Intergenerational Transmission of Body Mass Index and Associations with Educational Attainment

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Abstract

Background

Individual differences in educational attainment (EA) and physical health, as indexed by body mass index (BMI), are correlated within persons and across generations. The present aim was to assess these associations while controlling for parental transmission.

Methods

We analyzed BMI and EA obtained for 8,866 families from the Netherlands. Data were available for 19,132 persons, including 6,901 parents (mean age 54) and 12,234 of their adult offspring (mean age 32). We employed structural equation modeling to simultaneously model the direct and indirect transmission of BMI and EA from parents to offspring, spousal correlations, and the residual within-person BMI-EA association and tested for gender differences in the transmission parameters.

Results

We found significant intergeneration transmission of BMI and EA from parents to their adult offspring, and substantial spousal correlations (0.23 for BMI and 0.51 for EA). Cross-trait parent to offspring transmission was weak. The strength of transmission was largely independent of parent or offspring gender. About 60% of the EA-BMI correlation in the offspring persisted after taking into account the intergeneration transmission.

Conclusions

The intergenerational transmission for BMI and EA is mainly predictive within traits. Significant spousal and within person correlations in the parental generation are responsible for the effect of parental EA on offspring BMI. Offspring EA and BMI are further correlated beyond parental influences.

Introduction

Body Mass Index (BMI) is an important marker of overall health and is strongly associated with the risk of – and mortality from – chronic diseases [1]. In the last three decades, the prevalence of obesity has dramatically increased globally [2], and the burden of disease attributable to high BMI has more than doubled, although this trend differs across countries [3]. Within countries, overweight and obesity are not randomly distributed across the population, but tend to be more frequent among people with a lower educational attainment (EA) [4] and in low socio-economic strata [5].

One potential explanation for the association between EA and obesity is that a higher EA leads to better occupation, higher income and higher socioeconomic status (SES). In fact, many studies use EA – as well as occupation and income – either as proxy for, or a component of, SES measures. SES is inversely related to obesity risk in global north countries [6–9]. Higher education and socioeconomic status result in a better health knowledge, and provide the individual with resources that afford healthy food options [10] and free time to engage in physical exercise [11], i.e., behaviors associated with lower obesity risk. In addition, early research showed a residual link between obesity and cognitive deficiency, even after controlling for parental social class [12]. Conversely, obesity itself can influence EA [13], employment [14], future income [15], and other SES measures [16, 17]. Furthermore, genetic factors have been shown to affect both BMI and EA [18, 19], as well as related behavioral factors such as self-control and time preference (i.e. delayed gratification) [20]

EA and BMI are both subject to intergenerational transmission. The average correlation between parent and offspring EA in Western Europe and USA is 0.39 [21]. As for BMI, a recent meta-analysis showed that having a single parent with obesity is strongly associated with childhood obesity (average odds ratio of 3.49)[22]. While very few studies examine associations between parent and offspring BMI in adults, one study showed an increased association in older offspring compared to younger ones [23].

In addition to the effect of parental BMI on offspring BMI, studies have shown significant associations between parental EA and offspring BMI [24–26]. Of these studies, only one included parental BMI as a covariate, with the finding that only maternal education had a significant effect [24]. Furthermore, many studies show significant associations between parental SES and offspring BMI, yet none of these studies controlled for parental BMI [27–33].

We used structural equation modeling (SEM) to study the phenotypic transmission of BMI and EA from fathers and mothers to their adult offspring in a large population cohort from Netherlands, including families with at least one parent and one offspring. Our design allows us to take into account the correlation of BMI and EA within and between parents, while examining the simultaneous transmission of BMI and EA. The latter includes the prediction of offspring BMI and EA from parental BMI and EA within (e.g., BMI to BMI) and across traits (e.g., BMI to EA). Further, we tested whether the strength of the associations depend on the sex of the parent and the offspring. We examined each predictor's direct effects, defined as the transmission coefficient from parent to offspring within or across traits, and indirect effects, resulting from 1) the correlations between EA and BMI within each parent and 2) the spousal correlations between parents for these traits. For instance, maternal EA has a direct effect path on offspring BMI, an indirect effect path through maternal BMI proportional to the EA-BMI correlation in the mother, and two indirect effect paths through paternal EA and BMI, given non-zero spousal correlations. The SEM approach also allows us to calculate residual correlations between EA and BMI in the offspring generation in order to examine residual association while controlling for parental transmission. This examination is important to determine to what extent EA and BMI associations in the children are independent of the parental effects.

