Supplementary Material:

Materials and Methods

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References (36-42)

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Materials and Methods:

The Icelandic quantitative trait cohorts

For all the traits studied here, probands were restricted to those with all four grandparents listed in our genealogy database (see the paragraph on AGFC below), with at least one parent also genotyped, and with yob ≥ 1940. Because of the profound social changes that took place in Iceland in the period around 1940, particularly with respect to the access to education, we believe that this yob cutoff is appropriate for the investigation of genetic nurture related to EA. Moreover, with the criterion that at least one parent was also genotyped, even without this yob cutoff, only a small fraction of the potential probands would be born before 1940.

Educational Attainment (EA). The deCODE data on educational attainment were part of the published meta-analysis (*8*). While the original data were collected through various questionnaires, they were recoded to the format used for the meta-analysis. Responses to the questionnaires were mapped into the UNESCO ISCED classification (http://www.uis.unesco.org/Education/ISCEDMappings/Pages/default.aspx), resulting in a quantitative measure that ranges from a minimum of 10 years to a maximum of 20 years. For consistency, the same recoded data were used for the analyses presented here. Probands here are those used in the published meta-analysis (*8*) who have at least one parent was also genotyped and yob ≥ 1940. Maximum yob is 1983, which ensures that most of the probands would have already acquired their highest lifetime educational attainment.

Age at first child (AGFC). This information came from a genealogical database of Iceland that has been utilized for genetics studies performed by deCODE genetics (*36-38*). This database is constantly updated. Currently, the deCODE Genetics genealogical database contains essentially all of approximately 317,000 living Icelanders and the vast majority of their ancestors going back to about 1650 and a smaller portion of ancestors prior to that time. Here, probands, apart from having at least one child, were selected to have at least one parent also genotyped and yob between 1940 and 1975. The 1975 upper cutoff ensures that the number of children they have now would be very highly correlated with the number of children they would have lifetime.

HDL, BMI, FG, HT CPD. General descriptions about these quantitative traits and their ascertainment can be found in previous publications (*19-23*). The sample sizes here correspond to current data and the probands were selected to to have at least one parent also genotyped and yob between 1940 and 1989. The 1989 upper cutoff ensures that data were taken from adults.

Composite Health Trait (HLTH). The probands here are the union of the probands for the traits HDL, BMI, FG, HT and CPD. For HLTH, we think of the traits as HDL, -BMI, -FG, HT, and –CPD. The negative signs added to BMI, FG and CPD ensures that a positive value is associated with better health. For a proband, we summed the trait values for the traits with data, and divided it by the square root of the number of traits we summed. For example, if data were available for HDL, BMI and FG, we computed (HDL-BMI-FG)/$\sqrt{3}$. Because the individual traits have been standardized, this quantity has a nominal variance of one. The actual variance is higher (1.23) because the traits are positively correlated. When the association analyses with the

polygenic scores were performed, this composite trait was processed just like the other traits (see below), *i.e.* adjusted for sex, yob, sex/yob interactions, and 100 PCs and then standardized.

Meta-analysis and polygenic scores

A detailed description of the markers and associated weights used to compute the EA polygenic scores can be found in the Methods section of a recent publication (*39*). The only difference here is that the polygenic scores were computed for both the transmitted and non-transmitted alleles, and for the maternal and paternal alleles separately. A brief description of the calculations is given here. After the exclusion of the Icelandic samples and the samples from 23andMe, meta-analysis results based on 278,948 samples taken from (*8*) are used. The 23andMe results were excluded because their policy forbids the release of full GWAS results. The Icelandic results were excluded to avoid confounding/bias and/or overfitting. For the 120 genomewide significant markers, the estimated effects on educational attainment (used in Supplementary Table 1) did incorporate the 23andMe data, and were based on 355,103 samples. The basic method used to process the genotype data for Icelanders, including imputations based on full-genome sequencing results, was described by Gudbjartsson (*40*). A framework set of approximately 618,762 high quality SNPs covering the whole genome were used to compute the polygenic scores. The weights for computing the polygenic scores were based on results obtained from the meta-analysis as described above, and adjusted for linkage disequilibrium using LDpred (*14*). Linkage disequilibrium between markers were estimated using the Icelandic samples.

Results in Supplementary Tables S2 and S3 are for polygenic scores calculated in the same way as above, with the only difference that 21,411 SNPs that are in or close to known imprinted regions(*24*) were eliminated. The HT and BMI polygenic scores used to generate results in

Supplementary Tables S5 to S8 were also calculated in the same only. The only difference is that the association results were either taken from a height GWAS(*31*) or a BMI GWAS(*32*).

Regression analyses and missing data

We started by processing the trait values and the polygenic scores separately. For the quantitative traits, we began by calculating the values with adjustment for sex, yob up to the third power, interactions between sex and the yob terms, and 100 PCs. (The PC loadings were calculated based on the data of 71,510 uncorrelated/weakly-correlated SNPs of 10,000 Icelanders.) This was done through regressing the raw trait values on the other variables and taking the residuals. The adjusted values were then standardized to have variance one. The polygenic scores were processed in a similar way with two extra details. For the transmitted polygenic scores, the standardization was done so that $poly_T$ has variance 1. The adjusted (but before standardization) values of $poly_{TP}$ and $poly_{TM}$ were divided by the same constant used to standardize $poly_T$. As a result the final values of $poly_{TP}$ and $poly_{TM}$ each has variance approximately equal to one-half. For probands with the father also genotyped, $poly_{NTP}$ were similarly adjusted and standardized. A final value of zero was imputed for those whose father was not genotyped. Note that this means that the values of $poly_{NTP}$ have mean zero and variance approximately equal to one-half when restricted to probands with fathers genotyped, and have variance approximately equal to one-half times the fraction of fathers genotyped when all probands are considered. $Poly_{NTM}$ was calculated similarly. This way, the estimated effects of the non-transmitted polygenic scores would be directly comparable to the estimated effects of the transmitted polygenic scores. The association results given in the paper were obtained by applying regression to the adjusted and standardized values of the traits and the polygenic scores.

Genomic control

For the statistical analyses performed, to adjust for the relatedness between the probands, standard errors and *P-values* were computed/adjusted using a genomic control (*41*) method based on applying LD Score regression (*16*) to 1.1 million SNPs spanning the genome. Each SNP was processed in a way that matched the analyses of the corresponding polygenic scores. For example, to obtain adjustment factors for the results in Table 1, the transmitted and non-transmitted alleles/genotypes of a SNP, adjusted and standardized, were entered jointly in a multiple regression. Missing data were treated in the say way as with the polygenic scores. From these results, separate adjustment factors were computed for the transmitted and non-transmitted polygenic scores. *P* values given are 2-sided unless explicitly stated otherwise.

