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Associations between Birth Weight and Attention-Deficit/ Hyperactivity Disorder (ADHD) Symptom Severity: Indirect Effects via Primary Neuropsychological Functions

Burt Hatch[1], **Dione M. Healey**[1], and **Jeffrey M. Halperin**[2]

[1]Department of Psychology, University of Otago, Dunedin, New Zealand [2]Psychology Department, Queens College, NY, United States

Abstract

Background—ADHD has a range of aetiological origins which are associated with a number of disruptions in neuropsychological functioning. This study aims to examine how low birth weight, a proxy measure for a range of environmental complications during gestation, predicts ADHD symptom severity in preschool-aged children indirectly via neuropsychological functioning.

Methods—197 preschool-aged children were recruited as part of a larger longitudinal study. Two neuropsychological factors were derived from NEPSY domain scores. One, referred to as 'Primary Neuropsychological Function,' loaded highly with Sensorimotor and Visuospatial scores. The other, termed 'Higher-Order Function' loaded highly with Language and Memory domain scores. Executive functioning split evenly across the two. Analyses examined whether these neuropsychological factors allowed for an indirect association between birth weight and ADHD symptom severity.

Results—While both factors were associated with symptom severity, only the Primary Neuropsychological Factor was associated with birth weight. Furthermore, birth weight was indirectly associated to symptom severity via this factor.

Conclusions—These data indicate that birth weight is indirectly associated with ADHD severity via disruption of neuropsychological functions that are more primary in function as opposed to functions that play a higher-order role in utilising and integrating the primary functions.

Keywords

ADHD; neuropsychology; birth weight

Introduction

The symptoms characteristic of attention-deficit/hyperactivity disorder (ADHD) are often detected in the preschool years (Greenhill, Posner, Vaughan, & Kratochvil, 2008), are quite

Correspondence: Burt Hatch, Department of Psychology, University of Otago, PO Box 56, Dunedin 9054, New Zealand; hatbu157@student.otago.ac.nz.

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stable over time, and predictive of later functional difficulties and persistence of ADHD into the school-age years (Lahey et al., 2004; Riddle et al., 2013). It is associated with a variety of aetiological factors, both environmental and genetic, as well as a range of abnormalities in cortical and sub-cortical structures that involve the mesolimbic, mesocortical, and nigrostriatal pathways (Halperin & Schulz, 2006; Kieling, Goncalves, Tannock, & Castellanos, 2008; Sagvolden, Johansen, Aase, & Russell, 2005; Sonuga-Barke, 2005; Swanson et al., 2007). It is also associated with a range of neuropsychological difficulties. Investigations into the neuropsychological functioning of those with the disorder have focused largely upon executive functioning; where individuals with ADHD are often found to perform poorly on tasks such as behavioural inhibition, set shifting or working memory (Hall, Halperin, Schwartz, & Newcorn, 1997; Halperin et al., 1993; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Sergeant, Geurts, & Oosterlaan, 2002).

It is unlikely however, that ADHD can be accounted for solely by dysregulation in executive functioning (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). In a meta-analytic review, Willcutt et al. (2005) found that out of 83 studies, children with ADHD were only differentiated from the typically developing control groups on measures of executive functioning on 65% of comparisons, with effect sizes usually in the medium range (.46 to .69). Children with ADHD also tend to perform worse than their typically developing peers on tasks that involve other areas of neuropsychological functioning. For example, motor coordination (Carte, Nigg, & Hinshaw, 1996), perception (Mangeot et al., 2001), visuomotor integration (Raggio, 1999), and intra-individual variability of reaction time (Russell et al., 2006).

