NEW RESEARCH

Longitudinal Associations Between ADHD and Weight From Birth to Adolescence

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Objective: Attention-deficit/hyperactivity disorder (ADHD) is associated with lower birth weight, but also with obesity in childhood. Findings on the direction of this association are mixed. This study investigated the relationship between weight and ADHD from birth across development.

Method: We used data from the Millennium Cohort Study (MCS), collected at 7 time points between age 9 months and 17 years. ADHD diagnosis status and scores on the Strength and Difficulties Questionnaire (SDQ) were used to create an ADHD group and a control group. Random intercept cross-lagged panel models were conducted in female individuals (n = 4,051) and male individuals (n = 3,857) to examine bidirectional associations between body mass index (BMI) *z* scores and SDQ scores between ages 3 and 17 years. Analyses were adjusted for common risk factors for ADHD and obesity, such as sex assigned at birth, multiple births, and ADHD medication status.

Results: Children in the ADHD group were significantly lighter in weight at birth than the control group (t[5674] = 2.65, 95% CI = 0.02, 0.14, p = .008) and were significantly more likely to have obesity at age 5 years onward (odds ratio range = 1.57-2.46, relative risk range 0.98-2.29). Path analyses conducted separately for male and female individuals showed that higher ADHD symptoms in female individuals at ages 7, 11, and 14 years significantly predicted higher BMI *z* scores at ages 11, 14, and 17 years, respectively. In male individuals, this association was seen only between ages 11 and 14 years (β = 0.07; 95% CI = 0.04-0.10, $p < .001$).

Conclusion: Results suggest that interventions for children with ADHD, and their parents, should begin as soon as possible, ideally prenatally. Developmental sex differences should be considered.

Key words: ADHD; obesity; environmental factors; ADHD medication

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Attention-deficit/hyperactivity disorder (ADHD), characterized by persistent and impairing inattention and/or hyperactivity and impulsivity,[1] is the most common neurodevelopmental condition. Global prevalence estimates range between 5% and 11%[2-4] in childhood and adolescence. Diagnostic rates are more than twice as high in male individuals than in female individuals,[5] with possible reasons for the disparity including diagnostic bias, differing symptom presentation, or biological differences.[6]

The relationship between ADHD and body weight, despite being largely investigated, is still unclear.[7] Children with increased ADHD symptoms are typically lighter in weight at birth than their peers,[8] but are later more likely to have obesity.[9] Research into the "when and why" regarding this turning point is scarce. It is unclear when children with ADHD experience the shift from underweight to overweight during their development, and at what age preventive measures might be usefully implemented. As both ADHD and high body mass index (BMI) are associated with numerous negative health outcomes, including asthma, diabetes, and cardiovascular disorders,[10,11] the relationship between the 2 conditions has important clinical implications.

The longitudinal direction of the association between ADHD and overweight and the mechanisms involved is also unclear. Some studies have suggested that high BMI leads to an increase in ADHD risk,[12] whereas others have found that childhood ADHD symptoms predict obesity in adolescence.[9] One longitudinal cohort study[13] assessing the bidirectional relationship between body composition and ADHD in children aged 18 months to 9 years found that higher ADHD symptoms predicted higher weight from age 3 years onward; however, this was not investigated for boys and girls separately.

When investigating the relationship between weight and ADHD, it is important to take into account not only cohort members' sex but also common risk factors, including factors prior to conception, such as mothers' BMI,[14] and family demographics, such as socioeconomic disadvantage.[15] ADHD medication may also confound the relationship, as stimulants are known to have an effect on weight, particularly at the start of treatment.[16]

Importantly, although substantial previous literature describes the relationship between ADHD and numerous weight measures at different times during development, much of this research is cross-sectional or assesses only a short time period in childhood.

Shedding light on the longitudinal relationships between ADHD and body weight during the developmental period is crucial to inform preventive programs. In the present study, we used data from a large, population-representative, UK sample with data from birth to late adolescence (age 17 years), to explore the relationship between weight and ADHD in male and female individuals, across childhood and adolescence, while adjusting for relevant environmental risk factors. The main aims were to investigate the following: (1) at what age underweight shifts toward overweight in the ADHD group compared to the control group; (2) the bidirectional associations between ADHD symptoms and BMI across childhood; and (3) whether this association is different in male and female individuals.