Calculating the *P*-value for a result obtained for the 120 SNPs

For the 120 SNPs that are genomewide significant in the Iceland-excluded meta-analysis, fifteen (12.5%) have one-sided $P < 0.05$ (calculated with genomic control) for the non-transmitted allele, when six are expected under the null hypothesis. If the SNPs were uncorrelated, a binomial test would give a *P* of 9.9×10^{-4}. However, some of the SNPs are correlated. To calculate the proper *P*, we simulated a completely random trait for the 21,637 probands 100,000 times. For each of the simulation, we performed the same analysis that was performed for EA. For the 100,000 simulated traits, 147 have 15 or more SNPs with $P < 0.05$ for the non-transmitted alleles. Thus the empirical *P* is $147/100000 = 1.47 \times 10^{-3}$, which rounded to 1.5×10^{-3}.

Estimating the confounding effects induced by assortative mating

Here we show how the relative assortative-mating induced confounding effects ϕ_δ/δ and ϕ_η/η are estimated. The mathematical consequences of assortative mating have been well studied (*9*), but for outbred populations such as humans, estimating the effects related to assortative mating remains a major challenge as many key parameters/variables are unknown/unobserved. Moreover, the degree and nature of assortative mating could have been changing over time, *i.e.* different for different generations, further complicating estimation. In particular, note that while the associations between polygenic scores and EA in the main text are calculated for the probands, assortative mating is mainly focused on the parents. We start by establishing notation and terminology. The full genetic propensity to EA is partitioned into two orthogonal (e.g. independent within a person under a scenario with no assortative mating) components A and B, where A stands for the EA polygenic score that is used in the manuscript. Specifically, let A_{TP}, A_{NTP}, A_{TM}, and A_{NTM} denote respectively $poly_{TP}$, $poly_{NTP}$, $poly_{TM}$, and $poly_{NTM}$. Let B_{TP}, B_{NTP}, B_{TM}, and B_{NTM} be similarly defined. We use cor(.) to denote expected correlation, and *r*(.) to denote empirical correlations calculated from data. For here, correlations are assumed to be calculated for trait values with adjustment for sex, yob, and 100 principal components. We adopt a model that makes the following simplifying assumptions (ASMPs):

I. The P (paternal) genetic scores (A or B, T or NT) are conditionally independent of the M (maternal) genetic scores given some parental phenotypes Y_P and Y_M. As a consequence, for example, $cor(A_{TP}, B_{NTM}) = cor(A_{TP}, Y_P) \times cor(Y_P, Y_M) \times cor(Y_M, B_{NTM})$. Y_P and Y_M need not be equal to EA_P and EA_M, the educational attainments of

the father and mother. For the simplicity of calculation and presentation, we assume the Ys are scalars.

II. Symmetry between the P and M components. For example, cor(A_{TP}, Y_P) is the same as cor(A_{TM}, Y_M). To make this assumption more plausible, it is helpful to think of Y as having adjusted for covariates within sex.

III. Symmetry between the T and NT components w.r.t. their correlations with Y of the corresponding parent. For example, cor(A_{TP}, Y_P) = cor(A_{NTP}, Y_P). This symmetry follows from the assumption of random transmission.

IV. When Y_P and Y_M are not EA_P and EA_M, their correlations with the A and B scores can be bigger or smaller than the correlation between the A and B scores and EA_P and EA_M, but the relative effects of the A and B scores are the same. For example, cor(A_{TP}, Y_P) can be different from cor(A_{TP}, EA_P), but cor(B_{TP}, Y_P)/cor(A_{TP}, Y_P) = cor(B_{TP}, EA_P)/cor(A_{TP}, EA_P).

V. The ratio of the nurturing effect and the direct effect is the same for A and B. Specifically, we assume that $(\eta_B/\delta_B) = (\eta/\delta)$ where η_B and δ_B denote the nurturing and direct effects of the B components, and η and δ, as in the main text, denote the nurturing effects of the A components.

VI. Within the same parental origin, the T and NT components are independent, *i.e.* (A_{TP},B_{TP}) is independent of (A_{NTP},B_{NTP}), and (A_{TM},B_{TM}) is independent of (A_{NTM},B_{NTM}).

Given that the B components are not directly observed and Y_P and Y_M are unknown/unobserved, most of these assumptions are made to make estimation possible with limited data. We believe that modest deviations from these assumptions would only have a second order effect on the results. ASMP VI, however, is different. This assumption essentially implies that, while assortative mating with respect to (A, B) took place in the parents' generation, it did not or was negligible in the grandparents' generation. Using the language of the main text and Fig. 2, this corresponds to not having *trans* correlation in the parents' generation. Not making this assumption would introduce much complications to the mathematics. However, it is an assumption that has to be verified to be at least not clearly false. We did that by calculating the empirical correlations $r(A_{TP},A_{NTP})$ and $r(A_{TM},A_{NTM})$. While these correlations are positive without any principal component (PC) adjustment, their average is negative (-1.1×10^{-3}), but not significantly so, after adjustments for 100 PCs. This suggests that, in the grandparents' generation, assortative mating with respect to the EA propensity (A,B) was mainly driven by population structure such as geography. As we will see, that is not the case of for the parents' generation. For reference, it is noted that the average yob of the probands, their parents, and their grandparents is respectively, 1958.4, 1930.1 and 1898.2.

To simplify the mathematics, assume the B (T and NT) components are scaled to have the same variances as the corresponding A (T and NT) components. In particular, let

$$var(A_{TM}) = var(A_{TP}) = \cdots = var(B_{TP}) = var(B_{NTM}) = var(B_{NTP}) = v.$$

Let $A_T = A_{TM} + A_{TP}$ and $A_{NT} = A_{NTM} + A_{NTP}$. For simplicity, ignoring the nurturing effect for now, let

$$X = \delta(A_{TM} + A_{TP}) + \delta_B(B_{TM} + B_{TP}) + \epsilon$$

where ϵ is variation independent of the A and B components. Based on this model and the above assumptions,

$$var(A_T) = var(A_{NT}) = 2v[1 + cor(A_{TM}, A_{TP})],$$

$$cov(A_T, A_{NT}) = 2vcor(A_{TM}, A_{TP}),$$

$$cov(X, A_T) = 2v[\delta[1 + cor(A_{TM}, A_{TP})] + \delta_B cor(A_{TM}, B_{TP})],$$

and

$$cov(X, A_{NT}) = 2v[\delta cor(A_{TM}, A_{TP}) + \delta_B cor(A_{TM}, B_{TP})].$$

When X is regressed on A_T and A_{NT} jointly, going through the multiple regression algebra, it can be shown that the fitted coefficients for A_T and A_{NT} have expectations $(\delta + \phi_\delta)$ and ϕ_δ respectively, where

$$\phi_\delta = \frac{\delta_B cor(A_{TM}, B_{TP})}{1 + 2cor(A_{TM}, A_{TP})}.$$

The main term $\delta_B$cor(A_{TM},B_{TP}) is what one can easily appreciate by focusing on the correlation between A_{TM} and B_{TP} alone. The actual confounding effect is somewhat reduced due to the multiplicative term $[1 + 2cor(A_{TM}, A_{TP})]^{-1}$, which arises because the A components are correlated, usually weakly, with each other. This tends to be a very modest adjustment term, and so one should not be too distracted by it when trying to gain a basic understanding of what is going on, e.g. in our case, the results would not change meaningfully if this adjustment term is ignored. Let π be the ratio of the variance of EA explained by the direct effect of the B components versus the variance of EA explained by the direct effect of the A components.

