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

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Summary

Similarities between parents and offspring arise from nature and nurture. Beyond this simple dichotomy, recent genomic studies have uncovered "genetic nurture" effects, whereby parental genotypes influence offspring outcomes via environmental pathways rather than genetic transmission. Such genetic nurture effects also need to be accounted for to accurately estimate "direct" genetic effects (i.e., genetic effects on a trait originating in the offspring). Empirical studies have indicated that genetic nurture effects are particularly relevant to the intergenerational transmission of risk for child educational outcomes, which are, in turn, associated with major psychological and health milestones throughout the life course. These findings have yet to be systematically appraised across contexts. We conducted a systematic review and meta-analysis to quantify genetic nurture effects on educational outcomes. A total of 12 studies comprising 38,654 distinct parent(s)-offspring pairs or trios from 8 cohorts reported 22 estimates of genetic nurture effects. Genetic nurture effects on offspring's educational outcomes ($\beta_{\text{genetic nurture}} = 0.08$, 95% CI [0.07, 0.09]) were smaller than direct genetic effects ($\beta_{\text{direct genetic}} =$ 0.17, 95% CI [0.13, 0.20]). Findings were largely consistent across studies. Genetic nurture effects originating from mothers and fathers were of similar magnitude, highlighting the need for a greater inclusion of fathers in educational research. Genetic nurture effects were largely explained by observed parental education and socioeconomic status, pointing to their role in environmental pathways shaping child educational outcomes. Findings provide consistent evidence that environmentally mediated parental genetic influences contribute to the intergenerational transmission of educational outcomes, in addition to effects due to genetic transmission.

Introduction

Educational attainment is defined as the highest education level a person attains. A related construct is educational achievement, which refers to one's school performance. These two constructs are prospectively associated with major psychological, social, economic, and health milestones throughout the life course.[1–3] Parents' educational levels are important early predictors of their offspring's own educational attainment and achievement.[4] It is crucial to understand the processes underlying this transmission of educational attainment and achievement, which can lead to cycles of disadvantage across generations.

Positive associations between parents' education and their offspring's education are found in nearly every society.[5] For example, correlations between parents' and offspring's educational outcomes were consistent across 12 Western countries with estimates ranging from $r = 0.30$ (Denmark) to 0.46 (U.S.).[6] Parent-offspring resemblance in educational outcomes can be attributed to nature (genetic variants that offspring inherit from their parents) and nurture (the environment that parents provide for their offspring).[7] These nature and nurture effects are complex and intertwined. For example, the environment created by parents can be partly shaped by genetic influences; parents with a higher genetic propensity for learning may have a greater interest in activities such as reading that, in turn, nurture learning in their offspring.

"Genetic nurture" is used to describe the phenomenon by which parental nature (i.e., parental genotype) influences offspring outcomes by shaping the environment that parents provide.[8] Genetic nurture effects can therefore be considered to be indirect effects from parental genotype to offspring outcomes that are mediated through environmental pathways whereas "nature" effects correspond to the direct transmission of parental genotypes to the child. Importantly, such direct genetic transmission from parent to offspring can generate correlations between parental and child educational outcomes in the absence of any effect of parental nurture in shaping child outcomes (a phenomenon akin to passive gene-environment correlation). Conversely, genetic nurture effects are free from genetic confounding arising from genetic variants shared between parents and offspring. As such, evidence of genetic nurture effects suggests that environmental pathways matter when it comes to shaping children's educational outcomes, even after accounting for genetic transmission. The interpretation of genetic nurture effects must be considered in light

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https://doi.org/10.1016/j.ajhg.2021.07.010.

of some limitations and assumptions, outlined here and further developed in the discussion section. First, despite the term "nurture," genetic nurture may exist without actual parent-offspring nurturing behavior but instead operate through distal factors, inside or outside the home, that are correlated with parental genotypes, such as income or school quality. Thus, detecting genetic nurture effects does not, per se, identify which environmental pathways are implicated. In addition, genetic nurture effects only reflect genuine environmental pathways of transmission when population stratification and assortative mating are entirely accounted for. In the presence of population stratification and assortative mating, spurious genetic nurture effects may be detected even in the absence of what has been termed cultural transmission (i.e., the causal effect of the environment on child outcomes).[9]