METHOD

Sample

We used data from the Millennium Cohort Study (MCS), a longitudinal cohort study involving more than 19,000 families with children born between 2000 and 2002. Data have been collected at 7 time points to date, when cohort members were 9 months, and 3, 5, 7, 11, 14, and 17 years of age. Cohort members taking ADHD medications (n = 141) were excluded from the comparison analyses and adjusted for in the longitudinal analysis, as stimulants may lead to decreased weight.[16]

As is common in longitudinal cohort studies, participation rates varied across survey waves, with families joining and withdrawing at different time points. The random-intercept cross-lagged panel model (RI-CLPM) sample (combined N = 7,908) included only cohort members who had participated at every wave and excluded those missing all environmental risk data. Survey weights, stratification, and cluster variables were also included in the analysis because of the MCS complex sampling methods.[17] In addition to the informed consent procedures of the Millennium Cohort Study,[18] this research was approved by the University of Southampton Ethics Committee.

Measures

ADHD Diagnosis and Symptoms. As in previous studies,[19] cohort members were identified as having ADHD based on diagnosis and/or scores above the clinical threshold on the Strengths and Difficulties Questionnaire (SDQ)[20] hyperactivity/inattention subscale. At waves 3 to 6 (age 5-14 years), parents were asked if their child had been diagnosed with ADHD. The SDQ was completed by parents at waves 2 to 7 (age 3-17 years). The hyperactivity/inattention subscale requires parents to indicate whether the following 5 statements are "not true," "somewhat true," or "certainly true" about their child: "restless, overactive, cannot stay still for long," "constantly fidgeting or squirming," "easily distracted, concentration wanders," "thinks things out before acting," and "sees tasks through to the end, good attention span." Each of the 5 questions receives a score of 0, 1, or 2 (positive statements are reverse coded), resulting in a total score out of 10.

The ADHD group (n = 442) comprised cohort members with a reported ADHD diagnosis at any wave and/or SDQ scores ≥8 in at least 5 of 6 waves. This cut-off corresponds to the 4-band categorization of the SDQ, with a score of 8 considered "high" and scores of 9 or 10 considered "very high."[21] The control group (n = 5,398) comprised cohort members with no ADHD diagnosis and SDQ scores <8 at every wave.

Weight. Parents reported their child's birth weight in the first data sweep, and cohort members' weight was directly measured during each wave of data collection. As in previous research,[22] outliers 5 SDs above or below the mean were excluded from the data (3.1%). A binary variable was created to indicate whether cohort members had obesity or not, using the International Obesity Task Force (IOTF) cut-off points.[23]

Covariates. We created 4 cumulative risk indices (CRIs), each including 3 environmental factors identified in previous research as having an association with ADHD and/or obesity (Table 1[24-37]). Data were collected during interviews with parents at the first data sweep (age 9 months). For each of the 12 individual factors, cohort members were classified as "high risk" or "low risk." Cut-offs used in previous literature (Table 1) were used to create binary variables from continuous environmental factors. Individual risk factors were grouped and totalled to account for the accumulation of environmental risk at similar developmental stages. The 4 CRIs were as follows (high risk criteria in parentheses): (1) prenatal risk: mother's pre-pregnancy BMI (>24.9), antenatal blood pressure (preeclampsia/other related diagnosis), smoking status in pregnancy (≥1 cigarette); (2) birth and neonatal risk: birthweight (<2.5 kg), gestation (<37 weeks), breastfeeding (none attempted); (3) socioeconomic status: household income (below 60% median poverty indicator), household crowding (fewer rooms than people, excluding bathrooms),

TABLE 1 Weighted Frequencies of Individual Environmental Risk Factors and Physical Health Conditions in the Male and Female Individual Random Intercept Cross-Lagged Panel Model Samples Combined, With Reference to the Associated Literature (N = 7,908)