Methods

Participants. Participants were registered with the Netherlands Twin Register (NTR). The NTR collects longitudinal data on health, personality and lifestyle from twin families in the Netherlands. These families were recruited by NTR across the Netherlands through city councils, newsletters, and the NTR website. The present study is based on data collected between 2009 and 2012. Participants first received a written invitation including a link to a webpage, where they could log on to a web-based version of the survey with a unique, personal login name and password. If subjects did not access the web-based survey within the 6 weeks after the invitation, they received a paper version of the survey. Between 3–9 months after the paper versions of the survey were sent, subjects who had not responded received a reminder by post, or a reminder by email, if an email address was available. Several groups of non-responders (e.g. twins from incomplete twin pairs) were reminded in a phone call [34] We selected nuclear family members (parents and offspring), resulting in a sample of 19,135 participants. The survey was collected in two versions, a longer and a shorter version; a short version, filled out by 3,421 participants, did not include questions concerning EA. Our sample comprises twins registered with NTR and their parents and siblings. We refer to the twins and their siblings as the offspring generation. There are 12,234 offspring aged between 16 and 97 years and 6,901 parents (2,817 fathers and 4,084 mothers) aged between 21 and 94 years. Note that the age distributions overlap, as twins registered with the NTR include younger twins, often registered with their parents and older twins, usually registered without their parents.

The total number of families was 8,866 with an average of 1.4 offspring per family, where some families (18%) had respondents only from the parental generation. In addition, 45% of families included twins without their parents; 18% included twins and 1 parent; and 19% included twins and 2 parents (see supplementary table 1). There is information for

both parent and offspring generation on BMI in 3,214 families (with N = 10,650), and on EA in 1,089 families (with N = 3,563) (see supplementary tables 2 and 3). EA from participants below the age of 25 years was not included, as they may not have yet reached their highest educational level, resulting in a sample size of 12,078 for EA. The data were checked for outliers and 5 individuals were excluded due to extreme values for height or weight.

Measures/Phenotyping. BMI was based on self-reported height and weight, and was analyzed as a continuous variable. EA was self-reported and categorized according to the highest achieved educational level. The EA scale has 8 levels, and was analyzed as a continuous variable (see Table 1). EA and BMI were adjusted for age of participant at the time of completing the survey. Age adjusted measures were used in the analyses. Outlying BMI data were checked against measurements obtained for the same subject from other surveys in the NTR database. Participants were grouped by sex in the analysis, and separate means were estimated for fathers, mothers, sons and daughters.

Table 1
Age, BMI, and EA in parents and offspring

		Fathers n=2,817	Mothers n=4,084	Sons n=3,999	Daughters n=8,235
Age	N available data	2,817	4,084	3,999	8,235
	mean	55.88	53.01	31.64	31.91
	SD	7.90	8.04	14.74	14.35
BMI	N available data	2,758	3,960	3,865	7,924
	mean	26.10	25.66	23.53	23.06
	SD	3.27	4.46	3.49	3.84
EA	N available data	2,030	2,897	1,902	4,163
EA level	Dutch equivalent				
Elementary school	Basisschool	1.7%	2.3%	1.1%	1.3%
Lower vocational education	Vmbo/vocational stream	13.2%	11.3%	7.5%	%6.3
Lower general secondary school	Mulo, mavo, vmbo/theoretical stream	9.1%	19.5%	6.1%	9.2%
Intermediate vocational education	Mbo	20.0%	23.1%	20.8%	27.3%
Upper general secondary school	Havo, hbs, atheneum, gymnasium	5.3%	8.7%	2.9%	4.9%
Higher vocational education	Hbo	27.9%	25.5%	29.3%	29.1%
University degree	Post-hbo degree	18.8%	8.9%	25.7%	18.4%
Post-graduate degree	PhD degree	4.1%	0.8%	6.6%	3.6%
Abbreviations: BMI, body mass index; EA, educational attainment; SD, standard deviation					

