Title: The Impacts of Childhood Weight Status on Academic Achievement: Evidence from Mendelian Randomization

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Abstract

Objective: We investigated the causal link between childhood weight status and academic achievement across genders and different school subjects in Norway.

Study design and setting: We used data from the Norwegian Mother, Father and Child Cohort Study, which includes genetic data (N=13,648, 8-year-old children). We employed within-family mendelian randomization, using a BMI polygenic risk score as an instrument to address unobserved heterogeneity.

Results: Contrary to most previous findings, we observed that overweight status (including obesity) has more detrimental effects on reading achievement in boys than in girls; the test scores of overweight boys were about a standard deviation lower than those of normal weight boys, and the negative effects on reading achievement became stronger in the later grade.

Conclusion: Previous obesity prevention studies have mainly targeted girls, based on the assumption that the obesity penalty is greater for girls. Our findings highlight that particular attention to boys with overweight may help to reduce the existing gender gap in academic achievement.

Keywords: childhood obesity; academic achievement; polygenic risk scores; instrumental variable analysis; Mendelian Randomization; MoBa

Word count: 2,986

1. Introduction

Obesity has reached epidemic proportions globally, with childhood obesity becoming one of the most pressing issues in public health, clinical practice and research [1]. Childhood obesity is closely associated with adult obesity, morbidity and mortality [2] and also linked to important academic outcomes [3]. Previous studies have for example reported associations between childhood obesity and academic performance [4], negative teacher evaluation [5], and poor mental health [6]. Despite the suggestive findings, however, robust empirical evidence remains lacking [4, 7]. Child weight status is a function of parenting styles, eating habit/preferences, peer effects, and genetic factors [8], which are difficult to fully measure and control for in observational studies. Furthermore, the available evidence is mainly limited to North American and British contexts [4]. In this study, we present empirical evidence from Norway by exploiting the random segregation of genetic variants from parents to offspring to improve our current understanding of the relationship between childhood obesity and academic achievement.

Unobserved confounding is an enduring source of bias in observational studies [9]. Even if known, measuring them can be prohibitively time-consuming and costly in terms of data collection. Several previous studies on obesity and academic achievement have thus employed an instrumental variable (IV) analysis to make causal inference [10-13]. IV analyses rely on two key identifying requirements in seeking to make casual inferences [14] (for details on other IV assumptions see analytic strategy): first, the instrumental variable should be highly correlated with the key explanatory variable of interest; second, the instrumental variable should be uncorrelated with the error term of the dependent variable. Since it is challenging to identify appropriate IVs in observational studies, several social scientists have attempted to utilize

genetic markers to instrument for child weight status, an approach known as Mendelian randomization.

In particular, a polygenic risk score (PRS) is an individual-level sum-score of many single nucleotide polymorphisms (SNPs) (i.e., genetic variants of individual DNA letters), weighted by their association with a specific phenotype (trait or characteristic) from available genome-wide association studies (GWAS) [15]. It can be computed for any phenotype such as obesity, educational attainment, or well-being. Because of the small effect sizes of single genetic variants, the PRS generated by weighted multiple genetic variants is preferred. The underlying rationale of using a PRS as IV is that as the PRS is generated from available SNPs targeting a specific phenotype, it is likely to have a relatively strong predictive power. Moreover, since genes are randomly assigned within families, within-family PRS (i.e., child PRS adjusted for parental PRS) may not affect dependent variables via other channels [16]. While those IV studies using single or multiple SNPs tend to report small or null effects of weight status on academic performance [17-20], a recent IV study using PRS showed that childhood BMI may in fact have nontrivial effects on test scores [21].

Overall, although the previous IV studies suggest a negative association between child weight status and academic performance, the available evidence is mainly limited to US and UK contexts. Given the importance of considering social/cultural differences in obesity prevention [22], the relative paucity of research in other social contexts is striking. Related to this study, Hughes *et al.* [21] investigated the link between childhood BMI and educational attainment in the UK using BMI PRS. Although this study provides a comprehensive overview of the relationships between common health conditions and educational outcomes, it does not consider gender and subject differences or non-linear effects of BMI (e.g., overweight/obesity status).

More importantly, the study did not use *within-family* BMI PRS, meaning that its findings could still be explained by confounding factors such as parental effects.

This study fills the gap by exploiting the advantages of the rich Norwegian Mother, Father and Child Cohort Study. We instrument the endogenous weight status with *within-family* child BMI PRS. Since genes are randomly assigned at conception, our within-family approach conditioning on parental genotype will provide robust estimates against familial effects [16, 23]. The weight of a biological relative has been extensively employed as IV in econometric studies [10-12]; we add results obtained from the IV-BMI specification for comparison. Previous studies report the heterogenous effects of weight status across subjects [12], grade [4], and gender [11]. We thus carefully examine the link across reading, math, and English subjects measured at 5th and 8th grade between boys and girls, respectively. In many developed nations, boys have underperformed relative to girls, particularly in reading [24]. Our genetically informed methods may provide further insight into the gender gap in academic performance.

Norway has been considered an egalitarian and culturally/racially homogenous society compared to the US and UK, which previous IV studies have primarily focused on. In Norway, gender and sexual equality and redistributive policies have been core values of national identity [25]. As the "obesity penalty" tends to be more pronounced for female or minority groups [26], the detrimental effects of childhood obesity may be more salient in societies where systems of universal social welfare are not the norm and gender discriminations prevail. The prevalence of overweight (including obesity) in Norway is about 25% among boys and 23% among girls, which is similar to that of Eastern European countries, but much lower than in Southern Europe [27]. This study provides a unique opportunity to study how childhood weight status may

generate negative effects on academic outcomes even in such favorable and homogenous social contexts.

2. Methods

2.1 Data and sample

The Norwegian Mother, Father and Child Cohort Study (MoBa) is a prospective cohort study conducted by the Norwegian Institute of Public Health [28]. It includes mothers, fathers, and children related to 112,789 pregnancies sampled between 1999 and 2008. Parents reported on their own and their children's behaviors and health during early years at regular intervals. This study focused on the latest data from eight-year-old children (grade 2 or 3), which also provides baseline covariates information on children and families. From the data's total sample of 43,616, 13,648 children (excluding twins) linked to the genome-wide genetic data were selected. The analytic genetic data has similar characteristics to those of the original analytic sample (see Appendix A). We used version 12 of the quality-assured data.