Recent methodological advances combined with genome-wide data have enabled the estimation of genetic nurture and direct genetic effects. These methods rely on genome-wide association studies (GWASs) for educational attainment (EA) to generate polygenic scores. Specifically, polygenic scores (PGSs) can be derived from GWASs of EA 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). Two studies[8,10] adopted a novel design to assess the magnitude of genetic nurture effects by constructing a parental PGS based on alleles that are not transmitted to the offspring. The association of such a PGS with offspring outcomes cannot arise from genetic transmission but can occur through environmental pathways and thereby reflects genetic nurture effects by design. This approach is termed the "virtual parent design" (further description in supplemental note 1.1). Notably, because the effect of a child's genotype on their outcomes can reflect both direct and genetic nurture effects, the association between a child's PGS and their own outcomes can be overestimated when genetic nurture is not accounted for.[8] Direct genetic effects represent genetic influences that originate in the child genotype and must be corrected for genetic nurture effects. In addition to assessing non-transmitted and transmitted alleles, genetic nurture and direct genetic effects can also be obtained by estimating the effect of parental PGS(s) on offspring outcomes, while statistically controlling for the offspring PGS (for further description see supplemental note 1.2). This statistical control approach has been applied in several studies.[11,12] The statistical control approach requires genotyped trios (mother-father-child) to obtain unbiased estimates but can nonetheless provide an approximation of genetic nurture effects when only genetic data of parent-child pairs are available.[13,14]

Such approaches have now been implemented to estimate genetic nurture and direct effects on child educational outcomes in different contexts, such as using cohorts from different countries, using maternal and/or paternal PGS(s), or capitalizing on increasingly larger genomic datasets.[15,16] However, these findings have yet to be systematically appraised and moderators fully investigated. Here we present a meta-analysis of (1) genetic nurture effects on child educational outcomes, (2) direct genetic effects child educational outcomes, and (3) key moderators of these effects.

Subjects and methods

Search strategy and study selection

This systematic review and meta-analysis was performed in line with the Preferred Reporting Items for Systematic Reviews and the Meta-Analyses (PRISMA[17]) statement and Meta-Analyses of Observational Studies in Epidemiology (MOOSE[18]) guidelines (Tables S1 and S2). The protocol was registered on the Open Science Framework (https://osf.io/q8b25/). The literature search was performed in July 2020. We searched Ovid (MEDLINE, EMBASE, PsycINFO), Web of Science Core Collection, and PubMed for peer-reviewed articles written in English. To estimate genetic nurture effects on educational outcomes, we considered articles estimating genetic nurture in parent(s)-offspring samples using EA PGSs. Therefore, the publication period was limited to 2013 onward, when the first EA GWAS[19] became available. To retrieve relevant publications, the search included terms related to: (1) educational outcomes, (2) polygenic scores, and (3) genetic nurture effects. A detailed literature search strategy and terms are presented in supplemental note 2.1. Two authors (B.W. and T.S.) independently screened titles and abstracts of all articles retrieved during the search before reviewing the full text of potentially eligible studies (see criteria below). Disagreements were resolved through discussion with the senior researcher (J.-B.P.).

Eligible studies met the following criteria: (1) they assessed offspring educational attainment (e.g., years of education, highest degree obtained) or educational achievement (e.g., national test scores or levels, school grades) in the general population, (2) the exposure variable(s) included genomic proxies for education in parents and offspring, measured in the form of PGSs[20] derived from the EA GWASs, and (3) they derived estimates for genetic nurture effects on education based on one of the following designs that rely on genotype data from parents and their biological offspring: (a) virtual parent: testing whether the PGSs calculated from parents' non-transmitted alleles predict offspring educational outcomes; or (b) statistical control: testing whether parents' PGSs predict offspring educational outcomes over and above offspring's own PGS. For more information on inclusion criteria, see supplemental note 2.2.

Quality assessment, data extraction, and effect size calculation

The methodological quality of each included study was independently assessed by two of the authors (B.W. and one additional author among J.R.B., W.B., and R.C.) using an adapted version of the Newcastle-Ottawa scale (NOS[21]). The NOS was adapted for use on genetically informed studies and included nine questions tapping into four wider aspects relevant to study quality, including the quality of cohort selection, the assessment of exposure, the level of comparability of the cohort, and the assessment of outcomes. Overall study quality was indexed as a sum score ranging from 0 to 9 (see supplemental note 2.3 for detailed scoring criteria and Table S3 for scores of included studies).

Data extraction for each included study was independently performed by two of the authors (B.W. and one additional author among J.R.B., W.B., and R.C.). The following data were extracted: publication characteristics (study name, first author, year), sample characteristics (cohort name, sample size, population source, ethnicity, sex distribution), study design (virtual parent or statistical control), calculation of PGSs (the GWAS used to derive the PGS, PGS threshold, source/parent of origin of genotype, whether standardized), education-related outcomes assessed (educational outcome, outcome type, age at assessment, whether standardized), effect size (estimation type, estimation, 95% CI or standard error of the estimation), and confounding variables adjusted for. Where information was missing, original study authors were contacted to request the information.

