Longitudinal Associations Between ADHD and Weight from Birth to Adolescence

Running title: ADHD AND WEIGHT ACROSS DEVELOPMENT

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Funding for this study: This study received no funding.

Acknowledgements: We are grateful to the Centre for Longitudinal Studies (CLS), UCL Social Research Institute, for the use of these data and to the UK Data Service for making them available. However, neither CLS nor the UK Data Service bear any responsibility for the analysis or interpretation of these data.

Samuele Cortese, NIHR Research Professor (NIHR303122) is funded by the NIHR for this research project. The views expressed in this publication are those of the author(s) and not necessarily those of the NIHR, NHS or the UK Department of Health and Social Care. Samuele Cortese is also supported by NIHR grants NIHR203684, NIHR203035, NIHR130077, NIHR128472, RP-PG-0618-20003 and by grant 101095568-HORIZONHLTH- 2022-DISEASE-07-03 from the European Research Executive Agency.

Financial disclosures:

Valerie Brandt acknowledges financial support from the Academy of Medical Sciences. Claire Reed was awarded a travel grant from the European College of Neuropsychopharmacology (ECNP) for travel to the 36th Congress. Dr Golm receives honoraria from CoramBAAF as Editor-in-Chief of Adoption & Fostering. Prof. Cortese has declared reimbursement for travel and accommodation expenses from the Association for Child and Adolescent Central Health (ACAMH) in relation to lectures delivered for ACAMH, the Canadian AADHD Alliance Resource, the British Association of Psychopharmacology, and from Healthcare Convention for educational activity on ADHD, and has received honoraria from Medice.

Conflict of interest: SC, CR, DG and VB have no COIs to declare.

Keywords: ADHD, obesity, environmental factors, ADHD medication.

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Abstract

Objective: 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 seven time points between 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 females (N=4051) and males (N=3857) to examine bidirectional associations between BMI z-scores and SDQ scores between ages three and 17. 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 at birth than the control group (t(5674)=2.65, 95% CI [0.02, 0.14] p=.008) and significantly more likely to have obesity at age five onwards (OR range 1.57-2.46, RR range 0.98-2.29). Path analyses conducted separately for males and females showed that higher ADHD symptoms in females at ages 7, 11 and 14 significantly predicted higher BMI z-score at ages 11, 14 and 17 respectively. In males, this association was only seen between age 11 and 14 (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, prenatally if possible. Developmental sex differences should be considered.

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD), characterised by persistent and impairing inattention and/or hyperactivity and impulsivity,[1] is the most common neurodevelopmental condition. Global prevalence estimates range between 5-11%[2-4] in childhood and adolescence. Diagnostic rates are more than twice as high in males than females,[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 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 preventative 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 two 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 high BMI leads to an increase in ADHD risk,[12] whereas others have found 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 nine years found that higher ADHD symptoms predicted higher weight from age three onwards, but 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, while 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 only assessed 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 males and females, across childhood and adolescence, while adjusting for relevant environmental risk factors. The main aims were to investigate: 1) at what age underweight shifts towards overweight in the ADHD group compared to the control group, 2) the bidirectional associations between ADHD symptoms and BMI across childhood, 3) whether this association is different in males and females.

Methods

Sample

We used data from the Millennium Cohort Study (MCS), a longitudinal cohort study involving over 19,000 families with children born between 2000 and 2002. Data have been collected at seven time points to date, when cohort members were nine months, 3, 5, 7, 11, 14, and 17 years old. 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=7908) only included 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 due to 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 three to six (age five to 14) parents were asked if their child had been diagnosed with ADHD. The SDQ was completed by parents at waves two to seven (age three to 17). The hyperactivity/inattention subscale requires parents to indicate whether the following five 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 five 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 five out of six waves. This cut-off corresponds to the 4-band categorisation of the SDQ with a score of 8 considered 'high' and scores of 9 or 10 considered 'very high.'[21] The control group (N= 5398) 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 five standard deviations 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 four cumulative risk indices (CRIs), each including three environmental factors identified in previous research as having an association with ADHD and/or obesity (Table 1). Data were collected during interviews with parents at the first data sweep (age nine 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 four CRIs were as follows (high risk criteria in brackets):

1) *Prenatal risk:* Mother's pre-pregnancy BMI (>24.9), antenatal blood pressure (pre-eclampsia/other related diagnosis), smoking status in pregnancy (≥1 cigarette);

2) *Birth and neonatal risk:* birthweight (<2.5kg), gestation (<37 weeks), breastfeeding (none attempted);

3) *Socioeconomic status:* household income (below 60% median poverty indicator), household crowding (fewer rooms than people, excluding bathrooms), 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 categorised as scoring ≥4 on a 9-item short form version[24] of the Rutter Malaise Inventory[25]).

In the RI-CLPM, the four CRIs, along with the following, were included as time-invariant predictors due to 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 if 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"[26] to calculate BMI and associated z-scores, adjusted for age and sex assigned at birth, based on the World Health Organisation Child Growth Standards.[27]

Two independent samples t-tests were conducted to compare 1) mean birth weight and 2) mean weight at nine months in the ADHD group and control group. Chi-squared 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, Chicago, Illinois).

Extended versions of a RI-CLPM with time-invariant predictors[28] were conducted for males and females in MPlus version 8.10.[29] The models were used to explore bidirectional associations between SDQ scores and BMI z-scores at six time points (ages 3, 5, 7, 11, 14 and 17). The RI-CLPM builds on a traditional cross-lagged panel model by accounting for both within-person and between-person 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, for example age 3 SDQ score to age 5 SDQ score) and cross-lagged paths (associations between a variable at one time point and another variable at the subsequent time point, for example age three SDQ score to age five BMI z-score). The four CRIs plus ethnicity, multiple birth, and ADHD medication (in males 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 root mean square error of approximation (RMSEA) and comparative fit index (CFI). Values below .05 (RMSEA) and above .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 (M = 3.32 kg) had significantly lower birth weight than children in the control group (M = 3.39 kg); t(5674) = 2.65, CI 95% [0.020, 0.135] p = .008, although the effect size was very small (Cohen's d = 0.13). By nine months, the difference between the ADHD group (M = 8.79 kg) and the control group (M=8.77 kg) was no longer significant, t(5595) = -0.26, 95% CI [-0.147, 0.112] p = .79, Cohen's d = -.01. Children in the ADHD group (excluding those on medication) were significantly more likely to have obesity from age five onwards. 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 females (N = 4051) and males (N = 3857) separately. Only eight females (<2%) were taking ADHD medications; therefore, these cohort members were excluded, and ADHD medication was not entered as a covariate in the female analysis. In both models (Figure 2a and 2b), all autoregressive paths were significant (p < .001). Goodness of fit indices indicated a good model fit for both (Females: CFI = .988, RMSEA = .041; Males: CFI = .986, RMSEA = .045). BMI z-score did not significantly predict subsequent SDQ score at any time point in either model. In the females-only model, SDQ score at ages 7, 11 and 14 significantly predicted BMI z-score at ages 11, 14 and 17, respectively. In the males-only model SDQ score at age 11 significantly predicted BMI z-score at age 14 only. Additional model statistics and group demographics can be found in Tables S1 and S2, available online. A further RI-CLPM with males and females

combined can also be found in Table S3. Covariate statistics for this combined model are reported in Figures S1-S8.