Because variance explained is proportional to the effect squared, $\delta_B = \delta\sqrt{\pi}$. Similar arguments lead to

$$cor(A_{TM}, B_{TP}) = \sqrt{\pi} cor(A_{TM}, A_{TP}).$$

It follows that

$$\phi_\delta = \frac{\delta_B cor(A_{TM}, B_{TP})}{1 + 2cor(A_{TM}, A_{TP})} = \frac{\delta\sqrt{\pi}\sqrt{\pi} cor(A_{TM}, A_{TP})}{1 + 2cor(A_{TM}, A_{TP})} = \frac{\delta\pi cor(A_{TM}, A_{TP})}{1 + 2cor(A_{TM}, A_{TP})},$$

and

$$\phi_\delta/\delta = \frac{\pi cor(A_{TM}, A_{TP})}{1 + 2cor(A_{TM}, A_{TP})}.$$

Thus an estimate of ϕ_δ/δ can be obtained if we have estimates for π and cor(A_{TM}, A_{TP}). The direct effect of the full EA genetic component is estimated (*17*) to explain 17.0% of the variance of EA. Given that the direct effect of polygenic score (A) is estimated to explain 2.45% of the trait variance, B is estimated to explain 17.0%-2.45% = 14.55% of the variance. Hence (14.55/2.45) = 5.94 is an estimate of π. To estimate cor(A_{TM}, A_{TP}), we use data from the 21637 EA probands and their parents to calculate the empirical correlations *r*(A_{TP}, A_{TM}), *r*(A_{TP}, A_{NTM}), *r*(A_{NTP}, A_{TM}), and *r*(A_{NTP},A_{NTM}), which, because of ASMPs II and III, all have expectation equal to cor(A_{TM}, A_{TP}). The four *r*'s are calculated with sample sizes of 21637, 19012, 13948, and 11323. The sample sizes vary because, out of the 21637 probands, 19012 have the mother genotyped, 13948 have the father genotyped, and 11323 have both parents genotyped. Their weighted average, with the weights proportional to the sample sizes, is 0.0112. Combining this with the estimate of π, ϕ_δ/δ is estimated to be

$$\frac{5.94 \times 0.0112}{1 + 0.0224} = \frac{0.0665}{1.0224} = 0.065,$$

the value presented in the main text.

Here we explore what would result if we make the assumption that $Y_P = EA_P$ and $Y_M = EA_M$. For 6513 of the 21,637 probands, the EAs of both parents are known, and after adjusting for the yob and 100 PCs (of the proband), the $r(E_{AP}, E_{AM}) = 0.33$. For the 5384 unique fathers of the probands who are genotyped and for whom we have EA data, $r^2(A_{TP}+A_{NTP}, EA_P) = 0.0335$. Similarly, for the 7474 unique mothers of the probands who are both genotyped and for whom we have EA data, $r^2(A_{TM}+A_{NTM}, EA_M) = 0.0225$. Using these numbers to estimate $cor(A_{TP}+A_{NTP}, EA_P)$ and $cor(A_{TM}+A_{NTM}, EA_M)$,

$$\pi cor(A_{TM}, A_{TP}) = \pi cor(A_{TM}, Y_M) cor(Y_M, Y_P) cor(Y_P, A_{TP})$$

would be estimated as

$$\frac{5.94 \times \sqrt{0.0225/2} \times 0.33 \times \sqrt{0.0335/2}}{1 + 2 \times \sqrt{0.0225/2} \times 0.33 \times \sqrt{0.0335/2}} = \frac{0.0269}{1.009} = 0.027,$$

less than half the estimate 0.065 that we actually use. These results suggest that (i) the correlation between the genetic propensity to EA and EA has been increasing over time, and (ii) the correlations between the father's and mother's EA propensities cannot be fully accounted for by mating selection through the EA traits alone. Most importantly, these results suggest that if our estimate of ϕ_δ/δ (0.065) ends up to be on the low side, it is unlikely to be very far off.

The value of the ratio (ϕ_η/η) can be derived in the same way as above. Indeed, because in addition to B_{TP} and B_{TM}, B_{NTP} and B_{NTM} also have nurturing effects,

$$(\phi_\eta/\eta) = 2 \times (\phi_\delta/\delta).$$

Thus, for EA, (ϕ_η/η) is estimated as $0.065 \times 2 = 0.130$. Given that $\hat{\theta}_{NT}$ is an estimate of $\phi_\delta + \eta + \phi_\eta$, with estimates for (ϕ_δ/δ) and (ϕ_η/η), individual estimates for ϕ_δ, η, and ϕ_η can be calculated. For the other traits highlighted in Table 1, these estimates were similarly calculated using the specific data for each trait.

In Supplementary Tables S5 and S7, the estimates of the confounding effects for the height polygenic scores were similarly computed.

Estimating η_P and η_M

We started by estimating $(\eta_M - \eta_P)$ using a weighted average of $(\hat{\theta}_{TM} - \hat{\theta}_{TP})$ and $(\hat{\theta}_{NTM} - \hat{\theta}_{NTP})$. We could have used the simple average, but it is suboptimal because the effective sample size for estimating $(\hat{\theta}_{NTM} - \hat{\theta}_{NTP})$ is smaller than that for estimating $(\hat{\theta}_{TM} - \hat{\theta}_{TP})$ because some parents are not genotyped. We used weights proportional to (standard error)$^{-2}$. For the eight phenotypes studied, the weight of $(\hat{\theta}_{NTM} - \hat{\theta}_{NTP})$ is within $71.5 \pm 2.5\%$ of the weight for $(\hat{\theta}_{TM} - \hat{\theta}_{TP})$. Similarly, we treated $\hat{\eta}$ as an estimate of a weighted average of η_P and η_M with weights proportional to the number of fathers and number to mothers genotyped. Combining these results, we solved two linear equations with two unknowns to calculate $\hat{\eta}_P$and $\hat{\eta}_M$.