Statistical Analyses. We used SEM to simultaneously analyze the influence of parental EA and BMI on EA and BMI in their offspring, including BMI-EA correlations within person and across persons within each generation. Paternal and maternal transmission to sons and daughters were specified separately, i.e., we estimated 4 sets (mother-daughter, mother-son,

father-daughter, and father-son) of 4 coefficients (EA to EA, BMI to BMI and the cross-transmission coefficients). To accommodate multiple offspring, we included a maximum of 6 offspring (3 males and 3 females) We decided to limit the analysis to a maximum of 6 offspring on the basis of computational considerations. This did not result in an appreciable loss of data (we analyzed 99.37% of the original study sample). Thus, in the full model (figure 1), all coefficients were estimated separately for sons and daughters and for mothers and fathers. Subsequently we imposed equality constraints on transmission parameters across genders of parents and offspring to test the differences in influence across various parent-offspring gender combinations. For these tests, we applied a Bonferroni correction given a family-wise α of 0.01 and, given 8 tests, a test-wise alpha of .01/8 = 0.00125. We used IBM SPSS (version 26) to obtain frequency tables and descriptives. In the structural equation modeling, we used full information maximum likelihood estimation, as this exploits all available data and is more efficient than list- or pairwise deletion. We carried out the structural equation modeling in the Lavaan package (version 0.6-6) in R (version 3.6.1).

The following parameters were specified, with estimates for offspring of the same gender (3 sons and 3 daughters per family) constrained to be equal:

- 16 parent-offspring transmission parameters: 2 traits (EA & BMI) x 2 types of transmission (within- and cross-trait) x 2 parental genders x 2 offspring genders
- 4 spousal correlations: 2 measures (EA & BMI) x 2 types of correlation (within- and cross-trait)
- 2 within person EA/BMI correlations in the parental generation: fathers and mothers
- 2 residual EA/BMI correlations in the offspring generation: sons and daughters
- 4 variances in the parental generation: 2 traits x 2 genders (fathers and mothers)
- 4 residual variances in the offspring generation: 2 traits x 2 genders (sons and daughters)

In addition, we estimated all intercepts for EA and BMI (in 2 parents and 6 offspring) and the within and across-trait correlations for all residuals to account for the conditional association of BMI and EA in the offspring generation (i.e., conditional on parental effects). These included 15 within-trait sibling correlations for EA and 15 for BMI and 30 EA/BMI cross-trait sibling correlations.

Results

Descriptive statistics. Mean age of fathers was slightly higher than mothers (56 vs. 53 years), while offspring of both genders were both around 32 years. In both generations, the average male BMI was .5 points higher than the average female BMI. Mean BMI in parental generation was 26.1 for fathers and 25.6 for mothers, and in offspring generation it was 23.5 for sons and 23.1 for daughters. Correcting for age reduced the differences in BMI between the two generations (i.e., males: 2.6, females: 2.5) to 0.07 BMI points for males and 0.5 for females. Supplementary figure 1 shows BMI distribution across age for males and females, with higher BMI in older individuals and consistently higher BMI for males in all age groups. EA levels were higher in males than in females, although the difference was smaller in the offspring generation. Supplementary figure 2 shows EA distribution across age for males and females, with higher levels of education and a narrowing gender gap for younger individuals. BMI (age adjusted) showed higher variance in females than in males in the parental generation (19.63, CI 18.78-20.48 vs 10.49, CI 9.95-11.03) and in the offspring generation (13.27, CI 12.85-13.68 vs 9.30, CI 8.88-9.71) (Table 2). EA (age adjusted) showed slightly higher variance in males than in females in parental generation (3.35, CI 3.15-3.55 vs 2.59, CI 2.46-2.72) and in the offspring generation (2.83, CI 2.65-3.01 vs 2.33, CI 2.23-2.43). Negative within person EA-BMI correlations were observed for all family members (fathers, -0.102; mothers, -0.147; sons, -0.154; daughters, -0.173). Unadjusted correlations tended to be higher for most measures (see supplementary table 4).