As a common metric, we extracted (or converted effect sizes to) standardized beta coefficients and corresponding standard errors from all individual studies. These data were then included in our meta-analytical models to derive the pooled estimate of genetic nurture effects. For studies using the virtual parent design, we extracted standardized regression coefficients for the non-transmitted PGS. For studies using the statistical control design, we extracted adjusted standardized regression coefficients for the parental PGS(s), while controlling for the offspring's PGS. For studies reporting effect estimates in metrics other than standardized beta or without corresponding standard errors, we transformed the reported statistics using the formulae included in the R package compute.es_0.2-4.[22] One estimate of genetic nurture derived from an average parental PGS was recalibrated to be comparable with other studies using PGSs of individual parents (for justification see supplemental note 7.2). Estimates of direct genetic effects were extracted when available or imputable (i.e., the difference between standardized regression coefficients of transmitted PGS and non-transmitted PGS in the virtual parent design or adjusted standardized regression coefficients of offspring's PGS while statistically controlling for parental PGSs). Whenever applicable, we also derived unadjusted parental or child effects, namely unadjusted regression coefficients of the effect of parental or offspring's PGSs on offspring educational outcomes. For more information on the effect size transformation and calculation, see supplemental note 3.1.

With each article reviewed and coded by two authors, the two coders had inter-rater reliabilities of 92.6% on quality assessment and 97.8% on data extraction. Before moving on to analyses, discrepancies were reviewed and arbitrated by the two coders, and disagreements were resolved through discussion with the senior researcher (J.-B.P.).

Statistical analysis

Analyses were conducted in R v.3.6.1[23] using the *metafor* package (v.2.4-0).[24] Since multiple effect sizes were reported in individual studies and cohorts, we used three-level Multilevel Random-Effects Models (MREMs) to account for dependencies among effect sizes within single studies/cohorts (i.e., correlation between effect sizes). These models incorporate three variance components; namely sampling variance at level 1 (variance that is unique for each estimated effect size), within-cohort variance at level 2 (variance across outcomes within a cohort), and between-cohort variance at level 3 (variance across cohorts). For more information on multilevel random-effects models, see supplemental note 3.2. We assessed the heterogeneity between studies using the I^2 statistic and tested whether heterogeneity of effect sizes at level 2 (within-cohort heterogeneity) and level 3 (between-cohort heterogeneity) were statistically significant by conducting two separate one-sided log-likelihood ratio tests.[25] Publication bias was visually assessed by checking the asymmetry of funnel plots and more formally tested by using precision (sampling variance) as a moderator in meta-analysis models.[26]

Meta-regression analyses were performed to explore potential sources of heterogeneity in effect sizes. We tested four main categorical moderators: (1) whether the parental PGS was constructed based on maternal, paternal, or the mixture of both parents' genotypes, (2) the type of analytic method used to estimate the genetic nurture effects (virtual parent, partial or full statistical control), (3) the type of educational outcome assessed (educational attainment or educational achievement), and (4) the specific GWAS summary statistics used to derive PGSs (EA1 with $N = 101{,}069$,[19] EA2 with $N = 293{,}723$,[27] or EA3 with $N = 1{,}131{,}881$[28]). In addition, we tested the moderating role of study characteristics (i.e., methodological quality, sample size, and attrition in cohorts). For more information on moderator analyses, see supplemental note 5. To explore potential environmental pathways genetic nurture operates through, we tested the extent to which genetic nurture effects attenuated in estimates that adjusted for observed parental educational levels and family socioeconomic status (SES) (details in supplemental note 6).

Lastly, we undertook a series of sensitivity checks to evaluate the robustness of our results including computing robust confidence intervals, evaluating the impact of recalibrating effects derived from average parental PGS in one study,[29] assessing the impact of a potentially influential study,[8] performing jackknife leave-one-out analyses, and assessing the moderating effect of outcome type within studies (i.e., when educational attainment and achievement were measured in the same study). For more information on sensitivity analyses, see supplemental note 7. In all tests, a 2-tailed $p < 0.05$ was considered statistically significant.