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

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. While the rate of obesity did not differ between children with and without high ADHD symptoms at age three, children in the ADHD group were more likely to have obesity from the age of five onwards. As children with ADHD are typically lighter at birth than children without ADHD, our results suggest there may be a sensitive time period between the ages of three and five 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 seven in females and age 11 in males. This indicates that the shift from underweight to overweight during ages three-five years is likely not directly accounted for only by ADHD symptoms. Higher age five BMI was mainly significantly predicted by prenatal factors, i.e., 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,[30] or, as parents with ADHD are more likely to have children with ADHD,[31] this may impact 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 which 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 of the association.[32]

It has also been hypothesized that differences in brain energy consumption during early childhood may be responsible for later increased obesity risk.[33] Additionally, 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.[34] 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[35] 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 interaction between multiple mechanisms.

Longitudinally, we found that higher SDQ scores in girls predicted later higher BMI z-scores from age seven onwards, whereas in boys this association was only seen between ages 11 and 14. 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.[30] 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

over-eat, 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.[36] 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, while 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 programmes targeting healthy eating and weight management are more indicated for girls than boys with ADHD in order 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 five. 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 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 to encompass a wide age range and to differentiate between males and females.

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. Additionally, the SDQ subscale combines both hyperactive and inattentive traits and does not allow for an investigation of differential effects of individual symptom dimensions. While both domains are associated with emotional overeating[37], 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[38]. Future studies should therefore explore the contribution of both symptom domains on obesity and investigate relevant mediators of this effect such as exercise behaviours.

This study utilises 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 were unable to be included in this study due to 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 only available at age 14 and we were therefore unable to account for children who were not taking ADHD medication but had previously.

In summary, the current study shows that the weight shift from underweight to overweight in children with ADHD starts between the ages three to five. ADHD symptoms were directly associated with later obesity from age seven in girls and from age 11 in boys in the UK, while obesity did not affect ADHD symptoms. This study further clarifies the direction of the association between ADHD and weight, while the mechanisms underlying weight gain in children with ADHD remain to be further explored.

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Table 1. Weighted Frequencies of Individual Environmental Risk Factors and Physical Health Conditions in the Males and Females Random Intercept Cross Lagged Panel Model Samples Combined, with Reference to the Associated Literature.

Environmental risk factors	High Risk Criteria	N = 7908
Prenatal factors		**N (%)**
Mother's pre-pregnancy BMI[14]	>24.9	2180 (27.6)
Antenatal blood pressure[39]	Pre-eclampsia/Hypertension diagnosis	605 (7.7)
Smoking in pregnancy[40,41]	Mother smoked 1 or more cigarettes during pregnancy	1342 (17)
Birth and neonatal factors		
Birthweight[42]	< 2.5kg	556 (7)
Gestation[42,43]	<37 weeks (premature)	580 (7.3)
Breastfeeding[44]	No breastfeeding	1925 (24.3)
Socioeconomic status		
Household income[45]	Below 60% median poverty indicator	2043 (25.8)
Household crowding[46]	Fewer rooms than people (excluding bathrooms)	703 (8.9)
Housing tenure[47]	Social housing or renting from local authority	1398 (17.7)
Home environment		
Mother's education attainment[48,49]	<NVQ level 3 (2 A-levels)	3447 (43.6)
Number of parents in household[45]	1 parent living in household	796 (10.1)
Mother's mental health[50-52]	Depression/anxiety diagnosis/<4 on Rutter Malaise Inventory	2238 (28.3)

Note: BMI = Body Mass Index; NVQ = National Vocational Qualification.

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

		Females (n=4051)	Males (n=3857)
		M (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	2304 (56.9)	2161 (56)
	1 risk	1417 (35)	1370 (35.5)
	2 risks	320 (7.9)	308 (8)
	3 risks	10 (0.2)	18 (0.5)
Birth and Neonatal	0 risks	2699 (66.6)	2690 (69.7)
	1 risk	1103 (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	2642 (65.2)	2602 (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	1627 (40.2)	1589 (41.2)
	1 risk	1599 (39.5)	1536 (39.8)
	2 risks	710 (17.5)	615 (15.9)
	3 risks	115 (2.8)	117 (3)

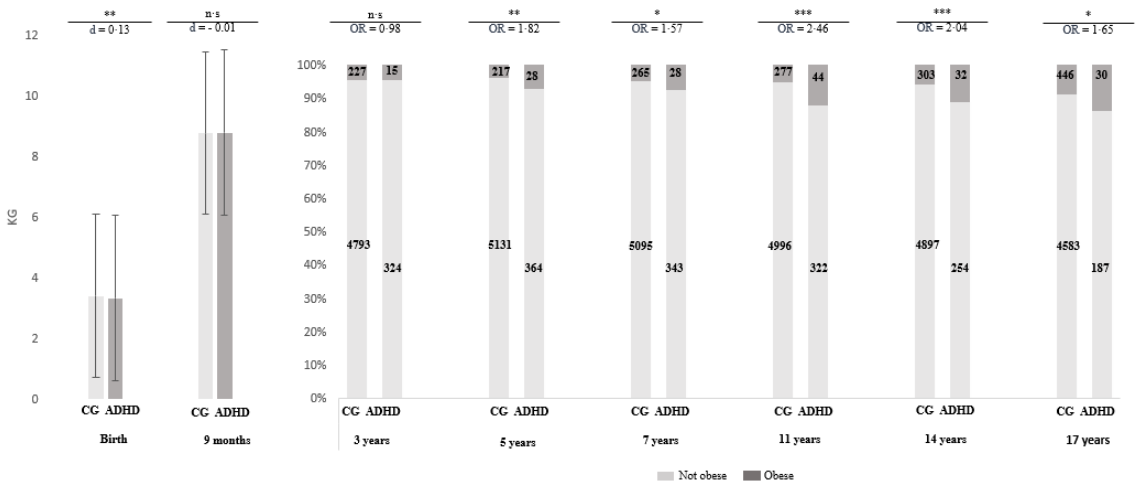
Note: BMI = Body Mass Index; M = Mean; SD = Standard Deviation; SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale).