Thus, ADHD is associated with deficits in both executive and non-executive neuropsychological functioning. This is in keeping with the range of aetiological factors associated with the disorder. One factor found to be associated with the emergence of ADHD is low birth weight (Banerjee, Middleton, & Faraone, 2007). It is likely that low birth weight is associated with ADHD because it is a proxy for a variety of environmental occurrences, such as maternal smoking during gestation and poor maternal nutrition (Kramer, 1987), that have adverse effects on neural development (Weinstock, 2005). Several studies have reported that children with a low or extremely low birth weight are as much as 3.8 times more likely to meet diagnostic criteria for ADHD (Botting, Powls, Cooke, & Marlow, 1997; Breslau, Chilcoat, DelDotto, Andreski, & Brown, 1996; Mick, Biederman, Prince, Fischer, & Faraone, 2002; Szatmari, Saigal, Rosenbaum, & Campbell, 1993). The association between low birth weight and ADHD remains even after controlling for parental ADHD, parental antisocial behavior disorders, maternal substance abuse while pregnant, social class, and heritability (Hultman et al., 2007; Mick et al., 2002).

While some studies indicate an association between lower birth weight and ADHD, others indicate that children who are born with a low birth weight are also at greater risk of having a range of neuropsychological deficits, many of which overlap with those found in children with ADHD. In particular, low birth weight has been associated with difficulties on verbal fluency, working memory, and cognitive flexibility tasks, which involve aspects of executive functioning; as well as other tasks that measure expressive or receptive language, visuo-spatial reasoning, and motor control (Aarnoudse-Moens, Weisglas-Kuperus, van

Goudoever, & Oosterlaan, 2009; Breslau et al., 1996; Breslau, Chilcoat, Johnson, Andreski, & Lucia, 2000). The association between lower birth weight and poorer neuropsychological functioning was found to be present even for children born within the normal birth weight range (Shenkin, Starr, & Deary, 2004).

Given that low birth weight is a risk factor for ADHD, and that both children with ADHD and those born with low birth weight perform worse than controls on a range of neuropsychological tasks, it is plausible that deficits in particular neuropsychological domains at least partially mediate the relationship between birth weight and ADHD symptoms. Following this reasoning, one study, using a sample of 823 six year-old children, found that perceptual sensitivity, motor speed, and motor coordination, partially mediated the association between birth weight and ADHD symptoms (Martel, Lucia, Nigg, & Breslau, 2007). It is of note that this study only included a limited range of neuropsychological measures although, as previously described, children with ADHD or those born with low birth weight perform worse than their peers on a variety of other cognitive domains such as language, working memory, and visual-spatial processing.

The variety of neuropsychological dysfunction found amongst those with ADHD has been explained within the framework of a neuropsychological/developmental perspective (Halperin, & Schulz, 2006). This view takes into account that, generally, the development of brain regions that underlie primary sensorimotor and visual functions precedes the development of regions that are involved in higher-order cognitive functions (e.g., language, memory). These higher-order functions rely on, as well as integrate, the primary functions (Gogtay et al., 2004). From a developmental view of ADHD, symptoms arise from etiological factors that occur early in development (e.g., genetic expression, gestation, or birth), and thus disrupt neuropsychological functions that develop earlier in life; while the persistence or remittance of symptoms is dependent upon the development of regions involved in higher-order neuropsychological functions that could potentially compensate for lower order deficits.

The aim of this study was to examine the associations between the hyperactive/impulsive and inattentive symptoms of ADHD, neuropsychological functioning, and birth weight. In this study birth weight is used as a proxy for early environmental insults (e.g., maternal health, smoking). In accordance with a developmental perspective of ADHD it was hypothesized that lower birth weight would be indirectly associated with hyperactivity/impulsivity and inattention via neuropsychological functions that develop earlier, such as visual-spatial and motor coordination, but would not be associated with higher-order neuropsychological functioning, including language and memory, that typically develops later. It was also hypothesized that, while not associated with birth weight, higher-order neuropsychological functioning would be associated with hyperactive/impulsive and inattentive symptom severity.