Table 2
Observed covariance and correlation table for age adjusted EA and BMI.

	Offspring BMI (male/female)	Offspring EA (male/female)	Paternal BMI	Maternal BMI	Paternal EA	Maternal EA
Offspring BMI (male/female)	9.298/13.268	-0.791/-0.965	1.804/2.264	1.927/3.432	-0.405/-0.434	-0.269/-0.416
Offspring EA (male/female)	-0.154/-0.173	2.796/2.329	-0.384/-0.436	-0.837/-0.410	0.860/0.629	0.597/0.651
Paternal BMI	0.183/0.192	-0.071/-0.088	10.492	3.277	-0.606	-0.583
Maternal BMI	0.143/0.213	-0.112/-0.061	0.228	19.630	-1.402	-1.050
Paternal EA	-0.073/-0.065	0.279 /0.225	-0.102	-0.173	3.350	1.509
Maternal EA	-0.055/-0.071	0.220/0.265	-0.112	-0.147	0.512	2.593
Upper triangle, covariance. Lower triangle: correlation. Diagonal: variance.						
Abbreviations: BMI, body mass index; EA, educational attainment						

Direct and indirect effects, full model. Table 3 shows results for the full model, and are summarized in Figure 2. A direct effect was defined as the transmission coefficient in the regression of parental BMI/EA on offspring BMI/EA. An indirect effect was calculated as the sum of three paths through correlations of one parental measures with the remaining three. For example, if we consider paternal BMI, there is one direct path and three indirect paths going through maternal BMI, paternal EA, and maternal EA. Total effects were calculated by adding up direct and indirect effects. In the full model, within-trait direct effects (parental BMI on offspring BMI and parental EA on offspring EA) were positive and significant for both parents, while cross-trait direct effects were insignificant, i.e., parental BMI did not have significant direct effects on offspring EA, and vice versa. Indirect effects were larger than direct effects for cross-trait transmission, due to significant cross-trait correlations at the parental level (see Table 2). For example, the standardized direct effect of maternal EA on male offspring BMI was -0.030, while the indirect effect was -0.072, adding to a total effect of -0.101). Residual within person EA - BMI correlations in the offspring generation remained significant after accounting for parental effects, which indicates EA and BMI are still correlated when controlling for parental influence. To check if extreme BMI values (due to disorders such as anorexia nervosa or monogenic causes of morbid obesity) influenced our results, analysis was repeated excluding 25 subjects with BMI values less than 15 or higher than 45. Parameter estimates obtained in the reduced sample hardly differed from those obtained in the full sample.

Table 3
Associations between Parent/Offspring BMI/EA (full model, see Figure 1).