2.2 Measures

2.2.1 Child academic development

We used national test scores obtained through linkage to Norway's National Education Database. These are reading, math, and English scores measured at 5th and 8th grade. Compulsory national assessments for children have been administered every fall in Norway since 2007 (about 96% of all students in Norway), in the subjects reading (Norwegian), math, and English. Test data are available only through 2017. As not all children had passed through the 8th grade tests at that time due to their age, valid test score observations vary across the two

grades. We standardized the test scores within year to capture cohort effects (raw scores ranged from 19 to 81 and the values of skewness and kurtosis ranged from –.50 to –.03 and 2.11 to 2.61, respectively).

2.2.2 Child weight status

We generated childhood BMI based on mother reports of child weight and height measured at eight years old. Additionally, to account for a nonlinear relationship between BMI and academic performance, we generated age- and sex-specific overweight status (=1) according to International Obesity Task Force definitions [29]. Due to the small number of obese children (1.72%) in our sample (BMI-for-age ≥ 2SD), we created the overweight status including obese children (ref=normal weight).

2.2.3 Instrumental variable

We instrumented child weight status with BMI PRS and maternal BMI, respectively. First, the PRS for BMI was constructed from the most recent and largest genome-wide association study [30]. The PRS was obtained for the 93,582 individuals (European ancestry) who passed quality control using PRSice software [31]. We used information from all available SNPs calculating scores (i.e., p-value threshold = 1.00). To remove potential parental confounding effects, we utilized residuals of child PRS after regressing out mother and father PRS (for the data generation process see Cheesman *et al.* [16]). PRS was standardized to zero mean and unit variance. Second, maternal BMI was measured using the Norwegian medical birth register data on BMI prior to pregnancy from 46,004 parents who have quality-control genotype and phenotype data on BMI.

2.2.4 Confounders

We selected basic demographic variables from MoBa [16, 32]. Gender was coded one for boys and zero for girls, and school grade and child age (by months) were also included. Parental educational level and annual household income were obtained from the Norwegian administrative register data at the baseline year. Given the sample size of each category, we created categorized father and mother education (0=middle school, 1=high school, 3=BA, 4=MA/PhD, as dummy variables). We also included parent non-native Norwegian speaker (=1), single (separated) mother (=1), cohort dummies, and number of children at home as baseline controls.

2.3 Analytic strategy

To investigate the causal effects of childhood weight status on academic performance, we employed IV and OLS estimators. There are four assumptions for an IV analysis which must be met. First, the instrument should be highly correlated with the variable of interest (i.e., instrument relevance). Second, there are no unmeasured confounders between the instrument and outcome variable (i.e., independence assumption). Third, the instrumental variable should affect the dependent variable only through the predictor (i.e., exclusion restriction). Lastly, it is assumed that individuals' response to the instrument is monotonic (i.e., monotonicity) [33, 34]. Figure 1 illustrates our IV research framework and related assumptions (dashed lines represent opened "backdoor paths" that should be closed). In Appendix B we discuss the rationale of our instrumental variable approach in detail. For comparison, we present results from the IV-PRS as well as IV-BMI specifications. Yet, due to the random nature of genetic inheritance within families, our preferred specification is the IV-PRS. The 10 genetic ancestry principal components (five based on maternal data and five based on paternal data) were included to account for potential population stratification [16]. We also included child sex and age to

account for the different sex- and age-specific distributions of BMI. We used the two-stage least squares (2SLS) estimator. Analyses were carried out in line with the *ivreg2* module, using the *cluster* option in Stata to adjust standard errors for the MoBa sampling design.

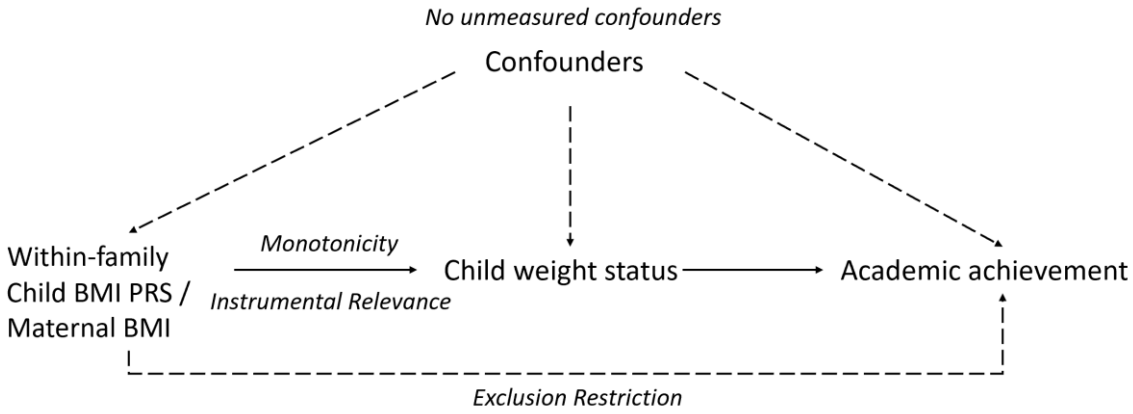

Fig 1. Diagram of instrumental variable assumptions

3. Results

3.1 Effects of childhood overweight on academic performance

The estimated coefficients and confidence intervals of overweight status on girls and boys are illustrated in Figures 2–4. Here we present results from IV models with a dichotomized weight variable (results obtained from the linear specification also showed similar patterns, see Appendix D-F including results for total sample). There were significant negative associations between BMI and reading and math achievements without controls (–.01 to –.03), yet we did not find clear evidence that child weight status has significant net effects on test scores, regardless of gender from OLS regression with controls [35, 36]. However, since unblocked backdoor paths from child weight status may offset the total effects of child weight status (see similar examples in Pearl [37]), we do not put much emphasis on the findings from the OLS estimator. We primarily focus on findings from the IV analyses.

