

Supplemental information

**Robust genetic nurture effects on education: A systematic review
and meta-analysis based on 38,654 families across 8 cohorts**

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Pingau**

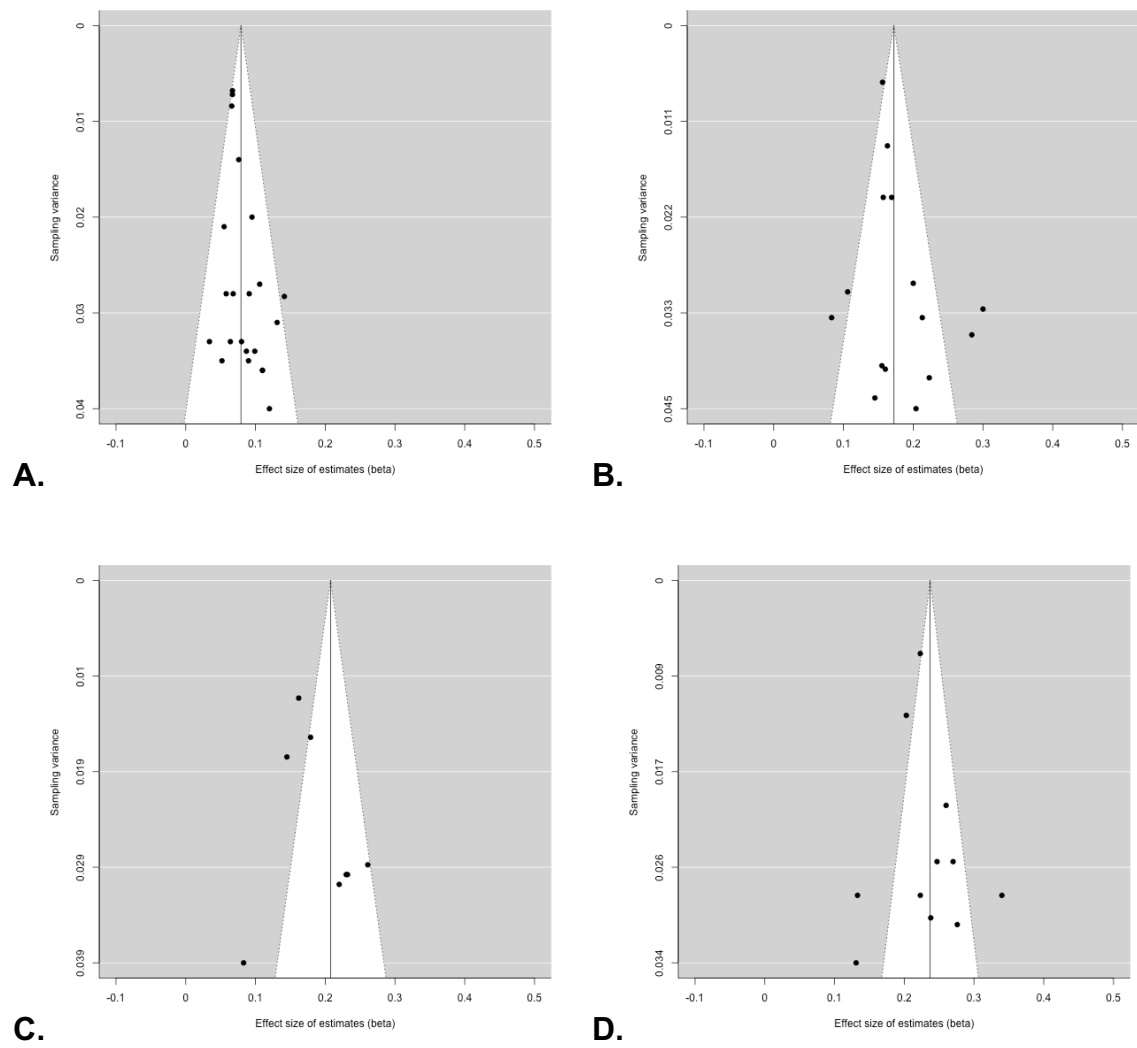


Figure S1. Funnel plots for effects on educational outcomes. A. Genetic nurture effects. **B.** Direct genetic effects. **C.** Unadjusted parental effects. **D.** Unadjusted child effects.