Environmental risk factors	High risk criteria	n (%)
Prenatal factors		
Mother's pre-pregnancy BMI[14]	>24.9	2,180 (27.6)
Antenatal blood pressure[24]	Pre-eclampsia/hypertension diagnosis	605 (7.7)
Smoking in pregnancy[25,26]	Mother smoked 1 or more cigarettes during pregnancy	1,342 (17)
Birth and neonatal factors		
Birthweight[27]	<2.5 kg	556 (7)
Gestation[27,28]	<37 wk (premature)	580 (7.3)
Breastfeeding[29]	No breastfeeding	1,925 (24.3)
Socioeconomic status		
Household income[30]	Below 60% median poverty indicator	2,043 (25.8)
Household crowding[31]	Fewer rooms than people (excluding bathrooms)	703 (8.9)
Housing tenure[32]	Social housing or renting from local authority	1,398 (17.7)
Home environment		
Mother's education attainment[33,34]	<NVQ level 3 (2 A-levels)	3,447 (43.6)
Number of parents in household[30]	One parent living in household	796 (10.1)
Mother's mental health[35-37]	Depression/anxiety diagnosis/<4 on Rutter Malaise Inventory	2,238 (28.3)

Note: BMI = body mass index; NVQ = National Vocational Qualification.

housing tenure (social housing/renting from local authority); (4) home environment: mother's educational attainment (<NVQ level 3), number of parents in household (single-parent family), mother's mental health (depression/anxiety diagnosis or high maternal distress; high maternal distress was categorized as scoring ≥4 on a 9-item short form version[38] of the Rutter Malaise Inventory[39]).

In the RI-CLPM, the 4 CRIs, along with the following, were included as time-invariant predictors because of their association with either ADHD or weight; ethnicity (coded as "any ethnicity other than White"/White), whether children were part of a multiple birth, and ADHD medication status. At the sixth data sweep only (age 14 years), parents were given a list including both stimulant and non-stimulant medications for ADHD and asked whether their child was taking any of the listed prescribed medications for ADHD. Responses were used to create a binary variable indicating ADHD medication status (yes/no).

Statistical Analysis

We used R packages "anthro" and "anthroplus"[40] to calculate BMI and associated z-scores, adjusted for age and sex assigned at birth, based on the World Health Organisation Child Growth Standards.[41]

Two independent-samples *t* tests were conducted to compare mean birth weight as well as mean weight at 9 months in the ADHD group and control group. χ^2 Tests were conducted to compare the number of cohort members with obesity in the ADHD group and control group. Assumptions were met and analyses were performed in IBM SPSS Statistics for Windows (Version 28.0).

Extended versions of a RI-CLPM with time-invariant predictors[42] were conducted for male and female individuals in MPlus version 8.10.[43] The models were used to explore bidirectional associations between SDQ scores and BMI *z* scores at 6 time points (ages 3, 5, 7, 11, 14, and 17 years). The RI-CLPM builds on a traditional cross-lagged panel model by accounting for both within-person and between-persons variance over time. The random intercepts also account for stable, trait-like differences between individual cohort members. The model determines both autoregressive paths (associations between different time points of a variable; eg, age 3 SDQ score to age 5 SDQ score) and cross-lagged paths (associations between a variable at 1 time point and another variable at the subsequent time point; eg, age 3 SDQ score to age 5 BMI *z* score). The 4 CRIs plus ethnicity, multiple births, and ADHD medication (in male individuals only) were specified as time-invariant predictors, constrained across waves. To account for missing data and non-normality of data, we used a maximum likelihood estimator with robust standard errors (MLR). Goodness of fit of the model was determined by the root mean square error of approximation (RMSEA) and the comparative fit index (CFI). Values

TABLE 2 Unweighted Statistics for Variables in the Random-Intercept Cross-Lagged Panel Models