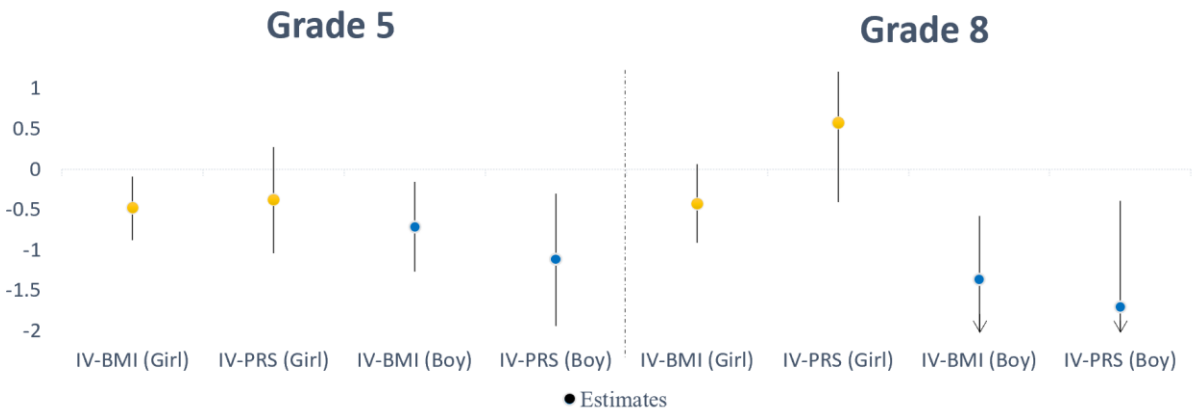

Fig 2. The effects of overweight on reading

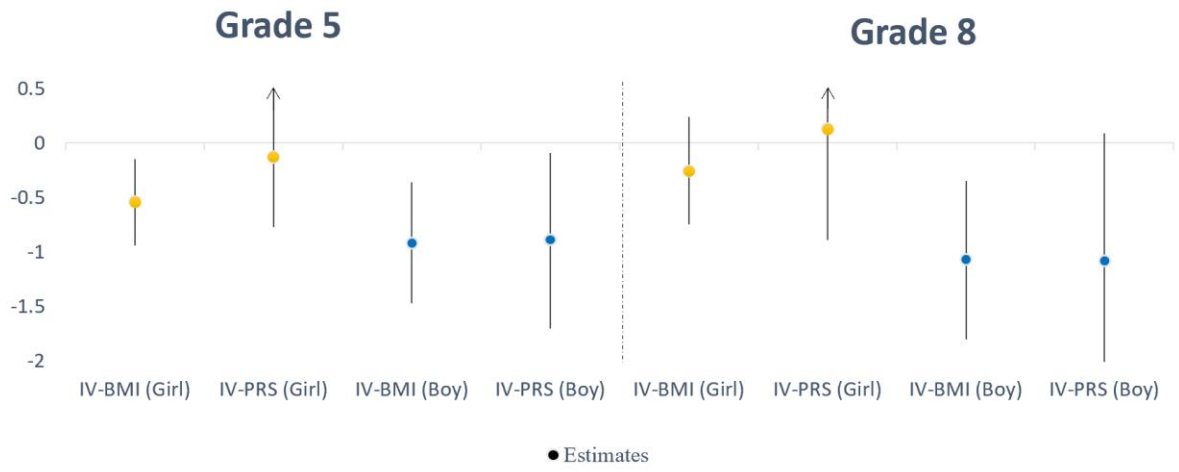

Fig 3. The effects of overweight on math

Returning to Figure 2, both the IV-BMI and IV-PRS specifications consistently report significant relationships of overweight status with boys' reading scores at 5th and 8th grades; The test scores of overweight boys were likely to be more than an SD lower than those of normal

weight boys. The observed patterns for boys were also evident in the 5th grade math scores in Figure 3. For girls, however, we only observed significant effects of overweight status on 5th grade reading and math scores from the IV-BMI specification. Overall, the observed coefficients were larger for boys, and the negative influences on reading became stronger in the later grade; the overlapping CIs suggest that the largest gender difference was found in 8th grade reading. However, we did not find any significant relationships of weight status with English in any of the specifications (see Figure 4).

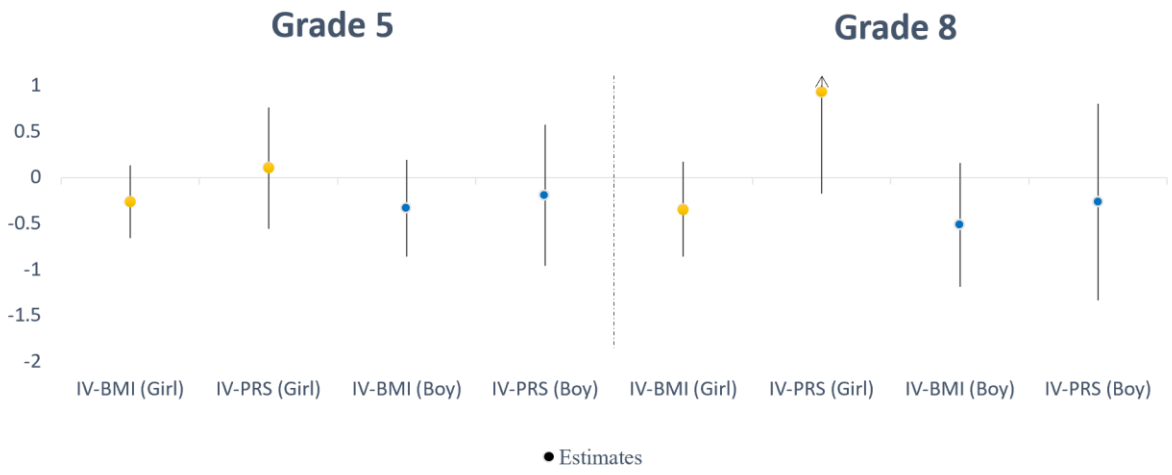

Fig 4. The effects of overweight on English

3.2 Robustness checks

We checked the robustness of our findings as follows. First, given the possibility that our continuous IVs may not serve as a good instrument for the dichotomized overweight status, we estimated the fitted value in the first stage using a probit model ($E(D_i|Z_i, X_i)$) instead, and then used the fitted value as IV [38]. The results, illustrated in Appendix G, showed very similar