Methods

Participants

The sample was recruited from the New York City metropolitan area as part of a larger longitudinal study. Of this sample all relevant information was available for 197 3–4 year-old children (mean age 4.32 yeas, SD=.439) of which 146 (74.1%) were boys. The mother's Socio-economic status (SES) was measured using the Nakao–Treas Socioeconomic Prestige Index (Nakao & Treas, 1994) and had a mean of 54.90 (SD=16.77), indicative of a middle-class sample, albeit with some variability. The study excluded children with IQ's below 80 (measured by the Wechsler Preschool and Primary Scale of Intelligence, 3rd edition; WPPSI-III), a pervasive developmental disorder, posttraumatic stress disorder, a diagnosed neurological disorder (e.g., epilepsy/seizures, hydrocephalus and cerebral palsy), or taking systematic medication for chronic medical conditions (including ADHD). Medication use was exclusionary to allow for a more accurate assessment of the children's baseline behavior. Given that the children were 3–4 years-old at the time of recruitment, very few were excluded because of medication treatment for ADHD ($n < 5$). Children participated based upon parent and teacher ratings on the Attention Deficit Hyperactivity Disorder-Rating Scale-Fourth Edition (ADHD-RS: DuPaul, Power, Anastopoulos, & Reid, 1998). The criteria categorised children as "at-risk" for ADHD if at least six symptoms of hyperactivity-impulsivity and/or inattention were rated as "Often" or "Very Often" across parent and/or teacher responses. Children were categorised as "typically developing" if fewer than 3 symptoms were rated as "Often" or "Very Often" by either parents or teachers. By design these criteria resulted in a sample with a wide range of symptom severity, and although a portion of the at-risk children did not meet diagnostic criteria for ADHD, many of these children were rated as presenting with several symptoms of ADHD in at least one setting. Of the children included in this study, 108 met criteria for a diagnosis of ADHD (10 Inattentive subtype, 45 Hyperactive/Impulsive subtype, 47 Combined subtype, and 6 ADHD-NOS). Of the remaining 89 children, 71 were initially recruited as "typically developing" and 18 recruited as "at-risk" for ADHD. It should be noted that these diagnoses are descriptive while measures of symptom severity were used for analytical purposes. With regard to race/ethnicity 40.1% of the sample was White, Non-Hispanic; 19.3% White Hispanic; 10.7% Black, Non-Hispanic; 1.5% Black, Hispanic; 10.2% Asian Non-Hispanic; and 18.3% reported mixed or "other" ethnicity/race. This distribution is representative of the community from which the sample was recruited. The majority (87.3%) of children had birth weights above 2500g, 9.7% were within the low birth weight range (1500g–2500), 1.5% were within the very low birth weight range (1000g–1499g), and 1.5% were within extremely low birth weight range (below 1000g).

Measures

The measures are described below and descriptive statistics for each of the measures are provided in Table 1.

Neuropsychological functioning—The NEPSY assesses neuropsychological functioning within five domains: Attention/Executive (e.g., ability to inhibit impulsive responses and visual attention), Language, Memory, Sensorimotor (e.g., fine motor

coordination), and Visuospatial (e.g., being able to construct two- and three-dimensional designs). While the NEPSY is reliable over time (r= .68–.90), recent revisions and data have lead to concerns about the construct validity of some of the domains (Korkman, Kirk, & Kemp, 1998). Thus, we conducted a Principal Components Analysis with Oblimin Rotation and Kaiser Normalization to assess the relations among the five domains in our sample of 3–4 year-old children. This yielded a two-factor solution: Language and Memory loaded onto one factor and Sensorimotor and Visuospatial loaded onto the other; the Attention/Executive domain loaded evenly onto both factors (see Table 2). The regression factor scores for each participant, calculated from all five domains, were saved into variables. We named the first factor "Primary Neuropsychological Function" because it has the greatest loadings of visuo-spatial perception and motor coordination. We named the second factor "Higher-Order Neuropsychological Function" because it has the greatest loadings of memory and language functioning.

Hyperactive/impulsive and inattentive symptom severity—Composite scores were calculated from parent and teacher responses on the ADHD-RS (DuPaul et al., 1998); in accordance with other studies from this cohort (Healey, Gopin, Grossman, Campbell, Halperin, 2010). This involved summing the highest of *either* the teacher *or* parent ratings for symptoms of inattention (termed here as the Inattentive Composite) or impulsivity and hyperactivity (termed here as the Hyperactive/Impulsive Composite). This allows for the maximum impact of symptoms that only occur in a single setting (e.g., severe attention difficulties that are reported by the teacher but not by the parent).

