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Article

Intergenerational Transmission of BMI and Educational Outcomes in Children and Adolescents

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Abstract

Individual differences in educational attainment (EA) and physical health, as indexed by body mass index (BMI), are correlated within individuals and across generations. The aim of our study was to assess the transmission of these traits from parents to their offspring in childhood and adolescence. We analyzed BMI and EA in 13,916 families from the Netherlands. Data were available for 27,577 parents (mean age 33) and 26,855 of their offspring at 4 and 12 years of age. We employed structural equation modeling to simultaneously estimate the phenotypic transmission of BMI and EA from parents to offspring, the spousal correlations, and the residual child BMI-EA associations after accounting for intergenerational transmission and testing for gender differences therein. We found a significant intergenerational transmission of BMI to BMI in childhood (age 4; standardized regression coefficient $\beta = .10$) and adolescence (age 12; $\beta = .20$), and of EA to academic achievement in adolescence ($\beta = .19$). Cross-trait parent-to-offspring transmission was weak. All transmission effects were independent of parent or offspring gender. We observed within-person EA-BMI correlations that were negative in parents ($\sim -.09$), positive in children ($\sim .05$) and negative in adolescents ($\sim -.06$). Residual EA-BMI were positive in children ($\sim .05$) and insignificant in adolescents. Spousal correlations were .46 for EA, .21 for BMI, and $\sim -.09$ cross-trait. After accounting for spousal correlations, the intergenerational transmission for BMI and EA is mainly predictive within, but not across, traits. The within-person correlation between BMI and EA can change in direction between childhood and adulthood.

Keywords: Parent-offspring transmission; cross-trait transmission; spouse correlation; structural equation modeling; childhood

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Childhood obesity is a growing problem worldwide, with adverse effects on virtually all organ systems, and serious short- and long-term sequelae (Daniels, 2009). The prevalence of overweight (including obesity) in children and adolescents — defined as body mass index (BMI) +1 standard deviations above the median — in the Netherlands is estimated to be slightly lower than the average for European countries. In the Netherlands, the World Health Organization estimates the prevalence at 26.9% in children aged 5–9 years and 23.7% in children aged 10–19 years, whereas the averages for European countries are 28.9% and 24.3% respectively. These rates vary across countries and regions, where, for example, in the United States the prevalence is 43% in 5- to 9-year-olds and 41.2% in 10- to 19-year-olds, with corresponding rates of 10.3% and 8.1% in South-East Asia. Globally, overweight prevalence is 20.9% in 5- to 9-year-olds and 17.3% in 10- to 19-year-olds (World Health Organization, 2017).

Within countries, the most important predictor of a child's BMI is the BMI of their parents (Butler et al., 2018; Vos & Welsh, 2010). In addition, low socioeconomic status (SES) (Newton et al., 2017) and lower educational attainment (EA) (Benson et al., 2018) are strong predictors, with overweight and obesity tending to be more prevalent in lower SES and EA. For example, in the Netherlands, the relative risk of overweight and obesity is twice as high for children with low maternal education and low household income (White et al., 2022).

While the association of parental EA and offspring BMI is well established (Benson et al., 2018; Lamerz et al., 2005; Matthiessen et al., 2014; Silventoinen et al., 2019), the etiology of the association is not. One the one hand, higher EA leads to better occupation and higher income and SES, and higher income enables individuals to afford food with higher nutritional value and lower caloric density (Darmon & Drewnowski, 2015; Drewnowski, 2010). On the other hand, Hanson and Conner (2014) showed that parents provide their offspring with food of high nutritional value regardless of income level. In addition to the possible role of nutrition, higher income is associated with many other factors; for example, it is associated with breastfeeding (Bartels et al., 2009) and may facilitate physical activity in children, insofar as such activity involves cost (e.g., club memberships, sportswear and material; Telford et al., 2016).

Many studies have established significant associations between parental SES and EA and offspring BMI, without controlling for

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parental BMI (Kleiser et al., 2009; Murasko, 2009; Nogueira et al., 2013; O'Dea & Dibley, 2010; Springer et al., 2015; Wang, 2001; Wang & Zhang, 2006). In a study of adult twins and their parents, we found a low negative, but statistically significant, correlation of −.06 between parental EA and offspring BMI. However, this correlation was not statistically significant, once we controlled for parental BMI (Alrouh et al., 2022).