patterns, particularly for boys. Further, we controlled for the Big Five personality traits (i.e., extraversion, benevolence, conscientiousness, neuroticism, and imagination) in the IV equation. Since individual personalities are significantly associated with educational outcomes [39], controlling for these traits may contribute to block opened backdoor paths in Figure 1. This might be a conservative approach since these personality items may partially mediate total effects of weight status. Although some of the observed significant effects of BMI became marginally significant at $p<.10$, the results showed similar patterns (see Appendix I). Additionally, we reassessed our findings using a recently developed sensitivity test [40] (for details see Appendix C). The results also showed that the observed significant effects of childhood weight status on test scores are robust to unobserved confounding. We interpret this relative consistency in the impacts of child weight status as evidence for the robustness of our results.

4. Discussion

Contrary to most previous findings, we showed that boys' overweight status has more detrimental effects on reading than that of girls. The observed effects were not negligible: the test scores of overweight boys were about an SD lower than those of normal weight boys (or .70 of an SD with the continuous BMI predictor), and the negative effects on reading became stronger in the later grade. More specifically, the results obtained from 2SLS with IV-BMI tended to be more significant and yielded similar effect sizes to those found in previous studies [11, 12]. Yet we still observed the significant effects from the IV-PRS specification particularly for reading, which were also robust to the violation of independence and exclusion restriction assumptions.

Nevertheless, we did not find any evidence that childhood weight status is linked to English achievement.

What might explain the underperformance of boys with overweight in Norway? It is often hypothesized that the obesity penalty is larger for girls due in part to greater gender discrimination based on body image [17, 41]. Yet, Black *et al.* [11] found that weight status is negatively related to academic performance for Australian boys but not girls. One explanation is that since gender and sexual equality has been a core value of Norwegian identity [25], the negative effects of social mechanisms related to stigma and discrimination around obesity may be less than in other societies. In addition, since boys tend to be associated with poorer academic performance and attitudes than girls [24], boys with overweight may experience "double jeopardy" due to their stigmatized identities and academic difficulties at school. It is also possible that the obesity penalty for girls may present later in adolescence, as children move to higher grades and participate in wider social interactions [11]. Further, obesity effects accumulate over time [4], and young children are generally less concerned about obesity than older children [42]. This may explain the observed stronger effects of overweight for boys in the later grade.

We also observed heterogeneous effects of childhood weight status across school subjects. A previous study reports similar correlations between BMI and test scores across different subjects [7]. In our study, we observed the significant relationships for reading and math but not English, regardless of gender and grade. Research suggests that the early stage is crucial in the development of foreign language skills [43]. Compared to other subjects, English skills developed early on may not be substantially affected by negative social interactions or handicaps in elementary schooling. However, we acknowledge that our findings may be specific

to Norwegian contexts, and further research is needed to explore the actual mechanisms of these observed findings.

4.1 Strengths and limitations

The key strength of our study is the use of a polygenic score for BMI as an IV. In particular, the rich MoBa data allowed us to employ a within-family genetic approach that shields against endogeneity due to family environmental effects, population stratification, and assortative mating. The MoBa is a national, population-based cohort study, and maternal BMI, test scores, basic demographic/SES characteristics were obtained from the Norwegian administrative registers. Further, we paid careful attention to potential heterogeneous effects of child weight status across different subjects as well as genders and grades. However, there are a few limitations that deserve mention. The MoBa may represent families that are above average in SES [32], and our analyses relied on a subsample of MoBa participants with European ancestries. A previous study also reports that the negative relationship between BMI and academic achievement is larger for American and European samples than Asian samples [7]. Therefore, our findings need to be carefully considered given the sample and national characteristics. Additionally, the potential mediating mechanisms of childhood obesity remain untested; further research is needed to explore the key pathways to develop more efficient intervention strategies.

4.2 Conclusion and implications

Evidence suggests that boys lag behind girls in school in terms of both academic performance and motivation [44]. Yet previous school-based obesity prevention strategies have mainly targeted girls [45, 46], based on the assumption that the obesity penalty is more severe for

girls. Our findings, however, indicate that boys' early-onset overweight may have profound impact on academic performance, which may in turn affect their long-term health and human capital outcomes. Childhood obesity is preventable and treatable, and interventions at different levels, including individual, school-based, and structural, have been shown to be effective (for a systematic review see Brown *et al.* [47]). Particular attention to boys with overweight may help to reduce the existing gender gap in academic achievement.

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Ethics Approval

The Norwegian Mother and Child Cohort Study has approvals from the Regional Ethics Committee and the Norwegian Data Inspectorate. All participants have provided signed informed consents.

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Supplemental Appendix A. Descriptive Statistics