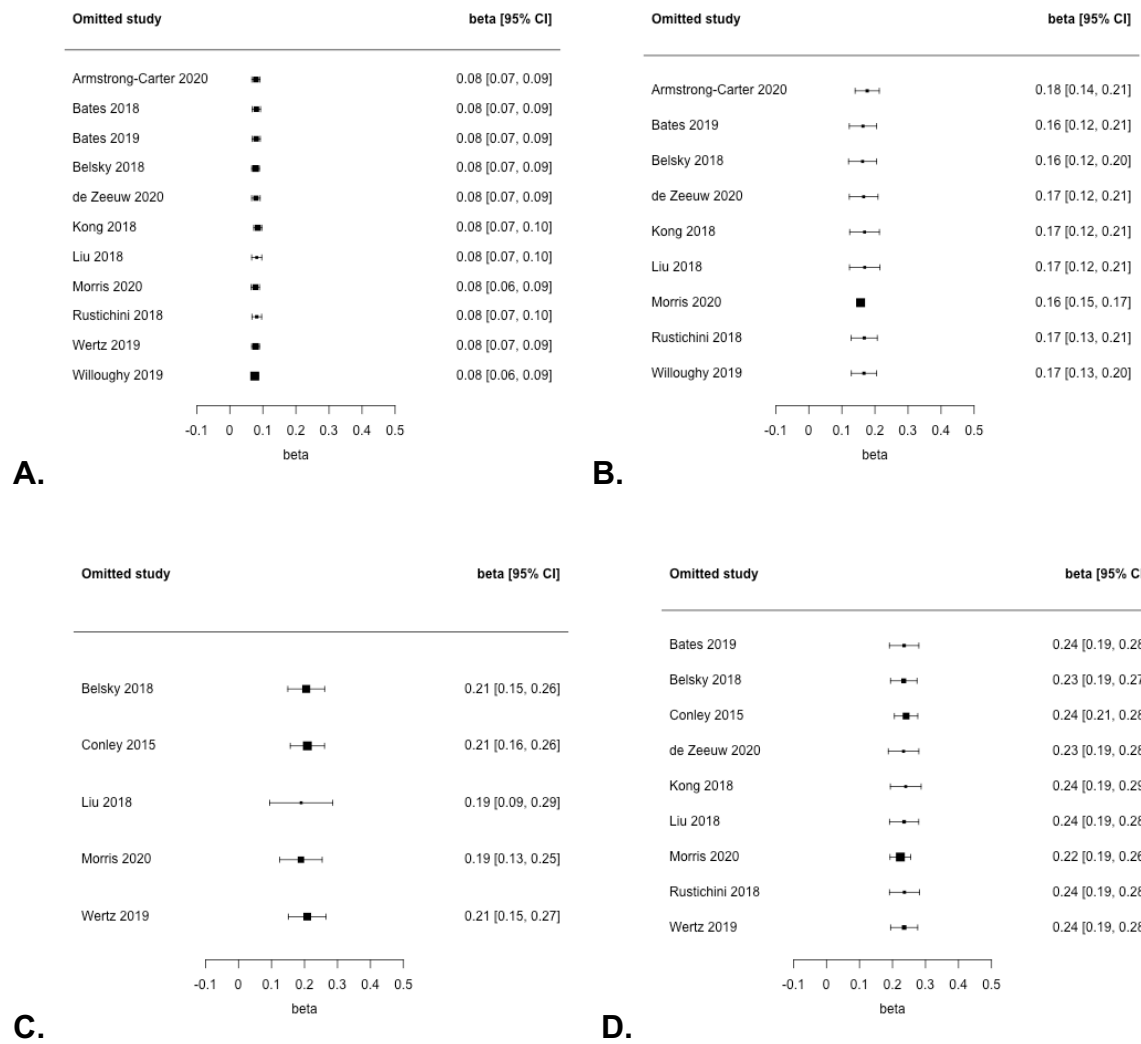
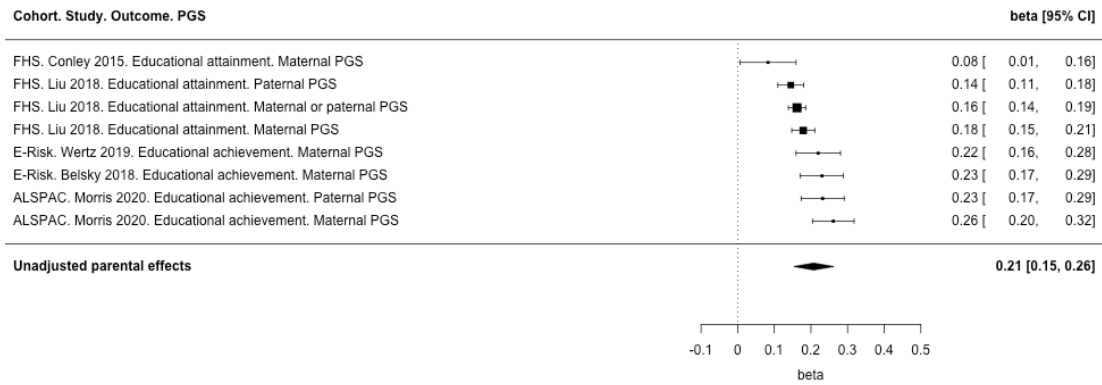
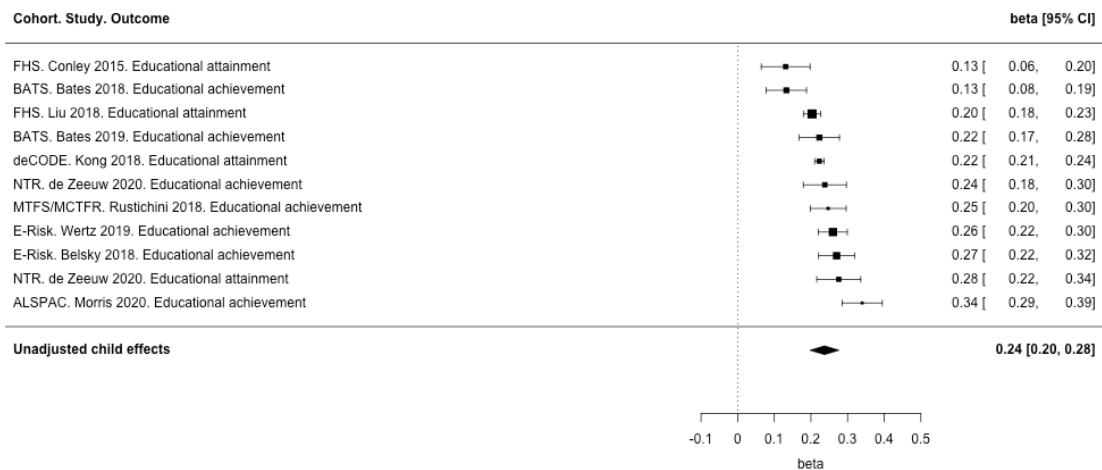


Figure S2. Jackknife sensitivity analyses for effects on educational outcomes.
A. Genetic nurture effects. **B.** Direct genetic effects. **C.** Unadjusted parental effects.
D. Unadjusted child effects. The estimate corresponding to each study listed reflects the pooled beta from a meta-analysis where that study was omitted.



A.



B.

Figure S3. Forest plot of multilevel random effects model for unadjusted effects on educational outcomes. A. Unadjusted parental effects. B. Unadjusted child effects. Effect sizes were standardised beta coefficients, which represent how many standard deviations of change in educational outcome occur per standard deviation of change in EA PGS.

	MREM of parental effects		MREM of child effects	
	Genetic nurture effects	Unadjusted parental effects	Direct genetic effects	Unadjusted child effects
k_{cohort}	7	3	7	6
k_{estimate}	19	8	15	10
β_{pooled}	0.08	0.21	0.17	0.24
$\beta_{95\% \text{ CI}}$	0.07-0.10	0.15-0.26	0.12-0.21	0.19-0.29
$\sigma^2_{\text{Level 2}}$	$\chi^2 < 0.01, p = .5000$	$\chi^2 < 0.01, p = .5000$	$\chi^2 < 0.01, p = .5000$	$\chi^2 = 1.68, p = .0977$
$\sigma^2_{\text{Level 3}}$	$\chi^2 < 0.01, p = .5000$	$\chi^2 = 5.73, p = .0083$	$\chi^2 = 5.12, p = .0118$	$\chi^2 = 1.40, p = .1187$
$I^2_{\text{Level 1}}$	>99.99%	19.14%	21.54%	14.57%
$I^2_{\text{Level 2}}$	<0.01%	<0.01%	<0.01%	24.11%
$I^2_{\text{Level 3}}$	<0.01%	80.86%	78.46%	61.32%
Publication bias	$Q = 0.88, p = .3486$	$Q = 3.22, p = .0727$	$Q = 0.02, p = .8787$	$Q = 0.90, p = .3427$

Table S5. Three-level random effects models after removing the potentially influential study (Kong et al. 2018)

Note. Sensitivity analysis to assess the role of a potentially influential study was performed by removing the largest included study Kong et al. 2018. MREM = Multilevel random effects model; β = standardized regression coefficients (i.e., the metric of effect sizes); CI = confidence interval; χ^2 Statistics from likelihood-ratio test to test within-cohort variance ($\sigma^2_{\text{Level 2}}$) and between-cohort variance ($\sigma^2_{\text{Level 3}}$) for significance; I^2 = % of the total variance accounted for by random sampling variance (Level 1), variation within cohorts (Level 2), variation between cohorts (Level 3); Publication bias, assessed by using precision (sampling variance) to predict the effect size.

