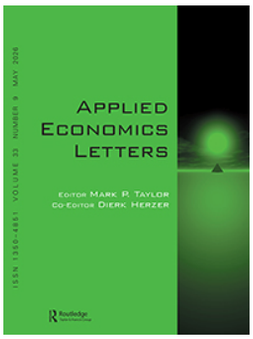

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Childhood adiposity and labor market outcomes: mendelian randomization evidence on the role of education

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ABSTRACT

We investigate whether childhood adiposity affects adulthood earnings via education, using Finnish genotyped data and a causal mediation design. A one unit increase in childhood BMI, which corresponds to transitioning from normal weight to overweight, or from overweight to obesity, reduces adult earnings by approximately 23% through lower educational attainment. Our results further suggest that early-life adiposity has long-term economic consequences that are independent of adult weight.

KEYWORDS

Mediation; childhood body mass index; educational attainment; adult earnings; polygenic risk score

JEL CLASSIFICATION

I12; I21; I26

I. Introduction

Economics literature has identified several mechanisms linking childhood body weight to human capital (e.g. Segal et al. 2021) and mechanisms linking human capital to earnings (e.g. Clark and Nielsen 2026). Overweight children face a higher risk of health problems and obesity-related diseases that may increase school absences and impair cognitive functioning, thereby reducing performance required for educational attainment (e.g. Cawley 2015). Social stigma may further weaken self-confidence and lower aspirations for post-compulsory schooling (e.g. Sarzosa, Carvalho, and Turley 2021). Human capital affects earnings through cognitive or social skills (e.g. Buser et al. 2024), signalling and networks (e.g. Tyler, Murnane, and Willett 2000), and technological complementarities (e.g. Autor et al. 2003). Lower levels of human capital in adolescence may also increase the risk of adulthood obesity (e.g. Khera et al. 2019), thereby reducing opportunities to higher earnings.

This study examines whether childhood body weight index (BMI) affects adult earnings through human capital. We contribute to the economics literature in two ways. First, to obtain causal evidence on mediated effects, we use genotyped data and multivariable Mendelian randomization (MVMR) design. Second, we provide policy-relevant insight into the timing and type of interventions, as our results indicate that early-life health promotion interventions could serve as a potential alternative to educational interventions introduced at later stages in shaping long-term socioeconomic outcomes.

II. Methods and data

Methods

We use genetic instruments for childhood BMI ($\hat{X}$) and educational attainment ($\hat{M}$) to estimate their causal effects on adult earnings. Two-stage least-squares models identify the effect of BMI on education (α_M) and the effect of education on earnings (β_M). We estimate:

$$M_i = \alpha_1 + \alpha_M \hat{X}_i + \alpha' Z_i + \nu_1 \quad (1)$$

and

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$$Y_i = \beta_1 + \beta_M \widehat{M_i} + \beta' Z_i + \nu_2 \quad (2)$$

To estimate the indirect effect of BMI via education, we apply a multivariable MVMR model:

$$Y_i = \pi_0 + \pi_1 \hat{X}_i + \pi_M \hat{M}_i + \pi' Z_i + \nu_3 \quad (3)$$

The mediated effect is calculated as the product of α_M and π_M, with standard errors obtained via bootstrapping. Under certain identifying assumptions, MVMR identifies the causal link between childhood BMI and earnings operating through education. The methodology, additional references, and approaches to address potential assumption violations are described in Online Appendix 2.

Data

The Cardiovascular Risk in Young Finns Study (YFS; (https://youngfinnsstudy.utu.fi/) is a population-based longitudinal study that commenced in 1980. It consists of randomly chosen boys and girls ($N = 3{,}596$) from six age cohorts (Raitakari et al. 2008). In 2009, 2,556 YFS participants were genotyped. In 2014, the YFS data were linked to administrative records from Statistical Finland. Table 1 and Online Appendix 1 report summary statistics and the sample flowchart.