	Full analytic sample					Genetic sample				
	N	Mean	SD	Min	Max	N	Mean	SD	Min	Max
Dependent variables										
English score 5th	38,278	.00	1.00	-2.80	2.14	12,745	.01	.99	-2.64	2.14
English score 8th	19,379	.00	1.00	-2.47	2.20	6,832	.00	1.00	-2.47	2.11
Math score 5th	38,518	.00	1.00	-3.19	2.13	12,827	.04	1.00	-3.19	2.13
Math score 8th	19,469	.00	1.00	-2.79	2.25	6,871	.03	1.00	-2.59	2.25
Reading score 5th	38,083	.00	1.00	-3.46	1.90	12,670	.02	1.00	-3.46	1.88
Reading score 8th	19,427	.00	1.00	-3.35	2.10	6,861	.01	1.00	-3.23	2.07
Independent variables										
BMI	35,162	16.25	2.08	6.63	33.77	11,741	16.24	2.04	7.34	32.65
Overweight/obesity	33,828	.13	.33	0	1	11,431	.13	.33	0	1
Instrumental variables										
Maternal BMI	39,977	23.88	4.10	13.21	59.16	13,382	23.94	4.09	13.21	54.08
BMI PRS	15,941	.00	1	-3.77	3.79	11,848	.00	1.00	-3.79	3.80
Confounders										
Maternal education	40,907	1.82	.76	0	3	13,589	1.83	.75	0	3
Paternal education	40,635	1.60	.89	0	3	13,543	1.64	.87	0	3
Family Income	41,074	719,953	297,729	0	9,452,458	13,631	724,524	285,468	0	7,363,115
Mother age	41,112	31.10	4.31	20	40	13,639	30.89	4.21	20	40
Father age	40,947	33.50	5.03	23	46	13,621	33.27	4.89	23	46
Number of children	40,749	2.40	.75	0	6	13,522	2.41	.73	0	6
Parent non-native speaker	41,113	.13	.33	0	1	13,639	.11	.32	0	1
Single mother	40,682	.13	.34	0	1	13,500	.12	.32	0	1
Gender (boy=1)	39,461	.51	.50	0	1	13,259	.52	.50	0	1
School grade	40,654	.60	.49	0	1	13,486	.59	.49	0	1
Child age (months)	41,145	97.55	1.51	91	104	13,648	97.55	1.46	92	103
Cohort (year)	41,145	2005.48	1.87	2002	2009	13,648	2005.42	1.87	2002	2009

Supplemental Appendix B. Instrumental variable analyses (Mendelian randomization in the context of this study)

To investigate the causal effects of childhood weight status on academic performance measured at 5th and 8th grade, we employed IV estimators. There are four assumptions for an instrumental variable analysis which must be met: First, the instrument should be highly correlated with the variable of interest (i.e., instrument relevance). Second, there are no unmeasured confounders between the instrument and outcome variable (i.e., independence assumption). Third, the instrumental variable should affect the dependent variable only through the predictor (i.e., exclusion restriction). The violation of the second and third assumptions implies that there are "backdoor paths" (dashed lines in Figure 1) from the instrument to the dependent variable. Lastly, it is assumed that individuals' response to the instrument is monotonic (i.e., monotonicity) (Von Hinke et al., 2016). In the following we discuss the rationale of our instrument variable approach in detail.

Instrument relevance. Our instrumental variables are PRS of BMI and mother's pre-pregnancy BMI. The weight of a biological relative (Averett & Stifel, 2010; Biener et al., 2020; Sabia & Rees, 2015; Scholder et al., 2012; Shi & Li, 2018) and BMI PRS or genetic risk score (Ding et al., 2009; Fletcher & Lehrer, 2011; Scholder et al., 2012; Von Hinke et al., 2016) have been widely employed as an instrument of child BMI related to educational outcomes. Those IV studies highlight the substantial genetic component in determining body weight. Indeed, our IVs have a strong explanatory power of childhood BMI and overweight, far exceeding the conventional norm of F=10 (Stock et al., 2002) (see also F-stat on 1st stage in Appendix D-F).

Independence and exclusion restriction assumptions. Previous studies using parents' BMI as IV have argued that a shared household environment has no detectable impacts on body weight (Black et al., 2018; Cawley & Meyerhoefer, 2012; Haberstick et al., 2010; Sabia & Rees, 2015; Shi & Li, 2018). Unobserved heterogeneity, however, may still be correlated with both a child's BMI and their relative's BMI (Böckerman et al., 2019). To strengthen the argument, those IV studies have employed various econometric techniques to account for potential unobserved confounding. That is, the previous IV studies rely on the conditional independence assumption that there are no unmeasured confounders after isolating the effects of key demographic and family factors. Yet, recent evidence from twin studies suggests that although shared environmental effects on BMI are no longer evident in late adolescence, environmental factors play certain roles in early childhood (e.g., Silventoinen et al., 2016). This implies that IV-BMI studies targeting infancy and early childhood may be more susceptible to the violation of independence and exclusion restriction assumptions.

More recently, PRS has been extensively employed as IV in investigating the relationship between various phenotypes and outcomes variables, which is called as Mendelian randomization in epidemiology (Raffington et al., 2020). The fact that PRS is a measure of an individuals' genetic predisposition to develop a certain phenotype (i.e., instrument relevance) and that genes are randomly assigned to offspring at conception (i.e., independence assumption) is compelling to many applied researchers who wish to find suitable IVs. Yet the effects of child PRS may capture non-genetic effects stemming from their parents. In our research framework, parents with obesity alleles may be more discriminated in the labor market or might be prone to unhealthy nutritional environment at home, which may in turn affect child educational opportunities (Milliken-Smith & Potter, 2021). To address this concern, we take the advantage of

MoBa data including parental genotype. Since genes are randomly assigned given parental genes, within-family approaches conditioning on parental genotype may provide robust estimates against such familial effects. Although the benefits of family-based study designs have been highlighted, it has been rarely exploited in IV studies (Davies et al., 2019). Here we utilized the residuals after the effects of parental PRS are regressed out from child PRS to account for potential unobserved confounding resulting from the correlation between child PRS and parental genotype (see similar approach in Cheesman et al. 2022).

Still, as most genetic loci tend also to be associated with other genetic traits (known as pleitropy e.g., BMI PRS may affect academic achievement via other genes), it is difficult to rule out the possibility of the violation of independence and exclusion restriction assumptions (Koellinger & De Vlaming, 2019). Thus, IV-PRS studies also have partially relied on the conditional independence assumption that after conditioning on child and family characteristics, PRS per se may not affect child academic performance via other channels. Unfortunately, in observational studies the violation of exclusion restriction or no unmeasured confounding assumption cannot be directly tested. To address the likely influences of unobserved confounding, we applied a recently developed sensitivity analysis to examine the robustness of our findings against potential unobserved confounding (see Appendix C).