SES is not the only possible pathway linking EA and obesity, as research has shown a link between obesity and cognitive performance, controlling for parental social class (Sørensen et al., 1983). Behavioral factors, such as self-control and delayed gratification, are associated with both EA (Golsteyn et al., 2014; Lee et al., 2012; Mischel et al., 1989) and with obesity (Caleza et al., 2016; Stoklosa et al., 2018). Decreased cognitive function is linked to impaired metabolic pathways, such as insulin signaling (Craft & Stennis Watson, 2004) and leptin regulation (Harvey, 2007), which are associated with obesity. Finally, obesity, as reflected by BMI, has significant genetic overlap with brain and cognitive measures (Vainik et al., 2018) and with EA (Li et al., 2021).

Parental BMI is the strongest predictor of offspring BMI. In a recent family-based study of Dutch adult twins, we found a parent-offspring BMI correlation of .20 (Alrouh et al., 2022). This finding is in line with meta-analytic results, which showed a mean parent-offspring correlation of .19 for BMI (Maes et al., 1997). The association is also manifest in terms of odds ratio: children with a single obese parent have an odds ratio of 3.49 for childhood obesity (Wang et al., 2017).

Prior studies generally examined the effects of parental EA and SES on offspring BMI without accounting for parental BMI, or the correlation between parental EA or SES and parental BMI. Therefore, it is possible that the finding that children who grow up in low EA households are more susceptible to obesity may be confounded by parental BMI. Indeed, we found this to be the case, when examining BMI of adult twins in the Dutch population (Alrouh et al., 2022). In the current study, we return to this possibility by now examining these pathways to offspring BMI at ages 4 and 12 years in a sample of young twins and their parents from the Dutch population and, at age 12, also include academic achievement in children. In short, we studied the within-trait and between-trait transmission of BMI and EA across generations, while controlling for the spousal correlations among BMI and EA.

Methods

Participants

Participants were registered with the Netherlands Twin Register (NTR). Since 1986, the NTR has approached parents of newborn twins born in the Netherlands in collaboration with a commercial 'birth felicitation' service. Additionally, recruitment is done with the support of the Dutch Society of Parents of Multiples (NVOM; https://www.nvom.nl). After obtaining informed consent, the family is registered with the NTR, and the parents receive a first survey on pre- and perinatal variables relating to their twins, as well as variables concerning the parent, including their height and weight. Subsequently, the family receives periodical surveys on a broad set of variables, including health variables and when the twins' developmental milestones were achieved. The NTR has registered about 52% of all Dutch twin-pairs born between 1987 and 2017 (Ligthart et al., 2019). For the present study, we selected twins and their parents who had completed surveys when their twins were age 5 and 12 years (referred to as survey 5 and survey 12). We excluded families in which zygosity of twins could not be determined (67 families), resulting in a sample of 54,432 participants. The sample comprises a total of 13,916 families, which includes 26,855 twins (13,347 males and 13,508 females) and 27,577 parents (13,739 fathers and 13,838 mothers), aged between 18 and 65 years at the time of birth of twins. The data were checked for outliers and inconsistent values (i.e., values more than 4 standard deviations above or below the mean, unexplained loss of height or weight in children during growth period, and large disagreement between mother and father reports). We excluded 26 BMI scores due to improbable values for height and weight. All measures of height and weight were obtained in centimeters (cm) and kilograms (kg).

Measures

Parental BMI was based on self-reported height and weight from the first survey sent after registration with the NTR, when the twins were 9 months old on average, and was adjusted for parents' age. The measure of the twins BMI was based on height and weight data as reported by the parent (mothers: 55%, fathers: 4%, both: 41%). When both reports were available, we randomly selected one of the parent reports. Survey 5 included questions about the twins' height and weight after the third birthday. We selected the data closest to the 4th birthday of the twins (Estourgie-van Burk et al., 2010). Survey 12, which was sent to parents when the twins were about 12 years, again included parental reports of the twins' height and weight. Parental EA was self-reported and categorized into four levels (primary, lower secondary, intermediate/higher secondary, and higher vocational/university). Child academic achievement was based on a standardized test for educational achievement, the CITO test (Citogroep, 2002), which is administered in the final grade (when children are 11 or 12 years old) of elementary school in February-March. Information on CITO scores was obtained from parents, teachers and/or self-report by twins at later ages (Bartels et al., 2002).