Birth weight—The child's birth weight was obtained from a form completed by parents and reviewed as part of a semi-structured interview.

Procedure

The procedure for data collection for this study has been described elsewhere (Healey, Gopin, Grossman, Campbell, & Halperin, 2010) and therefore will only be briefly described here. Data were collected in two phases. First, ratings on the ADHD-RS (among other scales) were collected from parents and teachers. Second, information was collected during an assessment session that lasted approximately four hours across two days. During this session, while parents completed a Kiddie-SADs semi-structured interview (Kaufman, Birmaher, Brent, Rao, & Ryan, 1996), children completed a number of laboratory tasks as well as the WPPSI-III (Wechsler, 2002) and the NEPSY (Korkman et al., 1998). A trained graduate student administered the tests to each child in the same order and in the same testing room.

Statistical Analysis

Preliminary analysis of the data was first conducted to examine the associations between variables and examine possible covariates. Based upon the preliminary analysis, path analysis was conducted.

Preliminary data analysis—Inter-correlations were examined among birth weight, neuropsychological functioning, and ADHD symptom severity to investigate whether there

were necessary significant associations between these variables to warrant conducting path analyses. Pearson correlations were run between age, maternal SES, birth weight, neuropsychological measures, and ADHD symptom severity to see whether age and maternal SES should be entered as covariates in further analyses. Age and birth weight were negatively skewed. Thus, before being being entered in the Pearson correlations, these variables were square-root transformed so that this skewness was no longer significant (Tabachnick & Fidell, 2006). To determine whether gender should be a covariate, independent sample t-tests were conducted.

Analysis of indirect effects—The hypotheses were tested using path analyses procedures, which test whether the association between independent and dependent variables is accounted for by other variables (often also referred to as mediation analysis). A requirement for traditional mediation analyses is significant correlations between the independent and dependent variables (Baron & Kenny, 1986). As indicated by our preliminary analysis, no significant correlations were found between birth weight and symptom severity measures. More recently it has been proposed that such a correlation is not necessary (Hayes, 2009; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002) as there may be multiple paths between the independent and dependent variables that cancel each other out. Tests of indirect effects that have an optimal balance between maximum power and reducing Type-I error either make use of the distribution of the product of the variables or bootstrapping (MacKinnon et al., 2002). Given that developmental trajectories of mental health typically involve a multitude of factors over time (Hackman, Farah, & Meaney, 2010) the use of such tests to examine the indirect relationships between early etiological factors and later functioning is particularly relevant. A method of analysis for indirect effect was conducted that examines confidence intervals around the indirect effect, with regression-based path analysis and bootstrapping (bootstrapping sample of 5000). This produces path coefficients as well as p-values for total and direct effects, and bias-corrected percentile confidence intervals (in which statistical significance is assumed if zero is not included in the interval) for the indirect effect (Hayes, 2012). This analysis procedure also has the advantage of being robust to violations in variable normality, thus non-transformed variables were entered. Before being entered in an analysis of indirect effects all variables (except gender) were converted to standardized z-scores.

Results

Preliminary Analyses

Correlations—Birth weight was significantly and positively correlated with Primary Neuropsychological Functions but not with Higher-Order Neuropsychological Functions or with Hyperactivity/Impulsivity and Inattention. Both Primary and Higher-Order Neuropsychological Functions were significantly negatively correlated with Hyperactivity/Impulsivity and Inattention. Age was significantly negatively correlated with Hyperactivity/Impulsivity. Maternal SES was significantly positively correlated with Higher-Order Neuropsychological Function and significantly negatively correlated with both hyperactivity/impulsivity and inattention (see Table 3).

T-tests—There was a significant difference between boys' (M=3347.77 gms, SD=648.94) and girls' (M=3115.11 gms, SD=837.66) birth weight; t(195)=2.04, p<.05. There were no other significant differences between the genders on the other study variables.