	Raw			Standardized				
	Direct			Direct			Indirect	Total
Sons								
BMI	Estimate	Lower CI	Upper CI	Estimate	Lower CI	Upper CI	Estimate	Direct + indirect
Paternal BMI	0.192*	0.140	0.244	0.207	0.151	0.263	0.047	0.254
Maternal BMI	0.105*	0.069	0.141	0.154	0.101	0.207	0.064	0.218
Paternal EA	-0.066	-0.195	0.064	-0.040	-0.119	0.039	-0.068	-0.108
Maternal EA	-0.055	-0.191	0.081	-0.030	-0.102	0.043	-0.072	-0.101
EA								
Paternal EA	0.247*	0.151	0.343	0.273	0.167	0.380	0.107	0.381
Maternal EA	0.165*	0.058	0.272	0.161	0.057	0.265	0.164	0.326
Paternal BMI	-0.030	-0.084	0.025	-0.059	-0.166	0.049	-0.074	-0.132
Maternal BMI	-0.031	-0.064	0.002	-0.083	-0.173	0.006	-0.091	-0.175
EA-BMI residual correlation	-0.405*	-0.663	-0.148	-0.097	-0.149	-0.044		
Daughters								
BMI	Estimate	Lower CI	Upper CI	Estimate	Lower CI	Upper CI	Estimate	Direct + indirect
Paternal BMI	0.235*	0.183	0.287	0.211	0.164	0.258	0.071	0.282
Maternal BMI	0.202*	0.168	0.236	0.248	0.206	0.289	0.065	0.313
Paternal EA	-0.065	-0.188	0.059	-0.033	-0.095	0.030	-0.089	-0.122
Maternal EA	-0.077	-0.203	0.048	-0.034	-0.090	0.022	-0.083	-0.118
EA								
Paternal EA	0.127*	0.054	0.200	0.155	0.066	0.243	0.168	0.323
Maternal EA	0.271*	0.199	0.344	0.290	0.212	0.367	0.097	0.387
Paternal BMI	-0.040	-0.077	-0.003	-0.085	-0.165	-0.006	-0.063	-0.148
Maternal BMI	-0.011	-0.035	0.013	-0.033	-0.104	0.039	-0.096	-0.129
EA-BMI residual correlation	-0.490*	-0.692	-0.289	-0.104	-0.144	-0.065		

Abbreviations: BMI, body mass index; EA, educational attainment; CI, confidence interval.

*p<0.01

Gender differences in transmission parameters. To assess gender differences in transmission from fathers and mothers to sons and daughters, we imposed equality constraints on transmission coefficients, and tested these constraints using

the likelihood ratio test. We found significant differences only for maternal BMI on offspring BMI (supplementary table 5), where the direct effect was almost double in females (males: b = 0.105 CI 0.061, 0.148; females: b = 0.202 CI 0.161, 0.243). Similar analysis for parental gender showed no significant differences between paternal and maternal transmission coefficients (supplementary table 6), i.e., transmission from that fathers and mothers were of equal magnitude. Based on the results of these tests, we arrived at the model denoted model 2. Parameter estimates for this model, shown in Table 4, indicate small, but significant, direct effects for cross-trait transmission from parents to offspring. Standardized within-trait transmission coefficients were similar for both EA and BMI, and generally did not depend on the gender of the parent or offspring, with the exception of maternal BMI on offspring BMI. Cross-trait transmission coefficients were larger for offspring BMI than offspring EA, i.e., parental EA had a larger effect on offspring BMI than parental BMI had on offspring EA. Finally, the within trait spousal correlations were 0.228 for BMI, and 0.512 for EA. Cross-trait spousal correlations were -0.112 for maternal EA/paternal BMI, and -0.173 for paternal EA/maternal BMI. Both models had good model fit measures, with model two scoring slightly higher than the full model (e.g. x^2 = 61.7 vs 52.1), model fit measures are presented in supplementary table 7.

Table 4
Associations between Parent/Offspring BMI/EA (model 2)

	Raw			**Standardized**			
				Sons		Daughters	
Offspring BMI	Estimate	Lower CI	Upper CI	Paternal	Maternal	Paternal	Maternal
Paternal BMI	0.212*	0.174	0.251	0.228	-	0.191	-
Maternal BMI (sons)	0.098*	0.064	0.133	-	0.145	-	-
Maternal BMI (daughters)	0.209*	0.177	0.241	-	-	-	0.257
Parent EA	-0.066*	-0.107	-0.026	-0.040	-0.035	-0.034	-0.029
Offspring EA							
Parent EA	0.201*	0.177	0.226	0.224	0.197	0.244	0.215
Parent BMI	-0.024*	-0.039	-0.009	-0.047	-0.064	-0.051	-0.070
Abbreviations: BMI, body mass index; EA, educational attainment; CI, confidence interval.							
*p<0.005							
Abbreviations: BMI, body mass index; EA, educational attainment; XT, cross-trait; SC, spousal correlation							
[a] separate coefficients for each gender							
Standardized transmission coefficients and correlations (male/female offspring). Solid lines: statistically significant at p<0.01. Dashed lines: statistically insignificant. Abbreviations: BMI, body mass index; EA, educational attainment							