Monotonicity. Lastly, in our research framework monotonicity means that individuals with more BMI related genes should have a higher (or lower) BMI compared to their counterparts (i.e., no defiers assumption). Although the monotonicity assumption is usually untestable, since genes are randomly assigned and individuals are not aware of their genotype, it is plausible to predict that they may not act to violate the assumption based on the knowledge of

their genes (Amin et al., 2020). There is, however, a possibility that visible genetic markers such as parents' BMI may not meet such assumption. For example, although children from obese families tend to prefer fatty foods and sedentary activities, they may desire to be thin to avoid obesity stigma and discrimination. Further, when gene-environment interactions are present, this assumption may be violated (Burgess & Thompson, 2015). Yet previous studies show that there is no strong or consistent evidence on gene-environmental interaction effects for BMI (Von Hinke et al., 2016; Yılmaz & Karadağ, 2021). As a supplemental analysis, following Von Hinke et al. (2016), we explored whether the associations between our IVs and child BMI significantly differ across demographic characteristics (see Appendix H). The results showed that although there are some variations, the observed overall patterns are similar thus providing little evidence on gene-environment interactions.[1]

In sum, we have a degree of confidence in the assumptions of instrumental relevance and monotonicity for our BMI PRS. Regarding maternal BMI, we are somewhat skeptical about the satisfaction of monotonicity assumption, due to its visible characteristics that may cause social interactions. As such, the estimates obtained from IV-BMI specification may be different from the local average treatment effects in IV analysis, and also be biased depending on the difference

[1] When the monotonicity assumption is met, an estimate in IV analysis is a local average treatment effect (LATE); that is, the average treatment effect for the compliers whose BMI are only affected by IVs. As such, our estimates may not be informative, since the LATE is instrument-specific, and it is difficult to identify the compliers. Yet, Dixon et al. (2020) argued that since BMI is continuous, the LATE may reflect the effects of the SNPs across the whole distribution of BMI. The estimates obtained from our models with BMI predictor may also be considered as the average treatment effect for the population. We also found sizeable effects of BMI predictor for boys' reading achievement. For example, the effect of boys' BMI on standardized reading achievement at grade 8 is –.17 (.08) (from 2SLS with IV-PRS in Appendix F), and in MoBa the mean and SD of BMI for boys are 16.21 and 2.05, respectively. Thus, the reading achievement gap between boys with obesity at 2 SD of BMI (20.31) and average boys is about .70 of a SD (4.1*–.17).

between the complier and defier average treatment effects (Huntington-Klein, 2020). The BMI PRS is thus the preferred instrument in our study, due to the random nature of genetic inheritance within families.

Supplemental Appendix C. Sensitivity test

Even if our IVs have a strong instrumental relevance, we cannot rule out the possibility of unobserved confounding or pleitropy. To test the robustness of our findings, we conducted sensitivity analyses in IV models suggested by Kang et al. (2020):

$$X_i = \gamma_0 + \delta\gamma_1 Z_i + \eta_i$$

$$Y_i = \beta_0 + \beta_1 X'_i + \varepsilon_i,$$

where Y_i represents the national test scores (reading, math, and English) of a child i; X_i represents the variable of interest (here child weight status); ε_i is the error term in the equation; Z_i represents the instrumental variables; and η_i is the error term in that equation. Based on the proposition, we assume that Z_i is independent of ε_i and η_i. Yet, under certain conditions, this assumption may be violated. δ captures such possibility that our instruments are invalid in the range of δ. It shows how much a unit change in the invalid instrument will affect Y_i, either through a direct effect of Z_i on Y_i or through correlation of Z_i with omitted determinants of Y_i. In testing the sensitivity of our IV specification, we begin with a correlation of .05 which is a similar size of the correlation between PRS and single family or maternal age among potential confounders.

We present the results for the sensitivity analyses in each table in Appendix D-F. The sensitivity analysis tests the null hypothesis of no effect due to unmeasured confounder that is correlated both with the IV and test scores at around δ in the IV equation. The estimated coefficients with $p < .05$ from the sensitivity analysis suggest that BMI and overweight effects are robust to unobserved confounding, which has a direct causal effect through IV to test scores

or through correlation of IV with omitted determinants of test scores. The results showed that the observed significant effects of childhood weight status on test scores are in general robust to unobserved confounders. Further, we increased the range of δ in the IV equation to examine the degree of robustness of our findings against unobserved confounding. In the sense that unobserved confounding presents as strongly as parental education effects, the significant effects observed were no longer evident in many cases. We think this is likely to be an uncommon situation in elementary schooling: unobserved confounding effects comparable to parental education above and beyond the controlled child and family characteristics as well as parental PRS are very unlikely to occur.

Supplemental Appendix D. Results for Total Sample

Total	Outcomes								
	Reading			Math			English		
Predictor	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)
5th grade									
BMI	.00	–.07**	–.09*	–.00	–.08***	–.07*	.00	–.03	.01
	(.00)	(.02)	(.03)	(.00)	(.02)	(.03)	(.00)	(.02)	(.03)
F-stat on 1st stage		414.97	194.46		440.27	186.79		424.59	185.86
Sensitivity test (p-value)		.02	.01		.00	.05		.40	.84
N	10,291	10,116	8,974	10,406	10,229	9,062	10,342	10,166	9,014
Overweight	–.03	–.59***	–.72**	–.06*	–.72***	–.50	–.05	–.31	–.02
	(.03)	(.17)	(.26)	(.03)	(.17)	(.26)	(.03)	(.16)	(.26)
F-stat on 1st stage		222.34	115.11		222.72	109.03		220.51	110.28
Sensitivity test (p-value)		.01	.01		.00	.07		.23	.99
N	9,089	8,934	7,903	9,182	9,025	7,971	9,132	8,976	7,935
8th grade									
BMI	–.01	–.10***	–.07	–.00	–.07**	–.07	.00	–.04	.03
	(.01)	(.02)	(.05)	(.01)	(.02)	(.05)	(.01)	(.02)	(.05)
F-stat on 1st stage		248.32	83.94		256.25	84.76		253.20	84.84
Sensitivity test (p-value)		.00	.13		.02	.18		.21	.49
N	5,609	5,497	4,659	5,623	5,512	4,675	5,591	5,478	4,644
Overweight	–.07	–.84***	–.50	–.06	–.61**	–.47	–.03	–.40	.35
	(.04)	(.22)	(.38)	(.04)	(.21)	(.38)	(.04)	(.21)	(.38)
F-stat on 1st stage		120.97	50.06		127.41	49.36		125.79	50.74
Sensitivity test (p-value)		.00	.20		.01	.22		.14	.37
N	4,973	4,875	4,114	4,984	4,886	4,126	4,957	4,858	4,100