	Female individuals (n = 4,051)		Male individuals (n = 3,857)	
	Mean (SD)			
SDQ score				
Age 3	3.43	(2.21)	4.00	(2.36)
Age 5	2.77	(2.18)	3.46	(2.41)
Age 7	2.75	(2.31)	3.62	(2.53)
Age 11	2.50	(2.19)	3.40	(2.52)
Age 14	2.37	(2.15)	3.26	(2.49)
Age 17	2.05	(2.04)	2.73	(2.35)
BMI *z* score				
Age 3	0.79	(1.01)	0.92	(1.05)
Age 5	0.51	(0.97)	0.63	(1.03)
Age 7	0.45	(1.08)	0.44	(1.16)
Age 11	0.51	(1.19)	0.55	(1.22)
Age 14	0.47	(1.11)	0.33	(1.23)
Age 17	0.50	(1.17)	0.30	(1.31)
	n (%)			
Part of multiple birth	104	(2.57)	82	(2.1)
Ethnicity other than White	620	(15.3)	563	(14.6)
Cumulative risk indices				
Prenatal: 0 risks	2,304	(56.9)	2,161	(56)
1 Risk	1,417	(35)	1,370	(35.5)
2 Risks	320	(7.9)	308	(8)
3 Risks	10	(0.2)	18	(0.5)
Birth and neonatal: 0 risk	2,699	(66.6)	2,690	(69.7)
1 Risk	1,103	(27.2)	960	(24.9)
2 Risks	198	(4.9)	172	(4.5)
3 Risks	51	(1.3)	35	(0.9)
Socioeconomic status: 0 risks	2,642	(65.2)	2,602	(67.5)
1 Risk	729	(18)	659	(17.1)
2 Risks	569	(14)	503	(13)
3 Risks	111	(2.7)	93	(2.4)
Home environment: 0 risks	1,627	(40.2)	1,589	(41.2)
1 Risk	1,599	(39.5)	1,536	(39.8)
2 Risks	710	(17.5)	615	(15.9)
3 Risks	115	(2.8)	117	(3)

Note: BMI = body mass index; SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale).

below 0.05 (RMSEA) and above 0.95 (CFI) are considered good.

RESULTS

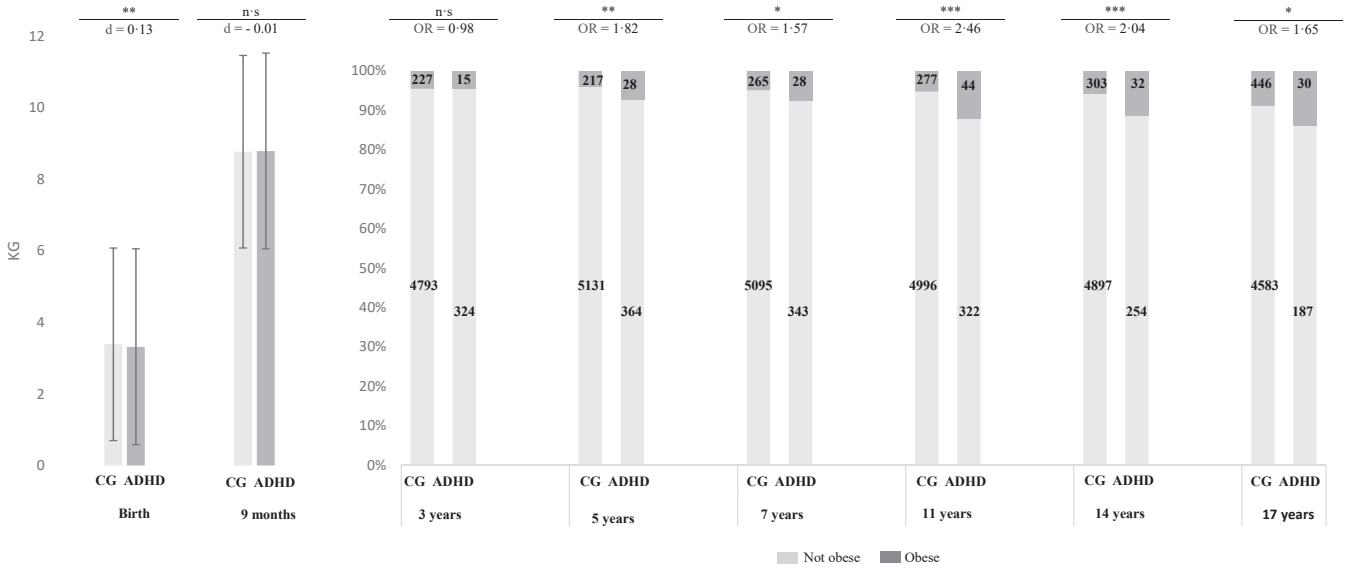
Descriptive statistics of participants can be found in Tables 1 and 2. Cohort members taking ADHD medication were excluded from the comparison analyses (n = 141) and adjusted for in longitudinal analyses (n = 83).