Discussion

To our knowledge, this is the first study in an adult population to examine the effects of parental EA on adult offspring BMI, while controlling for parental BMI. The average age of offspring in our sample is 32 years, which means that most will have left the parental home around 10 years earlier, as the average age at which offspring leave home in the Netherlands is 22.7 years for daughters and 24.2 for sons [35]. Parental BMI was correlated with their adult offspring BMI (r= ~.2). This is in line with previous studies examining parent-offspring BMI associations in isolation [36], which reported slightly lower magnitudes of association for younger offspring, and similar magnitudes for adults [23], i.e., parent-

offspring correlations increase as offspring grow older. This is in spite of the fact that older offspring share less environmental influence with their parents compared to younger offspring. The increase in correlation may be due to genetic effects as previous studies suggest greater genetic influences on BMI in later age compared to early childhood, especially in children of low educated parents [26]. Age adjusted parent-offspring BMI correlations were lower after accounting for parental EA, which shows that a portion of intergenerational BMI transmission is due to factors related to parental EA

Offspring BMI was negatively correlated with parental EA (r~-.07). However, this correlation was insignificant in the full model, when accounting for parental BMI. This suggests that the effect of parental EA on offspring BMI is mainly mediated by parental BMI. Similar trends were also observed when examining the influence of parental factors on offspring EA. The significant lowering of parental EA/BMI regression coefficients, when controlling for the other parental traits, supports earlier findings of shared factors influencing EA and BMI. This shared influence is classically explained in terms of the influence of the shared home environment. For example, parents who achieve higher education tend to have healthier food choices [10], which are shared with their children. However, recent studies suggest that certain genetically influenced characteristics, such as self-control and time preference[20], affect both EA and BMI.

Gender did not play a significant moderating role in our model. Transmission coefficients were largely similar for fathers and mothers as well as sons and daughters, with the exception of BMI transmission from mothers to sons, which was significantly lower than other parent-offspring combinations. The absence of these gender differences confirms findings of prior studies [36], although few studies have reported differences between fathers and mothers in BMI transmission [37, 38].

Parents in our sample exhibited moderate levels of spousal correlation for BMI and high levels for EA. The observed spousal correlation for BMI in our study (r=0.23) is somewhat higher than that reported in most other studies, averaging at 0.15[39]. Increased rates of spousal correlation over birth cohorts has been hypothesized to have contributed to the rise in obesity prevalence [40]. Indeed, odds of offspring obesity increase markedly when both parents have obesity. From our study design, it is unclear whether this correlation existed prior to marriage/cohabitation (i.e. due to phenotypic assortment or social homogamy) or developed with time (i.e. due to marital interaction). However, the latter scenario would involve an increase over time in spousal correlation for BMI, which is generally not found [39].

Within-person EA-BMI correlations were small in the offspring generation (r=-0.15 for sons and -0.17 for daughters), with similar level for mothers (-0.15) and slightly lower for fathers (-0.10). For offspring generation, these correlations were -0.10 after controlling for parental transmission, suggesting that the corresponding health inequalities across EA persist even after taking into account parental factors. This persistence of association suggests that the relationship between EA and BMI is largely independent of parental factors, which implies that interventions aimed at improving EA (and consequently SES) can translate into desirable changes in BMI as well, irrespective of parental EA and BMI.