** p<.05, ** p<.01, ***<.001 Note*: Robust standard errors are in parentheses.

Supplemental Appendix E. Results for Girls

Total	Outcomes								
	Reading			Math			English		
Predictor	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)
5th grade									
BMI	.00	–.06*	–.08	.00	–.07**	–.03	.00	–.03	.01
	(.01)	(.02)	(.04)	(.01)	(.02)	(.04)	(.01)	(.02)	(.04)
F-stat on 1st stage		206.88	102.51		218.55	100.48		210.76	99.69
Sensitivity test (p-value)		.04	.09		.01	.60		.33	.57
N	5,008	4,938	4,411	5,053	4,981	4,450	5,036	4,965	4,436
Overweight	–.06	–.48*	–.38	–.06	–.54**	–.13	–.08*	–.27	.10
	(.04)	(.20)	(.33)	(.04)	(.20)	(.32)	(.04)	(.20)	(.34)
F-stat on 1st stage		127.54	58.88		125.28	55.24		126.63	56.39
Sensitivity test (p-value)		.04	.28		.03	.81		.32	.63
N	4,428	4,366	3,894	4,465	4,401	3,923	4,448	4,385	3,911
8th grade									
BMI	.00	–.06*	.02	.01	–.04	–.00	.02	–.04	.08
	(.01)	(.03)	(.06)	(.01)	(.03)	(.06)	(.01)	(.03)	(.07)
F-stat on 1st stage		135.74	46.77		136.46	46.27		136.53	44.55
Sensitivity test (p-value)		.06	.76		.22	.94		.25	.19
N	2,722	2,676	2,303	2,730	2,684	2,315	2,722	2,676	2,302
Overweight	–.04	–.43	.57	–.03	–.26	.13	.01	–.35	.92
	(.05)	(.25)	(.50)	(.05)	(.25)	(.52)	(.05)	(.26)	(.56)
F-stat on 1st stage		76.04	25.77		79.47	24.05		78.43	24.45
Sensitivity test (p-value)		.13	.25		.35	.80		.26	.08
N	2,409	2,369	2,033	2,416	2,376	2,044	2,410	2,370	2,033

** p<.05, ** p<.01, ***<.001 Note*: Robust standard errors are in parentheses.

Supplemental Appendix F. Results for Boys

Total	Outcomes								
	Reading			Math			English		
Predictor	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)
5th grade									
BMI	.00	–.07*	–.10	–.00	–.10**	–.11*	–.00	–.02	–.01
	(.01)	(.03)	(.05)	(.01)	(.03)	(.05)	(.01)	(.03)	(.05)
F-stat on 1st stage		211.28	93.84		225.05	88.76		217.04	88.29
Sensitivity test (p-value)		.06	.06		.01	.03		.61	.80
N	5,283	5,178	4,563	5,353	5,248	4,612	5,306	5,201	4,578
Overweight	.01	–.72*	–1.12**	–.06	–.92**	–.89*	–.02	–.34	–.20
	(.04)	(.28)	(.42)	(.05)	(.28)	(.41)	(.05)	(.27)	(.39)
F-stat on 1st stage		94.57	55.68		97.20	53.79		93.47	53.64
Sensitivity test (p-value)		.03	.00		.00	.02		.34	.60
N	4,661	4,568	4,009	4,717	4,624	4,048	4,684	4,591	4,024
8th grade									
BMI	–.02	–.15***	–.17*	–.01	–.11**	–.13	–.01	–.05	–.02
	(.01)	(.04)	(.08)	(.01)	(.04)	(.08)	(.01)	(.04)	(.07)
F-stat on 1st stage		115.60	37.03		122.72	38.19		118.64	39.83
Sensitivity test (p-value)		.00	.03		.01	.10		.35	.80
N	2,887	2,821	2,356	2,893	2,828	2,360	2,869	2,802	2,342
Overweight	–.09	–1.36**	–1.71*	–.09	–1.07**	–1.08	–.07	–.52	–.27
	(.06)	(.40)	(.68)	(.06)	(.37)	(.60)	(.06)	(.35)	(.55)
F-stat on 1st stage		47.51	22.67		50.75	23.60		49.62	24.71
Sensitivity test (p-value)		.00	.01		.01	.06		.23	.66
N	2,564	2,506	2,081	2,568	2,510	2,082	2,547	2,488	2,067

* *p<.05, ** p<.01, ***<.001 Note:* Robust standard errors are in parentheses.

Supplemental Appendix G. Results for Girls and Boys (fitted value in the first stage as IV)

		Outcomes								
		Reading			Math			English		
	Predictor	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)	OLS	2SLS (BMI)	2SLS (PRS)
5th grade										
Girls	Overweight	–.06	–.54**	–.44	–.06	–.47*	.07	–.08*	–.30	.08
		(.04)	(.21)	(.35)	(.04)	(.20)	(.34)	(.04)	(.21)	(.35)
	F-stat on 1st stage		112.10	48.75		110.55	45.75		111.62	47.16
	N	4,428	4,366	3,894	4,465	4,401	3,923	4,448	4,385	3,911
	Overweight	.02	–.60*	–.75*	–.06	–.82**	–.78*	–.02	–.35	–.04
		(.04)	(.30)	(.37)	(.05)	(.31)	(.39)	(.05)	(.29)	(.37)
Boys	F-stat on 1st stage		67.77	53.97		68.65	51.76		66.37	50.97
	N	4,661	4,568	4,009	4,717	4,624	4,048	4,684	4,591	4,024
8th grade										
Girls	Overweight	–.05	–.49	.55	–.03	–.20	.19	.01	–.41	.77
		(.05)	(.26)	(.54)	(.05)	(.26)	(.56)	(.05)	(.27)	(.59)
	F-stat on 1st stage		66.49	19.49		69.67	17.50		68.95	18.27
	N	2,409	2,369	2,033	2,416	2,376	2,044	2,410	2,370	2,033
	Overweight	–.09	–1.54**	–1.39*	–.09	–1.16**	–.78	–.07	–.81	–.04
		(.06)	(.46)	(.63)	(.06)	(.43)	(.56)	(.06)	(.42)	(.51)
Boys	F-stat on 1st stage		31.26	21.08		33.74	22.39		33.49	23.89
	N	2,564	2,506	2,081	2,568	2,510	2,082	2,547	2,488	2,067