The cross-sectional results of each time point (Figure 1) showed that children in the ADHD group (mean = 3.32 kg) had significantly lower birth weight than children in the control group (mean = 3.39 kg); $t[5674] = 2.65$, 95% CI = 0.020-0.135, $p = .008$), although the effect size was very small (Cohen $d = 0.13$). By 9 months, the difference between the ADHD group (mean = 8.79 kg) and the control group (mean = 8.77 kg) was no longer significant ($t[5595] = -0.26$, 95% CI = −0.147 to 0.112, $p = .79$, Cohen $d = -0.01$). Children in the ADHD group (excluding those on medication) were significantly more likely to have obesity from age 5 years onward. Odds ratios ranged from 1.57 to 2.46, indicating small to medium effect sizes across time points (Table 3).

To explore the longitudinal associations between weight and ADHD, RI-CLPMs were conducted to explore the relationship between SDQ score and BMI *z* score in female (n = 4,051) and male (n = 3,857) individuals separately. Only 8 female individuals (<2%) were taking ADHD medications; therefore, these cohort members were excluded, and ADHD medication was not entered as a covariate in the analysis of female subjects. In both models (Figure 2a, 2b), all autoregressive paths were significant ($p < .001$). Goodness-of-fit indices indicated a good model fit for both (female individuals: CFI = 0.988, RMSEA = 0.041; male individuals: CFI = 0.986, RMSEA = 0.045). BMI *z* score did not significantly predict subsequent SDQ score at any time point in either model. In the female individuals–only model, SDQ score at ages 7, 11, and 14 years significantly predicted BMI *z* score at ages 11, 14, and 17 years, respectively. In the male individuals–only model, the SDQ score at age 11 years significantly predicted the BMI *z* score at age 14 years only. Additional model statistics and group demographics can be found in Tables S1 and S2, available online. A further RI-CLPM with male and female individuals combined can also be found in Table S3, available online. Covariate statistics for this combined model are reported in Figures S1 to S8, available online. The MPlus code for the combined model can also be found in Supplement 1, available online.

A sensitivity analysis, with the ADHD group consisting of only those cohort members with an ADHD diagnosis, found a similar pattern of results (Table S4, available online). Results at age 17 years did not reach a level of significance; however, effect sizes did mirror those in the main analyses.

FIGURE 1 Effect Sizes and Frequencies and Percentages of Children in the Attention-Deficit/Hyperactivity Disorder Group and Control Group at Different Ages



Note: *Weight in kilograms is displayed on the left for children in the ADHD group and the control group. The right side shows absolute numbers and the percentage of children in the ADHD group and control group with obesity at different ages. Error bars represent standard error. ADHD = attention-deficit/hyperactivity disorder; CG = control group; d = Cohen d effect size; OR = odds ratio.*
*p < .05; ** p < .01; *** p < .001.

DISCUSSION

The current study explored, for the first time, longitudinal associations between ADHD and weight using data from a large cohort sample at multiple time points between birth and 17 years. Overall, children with an ADHD diagnosis/consistently high ADHD symptoms had lower birth weight than children in the control group. Although the rate of obesity did not differ between children with and without high ADHD symptoms at age 3 years, children in the ADHD group were more likely to have obesity from the age of 5 years onward. As children with ADHD are typically lighter in weight at birth than children without ADHD, our results suggest there may be a sensitive time period between the ages of 3 and 5 years during which this association reverses and higher ADHD symptoms become associated with obesity.