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

	ADHD group (n=442)	**Control group (n=5398)**				
	M (SD)		**t**	***p***	**Cohen's d**	
Birth weight	3.32kg (0.62)	3.39kg (0.58)	2.65	**.008**	.13	
9 months weight	8.79kg (1.55)	8.77kg (1.23)	-0.26	.79	-.01	
Number 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 bold. ADHD = Attention Deficit Hyperactivity Disorder; CI = Confidence Interval; M = Mean; SD = Standard Deviation.

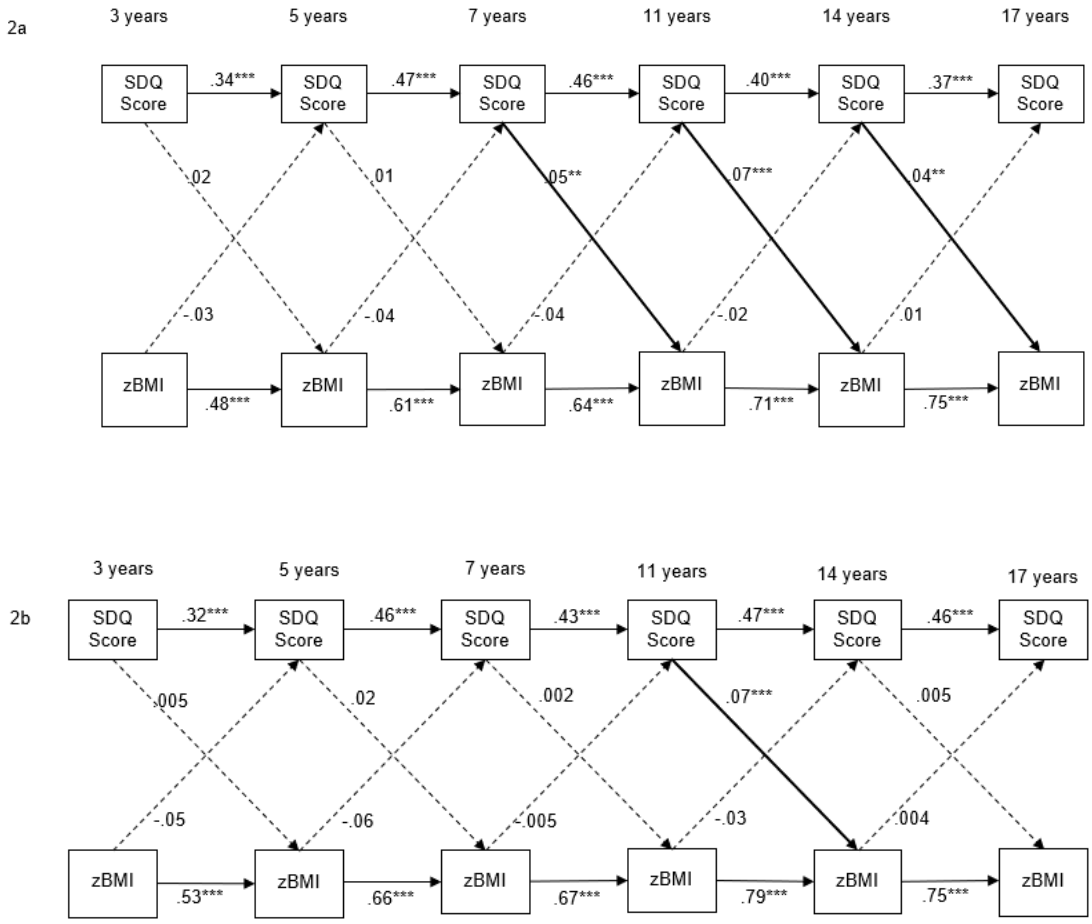
Figure 1. Effect Sizes and Frequencies and Percentages of Children in the ADHD Group and Control Group at Different Ages



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's d effect size; OR = Odds ratio.

*$p < \cdot 05$, ** $p < \cdot 01$, *** $p < \cdot 001$

Figure 2a and 2b: Auto-regressive and Cross-lagged Associations between BMI z-score and SDQ Score in Females (a) and Males (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.) SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale); zBMI = Body Mass Index z-score.

*$p < \cdot 05$, ** $p < \cdot 01$, *** $p < \cdot 001$

Supplementary Information

Table of Contents

Table S1. Standardised Model Statistics for the Weighted Random-Intercepts Cross-Lagged Panel Model- Females only.

	Estimate	95% CI	SE	*p*
Age 5 SDQ score on age 3 SDQ score	0.340	[0.29, 0.39]	0.024	**<.001**
Age 5 SDQ score on age 3 BMI z-score	-0.028	[-0.08, 0.03]	0.028	.33
Age 5 BMI z-score on age 3 SDQ score	0.024	[-0.03, 0.08]	0.027	.36
Age 5 BMI z-score on age 3 BMI z-score	0.480	[0.38, 0.58]	0.049	**<.001**
Age 7 SDQ score on age 5 SDQ score	0.470	[0.42, 0.52]	0.024	**<.001**
Age 7 SDQ score on age 5 BMI z-score	-0.041	[-0.09, 0.01]	0.026	.11
Age 7 BMI z-score on age 5 SDQ score	0.008	[-0.03, 0.05]	0.020	.67
Age 7 BMI z-score on age 5 BMI z-score	0.613	[0.53, 0.70]	0.045	**<.001**
Age 11 SDQ score on age 7 SDQ score	0.455	[0.41, 0.50]	0.024	**<.001**
Age 11 SDQ score on age 7 BMI z-score	-0.036	[-0.09, 0.01]	0.026	.16
Age 11 BMI z-score on age 7 SDQ score	0.048	[0.02, 0.08]	0.016	**.002**
Age 11 BMI z-score on age 7 BMI z-score	0.638	[0.57, 0.70]	0.034	**<.001**
Age 14 SDQ score on age 11 SDQ score	0.403	[0.35, 0.46]	0.027	**<.001**
Age 14 SDQ score on age 11 BMI z-score	-0.016	[-0.06, 0.03]	0.024	.51
Age 14 BMI z-score on age 11 SDQ score	0.066	[0.03, 0.10]	0.017	**<.001**
Age 14 BMI z-score on age 11 BMI z-score	0.714	[0.67, 0.75]	0.021	**<.001**
Age 17 SDQ score on age 14 SDQ score	0.373	[0.31, 0.42]	0.031	**<.001**
Age 17 SDQ score on age 14 BMI z-score	0.007	[-0.04, 0.05]	0.023	.75
Age 17 BMI z-score on age 14 SDQ score	0.044	[0.01, 0.07]	0.015	**.005**
Age 17 BMI z-score on age 14 BMI z-score	0.750	[0.72, 0.78]	0.015	**<.001**