Statistical Analyses

We applied structural equation modeling (SEM) to study the phenotypic transmission of BMI and EA from parents to BMI and EA of their twins at age 4 and 12 years. All measures (parental and child BMI and educational measures were analyzed as continuous variables. Our model includes the transmission of BMI and EA from parents to the twins, while including the correlation of BMI and EA within and between parents. The transmission includes the prediction of offspring BMI and EA from parental BMI and EA within (EA to EA, BMI to BMI) and across traits (e.g., BMI to EA, EA to BMI). We tested whether estimates of the parameters depended on the sex of the parent and the offspring. Frequency tables, descriptive statistics, and bivariate correlations were obtained using IBM SPSS (version 26). We carried out the structural equation modeling in the Lavaan package (version 0.6-6) in R (version 3.6.1). For all statistical tests, we used the Holm-Bonferroni correction method to control the family-wise error rate in multiple hypothesis testing (Holm, 1979). The Holm-Bonferroni correction is a post-hoc statistical procedure used to adjust p values in order to reduce the probability of type I errors when multiple statistical tests are performed simultaneously, and has a lower increase of type II error risk than the classical Bonferroni method (Aickin & Gensler, 1996). It works as follows:

1. All p values are sorted from smallest to largest. Let K be the number of the p values in a family of tests.

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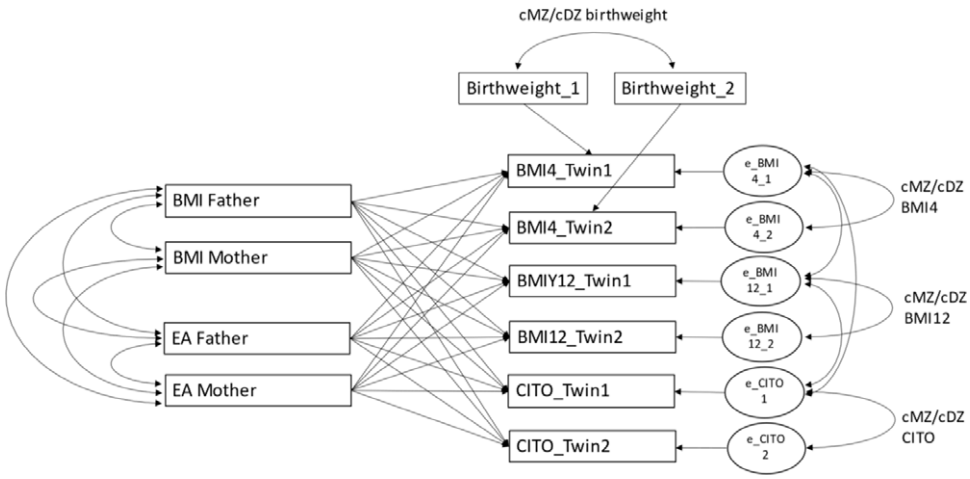

Figure 1. Intergenerational transmission of BMI and educational outcomes, model 1 (full model). The figure shows the full SEM. The separate transmission coefficients for sons and daughters, cross-trait correlations for twin 2 and cross-trait, cross-twin correlations are not shown in the figure to avoid clutter.
Note: EA, educational attainment; CITO, standardized educational achievement test; MZ, monozygotic; DZ, dizygotic.

2. If the first p value is greater than or equal to α/K, the procedure is stopped and no p values are significant. Otherwise, the first p value is declared significant, and the procedure continues.
3. The second p value is compared to α/(K-1). If the second p value is greater than or equal to α/(K-1), the procedure is stopped and no further p values are significant. Otherwise, the second value is declared significant, and we go on until the i-th ordered p value is such that: $p_{(i)} \geq \alpha /(\text{K-i} + 1)$.

Model Specifications

We initially specified separate transmission parameters from mothers and fathers to sons and daughters, to account for sex dependence of these parameters. That is, we estimated four sets (mother-daughter, mother-son, father-daughter, and father-son) of six coefficients (parent EA to offspring EA, parent BMI to offspring BMI at ages 4 and 12 (denoted BMI4 and BMI12), and all cross-transmission coefficients). The model included the following parameters:

- 24 parent-offspring transmission parameters: 2 parental traits (EA & BMI) x 3 offspring traits (EA, BMI4 and BMI12) x 2 parents x 2 offspring genders
- 4 spousal correlations: 2 measures (EA and BMI) x 2 types of correlation (within- and cross-trait)
- 2 within person EA/BMI correlations in the parental generation: fathers and mothers
- 6 residual within-person correlations in the offspring generation: 3 trait combinations (EA/BMI4, EA/BMI12, BMI4/BMI12) x 2 genders (sons and daughters)
- 4 variances in the parental generation: 2 traits (EA, BMI) x 2 genders (fathers and mothers)
- 6 residual variances in the offspring generation: 3 traits x 2 genders
- 4 intercepts in the parental generation: 2 traits x 2 genders
- 6 intercepts in the offspring generation: 3 traits x 2 genders
- 6 residual twin correlations: 3 traits (EA, BMI4, BMI12) x 2 zygosity groups (MZ/DZ)
- 6 cross trait residual twin correlations.