Variables

Childhood BMI

Weight and height were measured by trained nurses. To ensure comparability across ages and developmental stages, we averaged age- and sex-specific BMI z-scores calculated from measurements taken at ages 6, 9, and 12.

Table 1. Summary statistics.

	Mean	Standard deviation
BMI z-score	0.091	0.997
Years of education in 2007	14.01	2.751
Ln average earnings 2007–2019	7.111	1.735
Ln earnings in 2015	3.773	2.839
Grade point average (GPA) at age of 15	7.847	0.891
Female (share)	0.556	0.497
Age in 2007	34.63	3.335
High family education in 1985 (share)	0.146	0.353
Mother with high education in 1985 ($N = 797$) (share)	0.109	0.312
Father with high education in 1985 ($N = 797$) (share)	0.149	0.357
Mother's BMI in 1980 ($N = 797$)	23.28	3.545
Father's BMI 1980 ($N = 797$)	25.12	2.980

$N = 1{,}261$ unless otherwise stated. The GPA varies from 4 (the lowest score) to 10 (the highest score). The high family education variable equals 1 if at least one parent had completed higher education by 1985.

Education

Educational attainment was measured as completed years of schooling in 2007, when the youngest cohort was 30 years old. As an alternative mediator, we used grade point average (GPA) at age 15, measured at the end of compulsory schooling.

Earnings

Earnings were measured over a 12-year period (2007–2019), when participants were aged between 30 and 51. As an alternative outcome, we used earnings in 2015, when the average participant age was 42.

Instruments

For BMI, we used a polygenic risk score (PGS) based on single nucleotide polymorphisms (SNPs) associated with BMI at $p < .01$ in the GWAS of Locke et al. (2015). For education, we used SNPs associated with years of schooling at $p < .01$ in Okbay et al. (2016).

Variables addressing potential confounding and selection

First 10 principal components; Maternal and paternal BMI and education, and interactions between maternal and paternal variables, parental education coded 1 if at least one parent had completed a university degree by 1985; PGSs for type 2 diabetes, fasting insulin, total cholesterol, childhood IQ, autism, ADHD, and puberty timing.

III. Results on mediation through education

The results from Equations 1 and 2 show that a higher childhood BMI is negatively linked to educational attainment ($\alpha_M = -0.879$; $p < .01$). Including parental covariates and additional PGSs does not meaningfully alter this estimate ($\alpha_M = -0.946$; $p < .05$). Educational attainment is positively linked to earnings ($\beta_M = 0.261$; $p < .01$), and this estimate remains stable after including covariates ($\beta_M = 0.218$; $p < .01$). Instruments exceed or approach the conservative cut-off of

Table 2. Mediated effect of childhood BMI on adult earnings through education.

Specification	Model 1 Baseline	Model 2 Education measured by GPA	Model 3 Earnings measured in 2015	Model 4 Adult BMI controlled	Model 5 Additional family controls
Mediated effect	−0.235**	−0.215**	−0.322**	−0.283	−0.175
	(0.097)	(0.097)	(0.134)	(0.278)	(0.119)
	[0.016]	[0.026]	[0.016]	[0.310]	[0.140]
First-stage F-Stat.					
BMI	69.9	69.3	69.9	10.7	24.0
Education	52.5	46.3	52.5	46.4	27.6
First-stage S-W-Stat.					
BMI	117.4	118.1	117.4	20.9	47.1
Education	89.6	81.2	89.6	86.1	54.6
No of Obs.	1,261	1,261	1,261	1,261	797
Mean ln Earnings	7.111	7.111	3.773	7.111	7.252

The table reports coefficients, standard errors, and *p*-values. Significance at the ***$p < .01$, **$p < .05$, and *$p < .10$ levels. S-W refers to the Sanderson-Windmeijer multivariate F-test. Earnings were log-transformed. All models control for sex, birth cohort, birth month, region of residence in 1980, 10 principal components, parental education (equals 1 if at least one parent has high education), PGSs for type 2 diabetes, fasting insulin, total cholesterol, childhood IQ, autism, ADHD, and Tanner puberty scale. Model 2 includes an indicator, which equals one if the information on GPA was a self-reported measure. Model 3 measures earnings at the average age of 42. Model 4 uses BMI data from 2007, or from 2001 if 2007 data is missing. Model 5 includes indicators for parental BMI, parental education, and their interactions.