Effects of genetic nurture on phenotypic correlation between relatives

We consider the effect of an individual variant and consider the model

$$X = (\delta + \eta)a_P + (\delta + \eta)a_M + \eta a_{PNT} + \eta a_{MNT} + \epsilon$$

where a_P and a_M are respectively the transmitted paternal and maternal alleles, and a_{PNT} and a_{MNT} are the corresponding non-transmitted alleles. Here $\in$ includes both the non-genetic component and the genetic component excluding the as. The main simplifying assumptions are that ϵ is independent of the as, and the as are independent of each other (so assortative mating is not incorporated here). The purpose here is to calculate how much genetic nurture amplifies the contribution of the direct effect with respect to various measures. The first measure (measure i) is the variance explained by the two transmitted alleles, which is

$$2f(1-f) \times (\delta + \eta)^2 = 2f(1-f)\delta^2 \times (1+\rho)^2 = \Delta \times (1+\rho)^2$$

where $\rho = \eta/\delta$, and $\Delta = 2f(1-f)\delta^2$ is the contribution if $\eta = 0$. Thus $(1+\rho)^2$ is the multiplicative amplifying factor with a non-zero η. The second measure (measure ii) is the contribution of the all four alleles, the transmitted and the non-transmitted, which can be shown to be

$$\Delta \times [(1+\rho)^2 + \rho^2] = \Delta \times [1 + 2\rho + 2\rho^2].$$

The third measure (measure iii) is two times the 'induced' correlation, *i.e.* correlation that can be attributable to the variant, between the phenotypes of a child and a parent. Here we assume that the phenotypes are scaled to each have variance 1 (so covariance equals correlation). We define this measure as two times the induced correlation so that it will give the same value as the other measures when $\eta = 0$. To calculate this, note that a parent has two alleles (each having both a direct effect and a genetic nurturing effect for the parent), one transmitted to the offspring (having both a direct effect and a genetic nurturing effect for the offspring) and one non-transmitted (only having a genetic nurturing effect on the offspring). It is then not difficult to see that measure iii equals

$$2f(1-f)[(\delta+\eta)^2+(\delta+\eta)\eta] = \Delta[(1+\rho)^2+(1+\rho)\rho]$$

$$= \Delta[1+3\rho+2\rho^2].$$

The fourth measure (measure iv) is two times the induced correlation between (full) sibling pairs. Out of the four alleles in the parents, on average, one would be transmitted to both siblings, two would transmitted to one sib but not the other, and one would not be transmitted to either of the sib. It follows that measure iv equals

$$\Delta[(1+\rho)^2+2(1+\rho)\rho+\ \rho^2] = \Delta[1+4\rho+4\rho^2].$$

We note that Young et al (*17*) has derived a general expression for all relative types. Also, a fifth measure (measure v) not included in Fig. 3 is two times the induced correlation for MZ twins minus the induced correlation for DZ twins, one of the standard estimates of heritability. Here DZ twins are treated just like full sibs. For MZ twins, they share two transmitted alleles which have effects $(\delta+\ \eta)$ for both individuals, and two non-transmitted alleles which have effect η only for both. It follows that measure v equals

$$\Delta[2(1+\rho)^2+\ 2\rho^2-(\ 1+4\rho+4\rho^2)] = \Delta[2+4\rho+4\rho^2-(\ 1+4\rho+4\rho^2)] = \Delta.$$

So the genetic nurturing effect is cancelled out for this measure. This is not surprising as genetic nurture here is assumed to manifest through nurturing from the parents (and ancestors), and the twin-based estimate of heritability is designed to cancel out shared environmental effects that include such nurturing. However, this no longer holds if, as suggested a recent study (*26*), the outcome of a proband can be affected by the behaviour/outcome of a sibling. The latter is not necessarily that surprising. In addition to the human study, it has been observed that genotypes of cage mates can affect the outcomes of a mouse (*42*). In that case, the genetic nurturing effect manifested through a twin would not cancel for measure v. To see that, suppose there is no

genetic nurture through the parents, but there is a genetic nurturing effect η_S manifested though a twin/sibling, *i.e.* a parental allele that is not transmitted to the sib/twin of a proband will not have any genetic nurturing effect on the proband. In that case, MZ twins share two (transmitted) alleles which has effect $(\delta + \eta_S)$ for both twins. For DZ twins, they on average share one such alleles. On average, there are two alleles transmitted to one twin and not the other. They will have effect δ for one twin (the twin carrying it) and effect η_S for the other twin. On average, the one allele that is not transmitted to either twin would have no effect at all on both. In that case, denoting η_S/δ by ρ_S, measure v equals

$$\Delta\{\, 2(1+\rho_S)^2 - [(1+\rho_S)^2 + \, 2\rho_S]\} = \Delta\{(1+\rho_S)^2 - 2\rho_S\} = \Delta\{1+\rho_S^2\}.$$

So here the genetic nurturing effect does not cancel. This is merely a simplified example. If there is a genetic nurturing effect going through siblings and twins that is not negligible, then modelling heritability can become really complicated as, unlike the number of biological parents, the number of siblings and other related factors such as age and sex distributions vary. The most important point, however, is that for a trait like educational attainment, it could be a mistake to believe that the twin-based heritability estimate of heritability is always only capturing the direct effect. Indeed, the calculations here assume an additive model. For twins growing up together, the existence of interaction terms would not be surprising.

Supplementary Table S1. The associations with EA, in the Icelandic data, for the transmitted and non-transmitted alleles of 120 SNPs that are genomewide significant based on an EA meta-analysis that does not include Icelandic data. Results are presented for alleles with positive EA effect in the meta-analysis. P_{GWAS} is *P*-value from the meta-analysis. P_T and P_{NT} are one-sided *P*-values for the transmitted and non-transmitted alleles in the Icelandic data. Eff_T and Eff_{NT} are estimated effects, per allele, in the Icelandic data.