Note: BMI = Body Mass Index; CI = Confidence Interval; SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale); SE = Standard Error.

Table S2: Standardised Model Statistics for the Weighted Random-Intercepts Cross-Lagged Panel Model- Males only.

	Estimate	95% CI	SE	*p*
Age 5 SDQ score on age 3 SDQ score	0.319	[0.27, 0.37]	0.024	**<.001**
Age 5 SDQ score on age 3 BMI z-score	0.052	[-0.02, 0.12]	0.034	.13
Age 5 BMI z-score on age 3 SDQ score	0.005	[-0.05, 0.06]	0.026	.85
Age 5 BMI z-score on age 3 BMI z-score	0.527	[0.39, 0.66]	0.068	**<.001**
Age 7 SDQ score on age 5 SDQ score	0.459	[0.41, 0.51]	0.024	**<.001**
Age 7 SDQ score on age 5 BMI z-score	-0.060	[-0.12, -0.00]	0.031	.05
Age 7 BMI z-score on age 5 SDQ score	0.022	[-0.02, 0.06]	0.020	.25
Age 7 BMI z-score on age 5 BMI z-score	0.663	[0.56, 0.77]	0.052	**<.001**
Age 11 SDQ score on age 7 SDQ score	0.429	[0.38, 0.48]	0.027	**<.001**
Age 11 SDQ score on age 7 BMI z-score	-0.005	[-0.07, 0.06]	0.031	.88
Age 11 BMI z-score on age 7 SDQ score	0.002	[-0.04, 0.04]	0.019	.94
Age 11 BMI z-score on age 7 BMI z-score	0.668	[0.60, 0.74]	0.037	**<.001**
Age 14 SDQ score on age 11 SDQ score	0.471	[0.42, 0.52]	0.026	**<.001**
Age 14 SDQ score on age 11 BMI z-score	-0.031	[-0.07, 0.01]	0.022	.16
Age 14 BMI z-score on age 11 SDQ score	0.067	[0.04, 0.10]	0.014	**<.001**
Age 14 BMI z-score on age 11 BMI z-score	0.791	[0.76, 0.83]	0.018	**<.001**
Age 17 SDQ score on age 14 SDQ score	0.462	[0.41, 0.51]	0.026	**<.001**
Age 17 SDQ score on age 14 BMI z-score	0.004	[-0.04, 0.05]	0.022	.86
Age 17 BMI z-score on age 14 SDQ score	0.005	[-0.02, 0.03]	0.013	.70
Age 17 BMI z-score on age 14 BMI z-score	0.788	[0.76, 0.82]	0.015	<.001

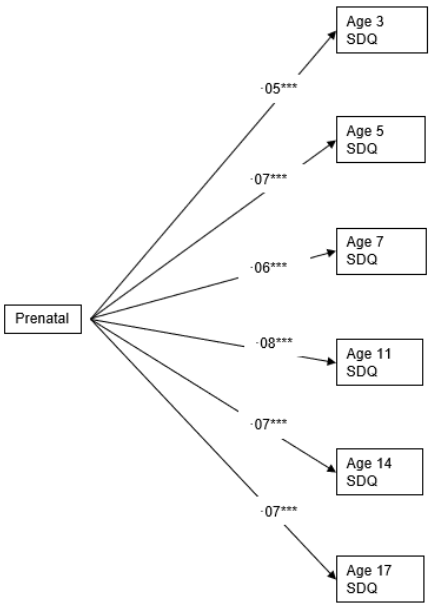
Note: BMI = Body Mass Index; CI = Confidence Interval; SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale); SE = Standard Error.

Table S3: Standardised Model Statistics for the Combined Sex Weighted Random-Intercepts Cross-Lagged Panel Model.

	Est	95% CI	SE	*p*
Age 5 SDQ score on age 3 SDQ score	0.327	[0.29, 0.36]	0.017	**<.001**
Age 5 SDQ score on age 3 BMI z-score	0.012	[-0.03, 0.06]	0.023	.60
Age 5 BMI z-score on age 3 SDQ score	0.013	[-0.02, 0.05]	0.018	.46
Age 5 BMI z-score on age 3 BMI z-score	0.499	[0.42, 0.58]	0.042	**<.001**
Age 7 SDQ score on age 5 SDQ score	0.462	[0.43, 0.50]	0.017	**<.001**
Age 7 SDQ score on age 5 BMI z-score	-0.052	[-0.09, -0.01]	0.020	**.009**
Age 7 BMI z-score on age 5 SDQ score	0.014	[-0.02, 0.04]	0.015	.34
Age 7 BMI z-score on age 5 BMI z-score	0.638	[0.57 0.71]	0.035	**<.001**
Age 11 SDQ score on age 7 SDQ score	0.441	[0.40, 0.48]	0.019	**<.001**
Age 11 SDQ score on age 7 BMI z-score	-0.021	[-0.06, 0.02]	0.020	.30
Age 11 BMI z-score on age 7 SDQ score	0.024	[-0.00, 0.05]	0.012	**.047**
Age 11 BMI z-score on age 7 BMI z-score	0.652	[0.60, 0.70]	0.025	**<.001**
Age 14 SDQ score on age 11 SDQ score	0.441	[0.40, 0.48]	0.020	**<.001**
Age 14 SDQ score on age 11 BMI z-score	-0.023	[-0.06, 0.01]	0.016	.15
Age 14 BMI z-score on age 11 SDQ score	0.067	[0.05, 0.09]	0.011	**<.001**
Age 14 BMI z-score on age 11 BMI z-score	0.756	[0.73, 0.78]	0.014	**<.001**
Age 17 SDQ score on age 14 SDQ score	0.424	[0.39, 0.46]	0.020	**<.001**
Age 17 SDQ score on age 14 BMI z-score	0.004	[-0.03, 0.04]	0.017	.81
Age 17 BMI z-score on age 14 SDQ score	0.021	[-0.00, 0.04]	0.010	**.030**
Age 17 BMI z-score on age 14 BMI z-score	0.770	[0.75, 0.79]	0.011	**<.001**

Note: BMI = Body Mass Index; CI = Confidence Interval; SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale); SE = Standard Error.