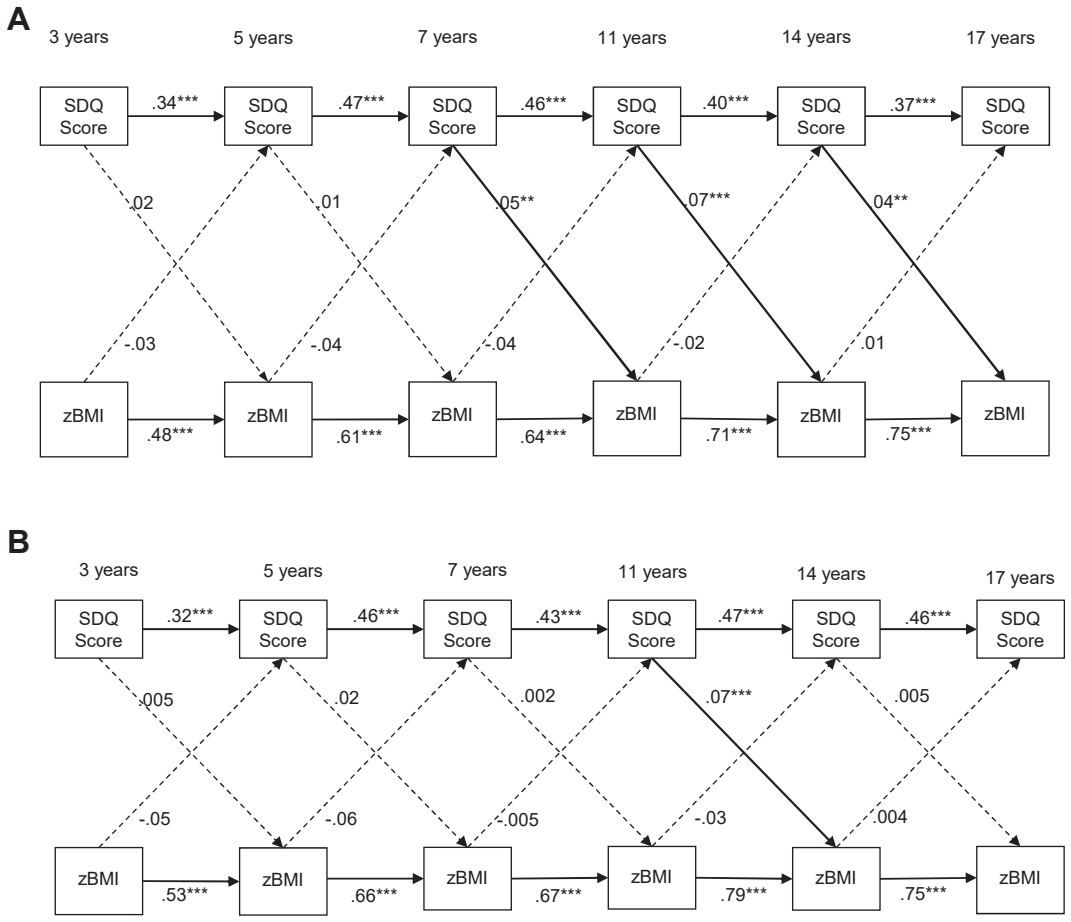
However, ADHD symptoms did not directly predict increased BMI until age 7 years in female and age 11 years

TABLE 3 Unweighted Descriptive Statistics and t/χ^2 Coefficients for Weight Variables

