dikmen, S. S., Heaton, R. K., ... Gershon, R. C. (2013). NIH toolbox cognition battery (CB): Introduction and pediatric data. *Monographs of the Society for Research in Child Development*, 78(4), 1–15. <https://doi.org/10.1111/mono.12031>
- Weintraub, S., Dikmen, S. S., Heaton, R. K., Tulsky, D. S., Zelazo, P. D., Slotkin, J., ... Fox, N. (2014). The cognition battery of the NIH toolbox for assessment of neurological and behavioral function: validation in an adult sample. *Journal of the International Neuropsychological Society*, 20(6), 567–578.
- Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. *Neurotherapeutics*, 9(3), 490–499.
- Willoughby, M. T., Kuhn, L. J., Blair, C. B., Samek, A., & List, J. A. (2017). The test–retest reliability of

the latent construct of executive function depends on whether tasks are represented as formative or reflective indicators. *Child Neuropsychology*, 23(7), 822–837.

Willoughby, M. T., Wylie, A. C., & Catellier, D. J. (2018). Testing the association between physical activity and executive function skills in early childhood. *Early Childhood Research Quarterly*, 44, 82–89.

Wolraich, M. L., Lambert, W., Doffing, M. A., Bickman, L., Simmons, T., & Worley, K. (2003). Psychometric properties of the Vanderbilt ADHD diagnostic parent rating scale in a referred population. *Journal of Pediatric Psychology*, 28(8), 559–568.

Table 1*Descriptive Statistics and Correlations for Study Variables*

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10	11	12
1.APQ positive parenting (W1)	131	66.19	5.98	-											
2.APQ negative parenting (W1)	131	28.92	3.75	-.17	-										
3.EF Touch EF (W1)	128	0.08	0.56	.13	-.26**	-									
4.NIH-TCB EF (W2)	118	108.74	13.92	.06	-.14	.45***	-								
5.SPSRQ RR (W1)	131	24.64	3.59	.12	.12	-.06	.11	-							
6.SPSRQ RR (W2)	128	24.20	3.23	.13	.00	-.13	-.08	.63***	-						
7.DISC ADHD-I symptoms (W2)	135	1.94	2.73	-.12	.20*	-.47***	-.27**	.23**	.37***	-					
8.DISC ADHD-H symptoms (W2)	135	1.97	2.58	-.02	.18*	-.41***	-.16	.30***	.43***	.62***	-				
9.DISC ADHD-C symptoms (W2)	135	3.85	4.77	-.07	.19*	-.47***	-.24**	.28**	.45***	.91***	.87***	-			
10.Vanderbilt ADHD-I symptoms (W2)	129	1.33	1.97	-.06	.08	-.46***	-.25**	.18*	.31***	.76***	.52***	.72***	-		
11.Vanderbilt ADHD-H symptoms (W2)	129	1.28	1.89	.05	.03	-.30***	-.11	.23**	.42***	.55***	.75***	.72***	.61***	-	
12.Vanderbilt ADHD-C symptoms (W2)	129	2.64	3.48	-.01	.07	-.42***	-.21*	.21*	.39***	.72***	.70***	.79***	.90***	.89***	-

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 2*Fit Indices for Model 1 and Model 2*

Model	χ^2	df	p	CFI	TLI	RMSEA (90% CI)	SRMR
Model 1: Predictor: Positive parenting							
Latent ADHD-I symptoms	22.366	17	.171	.983	.954	.052(.000-.104)	.048
Latent ADHD-H symptoms	29.286	17	.032	.957	.886	.078(.023-.124)	.051
Latent ADHD-C symptoms	23.176	17	.144	.981	.950	.055(.000-.106)	.048
Model 2: Predictor: Negative parenting							
Latent ADHD-I symptoms	26.947	17	.059	.969	.917	.070(.000-.118)	.052
Latent ADHD-H symptoms	36.769	17	.001	.925	.802	.106(.063-.149)	.057
Latent ADHD-C symptoms	30.537	17	.023	.960	.895	.082(.030-.128)	.053

Note. CFI = Comparative Fit Index (good fit>.95), TLI = Tucker-Lewis Index (good fit>.95), RMSEA = Root Mean Square Error Approximation (good fit<.05), SRMR = Standardized Root Mean Squared Residual (good fit<.08).