Figures S1-S8 : Beta Statistics for Covariates in the Combined Sex RI-CLPM



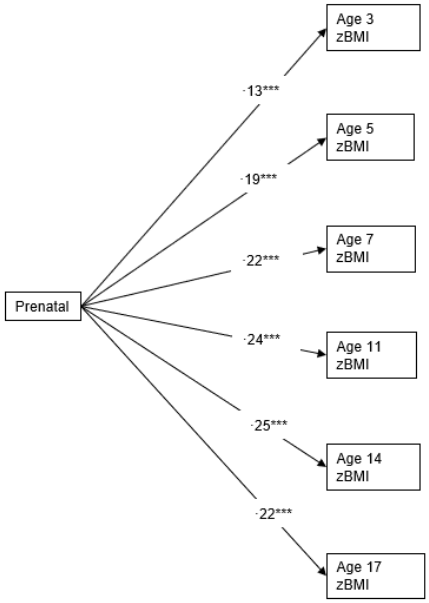
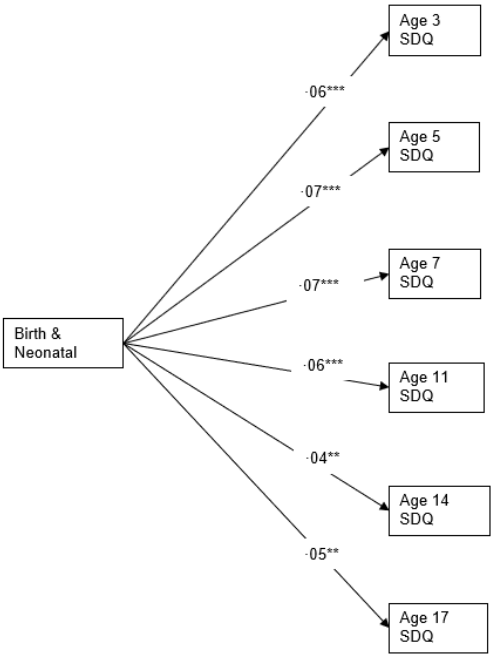


Figure S1. Prenatal Risk Factors.



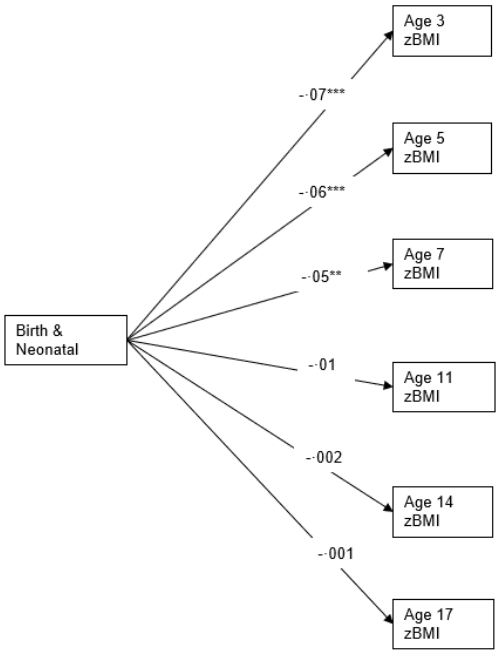


Figure S2. Birth and Neonatal Risk Factors.

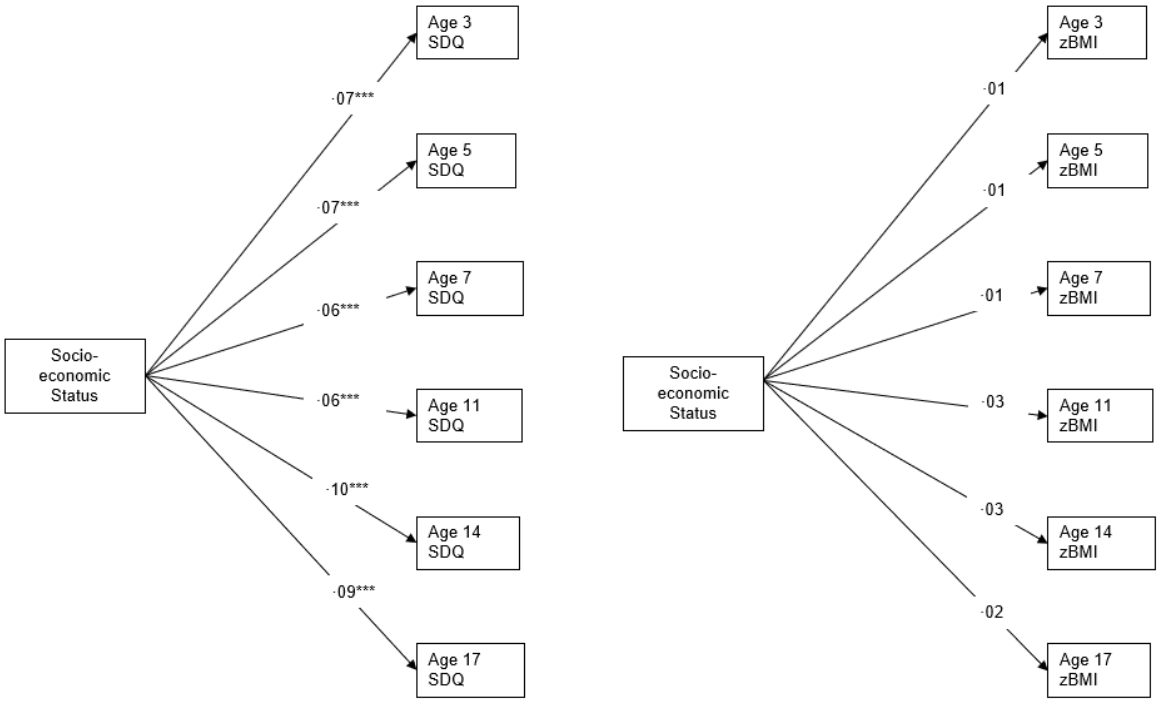


Figure S3. Socioeconomic Status Risk Factors.