** p<.05, ** p<.01, ***<.001 Note*: Robust standard errors are in parentheses

Supplemental Appendix H. Gene and Environment Interactions

Instruments	(1) Gender Boys (=1) Girls (=2)	(2) Grade G2 (=1) G3 (=2)	(3) Father's education Middle (=1) High (=2) BA (=3) MA/PhD (=4)	(4) Mother's education Middle (=1) High (=2) BA (=3) MA/PhD (=4)	(5) Immigrant background Non (=1) Immigrant (=2)	(6) Single family Non (=1) Single (=2)
BMI PRS, group 1	.28***	.32***	.39***	.40**	.32***	.32***
	(.03)	(.03)	(.08)	(.12)	(.02)	(.02)
BMI PRS, group 2	.34***	.31***	.30***	.33***	.22***	.24**
	(.03)	(.03)	(.03)	(.05)	(.06)	(.07)
BMI PRS, group 3			.30***	.29***		
			(.03)	(.03)		
BMI PRS, group 4			.28***	.28***		
			(.04)	(.04)		
p-value testing equality	.15	.67	.63	.63	.10	.26
Maternal BMI, group 1	.48***	.51***	.52***	.46***	.51***	.52***
	(.02)	(.03)	(.06)	(.09)	(.02)	(.02)
Maternal BMI, group 2	.56***	.51***	.47***	.44***	.50***	.42***
	(.03)	(.02)	(.03)	(.04)	(.06)	(.06)
Maternal BMI, group 3			.54***	.54***		
			(.03)	(.03)		
Maternal BMI, group 4			.53***	.49***		
			(.05)	(.05)		
p-value testing equality	.11	.99	.58	.27	.92	.14

Note: The estimates in each column are obtained from one regression with the ancestry principal components. It shows whether the association between instrument (BMI PRS and maternal BMI) and childhood BMI significantly differs by categorical covariates. A group in each row represents subgroups of each covariate. The "p-value testing equality" is the p-value of a chi-squared testing whether the coefficient on the different groups are significantly different from each other.

Supplemental Appendix I. Results for Girls and Boys controlled for Personality (i.e., Big Five traits)

		Reading		Math		English	
Predictor		2SLS (BMI)	2SLS (PRS)	2SLS (BMI)	2SLS (PRS)	2SLS (BMI)	2SLS (PRS)
5th grade							
Girls	BMI	–.06*	–.08	–.06**	–.03	–.03	.01
		(.02)	(.04)	(.02)	(.04)	(.02)	(.04)
	F-stat on 1st stage	207.52	102.78	219.28	100.24	211.18	99.39
	N	4,936	4,409	4,979	4,448	4,963	4,434
	Overweight	–.48*	–.41	–.48*	–.11	–.26	.08
		(.20)	(.33)	(.20)	(.32)	(.20)	(.33)
	F-stat on 1st stage	127.61	58.75	125.32	55.05	126.50	55.98
	N	4,365	3,893	4,400	3,922	4,384	3,910
Boys	BMI	–.07	–.09	–.09**	–.09	–.02	–.01
		(.03)	(.05)	(.03)	(.05)	(.03)	(05)
	F-stat on 1st stage	211.25	92.74	225.78	87.52	218.25	87.29
	N	5,174	4,560	5,244	4,609	5,197	4,575
	Overweight	–.66*	–1.09**	–.81**	–.82*	–.35	–.20
		(.28)	(.41)	(.28)	(.40)	(.27)	(.39)
	F-stat on 1st stage	93.68	54.84	96.67	53.02	92.95	52.91
	N	4,566	4,008	4,622	4,047	4,589	4,023
8th grade							
Girls	BMI	–.05	.02	–.03	–.01	–.03	.08
		(.03)	(.06)	(.03)	(.06)	(.03)	(.06)
	F-stat on 1st stage	133.39	46.84	133.74	46.09	134.14	44.66
	N	2,675	2,302	2,683	2,314	2,675	2,301
	Overweight	–.37	.52	–.20	.05	–.33	.80
		(.24)	(.48)	(.25)	(.50)	(.26)	(.54)
	F-stat on 1st stage	76.53	25.95	80.06	24.27	78.96	24.64
	N	2.369	2,033	2,376	2,044	2,370	2,033
Boys	BMI	–.15***	–.15	–.11**	–.09	–.06	–.00
		(.04)	(.08)	(.04)	(.07)	(.04)	(.07)
	F-stat on 1st stage	115.56	37.76	123.20	38.64	118.89	40.16
	N	2,819	2,354	2,827	2,359	2,801	2,341
	Overweight	–1.35**	–1.51*	–1.03**	–.90	–.55	–.19
		(.39)	(.64)	(.36)	(.57)	(.34)	(.53)
	F-stat on 1st stage	47.96	23.23	51.35	23.88	50.14	25.19
	N	2,505	2,080	2,510	2,082	2,488	2,067

** p<.05, ** p<.01, ***<.001 Note:* Robust standard errors are in parentheses. Short Norwegian Hierarchical Personality Inventory for the Assessment of Personality in Children are used. These are extraversion (e.g., “talk to people easily.”), benevolence (e.g., “takes himself/herself into consideration first.”), conscientiousness (e.g., “carries out work to the last detail.”), neuroticism (e.g., “doubt himself/herself.”), and imagination (“has a broad range of interests.”) that are measured by six items respectively asking “how well do these statements apply to your child’s behaviors over the past year?.

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