	Parental effects		Child effects	
	Joint parental model ^a	Unadjusted parental effects	Unadjusted child effects	Joint child model ^b
k _{cohort}	8	3	7	8
k _{estimate}	30	8	11	27
β_{pooled}	0.11	0.21	0.24	0.2
$\beta_{95\% \text{ CI}}$	0.08-0.14	0.15-0.26	0.20-0.28	0.16-0.24
$\beta_{\text{robust CI}}^c$	0.07-0.14	0.08-0.33	0.19-0.29	0.15-0.24
$\sigma^2_{\text{Level 2}}$	$\chi^2 = 45.23, p < .0001$	$\chi^2 < 0.01, p = .5000$	$\chi^2 = 1.55, p = .1067$	$\chi^2 = 60.17, p < .0001$
$\sigma^2_{\text{Level 3}}$	$\chi^2 = 1.23, p = .1339$	$\chi^2 = 5.73, p = .0083$	$\chi^2 = 1.27, p = .1298$	$\chi^2 = 5.46, p = .0097$
$I^2_{\text{Level 1}}$	10.17%	19.14%	11.57%	8.70%
$I^2_{\text{Level 2}}$	65.83%	<0.01%	28.51%	36.17%
$I^2_{\text{Level 3}}$	24.00%	80.86%	59.92%	55.13%
Publication bias	Q = 4.01, $p = .0453$	Q = 3.22, $p = .0727$	Q = 0.47, $p = .4917$	Q = 9.56, $p = .0020$

Table S6. Three-level random effects models of unadjusted parental and child effects on educational outcomes

Note. ^a Effect sizes of genetic nurture and unadjusted parental effects were jointly modelled. ^b Effect sizes of direct genetic and unadjusted child effects were jointly modelled. ^c Robust confidence intervals were cluster-robust variance estimations, for details see Supplemental Notes 7.1. MREM = Multilevel random effects model; β = standardized regression coefficients (i.e., the metric of effect sizes); CI = confidence interval; χ^2 Statistics from likelihood-ratio test to test within-cohort variance ($\sigma^2_{\text{Level 2}}$) and between-cohort variance ($\sigma^2_{\text{Level 3}}$) for significance; I^2 = % of the total variance accounted for by random sampling variance (Level 1), variation within cohorts (Level 2), variation between cohorts (Level 3); Publication bias, assessed by using precision (sampling variance) to predict the effect size.

Subgroup	k _{cohort}	Educational attainment			Educational achievement		
		k _{estimate}	β_{pooled}	$\beta_{95\% \text{ CI}}$	k _{estimate}	β_{pooled}	$\beta_{95\% \text{ CI}}$
Genetic nurture effects	2	3	0.11	0.08-0.14	3	0.05	0.01-0.09
Direct genetic effects	2	2	0.12	0.07-0.17	2	0.21	0.16-0.26
Unadjusted child effects	2	1	0.28	0.22-0.34	2	0.24	0.21-0.28

Table S9. Moderating role of educational outcome type within study

Note. Type of educational outcome moderation robustness check by examining studies assessing both educational attainment and achievement. Moderation analysis was performed with studies (de Zeeuw et al., 2020 and Rustichini et al., 2018) in which both educational attainment and achievement were assessed. No effects of unadjusted parental effects were available from these studies and therefore were not shown here.

Type of the outcome assessed as a dichotomized moderator [educational attainment (the highest level of education completed, e.g., year of schooling), educational achievement (how well performed at school, e.g., high school grades)]. Dummy variables were created for each category of the potential moderator. In order to obtain the mean effect (including significance and confidence interval) of all categories, separate meta-regressions were conducted, taking each category as the reference category in turn.

Supplemental Notes

1 Capturing genetic nurture effects with parent(s)-offspring genotype

1.1 Virtual-parent design

The parental genetic material transmitted to their offspring is randomly assigned during meiosis, with each allele having a 50% chance of being transmitted to the gamete (egg or sperm) and then to the offspring. Alleles that are not transmitted to offspring can nonetheless influence offspring outcomes through environmental rather than genetic pathways. The non-transmitted parental genotype can be considered as a “virtual parent” who is not genetically related to the offspring, whereas the transmitted parental genotype (50% from mother and 50% from father) makes up the child genotype. The effect of polygenic scores (PGSs) derived from non-transmitted parental genotype on offspring outcome is thus free from genetic confounding between parents and offspring due to shared genotypes.

In regressions with child education as the outcome, let PGS_T and PGS_{NT} be the standardised PGS of transmitted and non-transmitted parental genotypes respectively, C_p be the child phenotype, and β_T and β_{NT} be the corresponding respective coefficients. The model can be expressed as follows:

$$C_p = \beta_T * PGS_T + \beta_{NT} * PGS_{NT} + e$$

The estimated genetic nurture effects are:

$$\text{Genetic nurture effects} = \beta_{NT}$$

As both transmitted and non-transmitted parental genotype have nurturing effects, direct genetic effects originating in the child are:

$$\text{Direct genetic effects} = \beta_T - \beta_{NT}$$

For detailed decompositions of aforementioned equations see ¹. For more sophisticated decompositions of genetic influences using the virtual parent design, including genetic nurture effects, direct genetic effects, the assortative mating–induced confounding effect for the direct genetic effect component, and the confounding effect of the genetic nurturing component, see ².

1.2 Statistical control approach

The statistical control approach utilizes the complete parental genotype as an aggregation of transmitted and non-transmitted genotypes. Disentanglement of genetic nurture and direct genetic effects is achieved by modelling the association between parental PGS(s) and offspring phenotype while controlling for offspring PGS. As offspring genotype can fully mediate the effects of the parental transmitted genotype, the remaining effects of parental genotype on offspring outcome can be environmentally mediated, i.e., via genetic nurture effects. Similarly, since the effect of offspring genotype is controlled for parental genotype, it thus reflects direct genetic effects free from the inflation from genetic nurture.

Let PGS_C , PGS_M and PGS_P be the standardised PGS of child, maternal and paternal genotype respectively, and β_C , β_M and β_P be the corresponding coefficients. The full statistical control model can be expressed as follows when genotypes of both parents are available:

$$Cp = \beta_C * PGS_C + \beta_M * PGS_M + \beta_P * PGS_P + e$$

The partial statistical control model can be expressed as follows when genotypes of one parent is available:

$$Cp = \beta_C * PGS_C + \beta_M * PGS_M + e$$

or

$$Cp = \beta_C * PGS_C + \beta_P * PGS_P + e$$

The estimated genetic nurture effects are:

$$\text{Maternal genetic nurture effects} = \beta_M$$

$$\text{Paternal genetic nurture effects} = \beta_P$$

Importantly, estimates from the partial statistical control model can still be biased as they do not account for the confounding role of the other parent's PGS³. The estimated direct genetic effects are:

$$\text{Direct genetic effects} = \beta_C$$

2 Study selection and assessment

2.1 Literature search

Two search strategies were employed for study identification. First, we systematically searched the following databases:

1) Ovid:

i) MEDLINE In-Process & Other Non-Indexed Citations and Daily

ii) EMBASE

iii) PsycINFO

2) Web of Science Core Collection:

i) Sciences Citation Index Expanded (SCI-EXPANDED);

ii) Social Sciences Citation Index (SSCI);

iii) Arts & Humanities Citation Index (A&HCI);

iv) Emerging Sources Citation Index (ESCI).