SNP	Chr	Position	Allele	P_{GWAS}	$Eff_T \times 100$	P_T	$Eff_{NT} \times 100$	P_{NT}
rs10798888	chr1	31733498	G	2.4e-08	-0.75	0.68	-3.43	0.97
rs56044892	chr1	41364414	C	1.9e-09	0.52	0.36	-0.47	0.61
rs12076635	chr1	43560985	C	4.4e-12	0.36	0.40	-3.40	0.98
rs12410444	chr1	43723048	G	2.7e-12	1.88	0.07	-1.65	0.88
rs12143094	chr1	71639693	C	3.0e-08	3.86	0.057	8.21	0.0012
rs34305371	chr1	72267927	A	7.1e-17	6.97	2e-04	3.21	0.074
rs2568955	chr1	72296486	C	2.4e-08	3.63	0.0039	2.33	0.066
rs12142680	chr1	73150209	A	1.5e-08	3.45	0.034	0.42	0.42
rs1008078	chr1	90724174	C	7.1e-14	2.73	0.011	-0.01	0.50
rs12134151	chr1	95736887	G	5.9e-09	2.06	0.038	-1.12	0.81
rs4378243	chr1	97930325	T	5.0e-09	1.39	0.17	1.67	0.15
rs648163	chr1	199346870	T	9.6e-09	0.55	0.35	-1.47	0.83
rs11588857	chr1	204617919	A	4.8e-13	0.89	0.27	0.43	0.40
rs35771425	chr1	211436426	T	4.8e-09	3.93	0.0019	3.44	0.011
rs2992632	chr1	243340462	A	5.0e-10	1.62	0.10	0.65	0.33
rs76076331	chr2	10837459	T	8.6e-10	2.84	0.03	-2.87	0.96
rs17504614	chr2	50853343	T	3.8e-09	1.15	0.21	1.45	0.18
rs1606974	chr2	51646461	A	3.3e-12	0.10	0.48	0.16	0.47
rs7593947	chr2	60477798	A	5.9e-11	0.12	0.46	0.84	0.26
rs356992	chr2	60526458	C	1.4e-11	0.39	0.38	-0.06	0.52
rs6715849	chr2	99689916	G	2.7e-09	3.99	0.00033	-0.43	0.63
rs12987662	chr2	100205086	A	1.4e-22	4.83	3.1e-05	1.47	0.14
rs17824247	chr2	143394970	C	9.4e-10	3.20	0.0033	0.39	0.38
rs10178115	chr2	154595226	T	4.2e-10	0.51	0.33	0.76	0.28
rs16845580	chr2	161064373	T	4.7e-12	1.20	0.15	2.71	0.019
rs4500960	chr2	161962111	C	1.6e-08	2.06	0.038	2.00	0.06
rs1596747	chr2	192937752	A	1.8e-09	1.11	0.17	0.18	0.44
rs12694681	chr2	225744525	T	2.5e-08	1.35	0.14	-0.59	0.67
rs11687170	chr2	236149500	T	1.5e-08	4.20	0.0039	-0.11	0.52

rs7429990	chr3	47860313	C	1.2e-08	0.04	0.49	3.14	0.015
rs62263033	chr3	48328934	T	3.4e-08	1.66	0.29	1.85	0.28
rs3172494	chr3	48694054	T	4.8e-09	4.29	0.014	1.01	0.32
rs55786114	chr3	48944902	C	5.6e-10	0.52	0.41	2.51	0.16
rs13090388	chr3	49353649	T	6.4e-22	4.74	5.8e-05	1.00	0.23
rs11130222	chr3	49863627	A	8.7e-23	4.66	3.9e-05	1.24	0.17
rs112634398	chr3	50038061	A	5.2e-10	6.01	0.0049	0.30	0.46
rs7610856	chr3	71529871	A	3.4e-09	0.12	0.46	-0.93	0.76
rs62263923	chr3	85625640	G	1.6e-12	2.97	0.0059	1.40	0.14
rs9755467	chr3	127425042	T	1.6e-09	1.07	0.26	-1.73	0.83
rs12646808	chr4	3248101	T	3.8e-10	0.58	0.32	0.43	0.38
rs1967109	chr4	28719293	G	3.3e-09	-0.21	0.55	-0.53	0.61
rs4308415	chr4	66956156	G	5.2e-10	1.05	0.19	-0.07	0.52
rs6839705	chr4	105223578	A	2.8e-09	3.40	0.0031	-0.03	0.51
rs4863692	chr4	139842970	T	1.4e-10	2.51	0.023	0.73	0.30
rs1912528	chr4	140024812	T	3.7e-09	1.42	0.12	0.16	0.45
rs12640626	chr4	175705121	A	1.2e-08	-0.63	0.70	1.07	0.21
rs4493682	chr5	45187922	C	3.6e-10	2.93	0.047	0.42	0.41
rs1562242	chr5	58270667	C	2.3e-08	0.37	0.38	-0.09	0.53
rs61160187	chr5	60815752	G	1.1e-10	4.21	0.00018	-0.56	0.66
rs113474297	chr5	61259107	T	5.6e-09	3.59	0.017	2.59	0.081
rs10223052	chr5	61504509	A	5.1e-09	3.52	0.0016	0.41	0.38
rs12653396	chr5	88551455	T	3.2e-09	1.32	0.13	0.33	0.40
rs6882046	chr5	88673046	G	2.9e-12	5.32	8.9e-05	1.57	0.16
rs660001	chr5	114530901	G	1.9e-10	1.34	0.19	-0.27	0.57
rs7776010	chr6	14723377	C	6.6e-11	5.52	0.00019	2.29	0.095
rs6939294	chr6	16950400	T	3.1e-09	2.88	0.022	0.54	0.37
rs2179152	chr6	26325660	C	3.5e-08	3.25	0.0032	1.05	0.21
rs56231335	chr6	97739415	C	1.9e-12	2.53	0.019	0.75	0.29
rs1338554	chr6	97898925	A	1.9e-11	0.48	0.34	0.79	0.27
rs9401593	chr6	98101925	C	4.1e-27	2.02	0.042	-0.06	0.52
rs11756123	chr6	151896944	T	3.4e-11	2.22	0.032	1.30	0.16
rs12702087	chr7	44773008	A	7.5e-09	0.57	0.31	0.05	0.48
rs756912	chr7	72276812	C	1.8e-08	2.52	0.015	1.01	0.22
rs12534506	chr7	93033013	T	4.1e-10	0.70	0.27	0.71	0.29
rs148490894	chr7	99934132	A	1.5e-08	-1.01	0.61	0.56	0.45
rs11771168	chr7	114264006	C	4.4e-08	2.06	0.068	0.54	0.36
rs113520408	chr7	128762728	A	3.5e-08	1.47	0.13	-0.48	0.63
rs17167170	chr7	133617591	A	5.7e-12	0.28	0.42	0.44	0.39
rs7791133	chr7	135552348	C	3.5e-08	1.71	0.076	0.51	0.35
rs320700	chr7	137364731	A	4.1e-08	2.06	0.046	2.30	0.046
rs1106761	chr8	141609134	G	2.8e-10	3.29	0.0025	3.34	0.0056
rs11774212	chr8	144461122	T	8.0e-13	0.03	0.49	-1.46	0.87