Thus, in the full model, as shown in Figure 1, we estimated transmission parameters separately for sons and daughters and for mothers and fathers (24 parameters in all). Subsequently, we imposed equality constraints on these parameters across genders of parents and offspring to test the differences in influence across various parent-offspring gender combinations. Parameters were obtained by Full Information Maximum Likelihood (FIML). An advantage of FIML is that it handles missing data optimally: it exploits all available data and is more efficient than list- or pairwise deletion. Model fit was evaluated using the following fit indices: comparative fit index (CFI), root mean square error of approximation (RMSEA), and standardized root mean square residual (SRMR). CFI values range from 0 to 1, with larger values indicating better fit and a recommended cutoff of .95 (Xia & Yang, 2019). RMSEA is an absolute fit index, based on the fact that the fitted model is an approximation to the true model. Interpretation of RMSEA values is often considered according to the following: : 0 = perfect fit; <.05 = close fit; .05 to .08 = fair fit; .08 to .10 = mediocre fit; >.10 = poor fit (Byrne, 1998). SRMR is defined as the difference between the observed correlations and the model implied correlation matrix. As a rule of thumb, an SRMR ≤ .08 indicates an acceptable fit (Xia & Yang, 2019). Thus, it allows assessing the average magnitude of the discrepancies between observed and expected correlations as an absolute measure of (model) fit criterion.

Results

Descriptive Statistics

Parents. Table 1 contains a summary of the average age, BMI and EA for the parents. Mean age at first survey of fathers at was slightly higher than mothers (33.8 vs. 31.4 years). Mean BMI was 24.9 for fathers and 23.9 for mothers. BMI variance was higher for mothers (14.4, 95% CI [14.1, 14.8]) than fathers 9.0, 95% CI [8.8, 9.3]); age was unrelated to BMI in both parents. The EA level of fathers was comparable to the EA of mothers, with fathers having slightly higher percentage of higher vocational training and university education, and mothers having slightly higher percentage of intermediate and higher secondary schooling (see Table 1).

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Table 1. Age, BMI, and EA in parents

		Fathers $N = 13,739$	Mothers $N = 13,838$
Age	*n*	13,622	13,835
	Mean	33.84	31.43
	SD	4.65	3.89
BMI	*n*	11,364	11,716
	Mean	24.88	23.87
	SD	3.00	3.80
Educational attainment	*n*	13,036	13,310
	Primary school	5.4%	4.1%
	Lower vocational school and lower secondary school	25.3%	25.8%
	Intermediate vocational/ secondary school and higher secondary school	34.9%	40.3%
	Higher vocational school or university	34.5%	29.8%

Note: EA, educational attainment.

Table 2. BMI4, BMI12, CITO in offspring

		Boys $N = 13,347$	Girls $N = 13,508$
BMI4	*n*	9623	9709
	Age at measurement: mean (*SD*)	3.91 (.17)	3.91 (.17)
	Score: mean (*SD*)	15.35 (1.22)	15.16 (1.31)
BMI12	*n*	7611	7741
	Age at measurement: mean (*SD*)	12.2 (.35)	12.2 (.36)
	Score: mean (*SD*)	17.31 (2.38)	17.58 (2.57)
CITO	*n*	6984	7635
	Score: mean (*SD*)	538.5 (8.4)	537.3 (8.7)

Note: CITO, standardized educational achievement test.

The variance of EA was higher in fathers than in mothers (.82, 95% CI [.80, .84] vs. .73, 95% CI [.72, .74]).

Offspring. Table 2 summarizes sample characteristics in offspring. Mean BMI at age 4 was slightly higher in boys (15.35, 95% CI [15.32, 15.37]) than girls (15.16, 95% CI [15.14, 15.19]), while mean BMI at age 12 was slightly higher in girls (17.58, 95% CI [17.52, 17.64]) than boys (17.31, 95% CI [17.26, 17.37]). BMI4 variance was 1.50 for boys and 1.71 for girls, while BMI12 variance was 5.66 for boys and 6.58 for girls. Mean CITO scores were marginally higher in boys (538.5, 95% CI [538.3, 538.7], variance: 71.2) than girls (537.3, 95% CI [537.1, 537.5], variance: 76.5).

Full model. Table 3 shows results for the full model, which is depicted in Figure 1. Within traits, the transmission parameters were significant for all traits and all parent offspring combinations. In the regression of BMI4 on parental BMI, the standardized regression (i.e., transmission) coefficients (betas) ranged from .11 to .15. In the regression of BMI12 on parental BMI, the range was from .16 to .28. In the regression of the twins' CITO scores on parental EA, the coefficients ranged from .18 to .20. In the regression analysis concerning cross-trait transmission — that is, parental EA to offspring BMI and parental BMI to offspring EA — we obtained mixed results. In the regression of BMI4 on the parental EA, we found that maternal EA significantly predicted daughters' BMI, with a standardized beta of .04. In the regression of BMI12 on parental EA, we found that paternal EA significantly predicted BMI in both sons and daughters, with a standardized beta of −.04 and −.07 respectively; that is, a higher father's EA was associated with lower BMI at age 12. In the regression of CITO on parental BMI, we found that maternal BMI significantly predicted CITO scores in both sons and daughters, with a standardized beta of −.05; that is, a higher maternal BMI was associated with lower CITO scores in children.