Analysis of indirect effects

Analyses of indirect effects were conducted for two models. Because Higher-Order Neuropsychological Functions were not significantly associated with birth weight they were not entered in the models. Model 1 tested whether birth weight and Inattention were indirectly associated through Primary Neuropsychological Functions. Because Inattention was significantly correlated with maternal SES and because boys had significantly higher birth weights, both maternal SES and gender were entered as covariates. Model 2 tested whether birth weight and Hyperactivity/Impulsivity were indirectly associated via Primary Neuropsychological Functions. Because Hyperactivity/Impulsivity was significantly correlated with maternal SES and age, both were entered as covariates; as was gender due to the birth weight differences. Path coefficients, standard errors, and 95% bias corrected bootstrap confidence intervals for the models are presented in Table 4.

For Model 1, there was a significant association between birth weight and Primary Neuropsychological Functions (path coefficient=.215, p=.001) and between Primary Neuropsychological Functions and Inattention (path coefficient= −.438, p<.001). The indirect effect of birth weight on Inattention via Primary Neuropsychological Functions was statistically significant (path coefficient= −.094, 95% CI [−.156, −.039]). This indicates that for each standardized unit increase in birth weight there is a .094 standardized unit decrease in Inattention as a result of the effect of birth weight on Primary Neuropsychological Functions.

For Model 2, there was a statistically significant association between birth weight and Primary Neuropsychological Functions (path coefficient=.213 p=.002), and between Primary Neuropsychological Functions and Hyperactivity/Impulsivity (path coefficient= −.348, p<.001). The direct effect (path coefficient= .136, p=.021) was significant. This indicates that for each standardized unit increase in birth weight there is a significant increase in Hyperactivity/Impulsivity by 0.136 standardized units; that is independent of the effects of Primary Neuropsychological Functions on Hyperactivity/Impulsivity. The indirect effect via Primary Neuropsychological Functions (path coefficient= −.074, 95% CI [−.126, −.032]) was also statistically significant. This indicates that for each standardized unit increase in birth weight there is a .074 standardized unit decrease in Hyperactivity/Impulsivity, as a result of the effect of birth weight on Primary Neuropsychological Functions.

Additional analysis

Because the Attention/Executive function domain split variance across two factors, we tested the models using factors derived from the NEPSY domains but excluding the Attention/Executive Functioning domain. Two factors emerged; one with higher loadings from the Sensorimotor and Visuospatial domains, and one with higher loading from the Language and Memory domains. Like the initial analyses, the Memory/Language domain

was not associated with birth weight (r = .118, *p* =.099) while the Sensorimotor/Visuospatial domain was (r = .248, *p* <.001). Similar to initial analyses birth weight was indirectly associated via the Sensorimotor/Visuospatial domain with Inattention (indirect path coefficient = −.080, 95%CI [−.141, −.030]; direct path coefficient = −.037, p =.578) and Hyperactivity/Impulsivity (indirect path coefficient = −.060, 95%CI [−.112, −.022]; direct path coefficient = .122, p = .070).

While birth weight was used as a proxy for a range of events that could have affected neural development in gestation, it is possible that such factors could affect development without impacting upon birth weight. Similarly, perinatal factors could affect neural functioning without effecting birth weight. Therefore, post-hoc analyses of the indirect effects were conducted entering variables pertaining to prenatal/perinatal factors as covariates. In particular, maternal drug use (recreational and medication), tobacco use, alcohol use, maternal illness, and c-section (planned or emergency) were entered as dichotomous covariates. For both model 1 and 2 entering these variables did not result in any statistically significant results becoming non-significant or vice versa.

Discussion

Our primary prediction was that birth weight would be indirectly associated with hyperactive/impulsive and inattentive symptom severity via Primary Neuropsychological Functions; while Higher-Order Neuropsychological Functions would not be associated with birth weight, but would still be associated with symptom severity. Analyses of indirect effects supported this, indicating that birth weight was indirectly associated with symptomatology through Primary Neuropsychological Functions. There was a significant direct effect in Model 2 that was not hypothesised. In particular, there was a significant positive association between birth weight and Hyperactivity/Impulsivity when Primary Neuropsychological Functioning was held constant. According to contemporary interpretations of analyses of indirect effects such a finding does not negate the indirect effect, but may be illustrative of multiple indirect associations between birth weight and hyperactivity/impulsivity (MacKinnon et al., 2000).