The main strengths of this study are a large sample size and age range for parents and offspring, as well as use of multiple offspring within families. Our study sample covers different geographic areas and socioeconomic classes in the Netherlands. Recruitment of twins – considered representative of the general population [41] – and their families into the NTR was done through multiple channels including city council registries, leading to a sample that is reasonably representative of the Dutch population. There are also some limitations to the current study. The study relied on self-reported measures for height, weight, and EA for parents and offspring. In our study population, the correlation between self-reported and measured BMI was 0.83 [42]. While BMI is a common, convenient measure of obesity, other methods, such as skin fold thickness and percent body fat from dual energy X-ray absorptiometry, may provide more accurate estimates.

Our results pertain to offspring who are adults and generally have left the parental household. The estimates of parent-offspring BMI associations for adult offspring tended to be higher than those reported for younger offspring. A next step in future research is to examine whether the associations and transmission results based on adult offspring are also seen in younger offspring who still live with their parents, and likely share more of the home environment. This will facilitate the disentangling of the different aspects by which parents influence their offspring's probability of having overweight or obesity. This is important to inform policy and interventions aimed at reducing the prevalence of these conditions.

Conclusions

Our study highlights the importance of controlling for relevant parental factors when examining cross trait intergenerational transmission. This is particularly true for EA and BMI, where cross trait transmission became insignificant after controlling for the same trait in the parental generation. This pattern points towards shared factors that influence both measures. Furthermore, we demonstrate that while correlation between EA and BMI within individuals is partially due to parental factors, the majority portion of correlation is independent of parental influences.

Abbreviations

EA – educational attainment
BMI – body mass index
SES – socioeconomic status
NTR – Netherlands Twin Register
SEM – structural equation modeling

Declarations

Ethics approval and consent to participate

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Medical Ethics Review Committee of the Vrije Universiteit Medical Center Amsterdam (2008/244). Informed consent was obtained from all individual participants included in the study

Consent for publication

Not Applicable

Availability of data and materials

The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request. Code for SEM portion of the analysis is available at the following Github repository:

- Project name: SEM model of BMI/EA intergenerational transmission with multiple (6) offspring per family
- Project home page: https://github.com/hekmatov/EA-BMI-IGT-adult
- Archived version: 7978f29
- Operating system(s): Platform independent
- Programming language: R
- Other requirements: R 3.6.1 or higher. Lavaan package 0.6-6 or higher
- Licence: MIT
- Any restrictions to use by non-academics: licence needed

Competing interests

The authors have no relevant financial or non-financial interests to disclose.

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Authors' contributions

All authors contributed to the study conception and design. Data collection was realized by Boomsma and analyses were performed by Alrouh. The first draft of the manuscript was written by Alrouh and all authors commented on multiple versions of the manuscript. All authors read and approved the final manuscript.