Gender differences in transmission parameters. To assess gender differences in transmission from fathers and mothers to sons and daughters, we first estimated the transmission coefficients freely, and then imposed equality constraints on the transmission coefficients. We tested these constraints by means of likelihood ratio tests. The omnibus test of all 12 equality constraints for male and female offspring was not significant ($df = 12$, $p = .139$; see Supplementary Table S1). Subsequent individual (post hoc) tests of equality similarly were not significant. So, we concluded that the transmission coefficients are equal in male and female offspring. We then tested whether the transmission coefficients of the fathers equaled those of the mothers. The omnibus test was not significant ($df = 6$, $p = .074$; see Supplementary Table S2). Subsequent individual (post hoc) tests of equality similarly were not significant. Based on these results, we arrived at model 2. Parameter estimates in this model are shown in Table 4 and summarized in Figure 2. These results indicate small, but significant, effects for cross-trait transmission from parents to offspring BMI and CITO. The within-trait transmission coefficient of BMI4 (.10) was lower than other coefficients (BMI12: .20, CITO: .19). Estimates in this reduced model 2 were similar to those in the full model. Both models had good model fit measures, with negligible differences in fit indices: comparative fit index (CFI), .83; root mean square error of approximation (RMSEA) .068; and standardized root mean square residual (SRMR), .061. The SRMR and RMSEA indicate acceptable model fit, while the CFI is below the recommended cutoff of .95. The chi square likelihood ratio test of model 1 (χ^2: 5319.7, *df*: 374) vs model 2 (χ^2: 5348.5, *df*: 392) had a p-value of .0509, indicating no significant difference between the two models. Model fit measures are presented in Supplementary Table S3.

Within-person, spousal, and twin correlations. We observed negative within-person EA-BMI correlations for both parents (fathers: -.10; mothers: −.08) (see Table 5) (we obtained the same estimates in the SEM). The observed spousal correlation for EA was .46 (same estimate in the SEM). The observed spousal correlation for BMI was .22, and its estimate in the SEM was .21. Observed cross-trait spousal correlations were -.10 for father's EA with mother's BMI and −.08 for mother's EA with father's BMI, and we obtained the same estimates in SEM. In offspring, the observed correlations between BMI4 and CITO were positive (boys: .048; girls: .057), and the residual correlations (i.e., after taking parental EA/BMI into account) were estimated in the SEM at .057 for boys and .046 for girls. The observed correlations between BMI12 and CITO were negative (boys: −.052; girls: −.070). However, the residual correlations were not significant in the SEM after applying Holm-Bonferroni correction. The observed correlations between BMI4 and BMI12 were .43 (boys) and.46 (girls), and their residuals did not differ

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Table 3. Direct and indirect regression coefficients, full model

	Raw			Standardized		
Male offspring	Estimate	Lower CI	Upper CI	Estimate	Lower CI	Upper CI
BMI4						
Paternal BMI	.05*	.04	.06	.11*	.08	.13
Maternal BMI	.04*	.03	.05	.11*	.09	.13
Paternal EA	.01	−.03	.05	.01	−.02	.03
Maternal EA	.05	.01	.09	.03	.01	.06
BMI12						
Paternal BMI	.14*	.12	.16	.16*	.14	.19
Maternal BMI	.18*	.16	.20	.26*	.24	.29
Paternal EA	-.12*	-.20	−.04	−.04*	−.07	−.02
Maternal EA	−.06	-.15	.02	−.02	−.05	.01
CITO						
Paternal EA	1.94*	1.67	2.21	.20*	.17	.22
Maternal EA	1.85*	1.56	2.15	.18*	.15	.20
Paternal BMI	−.06	-.15	.03	−.02	−.05	.01
Maternal BMI	-.13*	-.20	−.06	−.05*	−.08	−.03
	Raw			Standardized		
Female offspring	Estimate	lower CI	Upper CI	Estimate	Lower CI	Upper CI
BMI4						
Paternal BMI	.05*	.04	.06	.12*	.09	.14
Maternal BMI	.05*	.04	.06	.15*	.13	.17
Paternal EA	−.02	−.06	.01	−.02	−.04	.01
Maternal EA	.06*	.02	.09	.04*	.01	.06
BMI12						
Paternal BMI	.18*	.16	.20	.22*	.19	.25
Maternal BMI	.17*	.16	.19	.28*	.25	.30
Paternal EA	-.18*	-.26	-.11	−.07*	-.10	−.04
Maternal EA	−.09	-.17	−.01	−.03	−.06	.00
CITO						
Paternal EA	1.81*	1.54	2.08	.19*	.16	.22
Maternal EA	1.92*	1.63	2.20	.19*	.16	.22
Paternal BMI	−.04	-.13	.04	−.02	−.05	.01
Maternal BMI	-.12*	-.19	−.05	−.05*	−.08	−.02