Results

Study description

Twelve studies met the inclusion criteria (see Figure 1 for the study selection procedure, Table 1 for a study summary, and Table S4 for further details). The studies comprised 38,654 distinct offspring individuals with at least one genotyped parent (for computation of total sample size, see supplemental note 5.4) across eight study cohorts from the United Kingdom, Australia, the United States, the Netherlands, and Iceland. We derived $k = 22$ estimates of genetic nurture effects on educational outcomes and $k = 16$ estimates of direct genetic effects. The majority of genetic nurture estimates were derived from studies using the statistical control approach (68.2% [$k = 15$]) and the rest from the virtual parent design (31.8% [$k = 7$]). Slightly more studies focused on educational achievement (54.5% [$k = 12$]) versus educational attainment (45.5% [$k = 10$]).

Genetic nurture effects on offspring educational outcomes

Genetic nurture had a small but robust effect on offspring educational outcomes ($\beta_{\text{genetic nurture}} = 0.08$, 95% CI [0.07, 0.09], robust CI [0.06, 0.10]; Table 2; Figure 2). Variances

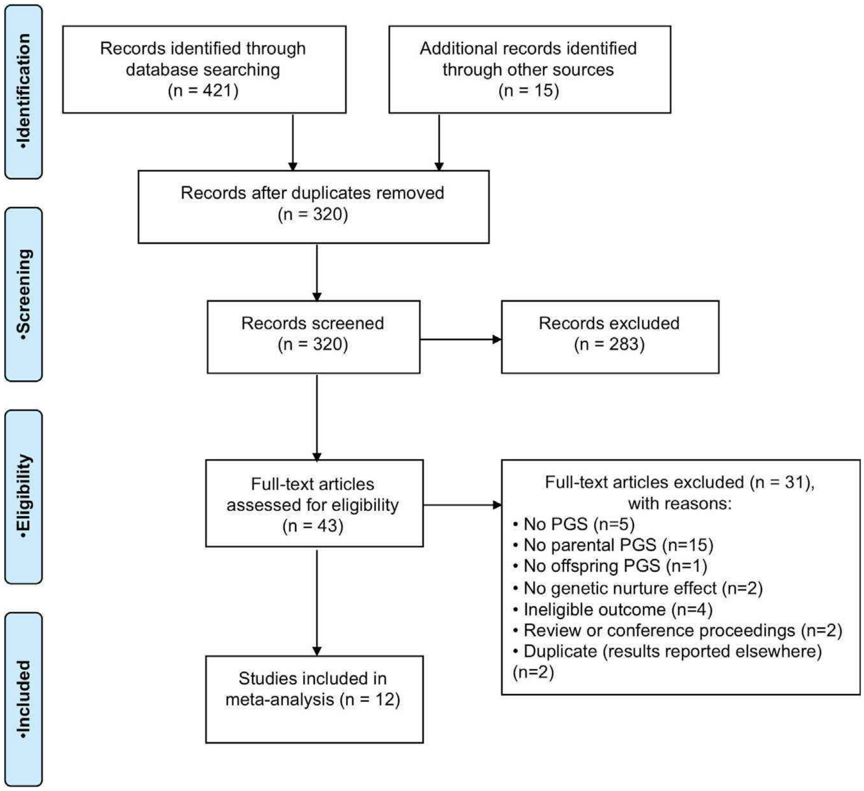

Figure 1. Flow chart of identification of eligible studies

among different estimates of genetic nurture effects was largely attributed to sampling differences ($I^2_{\text{Level 1}}$ = 76.80%). Within-cohort heterogeneity was close to null ($I^2_{\text{Level 2}}$ = < 0.01%) and between-cohort heterogeneity was minimal ($I^2_{\text{Level 3}}$ = 23.20%), suggesting largely homogeneous genetic nurture effects across studies. We found some evidence of publication bias in genetic nurture effects ($Q = 6.12$, $p = 0.0134$) although the funnel plot was visually symmetric (Figure S1). This bias was no longer present in the sensitivity analysis when excluding the potentially influential study[8] ($Q = 0.88$, $p = 0.3486$, see Table S5). Results from jackknife analyses suggested no unduly large effects arising from any individual study (Figure S2). The supplemental material includes more findings regarding unadjusted effects of parental PGS on offspring educational outcomes (supplemental note 4.1, Table S6, Figures S3, S5, and S6).