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Not Applicable

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Figures

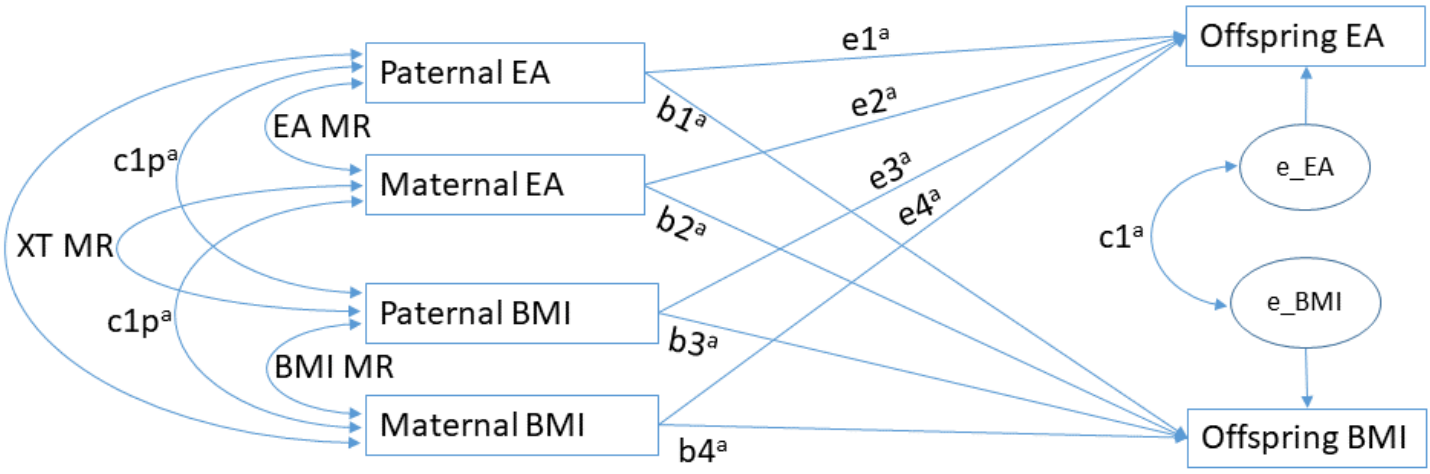

Figure 1

Full model outline Abbreviations: BMI, body mass index; EA, educational attainment; XT, cross-trait; SC, spousal correlation a separate coefficients for each gender

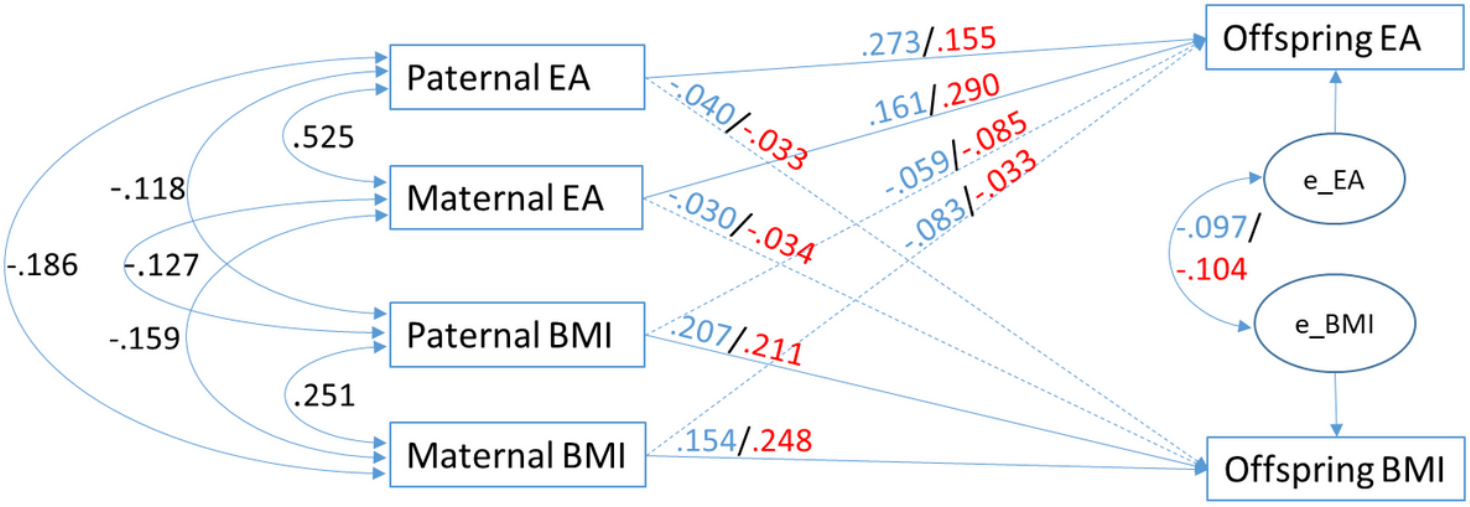

Figure 2

Full model results Standardized transmission coefficients and correlations (male/female offspring). Solid lines: statistically significant at p<0.01. Dashed lines: statistically insignificant. Abbreviations: BMI, body mass index; EA, educational attainment

Supplementary Files

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