3) PubMed.

The search terms are described below:

((education* [Title/Abstract]) OR (academic [Title/Abstract]) OR (college degree [Title/Abstract]) OR (college entry [Title/Abstract]) OR (university degree [Title/Abstract]) OR (university entry [Title/Abstract]) OR (school performance* [Title/Abstract]) OR (school achievement* [Title/Abstract]) OR (performance in school [Title/Abstract]) OR (schooling [Title/Abstract])

AND

(polygenic scor* [Title/Abstract]) OR (polygenic risk scor* [Title/Abstract]) OR (genetic scor* [Title/Abstract]) OR (genetic risk scor* [Title/Abstract]) OR (genomic scor* [Title/Abstract]) OR (genomic risk scor* [Title/Abstract]) OR (genome-wide polygenic scor* [Title/Abstract])

AND

(nature of nurture [Title/Abstract]) OR (genetic nurtur* [Title/Abstract]) OR (virtual-parent design [Title/Abstract]) OR (pseudo-control [Title/Abstract]) OR (dynastic effect* [Title/Abstract]) OR (intergeneration* [Title/Abstract]) OR (multigeneration* [Title/Abstract]) OR (transmit* allele* [Title/Abstract]) OR (nontransmit* allele* [Title/Abstract]) OR (non-transmit* allele* [Title/Abstract]) OR (passive gene–environment correlation [Title/Abstract]) OR (genetic inheritance [Title/Abstract]) OR

(genetic confounding* [Title/Abstract]) OR (genetic transmission [Title/Abstract]) OR (genetic influence* [Title/Abstract]) OR (parental transmission [Title/Abstract]) OR (maternal transmission [Title/Abstract]) OR (paternal transmission [Title/Abstract]) OR (parental influence* [Title/Abstract]) OR (maternal influence* [Title/Abstract]) OR (paternal influence* [Title/Abstract]) OR (social inheritance [Title/Abstract]) OR (social genetic effect* [Title/Abstract]) OR (environmental transmission [Title/Abstract]) OR (cultural transmission [Title/Abstract]) OR (vertical transmission [Title/Abstract]) OR (familial transmission [Title/Abstract]) OR (family-based stud* [Title/Abstract]) OR (trio* [Title/Abstract]) OR (triad* [Title/Abstract]) OR (dual* [Title/Abstract]) OR (dyad* [Title/Abstract]))

These search terms were translated into suitable terms for the Ovid database (MEDLINE, EMBASE and PsycINFO), Web of Science and PubMed, which can be made available upon request.

Second, we manually searched the reference list of relevant articles, including studies that used virtual parent and statistical control approaches to investigate the genetic nurture effects on education, as well as unpublished evidence in preprint platforms were screened to identify articles that were missed by the search. Two authors (B.W. and T.S.) independently screened all articles retrieved from the search on Rayyan (<http://rayyan.qcri.org>), a free platform for systematic review ⁴. Potentially eligible studies (see criteria below) were then reviewed in full text. This search resulted in n=12 studies eligible for inclusion.

2.2 Inclusion criteria

Studies were included if they assessed genetic nurture effects on educational outcomes, either educational attainment (e.g., years of education completed, highest degree obtained) or educational achievement (e.g., national tests scores or levels, school grades) in the general population. Due to lack of data in preliminary searches, it was part of the protocol to exclude studies that were conducted in clinically-referred sample or focused exclusively on specific types of educational outcomes (e.g., performances on math course or linguistics). No genetic nurture study on clinical sample or specific educational outcomes was present after the systematic

search. Studies were required to use one of the two designs that rely on genotype data from parents and their biological offspring: 1) virtual parent: testing genetic nurture effects on education by using parent(s)' non-transmitted genotype to predict children's education. In this case polygenic scores of educational attainment (hereafter referred as EA PGSs) calculated from at least one parent's non-transmitted alleles should be used; 2) statistical control: testing genetic nurture effects on education by using parent(s)' whole genotype to predict children's education over and above children's own genotype, in this case the EA PGSs of child and at least one parent should be used. Sibling^{5, 6} and adoption designs⁷ were not included in the meta-analysis to avoid introducing additional heterogeneity but served as measure of comparability and robustness when discussing results of our pooled genetic nurture effects.

2.3 Assessment of methodological quality

An adapted version of the Newcastle - Ottawa Quality Assessment Scale for Cohort Studies⁸ was applied to evaluate the methodological quality of the included studies.

The following scoring criteria was used:

Methodological quality assessment criteria of studies on genetic nurture effects on education

Note: A study can be awarded a maximum of one point for each numbered item within all categories.

Representativeness and attrition

1) Population representativeness of the cohort

- a) truly population-based on community, e.g. pregnancy/birth cohort or other population-based cohort (1)
- b) somewhat representative of the population , e.g., twin cohort (0.5)
- c) selected group of users, e.g., case-control, genomics company (0)
- d) no description of the cohort (0)

2) Attrition in the cohort (due to genotyping or outcome availability)

- a) complete cohort - all subjects of original cohort are used (1)
- b) subjects lost unlikely to introduce bias - small number lost - > 70 % of original cohort are used, or description provided of those lost¹ (1)

c) < 70% of original cohort are used and no description of those lost (0)

d) no statement (0)

¹only code 1 when loss of participants is unlikely to introduce bias, e.g., if description indicates random loss.