	ADHD group (n = 442)	Control group (n = 5,398)				
	Mean (SD)		*t*	*p*	Cohen *d*	
Weight at birth	3.32 kg (0.62)	3.39 kg (0.58)	2.65	**.008**	0.13	
Weight at 9 mo	8.79 kg (1.55)	8.77 kg (1.23)	−0.26	.79	−0.01	
No. of cohort members with obesity	**n (% of Valid data)**		χ^2	*p*	**Odds ratio (95% CI)**	**Relative risk (95% CI)**
Age 3	15 (4.42)	227 (4.21)	0.01	.93	0.98 (0.57, 1.67)	0.98 (0.59, 1.63)
Age 5	28 (7.14)	217 (4.06)	8.51	**.004**	1.82 (1.21, 2.73)	1.76 (1.20, 2.57)
Age 7	28 (7.55)	265 (4.94)	4.85	**.03**	1.57 (1.05, 2.35)	1.53 (1.05, 2.22)
Age 11	44 (12.02)	277 (5.25)	29.21	**<.001**	2.46 (1.76, 3.45)	2.29 (1.70, 3.09)
Age 14	32 (11.19)	303 (5.83)	13.59	**<.001**	2.04 (1.39, 2.99)	1.92 (1.36, 2.71)
Age 17	30 (13.82)	446 (8.87)	6.19	**.01**	1.65 (1.11, 2.45)	1.56 (1.11, 2.20)

Note: Significant associations indicated in boldface type. ADHD = attention-deficit/hyperactivity disorder.

FIGURE 2 Auto-Regressive and Cross-Lagged Associations Between Body Mass Index *z* Score and Strengths and Difficulties Questionnaire (SDQ [Hyperactivity/Inattention Subscale]) Score in Female Individuals (A) and Male Individuals (B) After Adjusting for Covariates



Note: *Significant paths are indicated by a solid line and non-significant paths by a dotted line. (Random intercepts and covariance parameters not shown.) zBMI = body mass index z score.*

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

in male individuals. This indicates that the shift from underweight to overweight during ages 3 to 5 years likely is not directly accounted for only by ADHD symptoms. Higher BMI at age 5 years was significantly predicted mainly by prenatal factors, namely, mothers' pre-pregnancy BMI, antenatal blood pressure, and smoking during pregnancy (Figure S1, available online). It is possible that there is a common genetic background to both ADHD and overweight,[44] or, as parents with ADHD are more likely to have children with ADHD,[45] this may have an impact on executive function skills involved in parenting, such as planning healthy meals, which in turn may influence weight status. Parents with an increased genetic risk may be influencing their child's home environment in ways that compound inherited risk. Future research should explore this interaction between direct heritability and genetic nurture, possibly through an adoption or twin study.

Several theories concerning underlying common mechanisms of ADHD and obesity have been discussed in previous literature. Genetic factors, executive function deficits, and environmental factors such as stress and disordered sleep may all offer potential explanations for the association.[46]

It has also been hypothesized that differences in brain energy consumption during early childhood may be responsible for later increased obesity risk.[47] In addition, children with ADHD may show altered function in the ability to convert glucose into energy for the brain, instead of storing it in fat cells.[48] As well as being a potential explanation for the executive function deficits associated with ADHD, this may also explain why higher obesity levels are

found in children with ADHD. Our results are also consistent with the thrifty phenotype hypothesis,[49] which suggests that prenatal factors, including poor nutrition, have long-lasting metabolic effects and may increase the risk of conditions such as diabetes. This suggests that future research into ADHD and weight may benefit from exploring insulin resistance resulting from prenatal factors. The complexity of the relationship posits that there is no simple explanation provided by a single factor, and further research is needed to determine the interactions among multiple mechanisms.

Longitudinally, we found that higher SDQ scores in girls predicted later higher BMI *z* scores from age 7 years onward, whereas in boys this association was seen only between ages 11 and 14 years. With increasing age, and attending school, children will gain increasing independence regarding food choices, and those with higher levels of impulsivity may be less likely to make healthier choices. The interaction between genes and environment may amplify this. Indeed, a recent study found a correlation between BMI polygenic risk scores and ADHD polygenic risk scores.[44] Comorbidity may be explained by a shared genetic risk if the same genes are implicated in multiple conditions. That study also reported that BMI and ADHD both demonstrated differences in brain areas responsible for reward processing, inhibitory control, and cognitive control. Children experiencing impairments in this area may be more likely to overeat, particularly at a time when they are undergoing significant changes in their lives, such as school transitions.