rs11998763	chr9	1787687	A	3.7e-14	0.00	0.50	2.53	0.026
rs4741343	chr9	14075096	G	4.4e-09	2.77	0.039	2.09	0.12
rs4741351	chr9	14222783	G	1.0e-10	1.28	0.15	-0.47	0.63
rs7029201	chr9	23358083	A	7.1e-23	4.15	2e-04	2.87	0.014
rs7033137	chr9	69440242	C	3.5e-09	0.70	0.30	-1.63	0.86
rs17425572	chr9	85391423	A	6.4e-10	1.09	0.17	-1.86	0.92
rs10818606	chr9	121856107	C	1.9e-08	3.06	0.005	-1.06	0.79
rs10430506	chr10	66100562	G	3.0e-08	-1.16	0.78	-0.71	0.67
rs149613931	chr10	101790524	G	1.6e-08	2.46	0.20	-0.41	0.55
rs73344830	chr10	102057071	A	8.5e-11	2.63	0.012	0.49	0.36
rs10786662	chr10	102230055	G	9.8e-15	1.14	0.16	0.43	0.37
rs12768205	chr10	102888092	C	1.3e-08	0.73	0.28	0.48	0.37
rs4930349	chr11	66252465	A	1.2e-08	-0.57	0.67	-0.90	0.74
rs76878669	chr11	66325096	C	2.7e-08	-0.69	0.70	1.05	0.24
rs644799	chr11	95831095	G	3.5e-08	0.26	0.41	-0.53	0.66
rs111321694	chr11	111079662	C	4.8e-08	1.07	0.26	0.13	0.47
rs79925071	chr11	122127545	T	2.5e-08	1.65	0.082	1.84	0.083
rs1550973	chr11	131421834	G	3.5e-09	1.17	0.17	1.32	0.17
rs7964899	chr12	14442822	A	1.7e-10	2.88	0.0069	2.49	0.029
rs67193874	chr12	15367795	G	9.4e-09	0.50	0.35	-1.16	0.80
rs2456973	chr12	56023144	C	6.3e-14	2.12	0.041	3.38	0.0064
rs10773002	chr12	123262414	A	4.7e-17	1.48	0.13	0.04	0.49
rs8002014	chr13	57784025	G	1.6e-18	4.65	0.00016	-1.80	0.89
rs9556958	chr13	98447792	C	3.7e-12	-0.77	0.75	-0.77	0.72
rs34344888	chr14	22918376	G	1.0e-10	2.74	0.01	3.53	0.004
rs1115240	chr14	26621182	G	1.3e-08	2.82	0.018	0.86	0.28
rs10483349	chr14	29160250	G	4.7e-10	0.61	0.34	2.46	0.067
rs242093	chr14	69014626	G	2.0e-08	0.35	0.38	-2.89	0.99
rs58694847	chr14	84450167	G	7.1e-10	3.29	0.0083	2.87	0.030
rs1378214	chr15	47286807	C	2.5e-09	3.23	0.0036	2.75	0.020
rs6493271	chr15	47321396	T	1.2e-09	2.01	0.094	-0.04	0.51
rs281302	chr15	47394465	G	5.3e-10	1.85	0.054	-1.69	0.90
rs12900061	chr15	65716910	A	2.3e-09	2.76	0.029	-0.22	0.55
rs4076457	chr15	77714871	T	1.3e-08	1.46	0.14	1.6	0.14
rs28420834	chr15	82220780	G	1.0e-09	0.99	0.20	1.01	0.22
rs4984541	chr15	96367910	G	4.1e-08	-0.03	0.51	1.22	0.20
rs8049439	chr16	28826194	T	2.3e-10	0.75	0.26	-0.74	0.71
rs11643654	chr16	51149817	A	3.4e-09	1.20	0.16	0.48	0.36
rs192818565	chr17	45914149	T	6.5e-11	2.75	0.034	-1.16	0.76
rs9964724	chr18	37579161	T	4.2e-14	2.15	0.041	-0.86	0.73
rs12956009	chr18	47241653	C	3.4e-09	0.23	0.42	0.62	0.32
rs62100765	chr18	53209048	C	2.7e-11	1.06	0.18	0.33	0.40
rs1382358	chr19	13060610	T	2.0e-08	1.32	0.20	1.72	0.16
rs111730030	chr19	13158012	T	4.3e-08	2.96	0.081	5.56	0.0089

rs78387210	chr20	49206904	T	2.2e-08	1.23	0.28	4.90	0.016
rs6065080	chr20	61257735	C	2.8e-08	-0.22	0.57	0.47	0.36
rs35532491	chr22	33933614	T	1.6e-08	2.80	0.044	0.97	0.30
rs7286601	chr22	50682988	G	1.0e-09	0.81	0.24	-0.35	0.61
Mean					1.84		0.63	

Supplementary Table S2. Association results for a modified EA polygenic score that excluded SNPs in or close to known imprinted regions

				Transmitted			Nontransmitted						
				T ($T = T_P + T_M$)			NT ($NT = NT_P + NT_M$)						
Trait	**N**	**N_{NTP}**	**N_{NTM}**	$\widehat{\theta}_T$	*P*	R^2 (%)	$\widehat{\theta}_{NT}$	*P*	R^2_δ (%)	$\widehat{\delta}\ /\widehat{\theta}_T$	$\widehat{\phi}_\delta / \widehat{\theta}_T$	$\widehat{\eta} / \widehat{\theta}_T$	$\widehat{\phi}_\eta / \widehat{\theta}_T$
EA	21637	13948	19012	0.223	1.6×10^{-173}	4.95	0.067	1.3×10^{-14}	2.42	0.700	0.04	0.234	0.027
AGFC	54372	35294	47052	0.108	9.4×10^{-110}	1.17	0.037	4.5×10^{-12}	0.51	0.659	0.051	0.251	0.039
HDL	46872	30855	40788	0.063	2.1×10^{-27}	0.40	0.025	3.1×10^{-5}	0.15	0.609	0.048	0.297	0.046
BMI	39078	26433	34533	-0.059	6.4×10^{-22}	0.35	-0.015	0.021	0.20	0.751	0.059	0.165	0.026
FG	34767	22959	30222	-0.051	1.9×10^{-17}	0.26	-0.018	0.0061	0.11	0.652	0.052	0.255	0.041
HT	39270	26563	34703	0.052	1.1×10^{-13}	0.27	0.032	6.6×10^{-6}	0.04	0.393	0.030	0.501	0.076
CPD	18887	12371	16589	-0.053	1.5×10^{-11}	0.28	-0.029	8.4×10^{-4}	0.06	0.454	0.032	0.450	0.064
HLTH	62328	41996	54546	0.080	8.6×10^{-58}	0.64	0.032	5.9×10^{-10}	0.23	0.602	0.053	0.294	0.052

Results here were obtained in the same way as those in Table 1 of the main text. The only difference is that, while the original EA polygenic score was computed based on 618,762 SNPs spanning the genome, here 21,411 of those SNPs which are in or close to known imprinted regions are eliminated.