100 in the baseline model ($N = 1271$; F = 134.4 for BMI and 95.5 for education) and remain strong in the augmented model ($N = 797$; F = 44.9 and 50.9).

MVMR estimates (Table 2) show that the genetic instruments are strong (F = 69.9 for BMI and 52.5 for education) and their strength increase with MVMR (S-W F-test is 117.4 and 89.6). The mediated effect through education is statistically significant ($\beta = -0.235$; $p = .016$) indicating that a one unit increase in childhood BMI, equivalent to moving from normal weight to overweight or from overweight to obesity, reduces earnings by about 23% via lower educational attainment.[1]

The estimate is robust when GPA is used as a mediator (Model 2: $\beta = -0.215$; $p = .026$) and increases in magnitude when earnings are measured cross-sectionally (Model 3: $\beta = -0.322$; $p = .016$). Including adult BMI leaves the effect unchanged although the estimate becomes statistically insignificant (Model 4: $\beta = -0.283$; $p = .310$). The result potentially reflects diminished instrument strength due to the high correlation between childhood and adult BMI ($r = .55$) rather than mediation through adult BMI. Adding familial controls yields a lower estimate (Model 5: $\beta = -0.175$; $p = 0.140$). The baseline model, estimated on the same smaller sample, yields a similar estimate ($\beta = -0.188$; $p = 0.048$). This implies that the insignificant estimate for Model 5 reflects reduced residual variation rather than a substantive change in the effect. MVMR results by sex are presented in Appendix 4. Although the estimates differ somewhat by sex, the limited statistical power means these results should be interpreted as suggestive.

IV. Conclusions

We employ genetic instruments to estimate the mediated effect of childhood BMI on adult earnings through education. To reduce the possibility that instruments affect earnings through alternative pathways, we conditioned on parental characteristics and genetic variants associated with metabolic diseases, cognitive skills, and neuropsychiatric traits. A limitation of our study is that the BMI and education PGSs are older versions, and the latest ones are unavailable.

Our analysis identifies two causal pathways: from childhood BMI to education and from education to adult earnings. The MVMR analysis shows that a one unit increase in childhood BMI translates into a 23% reduction in earnings through reduced educational attainment. This corresponds to a change from the upper end of normal weight to the upper end of overweight, or from the lower end of overweight to obesity. We also show that adulthood BMI does not mediate this relationship.

Our findings align with earlier evidence showing long-term consequences of childhood growth pat-

[1]Online Appendices 3 and 4 report causal estimates from Equations 1–3 and baseline model results by sex (Table 2), respectively.

terns (Buser et al. 2024; Davies et al. 2023; Fan, Yanhong, and Zhang 2023) and provide policy-relevant insight into intervention timing. The results suggest that childhood BMI influences educational outcomes, indicating that early-life health promotion interventions could be an alternative to educational interventions at later ages for promoting children's long-term socioeconomic outcomes. Robustness of the evidence could be further assessed using alternative PGSs (e.g. Lee et al. 2018; Yengo et al. 2018).

Author contributions

CRediT: **Jaakko Pehkonen:** Conceptualization, Investigation, Methodology, Supervision, Writing – original draft, Writing – review & editing; **Jutta Viinikainen:** Conceptualization, Formal analysis, Investigation, Writing – review & editing; **Jaana T. Kari:** Data curation, Formal analysis, Writing – review & editing; **Petri Böckerman:** Conceptualization, Investigation, Methodology, Writing – review & editing; **Terho Lehtimäki:** Data curation, Methodology; **Olli Raitakari:** Conceptualization, Data curation, Supervision, Writing – review & editing.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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