Interestingly, we found an earlier association between ADHD symptoms and obesity in girls than in boys. Obesity is known to affect the age of onset of puberty, with the mechanisms and effects differing between boys and girls.[50] Differences in body composition between boys and girls in puberty may be amplified in children with ADHD, who may show abnormal functioning in converting glucose to energy. The weight changes in girls additionally seemed to be more long lasting, whereas the observed changes in boys seemed to be transitory. However, follow-up data are missing, so it is unclear whether these associations continue into young adulthood. The current findings, however, suggest that early intervention programs targeting healthy eating and weight management are more indicated for girls than for boys with ADHD to inform prevention strategies. A stepped-care approach may be beneficial and could consider targeted interventions at critical stages. Results suggest that for prospective parents, support maintaining a healthy weight before pregnancy is important, as prenatal factors, including mothers' pre-pregnancy BMI, had a significant impact on children's weight at age 5 years. Given the heritability of ADHD and parental ADHD as a risk factor for child ADHD, this may be a particularly relevant secondary prevention strategy for parents with ADHD. Increasing the support for adults with ADHD in maintaining a healthy weight may benefit their future children. Continuing this support into the postnatal period could include offering increased weight monitoring for children during infancy and education on healthy eating habits for parents and caregivers. For children diagnosed with ADHD, clinicians should consider incorporating weight interventions or increased monitoring into treatment plans to ensure that the specific needs of each individual are considered at each stage of development.

This study has several strengths. We explored the relationship between ADHD and weight at multiple time points, from birth to adolescence. We used a large sample, weighted to be representative of the UK population, with relevant environmental risk accounted for in the longitudinal analyses. To our knowledge, this is the first study on the relationship between ADHD and weight to encompass a wide age range and to differentiate between male and female individuals.

It is important to note several limitations. As with all secondary research, frequencies in the sample do not always correspond with population estimates, as reflected in the low number of cohort members with an ADHD diagnosis. Although we attempted to mitigate this by including children with consistently high SDQ scores in the group analyses and by using SDQ score in the path analyses, our results should be interpreted with caution, as the SDQ is not a diagnostic tool. In addition, the SDQ subscale combines both hyperactive and inattentive traits and does not allow for an investigation of differential effects of individual symptom dimensions. Although both domains are associated with emotional overeating,[51] hyperactivity and inattention may have differential effects on obesity via lifestyle factors. A large Swedish cohort study found that inattention symptoms in childhood predicted less physical activity in adolescence, with the opposite pattern found for hyperactivity.[52] Future studies should therefore explore the contribution of both symptom domains on obesity and should investigate relevant mediators of this effect, such as exercise behaviors.

This study uses data across childhood; however, interpretation of the results should take into consideration the gap between each time period. Although the results shed light on the developmental period in which associations occur, we acknowledge that smaller gaps between time points would allow for a more precise interpretation. Future research may also adjust analyses for additional environmental factors, which we were unable to include in this study because of model complexity, such as comorbid child psychopathology. Further information about long-term medication use would also be beneficial in future studies, as ADHD medication status was available only at age 14 years, and we were therefore unable to account for children who were not taking ADHD medication but who had done so previously.

In summary, the current study shows that the weight shift from underweight to overweight in children with ADHD starts between the ages of 3 and 5 years. ADHD symptoms were directly associated with later obesity from age 7 years in girls and from age 11 years in boys in the United Kingdom, whereas obesity did not affect ADHD symptoms. This study further clarifies the direction of the association between ADHD and weight, although the mechanisms underlying weight gain in children with ADHD remain to be further explored.

CRediT authorship contribution statement

Claire Reed: Writing – original draft, Methodology, Formal analysis, Conceptualization. **Samuele Cortese:** Writing – review & editing, Methodology, Conceptualization. **Dennis Golm:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Valerie Brandt:** Writing – review & editing, Methodology, Formal analysis, Conceptualization.

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