Note: EA, educational attainment; CITO, standardized educational achievement test. *significant p value based on a Holm-Bonferroni corrected target α of .05 with 24 correlated tests

appreciably in the SEM. The observed MZ twin correlations were less than twice DZ correlations for all traits. The MZ and DZ correlations for BMI at age 4 equaled .84 and .47 respectively. For BMI at age 12, these are .90 and .50, respectively. For CITO, the correlations are .81 and .45, respectively (see Table 6).

Discussion

In a large number of two-generation Dutch nuclear families we looked at the transmission from parents to offspring for BMI and educational attainment. Within traits, transmission for BMI induced a positive association between parental and offspring BMI at age 4 and BMI at 12 in the SEM. The association was twice as strong in 12-year-olds than in 4-year-olds, that is, $r = .20$ versus .10 (based on a reduced model, in which transmission coefficients were equal for all parent-offspring gender combinations). The parent-adolescent BMI transmission coefficient (.20) is in the same range of transmission coefficients observed in our prior study in an independent sample from the Netherlands of parents and their adult offspring sample (i.e., .15 to .26; Alrouh et al., 2022). For educational attainment, the observed parent-offspring correlation ($r = \sim.28$) was higher than the standardized transmission coefficient implied by SEM (.19), suggesting that parental BMI does contribute to the correlation between parental EA and academic achievement in adolescents.

Across traits, offspring BMI at 4 years was positively correlated with parental EA, while offspring BMI at 12 years was negatively correlated with parental EA. In 4-year-olds, the observed correlation was ∼.02 in mothers and negligible in fathers, and

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Table 4. Regression coefficients of parental on offspring measures and residual correlations, reduced model

	Raw			Standardized		
	Estimate	Lower CI	Upper CI	estimate	Lower CI	Upper CI
Offspring BMI4						
Parent BMI	.05*	.04	.05	.10*	.09	.11
Parent EA	.02*	.01	.04	.02*	.01	.03
Offspring BMI12						
Parent BMI	.17*	.16	.18	.20*	.19	.21
Parent EA	−.12*	−.15	−.08	−.04*	−.05	−.03
Offspring CITO						
Parent EA	1.88*	1.76	1.99	.19*	.18	.20
Parent BMI	−.09*	−.13	−.06	−.03*	−.04	−.02
Residual correlations						
Male offspring						
BMI4-CITO	.62*	.30	.95	.06*	.03	.09
BMI12-CITO	−.53	−1.13	.06	−.03	−.06	.00
BMI4-BMI12	1.36*	1.27	1.44	.44*	.41	.46
Female offspring						
BMI4-CITO	.43*	.15	.71	.05*	.02	.08
BMI12-CITO	−.13	−.62	.36	−.01	−.04	.02
BMI4-BMI12	1.01*	.93	1.08	.39*	.37	.42

Note: EA, educational attainment; CITO, standardized educational achievement test.
*significant *p* value based on a Holm-Bonferroni corrected target α of .05 with 12 correlated tests.