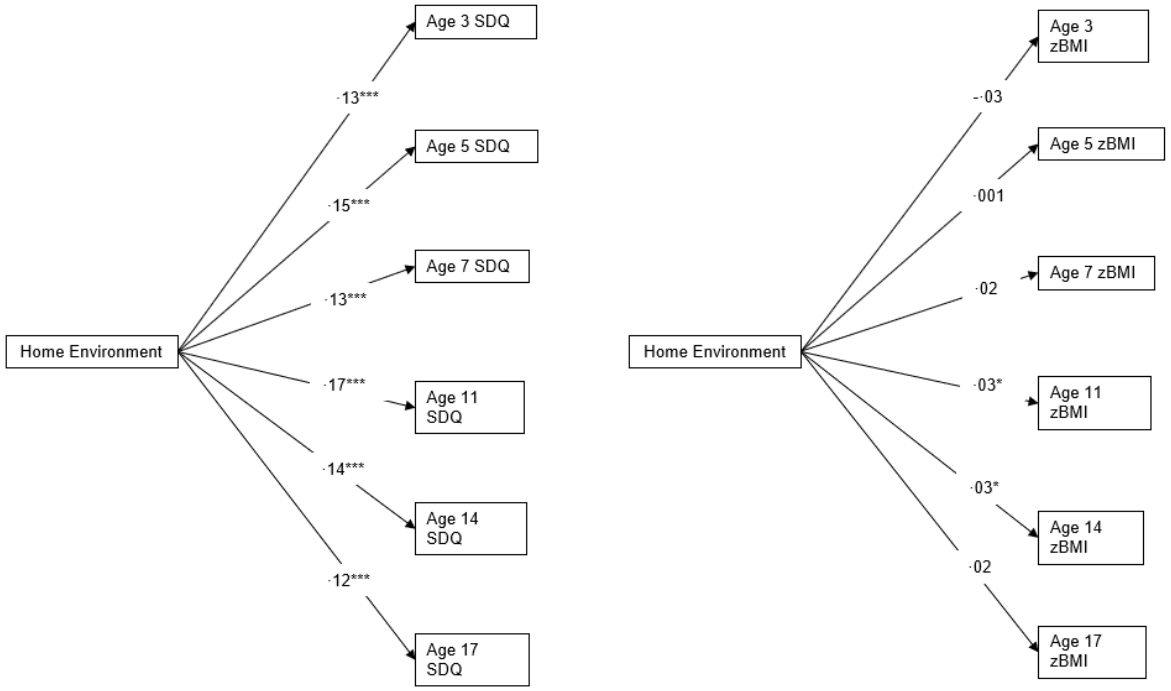


Figure S4. Home Environment Risk Factors.

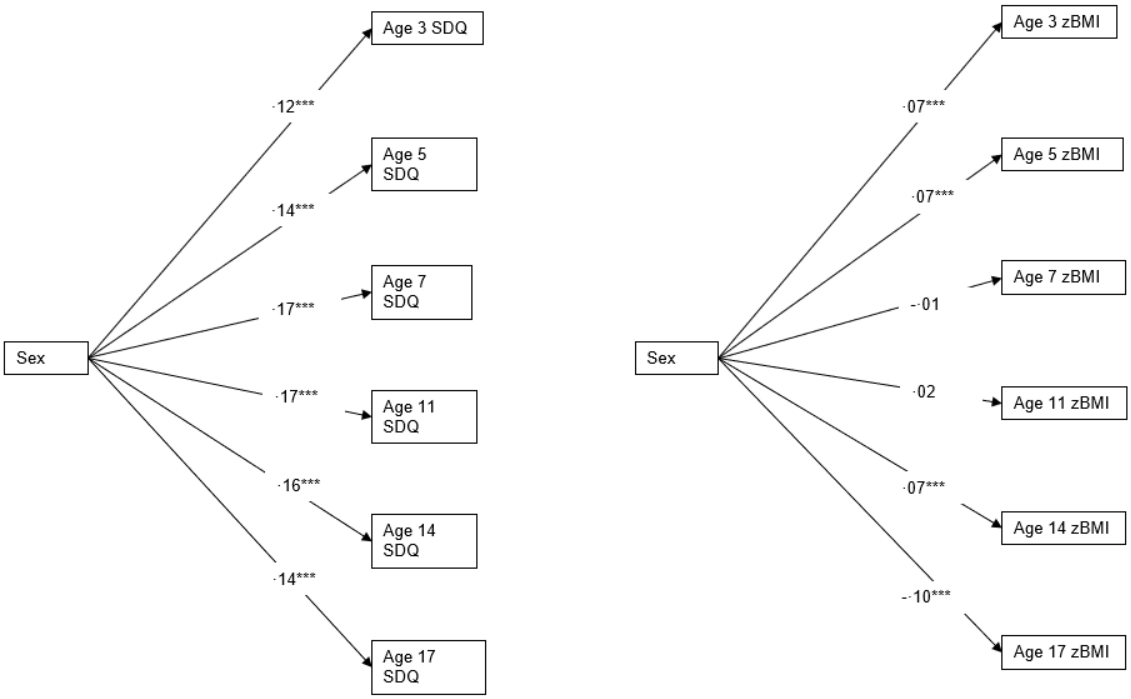


Figure S5. Sex entered as Time Invariant Predictor.