Supplementary Table S3. Parent-of-origin specific effects for a modified EA polygenic score that excluded SNPs in or close to known imprinted regions

	Transmitted				Non-Transmitted						
Trait	$\widehat{\theta}_{TP}$	***P***	$\widehat{\theta}_{TM}$	***P***	$\widehat{\theta}_{NTP}$	***P***	$\widehat{\theta}_{NTM}$	***P***	**Estimate of $\eta_M - \eta_P$**	***P***	$\widehat{\eta}_M / \widehat{\eta}_P$
EA	0.2129	4.3×10^{-89}	0.2322	8.4×10^{-103}	0.0676	2.8×10^{-7}	0.0663	5.1×10^{-9}	0.0109	0.33	1.2
AGFC	0.1002	2.2×10^{-52}	0.1160	6.9×10^{-69}	0.0324	5.8×10^{-5}	0.0402	1.0×10^{-8}	0.0125	0.07	1.6
HDL	0.0608	1.5×10^{-15}	0.0657	1.2×10^{-17}	0.0116	0.19	0.0347	8.6×10^{-6}	0.0126	0.10	2.1
BMI	-0.0584	9.7×10^{-13}	-0.0599	5.3×10^{-13}	-0.0192	0.047	-0.0113	0.18	0.0025	0.76	0.78
FG	-0.0428	1.4×10^{-7}	-0.0584	9.3×10^{-13}	-0.0091	0.35	-0.0241	0.0047	-0.0153	0.07	4.6
HT	0.0364	5.0×10^{-5}	0.0677	8.0×10^{-14}	0.0288	0.005	0.0337	2.4×10^{-4}	0.0202	0.02	2.4
CPD	-0.0376	5.2×10^{-4}	-0.0675	5.6×10^{-10}	-0.0342	0.01	-0.0246	0.032	-0.0132	0.25	1.8
HLTH	0.0691	8.7×10^{-26}	0.0909	1.5×10^{-42}	0.0251	0.0011	0.0371	4.7×10^{-8}	0.0176	0.01	2.3

Results here were obtained in the same way as those in Table 2 of the main text. The only difference is that, same as Supplementary Table S2, the modified polygenic score excluded SNPs in or close to known imprinted regions.

Supplementary Table S4. Effects of the non-transmitted EA polygenic score without and with adjustment for the educational attainment of the parent

				Without adjustment for parents' educational attainment		With adjustment for parents' educational attainment		
Trait	**N**	**N_{NTP}**	**N_{NTM}**	$\widehat{\theta}_{NT}$	***P***	$\tilde{\theta}_{NT}$	***P***	$\widehat{\theta}_{NT}/\tilde{\theta}_{NT}$
EA	17802	10009	14161	0.069	1.6×10^{-11}	0.032	0.0013	0.467
AGFC	35951	19421	26675	0.035	1.0×10^{-6}	0.022	0.0021	0.630
HDL	31209	16978	23283	0.026	9.0×10^{-4}	0.017	0.037	0.634
BMI	29025	16398	22381	-0.027	7.4×10^{-4}	-0.018	0.029	0.652
FG	22484	12123	16636	-0.010	0.25	-0.003	0.69	0.353
HT	29175	16476	22499	0.034	1.2×10^{-4}	0.027	0.0022	0.803
CPD	14397	8004	11184	-0.032	0.0027	-0.026	0.015	0.819
HLTH	41681	22926	31239	0.038	3.7×10^{-8}	0.026	0.00018	0.686

Here, for the NT polygenic score of a parent to be used, the parent had to be both genotyped and with known value for his/her educational attainment. Thus the smaller sample sizes compared to Table 1 in the main text. Otherwise, $\hat{\theta}_{NT}$ and the associated P values were calculated in the same way. Same samples were used to obtain $\tilde{\theta}_{NT}$. The difference is that the educational attainment of the parent was added to the explanatory variables in the regressions. Thus $\tilde{\theta}_{NT}$ is the estimated effect of the non-transmitted EA polygenic score with adjustment for the educational attainment of the parents.

Supplementary Table S5. Association results for the transmitted and non-transmitted HT polygenic scores.

				Transmitted			Nontransmitted						
				$T\ (T = T_P + T_M)$			$NT\ (NT = NT_P + NT_M)$						
Trait	**N**	N_{NTP}	N_{NTM}	$\widehat{\theta}_T$	*P*	R^2 (%)	$\widehat{\theta}_{NT}$	*P*	R^2_{δ} (%)	$\widehat{\delta}\ /\ \widehat{\theta}_T$	$\widehat{\phi}_{\delta}\ /\ \widehat{\theta}_T$	$\widehat{\eta}\ /\ \widehat{\theta}_T$	$\widehat{\phi}_{\eta}\ /\ \widehat{\theta}_T$
EA	21637	13948	19012	0.020	0.012	0.04	0.022	0.016	NA	NA	NA	NA	NA
AGFC	54372	35294	47052	0.012	0.014	0.01	0.009	0.088	NA	NA	NA	NA	NA
HDL	46872	30855	40788	0.005	0.4	0.00	0.009	0.150	NA	NA	NA	NA	NA
BMI	39078	26433	34533	-0.017	0.005	0.03	-0.003	0.630	NA	NA	NA	NA	NA
FG	34767	22959	30222	0.002	0.77	0.00	-0.001	0.890	NA	NA	NA	NA	NA
HT	39270	26563	34703	0.430	$< 10^{-200}$	18.47	0.027	3.4×10^{-5}	16.24	0.938	0.055	0.006	0.001
CPD	18887	12371	16589	-0.009	0.24	0.01	-0.004	0.660	NA	NA	NA	NA	NA
$HLTH_{-HT}$	62300	41974	54522	0.009	0.079	0.01	0.006	0.240	NA	NA	NA	NA	NA

Results here correspond to Table 1 of the main text with three differences. The polygenic scores were computed based on results of a GWAS on height (as opposed to EA). The composite health trait here, $HLTH_{-HT}$, does not include HT, i.e. only values HDL, BMI, FG and CPD have been incoporated. The decomposition results on the right hand of the table are NAs except for the HT trait. This is because for the other traits, the uncertainties (standard errors) associated with $\hat{\theta}_{NT}$ are too large for the decompositions to be meaningful.

Supplementary Table S6. Association results for the transmitted and non-transmitted BMI polygenic scores.

				Transmitted			Nontransmitted						
				T ($T = T_P + T_M$)			NT ($NT = NT_P + NT_M$)						
Trait	**N**	**N_{NTP}**	**N_{NTM}**	$\widehat{\theta}_T$	*P*	R^2 (%)	$\widehat{\theta}_{NT}$	*P*	R^2_δ (%)	$\widehat{\delta}\ /\widehat{\theta}_T$	$\widehat{\phi}_\delta / \widehat{\theta}_T$	$\widehat{\eta} / \widehat{\theta}_T$	$\widehat{\phi}_\eta / \widehat{\theta}_T$
EA	21637	13948	19012	-0.059	2.2×10^{-13}	0.35	-0.021	0.019	NA	NA	NA	NA	NA
AGFC	54372	35294	47052	-0.033	1.1×10^{-11}	0.11	-0.012	0.026	NA	NA	NA	NA	NA
HDL	46872	30855	40788	-0.102	2.5×10^{-69}	1.05	-0.004	0.530	NA	NA	NA	NA	NA
BMI	39078	26433	34533	0.313	$< 10^{-200}$	9.82	0.002	0.780	NA	NA	NA	NA	NA
FG	34767	22959	30222	0.088	3.4×10^{-49}	0.77	0.001	0.930	NA	NA	NA	NA	NA
HT	39270	26563	34703	-0.017	0.013	0.03	0.000	0.980	NA	NA	NA	NA	NA
CPD	18887	12371	16589	0.049	4.8×10^{-10}	0.24	0.017	0.053	NA	NA	NA	NA	NA
$HLTH_{-BMI}$	62328	41996	54546	-0.079	1.2×10^{-56}	0.63	-0.005	0.330	NA	NA	NA	NA	NA

Results here correspond to Table 1 of the main text with two differences. The polygenic scores were computed based on results of a GWAS on BMI (as opposed to EA). The composite health trait here, $HLTH_{-BMI}$, does not include BMI, i.e. only values HDL, FG, HT, and CPD have been incoporated. The decomposition results on the right hand of the table are all NAs. This is because the uncertainties (standard errors) associated with $\hat{\theta}_{NT}$ are too large for the decompositions to be meaningful.