Exposure: polygenic scores (PGSs) of educational attainment

3) Power/size of the genome-wide association studies (GWASs) used to compute the PGSs

a) Lee et al. et al. (2018, N = 1,131,881 (1)

b) Okbay et al. et al. (2016, N = 293,723 (0.5)

c) Rietveld et al. et al. (2013, N = 101,069 (0)

4) Sample overlap

a) the cohort does not overlap with the sample used to derive the GWAS (1)

b) an updated GWAS excluding the cohort is used (1)

c) the cohort is used to derive the GWAS (0)

5) Genetic ancestry

a) the cohort is from the same genetic ancestry with the GWAS² (1)

b) the cohort is from different genetic ancestry with the GWAS² (0)

²European descent

Comparability/confounding

6) Study fully accounts for genetic nurture effects

a) yes, study uses control by design, i.e., virtual parent design (1)

b) yes, study uses statistical control and accounted for PGSs of both parents and child (1)

c) yes, study uses statistical control and accounted for PGSs of one parent and child (0.5)

d) no (0)

7) Study accounts for the confounding of age, sex and principal components (PCs)

a) yes, study controls for age, sex and PCs (1)

b) yes, study controls for sex and PCs and all participants were the same on age (1)

c) yes, study partly controls for age, sex and PCs (0.5)

d) no (0)

Outcome: Educational attainment and educational achievement

8) Assessment of outcome

- a) official record (1)
 - b) instrument tested for validity and reliability (1)
 - c) self-report (0.5)
 - b) no description (0)
- 9) Same underlying phenotype of outcome and GWAS
- a) the outcome represents the same underlying phenotype as measured by the GWAS used, e.g., years of education, highest degree obtained (1)
 - b) the outcome represents somewhat the same underlying phenotype as measured by the GWAS used, e.g., national tests scores, school grades (0.5)
 - c) the outcome presents different phenotype as measured by the GWAS used, e.g., cognitive performance, math course level, EA difference between twins (0)

Detailed methodological score of each included study see Table S3. It should be noted that most of the included studies (9 out of 12) had limited representativeness of their original cohort. Taking the minimal attrition rate per study on genetic nurture estimates, the median attrition rate was 55.6% (attrition rate for each estimate see Table S4). This substantial rate in attrition among the included studies is mainly due to missingness of genetic data (i.e. samples not genotyped within cohorts) or ancestry restriction due to predominantly European-descent samples of GWASs ⁹.

3 Data extraction and synthesis

3.1 Effect size calculation

Standardised beta coefficients, which constituted the most commonly reported metric among the included studies, were used to measure effect sizes. One study¹⁰ reported unstandardised betas, which were manually standardised by multiplying unstandardised coefficients by the ratio of standard deviations of the corresponding independent variable and dependent variable. Some studies did not report the standard error of estimates, which was necessary for estimating the pooled effect size. For studies that reported 95% confidence intervals¹¹⁻¹⁵, standard errors were manually calculated by dividing corresponding confidence intervals (upper limit – lower limit) by 3.92. For studies that reported t-test statistics¹⁶, standard errors were manually calculated by dividing corresponding standardised betas by t-test statistics. For studies that did not report data allowing for direct calculation of standard errors², following non-response after contacting the author, we imputed standard errors from corresponding estimates and sample sizes by using the `compute.es_0.2-4` package¹⁷ in R version 3.6.1¹⁸.

Genetic nurture effects in one of the included studies¹⁹ using average parental PGS was recalibrated to improve comparability with other studies using individual parental PGS. Details see in Supplemental Notes 7.2.

For studies using the virtual parent design^{2, 12-14}, estimates of unadjusted child effects (based on transmitted PGS) and genetic nurture effects (based on non-transmitted PGS) were reported. The magnitude of direct genetic effects can be imputed as follows:

In regressions of the child education on both parental transmitted polygenic score (PGS_T) and non-transmitted polygenic score (PGS_{NT}), let N be the sample size, β_T be the coefficient of PGS_T and β_{NT} the coefficient of PGS_{NT} , σ_T be the standard error of β_T and σ_{NT} be the standard error of β_{NT} . β_T is the effect of the children's PGS on their own phenotype and β_{NT} is the effect of genetic nurture. Thus, estimated direct genetic effects $\beta_{direct} = \beta_T - \beta_{NT}$ ². The variance of β_{direct} is equal to the sum of variance of β_T and β_{NT} . Let σ_{direct} denotes the standard error of direct genetic effects, then it can be expressed as: $\sigma_{direct} = \sqrt{(\sigma_T^2 + \sigma_{NT}^2)}$.

3.2 Multilevel Random Effects Model (MREM)

The three-level MREM was performed following the tutorial of Assink and Wibbelink²⁰ and incorporated variation in effect sizes from three sources: Level 1 variance attributed to random sampling, which corresponds to the standard error of each individual effect size; Level 2 variation between estimates within a single cohort; Level 3 variation in estimates between different cohorts. Here the cohort level was defined as the original population-based sample on which the data about genotype and educational outcomes was collected. In this meta-analysis, we included 12 studies from eight independent cohorts (see Table 1, column 'Cohort'). This level was employed to account for the violation of independence due to multiple estimates and studies from the same cohort. For example, two studies^{10, 21} were both from the Framingham Heart Study (FHS), and in one study²¹ several estimates of genetic nurture effects were reported using genotypes from mothers and fathers. One special case was that participants in the Minnesota Center for Twin and Family Research (MCTFR) cohort were drawn from several longitudinal studies including the Minnesota Twin Family Study (MTFS) cohort, thus in the meta-analysis they were considered as the same cohort.

4 Unadjusted parental and child effects

4.1 Unadjusted parental effects

We derived $k = 8$ estimates of unadjusted parental effects on offspring educational outcomes (i.e., without adjusting for genetic nurture effects). Table S6 “Joint parental model” column shows MREM findings when jointly meta-analysing genetic nurture effects and unadjusted parental effects ($\beta_{\text{mixture}} = 0.11$, 95% CI [0.08, 0.14], robust CI [0.07, 0.14]). When evaluating the heterogeneity in effect sizes, we found that a substantial proportion of variance reflected within-cohort heterogeneity ($I^2_{\text{Level 2}} = 65.83\%$). This indicates that within-cohort factors (i.e., differences in effect sizes between outcomes within the same cohort) may account for some of the variation in effect sizes. Results of subgroup analysis showed that the magnitude of genetic nurture effects and unadjusted parental effects were highly significantly different ($Q = 20.58$, $df = 1$, $p < .0001$). As is shown in Table S6 and Figure S3, estimates from unadjusted parental effects ($\beta_{\text{parental unadjusted}} = 0.21$, 95% CI [0.15, 0.26], robust CI [0.08-0.33]) only were larger than genetic nurture. The variance among effect sizes of unadjusted parental effects mainly resulted from the between-cohort heterogeneity ($I^2_{\text{Level 3}} = 86.86\%$), suggesting that factors in which the cohorts may differ (e.g., type of the educational outcome, age when the outcome was assessed, accuracy of the GWASs) accounted for some of the variation in effect sizes. The funnel plot (see Figure S1) and formal test with precision as a moderator ($Q = 3.22$, $p = .0727$) suggested no publication bias in estimates of unadjusted parental effects. Results of jackknife leave-one-out analysis (see Figure S2) suggested no substantial role of a single influential study.