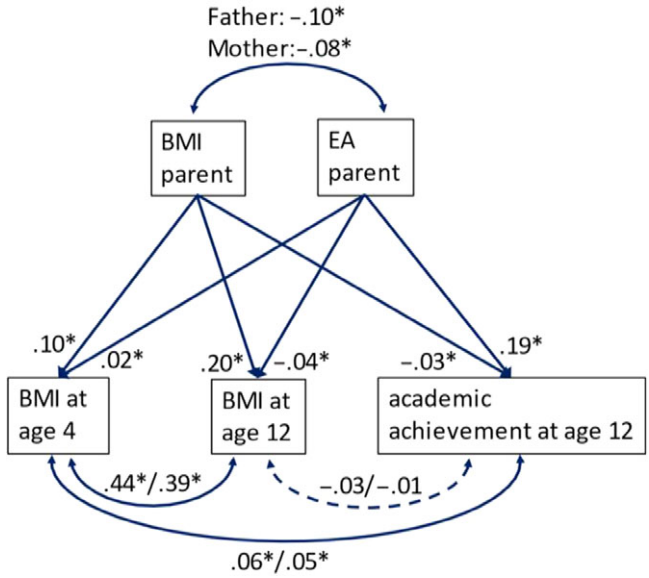

Figure 2. Intergenerational transmission of BMI and educational outcome, reduced model. The figure displays the transmission of parental BMI and educational attainment to children's BMI and academic achievement, based on data from 13,916 families. The figure shows the estimated effects (in standardized units) of parental BMI and educational attainment on their children's BMI (at ages 4 and 12) and academic achievement (nationally standardized test at age 12), as well as the residual correlations between BMI and academic achievement. The paths with an asterisk (*) had a significant *p* value based on a Holm-Bonferroni corrected target α of .05 with 12 correlated tests. Paths from parents to children were similar in boys and girls as well as fathers and mothers. The figure shows this reduced model, without gender differences in intergenerational transmission. The residual correlations are given separately for boys/girls.
Note: EA, educational attainment.

the standardized transmission coefficient (beta) was .02 in the reduced model. This weak correlation parallels findings in previous twin and population-based studies, which show no association (Morgen et al., 2017) or slightly lower BMI in the offspring of mothers with lower education levels (Howe et al., 2011; Silventoinen et al., 2019). In 12-year-olds, the observed correlation was about -.10, and the standardized beta was about −.04. After accounting for parental BMI, we saw a slight decrease in the strength of correlation between parental EA and BMI in adolescents. This decrease is in line with our earlier study of adult offspring, where we found that accounting for parental BMI all but eliminated the effect of parental EA on offspring BMI. In our current study, however, the association between parental EA and offspring BMI at age 12 remained statistically significant. This suggests that while parental BMI may drive some of the association between parental EA and offspring BMI, it does not account for the entire association. Parental EA does in fact have a distinctive effect (i.e., after taking parental BMI into account) on increasing the risk of obesity in adolescence, even if this effect decreases as children reach adulthood. This association is possibly due, but not limited, to the effects of parental EA on household income and associated consequences for breastfeeding (Bartels et al., 2009), nutrition (Darmon & Drewnowski, 2015) and physical activity (Telford et al., 2016).

There were generally no significant differences in transmission parameters between fathers and mothers to sons and daughters. At the offspring level, the lack of sex differences in the transmission parameters might be explained by the relatively young age of offspring (Silventoinen et al., 2007), where gender specific differences have yet to develop to be seen in adulthood (Schousboe et al., 2003). On the parental level, the lack of differences in transmission between fathers and mothers might be due to the relatively high level of gender equality in the Netherlands, which would result in a more comparable effect of the EAs of father and mother on household income.

Observed correlations and residual correlations (after accounting for transmission in the SEM) of academic achievement in adolescence and BMI in childhood were positive at $r = \sim.05$, indicating that a higher BMI in childhood was associated with better academic achievement later in life. In 12-year-olds, however, the observed correlation between academic achievement and BMI was negative ($r = \sim-.06$), but the residual correlation was not significant. This result contrasts with our prior results in adults, where the residual correlation between BMI and EA remained significant at r = -.10. Whereas our measure of EA at age 12 is mainly a reflection of cognitive ability (Bartels et al., 2002), it is likely that additional factors influence the highest educational degree attained in adulthood.

Our study design allowed us to estimate the phenotypic intergenerational transmission of BMI and EA. The present phenotypic model does not address environmental or cultural transmission in the presence of genetic transmission. This question can be addressed by applying the parents and twins design, which specifies both association between parents and their twins stemming from both types of transmission, in the presence of phenotypic assortative mating (Fulker, 1988; Keller et al., 2009).

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Table 5. Observed covariance and correlation table for parental and offspring variables

	BMI4 (male/female)	BMI12 (male/female)	CITO (male/female)	Father BMI	Mother BMI	Father EA	Mother EA
BMI4 (male/female)	1.50/1.71	1.21/1.52	.48/.65	.51/.59	.77/.87	.00/.00	.02/.03
BMI12 (male/female)	.43/.46	5.66/6.58	−.98/−1.5	1.78/1.87	2.68/3.06	−.21/−.27	−.17/−.21
CITO (male/female)	.05/.06	−.05/−.07	71.2/76.5	−1.42/−1.45	−2.59/−2.96	2.14/2.14	1.91/2.09
Father BMI	.09/.09	.25/.25	−.060/−.059	9.02	2.38	−.27	−.20
Mother BMI	.16/.17	.29/.33	−.09/−.09	.21	14.43	−.35	−.26
Father EA	.00/.00	−.10/-.12	.29/.27	−.10	−.10	.81	.35
Mother EA	.02/.03	−.09/-.10	.27/.28	−.08	−.08	.46	.72

Note: EA, educational attainment; CITO, standardized educational achievement test at age 12. Upper triangle, covariance. Lower triangle: correlation. Diagonal: variance.