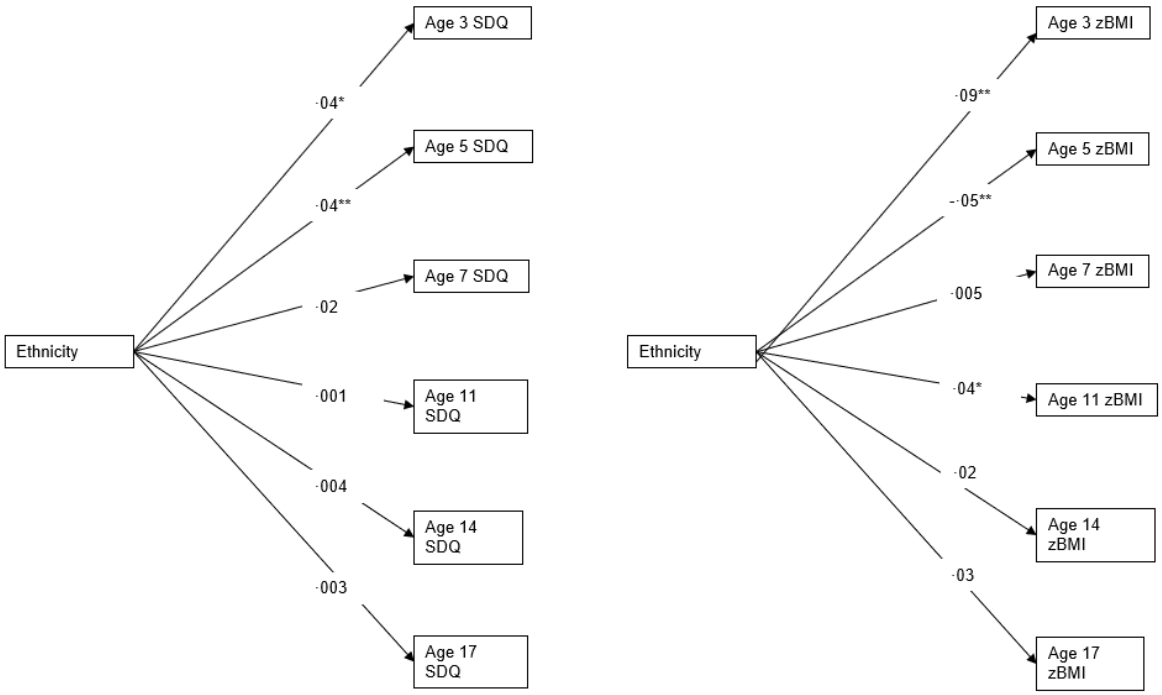


Figure S6. Ethnicity entered as Time Invariant Predictor.

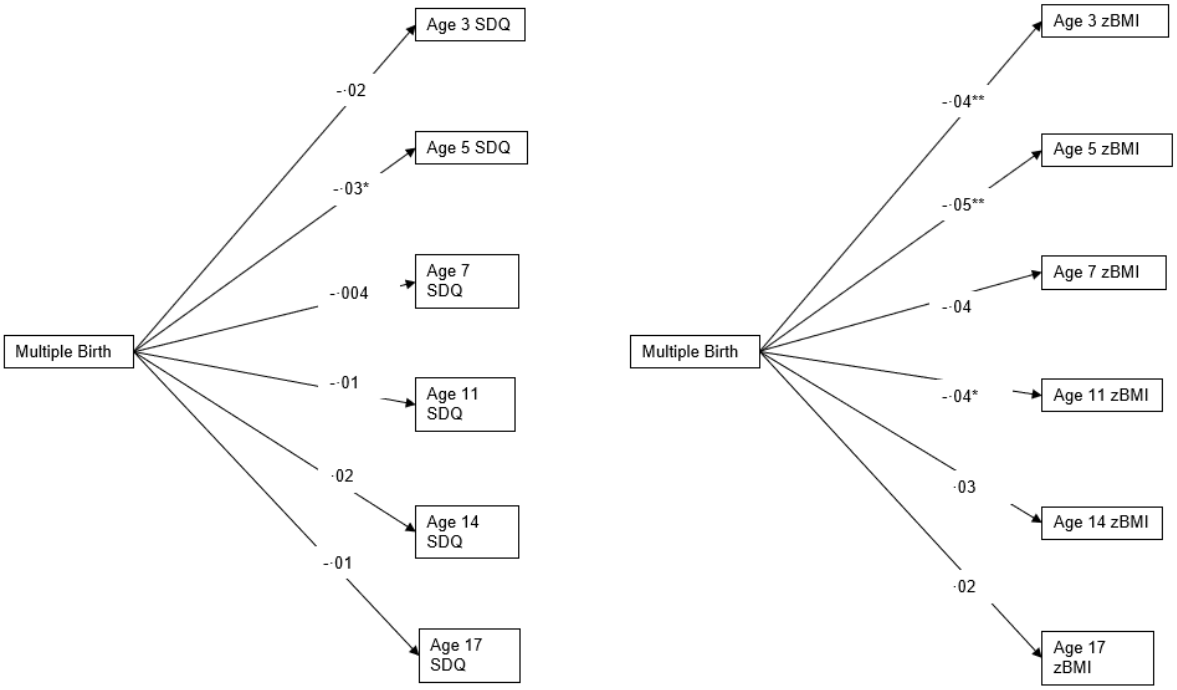


Figure S7. Multiple Birth Status entered as Time Invariant Predictor.

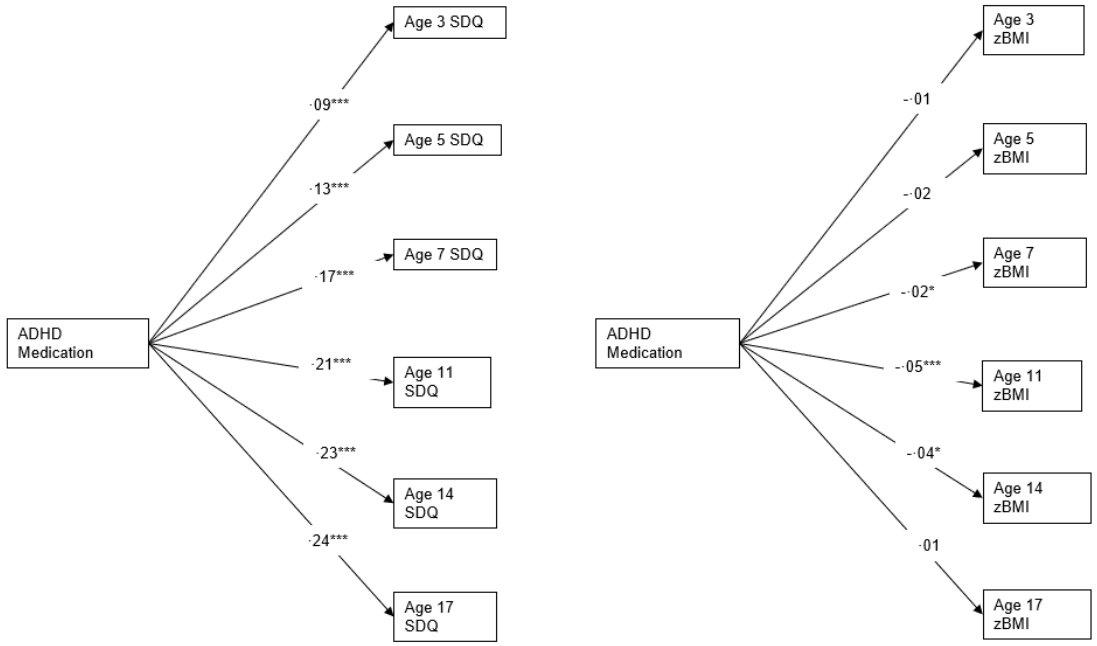


Figure S8. ADHD Medication Status entered as Time Invariant Predictor.
Note: SDQ = Strengths and Difficulties Questionnaire (Hyperactivity/Inattention subscale); zBMI = Body Mass Index z-score. * $p < .05$, ** $p < .01$, *** $p < .001$