4.2 Unadjusted child effects

We derived $k = 11$ estimates of unadjusted child effects on their own educational outcomes, i.e., effects of child PGS without considering genetic nurture effects. Table S6 “Joint child model” column shows MREM findings of child effects on education when jointly meta-analysing direct genetic effects and unadjusted child effects ($\beta_{\text{mixture}} = 0.20$, 95% CI [0.16, 0.24], robust CI [0.15, 0.24]). More variance among effect sizes in the joint model was attributable to between-cohort heterogeneity than within-cohort heterogeneity ($I^2_{\text{Level 2}} = 36.17\%$ versus $I^2_{\text{Level 3}} = 55.13\%$). Results of subgroup analysis showed that magnitudes of direct genetic

effects and unadjusted child effects were significantly different ($Q = 6.39$, $df = 1$, $p = .0115$). As is shown in Table S6 and Figure S3, the magnitude of unadjusted child effects ($\beta_{\text{child unadjusted}} = 0.24$, 95% CI [0.20, 0.28], robust CI [0.19, 0.29]) was larger than direct genetic effects and comparable to estimates obtained from studies assessing the explanatory power of EA PGSs on one's educational outcome without accounting for genetic nurture effects, which typically range between $\beta = 0.15$ and $\beta = 0.39$ ^{16, 22-24}. More variance among effect sizes in unadjusted child effects was attributable to between-cohort heterogeneity than within-cohort heterogeneity ($R^2_{\text{Level 2}} = 28.51\%$ versus $R^2_{\text{Level 3}} = 59.92\%$). The funnel plot (see Figure S1) and formal test with precision as a moderator suggested no publication bias in estimates of unadjusted child effects ($Q = 0.47$, $p = 0.4917$). Results of jackknife leave-one-out analysis (see Figure S2) suggests no substantial role of a single influential study.

5 Other sources of heterogeneity in genetic nurture effects

The magnitude of genetic nurture effects on children's educational outcomes may vary according to several factors other than parent of origin, we examined the sources of heterogeneity in genetic nurture, direct genetic as well unadjusted effects by several meta-regression analyses (see Table S7).

5.1 Study design

It is unclear whether the magnitude of genetic nurture effects in empirical studies differs depending on the analytic method used (i.e., virtual parent or statistical control). Moreover, due to lack of complete trio data (i.e. child and both parents), genetic nurture effects have often been estimated among parent-offspring pairs. It is unclear to what extent the missing parental genotype bias estimates. Therefore, we examined whether using different designs moderated effect sizes by comparing estimates relying on virtual parent (using parental non-transmitted PGS to predict children's education), partial statistical control (using PGS of one parent to predict children's education while controlling for the child's PGS) and full statistical control (using PGS of one parent to predict children's education while controlling for the child's and the other parent's PGS).

Genetic nurture effects detected by the virtual parent design ($\beta_{\text{virtual parent}} = 0.07$, 95% CI [0.06, 0.08]) were lower than those obtained from the statistical control approach ($\beta_{\text{partial control}} = 0.09$, 95% CI [0.07, 0.10], $\beta_{\text{full control}} = 0.09$, 95% CI [0.06, 0.11], $p = .0443$). In contrast, different designs captured similar effect sizes for direct genetic effects ($\beta_{\text{virtual parent}} = 0.15$, 95% CI [0.08, 0.21], $\beta_{\text{partial control}} = 0.18$, 95% CI [0.13, 0.24], $\beta_{\text{full control}} = 0.15$, 95% CI [0.08, 0.22], $p = .5039$).

Here, the magnitude of genetic nurture effects was slightly smaller in the virtual parent design versus the statistical control approach. Although we found no strong evidence for differences based upon findings from partial control (one parent) or full control (two parents), this may however reflect that these estimates were from different samples, hiding true differences. A recent study has shown the importance of using complete trio data, as missing the genotype of one parent can bias direct genetic effects and genetic nurture effects³. Evidence from one of the included

studies²⁵ using both partial and full statistical control approach echoed this view. Additional work is needed to compare estimates of genetic nurture using different analytical methods within the same sample to better understand the equivalence and comparability of different approaches.

5.2 Type of educational outcome

Previous genetically informed studies on educational outcomes have focused on attainment or achievement interchangeably^{23, 24, 26, 27}. However, both efforts relied on the GWASs of educational attainment, which is likely to more strongly correlate with educational attainment than achievement. Therefore, studies examining educational attainment may capture genetic nurture effects more accurately than those examining educational achievement. We thus considered whether the type of educational outcome moderated effect sizes by comparing studies assessing educational attainment and educational achievement.

Similar effect sizes of genetic nurture effects were found for educational attainment and achievement ($\beta_{\text{attainment}} = 0.09$, 95% CI [0.07, 0.11], $\beta_{\text{achievement}} = 0.07$, 95% CI [0.05, 0.10], $p = .3079$). However, when restricting the analysis to studies where both educational attainment and achievement were assessed^{14, 28}, larger genetic nurture effects on educational attainment relative to educational achievement were found (see Supplemental Notes 7.5, Table S9). Another explanation of larger genetic nurture effects for educational attainment compared to educational achievement is that attainment may be more socially influenced than achievement²⁹. That is, it may be easier for parents to influence attainment (e.g. by accessing more exclusive schooling or financially supporting further education). However, developmental trends in genetic nurture effects warrant more investigation. Heritability of educational outcomes increases with age³⁰. Conversely, the nurturing behaviours from parents may impact offspring more at earlier ages, as they spend more time at home rather than school, spend more time with their parents rather than peers, which might lead to genetic nurture effects decreasing with age. Moreover, genetic nurture may act distinctively over time through different pathways as suggested by a recent study showing parent non-cognitive but not cognitive related characteristics were more important for educational achievement at age 16 than age 12³¹.

Direct genetic effects were larger for educational achievement ($\beta_{\text{achievement}} = 0.19$, 95% CI [0.14, 0.24]) than for educational attainment ($\beta_{\text{attainment}} = 0.14$, 95% CI [0.08, 0.19]) and there was evidence of a moderating effect ($p = .0466$). The robustness of this finding was confirmed by restricting the analysis to studies reporting effects for both attainment and achievement (see Supplemental Notes 7.5, Table S9). This finding agrees with previous twin evidence, which suggested ~60% heritability for educational achievement measured in childhood and adolescence³² and ~40% for educational attainment measured in adulthood^{33, 34}. Several plausible explanations might account for the consistently higher heritability/direct genetic effect in educational achievement. One is that educational achievement is measured during compulsory schooling. The difference thus may reflect more genetically influenced traits in children like intelligence, personality and psychopathology³⁵. In contrast, years of education one completed is likely to be influenced by a wider range of factors, such as career plan or financial situation^{29, 36, 37}. Another explanation is that educational achievement is often measured with standard tests/scores which reflect one's relative ranking/decile among peers. Education years, however, can be more ambiguous as essentially different routes, such as academic and vocational, are not distinguished, which may introduce heterogeneity and measurement error. Future studies should examine genomic predictions with consistent measures of educational achievement across different developmental stages in order to capture dynamic changes. Evidence from one study, the Twins Early Development Study (TEDS), suggested that the predictive precision of PGSs (i.e., unadjusted child effects) on educational achievement increased from ages 7 to 16³⁸.