Table 6. Observed/residual twin correlations

		BMI4	BMI12	CITO
MZ	BMI4	.84/.85	–	–
	BMI12	.43/.40	.90/.89	–
	CITO	.03/.03	−.09/-.02	.81/.79
DZ	BMI4	.47/.45	–	–
	BMI12	.23/.17	.50/.42	–
	CITO	.01/.01	−.03/.02	.45/.38

Note: MZ, monozygotic; DZ, dizygotic; CITO, standardized educational achievement test at age 12. Lower triangle, cross-trait correlations. Diagonal, within-trait correlations.

The present data are suitable to conduct such a follow-up study. Alternatively, polygenic scores can be used to detect phenotypic transmission in the presence of genetic transmission. This approach requires genetic data in the in the parents and their twins, and phenotypic data in the twins (Bates et al., 2018; Hart et al., 2021; Kong et al., 2018; Okbay et al., 2022).

It is well known that positive cultural transmission is a source of additive genetic (A) and common environment (C) covariance, and that A-C covariance biases upwards the estimated C variance component in the classical twin design (Purcell, 2002). The C component of the CITO scores is known to be low in the Netherlands. For CITO, we have previously reported standardized variance components of 0.74 (A), 0.08 (C), and 0.18 for unique environment (E; de Zeeuw et al., 2016). In contrast, the twin correlations for BMI suggest a substantial C component. We have previously reported on BMI standardized variance components of ∼.65(A), ∼.16(C), and ∼.19(E) at age 4 and ∼.78 (A), ∼.11(C) and ∼.10(E) at age 12 (Silventoinen et al., 2007). The small C component of CITO does not rule out cultural transmission, but does suggest that the effect of cultural transmission is likely to be small. Indeed, in previous work we found for children's CITO scores no genetic-nurture effect (de Zeeuw et al., 2020). The larger C component of BMI could well be attributable partly to cultural transmission.

The main strengths of this study are a large sample size and a wide age range for parents. Our study sample covers different geographic areas and socioeconomic classes in the Netherlands. Recruitment of twins — who are generally considered representative of the general population to which they belong (Hur et al., 2019) — and their families into the NTR was done through multiple channels, leading to a sample that is reasonably representative of the Dutch population. Although the study relied on self-reported measures for height, weight and EA for parents and offspring, the reliability in self-reporting of height and weight is good: in an analysis of a subsample of 6026 individuals, we observed a correlation of .93 between self-reported BMI and BMI measured by a research nurse or assistant (Willemsen et al., 2010). We analyzed BMI as a continuous variable rather than a (clinical) binary variable (obese/overweight vs. normal weight), because we wanted to address the process of transmission as it pertains to the full range of BMI in the general population in the Netherlands. In this approach, we assume that the process of transmission as we identify it is relevant to the full range of BMI and EA. We note that it is possible to fit our present model (see Figure 1) to a binary BMI variable, but this would merely result in a loss of information. Specifically, the analysis in the case of a binary BMI variable is based on the liability threshold model, in which the model is fitted to latent continuous BMI variables. There is no advantage to this compared to fitting the directly to observed continuous BMI variables, but there is the disadvantage of a loss of power associated with loss of information. For this reason, we opted to use BMI as a continuous variable in our analysis.

There are also some limitations to the current study. While BMI is a common, convenient measure of obesity, other methods, such as skinfold thickness and percent body fat from dual energy X-ray absorptiometry, may provide more accurate estimates. In our analyses, we have modeled the process of transmission of EA and BMI from adult parents to adult offspring. Our resulting model is supposed to represents this process as it takes place in the general Dutch population with respect to the full range of BMI. As such, this model assumes that the process is the same regardless of the actual level of BMI in the parents or their offspring. We recognize that it is possible that the transmission process may differ in extremes of the BMI distribution. Detecting such heterogeneity is a statistically challenging task, which is beyond our present aim.

Our results pertain to young offspring who are still in the parental household, whereas our earlier study examined intergenerational transmission in adult offspring. The differences between findings in young and adult offspring may highlight different parental effects on child and adult obesity, especially given that younger offspring share the same household environment with their parents while adult offspring do not. One of the striking findings is the persistent association between low BMI in childhood and low academic achievement in adolescence, even after accounting for parental transmission. This highlights the need to address childhood underweight as well as overweight, as either condition can cause a different set of adverse outcomes later on in life.

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Ethical standard. The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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