Table 3*Standardized Results and 95% CIs of the Total and Specific Indirect Effects for Model 1 and Model 2*

	Model 1		Model 2	
	Predictor: Positive parenting		Predictor: Negative parenting	
	Std.Est	95% CI	Std.Est	95% CI
Outcome: Latent ADHD-I				
Total indirect effects	-.027	[-.192, .138]	.124	[.001, .246]
W1 Parenting → W1 EF → W2 ADHD-I	-.071	[-.200, .059]	.132	[.036, .228]
W1 Parenting → W2 EF → W2 ADHD-I	.001	[-.024, .026]	.000	[-.003, .004]
W1 Parenting → W1 EF → W2 EF → W2 ADHD-I	-.001	[-.027, .024]	.000	[-.041, .041]
W1 Parenting → W1 RR → W2 ADHD-I	.007	[-.018, .032]	.003	[-.010, .016]
W1 Parenting → W2 RR → W2 ADHD-I	.023	[-.020, .066]	-.041	[-.087, .004]
W1 Parenting → W1 RR → W2 RR → W2 ADHD-I	.025	[-.011, .061]	.010	[-.021, .041]
W1 Parenting → W1 EF → W2 RR → W2 ADHD-I	-.011	[-.034, .011]	.020	[-.001, .041]
W1 Parenting → W1 RR → W2 EF → W2 ADHD-I	.000	[-.005, .005]	.000	[-.002, .002]
Outcome: Latent ADHD-H				
Total indirect effects	-.024	[-.205, .157]	.093	[-.041, .228]
W1 Parenting → W1 EF → W2 ADHD-H	-.082	[-.235, .070]	.133	[.036, .229]
W1 Parenting → W2 EF → W2 ADHD-H	-.012	[-.045, .022]	-.002	[-.023, .019]
W1 Parenting → W1 EF → W2 EF → W2 ADHD-H	.012	[-.023, .048]	-.021	[-.069, .028]
W1 Parenting → W1 RR → W2 ADHD-H	.002	[-.023, .028]	.000	[-.011, .010]
W1 Parenting → W2 RR → W2 ADHD-H	.033	[-.027, .094]	-.066	[-.131, .000]
W1 Parenting → W1 RR → W2 RR → W2 ADHD-H	.036	[-.014, .087]	.016	[-.034, .065]
W1 Parenting → W1 EF → W2 RR → W2 ADHD-H	-.016	[-.048, .015]	.032	[.003, .061]
W1 Parenting → W1 RR → W2 EF → W2 ADHD-H	.002	[-.004, .009]	.001	[-.003, .005]
Outcome: Latent ADHD-C				
Total indirect effects	-.025	[-.206, .157]	.111	[-.020, .241]
W1 Parenting → W1 EF → W2 ADHD-C	-.078	[-.221, .065]	.135	[.039, .231]

	Model 1		Model 2	
	Predictor: Positive parenting		Predictor: Negative parenting	
	Std.Est	95% CI	Std.Est	95% CI
W1 Parenting → W2 EF → W2 ADHD-C	-.004	[-.021, .013]	-.001	[-.009, .008]
W1 Parenting → W1 EF → W2 EF → W2 ADHD-C	.004	[-.013, .022]	-.008	[-.034, .018]
W1 Parenting → W1 RR → W2 ADHD-C	.002	[-.021, .025]	.000	[-.009, .010]
W1 Parenting → W2 RR → W2 ADHD-C	.032	[-.026, .089]	-.061	[-.121, -.001]
W1 Parenting → W1 RR → W2 RR → W2 ADHD-C	.034	[-.013, .082]	.015	[-.031, .060]
W1 Parenting → W1 EF → W2 RR → W2 ADHD-C	-.016	[-.045, .014]	.030	[.003, .057]
W1 Parenting → W1 RR → W2 EF → W2 ADHD-C	.001	[-.003, .004]	.000	[-.001, .002]

Note. Bolded font represents statistically significant indirect pathways.

Figure 1*Structural Equation Model of Positive Parenting Predicting Child ADHD Symptoms*

a) Outcome: ADHD-I Symptoms

b) Outcome: ADHD-H Symptoms

c) Outcome: ADHD-C Symptoms

Note. Statistics are standardized regression coefficients. Dotted lines represent nonsignificant relations; solid lines represent significant relations; bolded lines represent significant indirect paths. * $p < .05$, ** $p < .01$, *** $p < .001$.

Figure 2*Structural Equation Model of Negative Parenting Predicting Child ADHD Symptoms*

a) Outcome: ADHD-I Symptoms

b) Outcome: ADHD-H Symptoms

c) Outcome: ADHD-C Symptoms

Note. Statistics are standardized regression coefficients. Dotted lines represent nonsignificant relations; solid lines represent significant relations; bolded lines represent significant indirect paths. $*p < .05$, $**p < .01$, $***p < .001$.