5.3 Predictive accuracy of the GWAS used to derive the PGSs

Genome-wide association studies (GWASs) have advanced rapidly in the last decade (Visscher et al., 2017). The first GWAS of educational attainment EA (hereafter referred to as "EA GWAS") was conducted in 2013 (EA1) in a discovery sample of 101,069 individuals, and found three independent SNPs with genome-wide significance (i.e., p value threshold of 5×10^{-8})³⁹. The discovery sample was extended to 293,723 individuals in 2016 by the second EA GWAS (EA2), which identified 74 genome-wide significant loci associated with years of schooling

completed ⁴⁰. The most recent EA GWAS (EA3) was conducted in 2018 in a sample of approximately 1.1 million individuals ($N = 1,131,881$), the marked increase in sample size boosted the predictive accuracy/statistical power to detect genetic associations and resulted in identifying 1,271 independent genome-wide-significant SNPs ⁴¹. Based on the EA GWASs, polygenic scores (PGSs)^{42, 43} can be derived to provide a single value reflecting an individual's genetic propensity to educational attainment (referred to as "EA PGS"; it is a sum of an individual's effect alleles weighted by effect sizes obtained from the EA GWAS). For example, EA PGSs derived from EA1, EA2 and EA3 explained about 2%, 3.2% and 11-13% of the variance in educational attainment, respectively. As studies have derived individual's genetic propensity depending on the most powerful data at that time of publication, to what extent the estimated genetic nurture effects differ depending on the accuracy of EA GWASs used remains untested. Therefore, we compared effect sizes from PGSs based on different EA GWASs.

As expected and consistent with previous studies ^{12, 38}, the predictive accuracy of the GWASs used to construct the PGS significantly moderated effect sizes of genetic nurture. Estimates of genetic nurture effects based on more accurate GWASs were significantly larger ($\beta_{EA3} = 0.09$, 95% CI [0.08, 0.11], $\beta_{EA2} = 0.07$, 95% CI [0.06, 0.08], $p_{\text{genetic nurture}} = .0066$). Similar results were found for unadjusted parental effects ($\beta_{EA3} = 0.24$, 95% CI [0.21, 0.27], $\beta_{EA2} = 0.16$, 95% CI [0.15, 0.18], $\beta_{EA1} = 0.08$, 95% CI [0.01, 0.16], $p_{\text{unadjusted parental}} < .0001$) and unadjusted child effects ($\beta_{EA3} = 0.27$, 95% CI [0.241, 0.31], $\beta_{EA2} = 0.20$, 95% CI [0.16, 0.24], $\beta_{EA1} = 0.13$, 95% CI [0.05, 0.20], $p_{\text{unadjusted child}} = .0010$). However, there was no significant difference for direct genetic effects due to the larger uncertainty in estimates ($\beta_{EA3} = 0.18$, 95% CI [0.14, 0.23], $\beta_{EA2} = 0.14$, 95% CI [0.08, 0.20], $p_{\text{direct genetic}} = .1783$).

5.4 Study characteristics

We also tested the moderating roles of a number of study characteristics reflecting methodological quality and sample representativeness, including study quality, sample size and attrition rate of the cohort.

Study quality was indexed by the total score of the methodological quality described in Supplemental Notes 2.3. Detailed score of each included study see Table S3. Methodological quality was negatively associated with the magnitude of genetic nurture effects ($slope = -0.02$, $p_{\text{genetic nurture}} = .0072$) and unadjusted parental effects ($slope = 0.05$, $p_{\text{unadjusted parental}} = .0353$).

The sample size was tested in the unit of 1,000 participants due to the relatively large sample size in studies (mean = 3,372, median = 1,626). For the sample size of which each effect size was based on, see Table S4. To note, only the largest sample size assessing genetic nurture effects in each cohort was used to compute the total sample size of the current meta-analysis (i.e., 38,654) in a conservative manner to preclude any overlap within the cohort. In Table 1, sample sizes were reported per study and outcome category (i.e., educational attainment vs. educational achievement) as part of the study summary. Sample size ($slope = -0.001$, $p = .0225$) was negatively associated with the magnitude of genetic nurture effects in a modest manner.

Both moderating effects of methodological quality and sample size can be attributed to the potentially influential study ² with the highest quality score and sample size (see Table S8). Nevertheless, it suggests that more reliable studies, namely with more rigorous methodology and larger sample size, may produce more conservative estimates of genetic nurture effects on educational outcomes.

Considering the prevalent attrition in original cohorts across studies (details see Supplemental Notes 2.3), the moderating role of attrition rate was tested. For the attrition rate of each effect size, see Table S4. Among four MREM of effects on educational outcomes, attrition in the cohort did not clearly moderate any estimate ($p > .05$).

6 Family-level adjustment

We tested the moderating role of family-level adjustment, including parental education level and family socioeconomic status (SES) to quantify the extent to which genetic nurture effects can be attributed to these distal family-level factors. As shown in the last panel of Table S7, we compared effect sizes with and without family-level adjustments. Effect sizes included in the main meta-analysis were unadjusted for family-level adjustment ($k_{\text{genetic nurture}} = 22$, $k_{\text{direct genetic}} = 16$, $k_{\text{parental unadjusted}} = 8$, $k_{\text{child unadjusted}} = 11$). All available effect sizes in the included studies with family-level adjustment of parental education or family SES were extracted as adjusted estimates ($k_{\text{genetic nurture}} = 18$, $k_{\text{direct genetic}} = 11$, $k_{\text{parental unadjusted}} = 4$, $k_{\text{child unadjusted}} = 3$). Effect sizes adjusted for family-level factors were only used to test the moderating role of family-level adjustment and not included in the main meta-analysis as they were fundamentally different from unadjusted ones. It should be noted that one study¹⁰ reported genetic nurture and direct genetic effects only with family-level adjustment, and unadjusted parental and child effects both with and without family-level adjustment. Therefore, for this particular study¹⁰, only effect sizes of unadjusted parental and child effects were included in the main meta-analysis, and all effect sizes were used for the moderator analysis of family-level adjustment.

The unadjusted effects were visually larger than the family-level adjusted effects for both genetic nurture and direct genetic effects. The largest decrease in effect sizes attributable to adjustment was present for genetic nurture effects ($\beta_{\text{unadjusted}} = 0.07$, 95% CI [0.07, 0.08] vs. $\beta_{\text{adjusted}} = 0.02$, 95% CI [0.01, 0.03]), which was supported by a highly significant moderating effect ($p_{\text{adjustment}} < .0001$) and remained robust when tested in a sensitivity analysis (see Table S8). Smaller changes in effect sizes following family-level adjustment were present for direct genetic effects ($\beta_{\text{unadjusted}} = 0.17$, 95% CI [0.13, 0.20] vs. $\beta_{\text{adjusted}} = 0.14$, 95% CI [0.10, 0.18]), in which case the moderating effect was also significant ($p_{\text{adjustment}} = .0098$). After accounting for parental education level or family SES, the effect of unadjusted parental and child effects on children's educational outcomes were both attenuated by ~30%. ($p_{\text{unadjusted parent}} = .0001$, $p_{\text{unadjusted child}} = .0223$).