MPlus Syntax for Path Analyses

```
TITLE: ZBMI RI_CLPM
DATA: FILE IS {REDACTED}


VARIABLE:
NAMES ARE
MCSID
CNum
Ethnicity
Multiples
W7_WGHT
W2_SDQ
W3_SDQ
W4_SDQ
W5_SDQ
W6_SDQ
W7_SDQ
W2_BMI
W3_BMI
W4_BMI
W5_BMI
W6_BMI
W7_BMI
W2_zbmi
W3_zbmi
W4_zbmi
W5_zbmi
W6_zbmi
W7_zbmi
ADHD_Med
Prenatal
BirthNeo
SES
HomeEnv
PTTYPE2
SPTN00;


USEVARIABLES ARE

Ethnicity
Multiples
W7_WGHT
W2_SDQ
W3_SDQ
W4_SDQ
W5_SDQ
```

```
W6_SDQ
W7_SDQ
W2_zbmi
W3_zbmi
W4_zbmi
W5_zbmi
W6_zbmi
W7_zbmi
ADHD_Med
Prenatal
BirthNeo
SES
HomeEnv
PTTYPE2
SPTN00;

MISSING ARE ALL (-999);
WEIGHT = W7_WGHT;
STRATIFICATION IS PTTYPE2;
CLUSTER IS SPTN00;

ANALYSIS:
TYPE = COMPLEX;
ESTIMATOR = MLR;
MODEL = NOCOV;


MODEL:
! Create between components (random intercepts)
RISDQ BY W2_SDQ@1 W3_SDQ@1 W4_SDQ@1 W5_SDQ@1 W6_SDQ@1
W7_SDQ@1;
RIBMI BY W2_zbmi@1 W3_zbmi@1 W4_zbmi@1 W5_zbmi@1 W6_zbmi@1 W7_zbmi;

! Estimate covariance between random intercepts
RISDQ WITH RIBMI;

! Create within-person centered variables
wW2_SDQ BY W2_SDQ@1;
wW3_SDQ BY W3_SDQ@1;
wW4_SDQ BY W4_SDQ@1;
wW5_SDQ BY W5_SDQ@1;
wW6_SDQ BY W6_SDQ@1;
wW7_SDQ BY W7_SDQ@1;
wW2_zbmi BY W2_zbmi@1;
wW3_zbmi BY W3_zbmi@1;
wW4_zbmi BY W4_zbmi@1;
wW5_zbmi BY W5_zbmi@1;
wW6_zbmi BY W6_zbmi@1;
wW7_zbmi BY W7_zbmi@1;
```

```
! Constrain measurement error variances to 0
W2_SDQ-W7_zbmi@0;

! Regression of observed variables on covariates
W2_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W3_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W4_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W5_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W6_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W7_SDQ ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;

W2_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W3_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W4_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W5_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W6_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;
W7_zbmi ON Sex Ethnicity Multiples ADHD_Med Prenatal BirthNeo SES HomeEnv;

! Estimate lagged effects between within-person centered variables
wW3_SDQ wW3_zbmi ON wW2_SDQ wW2_zbmi;
wW4_SDQ wW4_zbmi ON wW3_SDQ wW3_zbmi;
wW5_SDQ wW5_zbmi ON wW4_SDQ wW4_zbmi;
wW6_SDQ wW6_zbmi ON wW5_SDQ wW5_zbmi;
wW7_SDQ wW7_zbmi ON wW6_SDQ wW6_zbmi;

! Estimate covariance between within-person components at first wave
wW2_SDQ WITH wW2_zbmi;

! Estimate covariances between residuals of within-person component

wW3_SDQ WITH wW3_zbmi;
wW4_SDQ WITH wW4_zbmi;
wW5_SDQ WITH wW5_zbmi;
wW6_SDQ WITH wW6_zbmi;
wW7_SDQ WITH wW7_zbmi;

OUTPUT:     TECH1 STDYX SAMPSTAT CINTERVAL;
```

Table S4. Sensitivity Analysis- t/ χ^2 Coefficients for analyses with ADHD group only consisting of cohort members with a diagnosis.

	ADHD group (n=442)	**Control group (n=5398)**				
	M (SD)		**t**	***p***	**Cohen's d**	
Birth weight	3.27 (0.70)	3.38 (0.65)	3.18	**.002**	0.17	
9 months weight	8.38 (3.10)	8.53 (8.38)	1.10	.270	-0.06	
Number of cohort members with obesity	**N (% of valid data)**		**χ^2**	***p***	**Odds Ratio (95% CI)**	**Relative Risk (95% CI)**
Age 3	14 (4.76)	227 (4.52)	0.04	.848	1.06 (0.61, 1.84)	1.05 (0.62, 1.78)
Age 5	25 (7.27)	217 (4.06)	8.18	**.004**	1.85 (1.21, 2.85)	1.79 (1.20, 2.67)
Age 7	25 (7.72)	265 (4.94)	4.85	**.028**	1.61 (1.05, 2.46)	1.56 (1.05, 2.32)
Age 11	38 (11.84)	277 (5.25)	24.7	**<.001**	2.42 (1.69, 3.47)	2.25 (1.64, 3.10)
Age 14	27 (11.25)	303 (5.83)	11.8	**<.001**	2.05 (1.35, 3.11)	1.93 (1.33, 2.80)
Age 17	23 (12.9)	446 (8.87)	3.45	.063	1.52 (0.97, 2.39)	1.46 (0.99, 2.16)

ADHD: Attention Deficit Hyperactivity Disorder; M: Mean; SD: Standard Deviation; CI: Confidence Interval.
Significant associations indicated in bold.