The Primary Neuropsychological Functions factor had high loadings from the NEPSY Sensorimotor and Visuospatial domains and the Higher-Order Neuropsychological Functions factor had high loadings from the NEPSY Language and Memory domains. It is notable that the Attention/Executive domain split across the two factors. This may be due to a number of factors. First, it is unclear that the two subtests that comprise this domain truly assess executive functioning in this young age range. Second, scores on these subtests are not significantly correlated (Korkman, Kirk, Kemp, 2007) one primarily assesses visual searching/scanning while the other assesses the ability to stand stationary with closed eyes. Thus, it is not surprising that they do not load onto on a single factor.

The results of this study are consistent with research indicating that children with ADHD tend to have trouble on a wide range of neuropsychological functions, including basic primary neuropsychological functions such as visuosmotor integration (Raggio, 1999), perception (Mangeot et al., 2001), and motor control (Carte et al., 1996); as well as higher

order functions such as language (Bruce, Thernlund, & Nettelbladt, 2006) and memory (Martinussen et al., 2005). Beyond investigating the neuropsychological correlates of hyperactivity/impulsivity and inattention, this study builds upon previous findings by investigating how neuropsychological functioning relates to birth weight, one of several aetiological factors of ADHD. The findings are similar to those of Martel et al. (2007) who, in a sample of 6 year-olds, found that an association between birth weight and ADHD symptoms was mediated by motor speed, motor coordination, and perceptual sensitivity (the ability to distinguish between targets and non-targets on a continuous performance task). By assessing a wider range of neuropsychological functioning in younger children, this study was able to extend these findings. While we found that birth weight was associated with hyperactive/impulsive and inattentive symptomatolgy via primary neuropsychological functions, birth weight was not associated with higher-order neuropsychological functions, even though these functions were still associated with ADHD symptoms. This is consistent with the view that ADHD arises out of disruptions to the neurobiological systems primarily involved in more basic neuropsychological functions rather than higher order/executive functions (Halperin & Schulz, 2006).

While this study examines some particular examples of indirect effects, these associations are likely to occur within a wider context of other factors and associations. For instance, with regard to aetiology, heritability/genetic mechanisms play a large role in the presentation of ADHD symptoms (Faraone et al., 2005), something not accounted for in the models tested here. It is possible that particular genetic expressions, with or without interactions with environmental factors, lead to the development of ADHD symptomology (Todd & Neuman, 2007). Further, beside hyperactivity/impulsivity and inattention, there may be other outcome variables associated with birth weight or primary neuropsychological functions. These may be detectable in the younger ages (e.g., symptoms of autism spectrum disorder) or in later years (e.g., learning difficulties).

A number of study limitations need be acknowledged. First, the data regarding birth weight was obtained retrospectively. While this is not as accurate as the collection of birth weight data from health records or at the time of birth, the recollection of birth weight by maternal reports has been found to be a reliable and valid method of data collection (Olson, Shu, Ross, Pendergrass, & Robison, 1997). Second, although this study was able to consider some particular environmental factors and excluded children with diagnosed neurological problems, there are other prenatal, perinatal, or postnatal environmental factors that could be associated with birth weight, neuropsychological functioning, and/or ADHD symptom severity, that are not accounted for in the model (e.g., perinatal hypoxia, postnatal icterus, catch-up growth, or nutritional interventions). Finally, because neuropsychological functioning and ADHD symptom severity were assessed at the same time point, causal inferences between the two should be interpreted with a level of caution.

Despite these limitations this study has a number of strengths. First, the sample was ethnically diverse and children displayed a wide range of ADHD symptoms, allowing for better generalization of results. Second, by using a bootstrapping approach, path analysis could be conducted in the absence of a direct association between dependent and independent variables. This decreases Type-II error while limiting inflation of Type-I error.