7 Sensitivity analyses

7.1 Robust confidence intervals of dependent estimates

Among some included studies, we extracted multiple, statistically dependent effect size estimates from the same cohort. We utilized MREM to handle the dependence of effect sizes. As sensitivity checks, we also reported, robust confidence intervals (robust CI) of cluster-robust variance estimations, which is out of the MREM framework and obtained using the package clubSandwich version 0.5.0⁴⁴ in R version 3.6.1¹⁸.

7.2 Impact of recalibrating estimates using the average parental PGS

In general, comparing estimates using maternal, paternal, maternal and/or paternal genomic measures should be straightforward by directly compared their absolute values. Caution is warranted when using the average parental PGS, in which case the R^2 (variance explained) is unbiased but the estimate is inflated (compared to using individual parental PGS):

Let PGS_M be the maternal polygenic score and PGS_P be the paternal polygenic score, and let R^2_M and R^2_P be the variance explained by PGS_M and PGS_P , respectively. The variance explained by the average parental PGS, $R^2_{ave\ parent}$, equals to the addition of variance of mother and father ($R^2_{ave\ parent} = R^2_M + R^2_P$) when assuming PGS_M and PGS_P are uncorrelated. Thus, the standardised estimated genetic nurture effects from the average parental PGS, $\beta_{ave\ parent}$ is equal to the square root of $R^2_M + R^2_P$. Assuming that genetic nurture effects from mother and father are equal ($R^2_M = R^2_P = R^2_{ind\ parent}$), and let $\beta_{ind\ parent}$ be the standardised estimate of genetic nurture effects from the individual parental PGS, the relationship between $\beta_{ave\ parent}$ and $\beta_{ind\ parent}$ are:

Genetic nurture effects from the average parental PGS:

$$\beta_{ave\ parent} = \sqrt{(2R^2_{ind\ parent})} = \sqrt{2} \beta_{ind\ parent}$$

Genetic nurture effects from the individual parental PGS:

$$\beta_{ind\ parent} = \sqrt{(R^2_{ave\ parent}/2)} = \beta_{ave\ parent}/\sqrt{2}$$

Due to the abovementioned reason, one of the included studies¹⁹ utilizing the average parental PGS to capture genetic nurture effects had an outlying estimate ($\beta_{Willoughby\ original} = 0.20$) relative to other estimates included in our study. We thus used the recalibrated estimate ($\beta_{Willoughby\ adjusted} = 0.20/\sqrt{2} = 0.14$) for the main meta-

analysis to obtain better comparability with other studies using individual parental PGS.

The impact of this adjustment is examined by meta-analysing genetic nurture effects with the originally reported effect size. Using the original estimate of average parental PGS resulted in similar genetic nurture effects ($\beta_{\text{Willoughby original}} = 0.08$, 95% CI [0.07, 0.09], robust CI [0.06, 0.10]) but introduced more publication bias ($Q = 8.59$, $p = .0034$). The moderating effect of analytical design was still significant ($Q = 6.17$, $p = 0.0457$), with smaller estimates from the virtual parent design than the statistical control approach. Such results were expected, as the adjusted average parental PGS was derived from the statistical control approach and the recalibration decreased that estimate.

In our meta-analytic pooled estimate, the magnitude of genetic nurture represents the effects from an individual parent for easier comparison between studies. However, when comparing the relative contribution of genetic nurture and direct genetic effects, genetic nurture effects from both parents should be considered. With genetic nurture of an individual parent explaining $\beta_{\text{genetic nurture}} = 0.08^2 = 0.64\%$ of variance in offspring educational outcomes and assuming effects from both parents are equal and independent, genetic nurture of both parents explains $0.64\% \times 2 = 1.28\%$ of variance in offspring educational outcomes. As such, the magnitude of genetic nurture from both parents $\beta_{\text{both parents}} = \sqrt{1.28\%} = 0.11$, and the genetic nurture effects/ direct genetic effects ratio $= 0.11/0.17 = 0.65$. This finding is consistent with recent Relatedness Disequilibrium Regression (RDR) evidence ⁴⁵, in which genetic nurture effects originating in both parents explained 6.6% of the variance in EA, corresponding to an effect size of approximately 0.26 (the square root of 0.066). RDR-estimated direct genetic effects/heritability explained 17% of the variance in EA, corresponding to an effect size of approximately 0.41 (the square root of 0.17). The ratio of genetic nurture/direct genetic effects derived from the RDR method is thus $0.26/0.41=0.63$.

7.3 Impact of a potentially influential study

Due to the Inverse-variance weighting strategy adopted in our meta-analysis, one of the included studies ² might be more influential than others since standard errors in that study (imputed based on their corresponding effect sizes and sample sizes) were very small. Therefore, we tested the impact of this potentially influential study by re-running the meta-analysis omitting its estimates. We tested the robustness of our pooled effects as well as the distribution of variance in the MREM to see whether the narrow confidence intervals and approximate homogeneity of genetic nurture effects were exclusively attributed to this study, e.g., the Kong et al. 2018 study reported three estimates of genetic nurture effects using maternal, paternal and parental non-transmitted PGS. In addition, we performed meta-regression without estimates from this study for all the moderators that were potentially impacted, since the Kong et al. 2018 study may have independently influenced the moderating effects in some cases. For example, multiple genetic nurture effects from the Kong et al. 2018 study using maternal, paternal and parental non-transmitted PGS) may unduly impact on the moderating effect of parent of origin and mask effects from other studies.

The potentially influential study of Kong et al. 2018 did not show substantial impact on distributions of variance, pooled estimates, but resulted in some publication bias and changes in moderating effects of methodological quality and sample size on the magnitude of genetic nurture effects. For details see Tables S5 and S8.

7.4 Jackknife leave-one-out analyses

To account for any other potential influences from included studies, we assessed the undue effect of individual studies on our pooled estimates through jackknife leave-one-out analyses, by testing changes in the estimate across permutations in which each study was omitted in turn. For visualization of results see Figure S2.

7.5 The moderating effect of outcome type within study

Two of the included studies ^{14, 28} assessed both educational attainment and achievement, we thus checked the robustness of moderating effect of outcome type within study by running the meta regression within these two particular studies. As

shown in Table S9, the moderation role for outcome type on genetic nurture effects became statistically significant ($\beta_{\text{attainment}} = 0.11$, 95% CI [0.08, 0.14], $\beta_{\text{achievement}} = 0.05$, 95% CI [0.01, 0.09], $p = .0228$). The difference in direct genetic effects was larger in the opposite direction ($\beta_{\text{attainment}} = 0.12$, 95% CI [0.07, 0.17]), $\beta_{\text{achievement}} = 0.21$, 95% CI [0.16, 0.26], $p = .0144$).

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