Supplementary S7. Association results for the transmitted and non-transmitted HT polygenic scores with adjustment for the EA polygenic scores.

				Transmitted			Nontransmitted						
				T ($T = T_P + T_M$)			NT ($NT = NT_P + NT_M$)						
Trait	**N**	**N_{NTP}**	**N_{NTM}**	$\widehat{\theta}_T$	***P***	R^2 (%)	$\widehat{\theta}_{NT}$	***P***	R^2_δ (%)	$\widehat{\delta}\ /\ \widehat{\theta}_T$	$\widehat{\phi}_\delta\ /\ \widehat{\theta}_T$	$\widehat{\eta}\ /\ \widehat{\theta}_T$	$\widehat{\phi}_\eta\ /\ \widehat{\theta}_T$
EA	21637	13948	19012	0.002	0.76	0.00	0.016	0.07	NA	NA	NA	NA	NA
AGFC	54372	35294	47052	0.003	0.50	0.00	0.006	0.27	NA	NA	NA	NA	NA
HDL	46872	30855	40788	0.000	0.96	0.00	0.006	0.29	NA	NA	NA	NA	NA
BMI	39078	26433	34533	-0.013	0.04	0.02	-0.002	0.78	NA	NA	NA	NA	NA
FG	34767	22959	30222	0.006	0.32	0.00	0.001	0.93	NA	NA	NA	NA	NA
HT	39270	26563	34703	0.427	$< 10^{-200}$	18.24	0.025	1.1×10^{-4}	16.17	0.942	0.053	0.005	0.001
CPD	18887	12371	16589	-0.005	0.53	0.00	-0.001	0.88	NA	NA	NA	NA	NA
$HLTH_{-HT}$	62300	41974	54522	0.003	0.57	0.00	0.004	0.44	NA	NA	NA	NA	NA

The results here correspond to those in Supplementart Table S5. The only difference is that, for both the transmitted and non-transmitted polygenic scores, we first regressed the HT polygenic score on the EA polygenic score and used the residuals for the analyses.

Supplementary Table S8. Association results for the transmitted and non-transmitted BMI polygenic scores with adjustment for the EA polygenic scores.

				Transmitted			Nontransmitted						
				T ($T = T_P + T_M$)			NT ($NT = NT_P + NT_M$)						
Trait	**N**	N_{NTP}	N_{NTM}	$\widehat{\theta}_T$	*P*	R^2 (%)	$\widehat{\theta}_{NT}$	*P*	R^2_δ (%)	$\widehat{\delta}\ /\widehat{\theta}_T$	$\widehat{\phi}_\delta / \widehat{\theta}_T$	$\widehat{\eta} / \widehat{\theta}_T$	$\widehat{\phi}_\eta / \widehat{\theta}_T$
EA	21637	13948	19012	-0.029	3.4×10^{-4}	0.08	-0.011	0.21	NA	NA	NA	NA	NA
AGFC	54372	35294	47052	-0.018	1.9×10^{-4}	0.03	-0.006	0.25	NA	NA	NA	NA	NA
HDL	46872	30855	40788	-0.094	7.0×10^{-59}	0.89	0.000	1	NA	NA	NA	NA	NA
BMI	39078	26433	34533	0.308	$< 10^{-200}$	9.49	0.000	0.94	NA	NA	NA	NA	NA
FG	34767	22959	30222	0.081	1.3×10^{-42}	0.66	-0.002	0.75	NA	NA	NA	NA	NA
HT	39270	26563	34703	-0.010	0.14	0.01	0.004	0.55	NA	NA	NA	NA	NA
CPD	18887	12371	16589	0.041	1.2×10^{-7}	0.17	0.013	0.14	NA	NA	NA	NA	NA
$HLTH_{-BMI}$	62328	41996	54546	-0.075	5.8×10^{-51}	0.56	-0.004	0.48	NA	NA	NA	NA	NA

The results here correspond to those in Supplementart Table S6. The only difference is that, for both the transmitted and non-transmitted polygenic scores, we first regressed the BMI polygenic score on the EA polygenic score and used the residuals for the analyses.

Supplementary Table S9. Summary of data used in the manuscript.

Data	Phenotype	Publication
SNP effects from GWAS used for determining weightings in	EA	A. Okbay *et al.* , Genome=wide asociation study identifies 74 loci associated with education attainment. *Nature* **533**, 539-542 (2016).
	HT	A.R. Wood *et al.* , Defining the role of common variation in the genomic and biological architecture of adult human height. *Nat Genet* **46**, 1173-1186 (2014).
	BMI	A.E. Locke *et al.* , Genetic studies of body mass index yield new insights for obesity biology. *Nature* **518**, 197-206 (2015).
phenotype descriptions	AGFC	N. Barban et al., Genome-wide analysis identifies 12 loci influencing human reproductive behavior. Nat Genet 48, 1462-1472 (2016).
	HDL	A. Helgadottir et al., Variants with large effects on blood lipids and the role of cholesterol and triglycerides in coronary disease. Nat Genet 48, 634-639 (2016).
	BMI	G. Thorleifsson et al., Genome-wide association yields new sequence variants at seven loci that associate with measures of obesity. Nat Genet 41, 18-24 (2009).
	FG	J. Flannick et al., Loss-of-function mutations in SLC30A8 protect against type 2 diabetes. Nat Genet 46, 357-363 (2014).
	HT	D.F. Gudbjartsson et al., Many sequence variants affecting diversity of adult human height. Nat Genet 40, 609-615 (2008).
	CPD	T.E. Thorgeirsson et al., A variant associated with nicotine dependence, lung cancer and peripheral arterial disease. Nature 452, 638-642 (2008).
analysis of educational attainment data with respect to selection	EA	A. Kong et al., Selection against variants in the genome associated with education attainment. Proc Natl Acad Sci U S A 114, E727-E732 (2017).