Finally, by using continuous measures of birth weight and ADHD symptom severity the study was able to quantify the indirect association birth weight had with symptom severity through neuropsychological functioning.

In conclusion, this study demonstrates that, while a range of disruptions to neuropsychological functioning are associated with greater symptomatology in 3–4 year-old children, it is only the disruption of primary functions, rather than higher-order functions, that is associated with birth weight, a proxy for a range of insults to neural development. These findings could be extended in several ways in the future. First, replication is required. Second, the specificity of the aetiological variables should be examined. In particular, birth weight is likely to be a proxy for many different neuronal insults during gestation. Hence, examination of which insults lead to lower birth weight and also cause the most dysregulation to brain development would have obvious benefits with regard to prevention of symptom development (i.e., what events during gestation are most likely to result in ADHD). Third, the models could be extended by the inclusion of other factors associated with ADHD, such as genetics, heritability, and measures of brain physiology.

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Key points

1. Investigated neuropsychological mediators of the associations between birth weight and ADHD symptom severity in 3–4 year-old children.
2. Consistent with previous research, findings indicated ADHD symptom severity was associated with poorer overall neuropsychological functioning.
3. Lower birth weight was only associated with lower scores on Primary Neuropsychological Functions but not Higher-Order Functions.
4. Lower birth weight was indirectly associated with greater ADHD symptom severity via Primary Neuopsycholgical Functions.
5. Results are consistent with neurodevelopmental theories that suggest etiological factors of ADHD disrupt Primary Neuopsychological Functions while the persistence or remittance of symptoms is associated with Higher-Order Functions.

Table 1

Descriptive statistics for study variables and demographic variables

	Mean	SD	Range
Birth weight	3287.53	707.80	510.29–4592.62
Higher-Order Neuropsych. Functions	0	1	−3.41–2.37
Primary Neuropsych. Functions	0	1	−.259–2.86
Hyperactive/Impulsive Composite	15.37	8.26	0–27
Inattentive Composite	13.60	7.73	0–27

Table 2

NEPSY Principal components derived from factor analysis

NEPSY Domains	Domain Score: Mean (SD)	Factor 1: Primary Neuropsych. Functions	Factor 2: Higher-Order Neuropsych. Functions
Attention/Executive	99.22 (13.38)	0.441	0.449
Language	101.07 (11.80)	0.018	0.874
Sensorimotor	92.59 (15.05)	0.890	−0.033
Visuospatial	105.85 (13.00)	0.871	−0.045
Memory	93.02 (15.13)	−0.099	0.917

Table 3

Correlations between study variables and demographic variables

	Birth weight[#]	Higher-Order Neuropsych Function	Primary Neuropsych Functions	Hyperactive/Impulsive Composite	Inattentive Composite
Higher-Order	.127	—			
Primary	.155*	.30**	—		
Hyperactive/Impulsive	.046	−.23**	−.33**	—	
Inattentive	−.104	−.39**	−.46**	.82**	—
Age[#]	−.008	.096	−.096	−.146*	.115
Maternal SES	.045	.190**	.026	−.271**	−.173*

*$p < 0.05$ level;

**$p < 0.01$;

[#]Entered in correlation as transformed variable

Table 4

Path Coefficients and 95% Percentile Confidence Intervals for Model One and Model Two

	Path Coefficients	s.e.	p value	95% Bootstrap CI Confidence Interval
Model 1				
Path A: birth weight to Primary Neuropsych. Functions	.215	.066	.001	-
Path B: Primary Neuropsych Functions to Inattentive Composite	−.438	0.64	.000	-
Total effect	−.117	.065	.072	-
Direct effect	−.023	.058	.694	-
Indirect effect	−.094	.030	-	−.156, −.039
Model 2				
Path A: birth weight to Primary Neuropsych. Functions	.213	.066	.002	-
Path B: Primary Neuropsych. Functions to Hyper/Impulsive Composite	−.348	.065	.000	-
Total effect	.062	.062	.318	-
Direct effect	.136	.058	.021	-
Indirect effect	−.074	.025	-	−.127, −.032