Direct genetic effects on offspring educational outcomes

Direct genetic effects on offspring educational outcomes were greater in magnitude than genetic nurture effects ($\beta_{\text{direct genetic}}$ = 0.17, 95% CI [0.13, 0.20], robust CI [0.12, 0.21]; Table 2; Figure 2). Variance among estimates of direct genetic effects was largely attributable to between-cohort heterogeneity ($I^2_{\text{Level 3}}$ = 82.33%), with 17.67% (i.e., $I^2_{\text{Level 1}}$) explained by random sampling and negligible within-cohort heterogeneity ($I^2_{\text{Level 2}}$ = < 0.01%). The funnel plot (Figure S1) and formal test with precision as a moderator (Table 2) suggested no publication bias in estimates of direct genetic effects. Jackknife analyses suggested that no single study unduly influenced meta-analysis estimates (Figure S2). For findings regarding unadjusted effects of child PGS on educational outcomes, see supplemental note 4.2, Table S6, Figures S4–S6.

Sources of heterogeneity in genetic nurture and direct genetic effects on educational outcomes

Moderator analyses (Table 3) suggested similar effects of genetic nurture on educational outcomes regardless of whether effect sizes were obtained using polygenic scores derived from mothers only (β_{mother} = 0.08, 95% CI [0.07, 0.10]), from fathers only (β_{father} = 0.07, 95% CI [0.06, 0.09]), or from either parent or a mean parental PGS (β_{parents} = 0.08, 95% CI [0.06, 0.10]). Likewise, whether PGSs were based on mothers, fathers, or the mixture of both did not moderate direct genetic effects (β_{mother} = 0.17, 95% CI [0.12, 0.23], β_{father} = 0.20, 95% CI [0.13, 0.27], β_{parents} = 0.16, 95% CI [0.12, 0.20]). There was no evidence for moderating effects of parent of origin ($p_{\text{genetic nurture}}$ = 0.6680 and $p_{\text{direct genetic}}$ = 0.4885). These findings were robust to the removal of the potentially influential study[8] (Table S8). Results for other potential moderators are reported in supplemental note 5 and Table S7. After adjusting for phenotypic family-level factors (i.e., parental educational level or family SES), genetic nurture effects attenuated to a large extent ($k_{\text{unadjusted}}$ = 22, $\beta_{\text{unadjusted}}$ = 0.07, 95% CI [0.07, 0.08] versus k_{adjusted} = 18, β_{adjusted} = 0.02, 95% CI [0.01, 0.03], $p_{\text{adjustment}} < 0.0001$); for more details, see supplemental note 6.

Discussion

Across 12 studies that included 38,654 distinct parent(s)-offspring pairs or trios from 8 cohorts, we found strong evidence to support the notion that genetic nurture plays an important role in children's educational outcomes. The magnitude of genetic nurture effects was largely consistent across studies, was similar in both parents, and was largely explained by parental educational level and family socioeconomic status. After accounting for genetic nurture, we also observed substantial direct genetic effects on offspring education, due to genetic inheritance.

Table 1. Studies investigating genetic nurture effects on educational outcomes

Cohort[a]	Publication	Outcome[b]	Effective N[c]	Design	GWAS[d]	NOS score[e]
Born in Bradford birth cohort (BiB), UK	Armstrong-Carter et al.[13]	key stage 1 school-based exam score	1,267	statistical control	EA3	6.5
The Brisbane Adolescent Twin Study (BATS), Australia	Bates et al.[10]	The Queensland Core Skills Test	2,335	virtual parent	EA2	7.5
The Brisbane Adolescent Twin Study (BATS), Australia	Bates et al.[15]	The Queensland Core Skills Test	2,335	virtual parent	EA3	7.5
The Environmental Risk Longitudinal Twin Study (E-Risk), UK	Belsky et al.[30]	GCSE academic qualification level	1,574	statistical control	EA3	7.0
The Framingham Heart Study (FHS), USA	Conley et al.[12]	years of schooling	968	statistical control	EA1	5.0
The Netherlands Twin Register (NTR), the Netherlands	de Zeeuw et al.[16]	highest obtained degree; nationwide educational achievement test	1,931;1,120	virtual parent	EA3	7.0
The Icelandic quantitative trait cohorts (deCODE), Iceland	Kong et al.[8]	years of education completed	21,637	virtual parent	EA2	7.5
The Framingham Heart Study (FHS), USA	Liu[14]	years of education completed	6,298	statistical control	EA2	6.5
The Avon Longitudinal Study of Parents and Children (ALSPAC), UK	Morris et al.[11]	key stage 4 school-based exam score	1,095	statistical control	EA3	7.0
The Minnesota Twin Family Study (MTFS), USA	Rustichini et al.[31]	years of education completed;high school grades	1,690; 1,583	statistical control	EA3	6.0
The Environmental Risk Longitudinal Twin Study(E-Risk), UK	Wertz et al.[32]	GCSE academic qualification level	860	statistical control	EA3	6.0
The Minnesota Center for Twin and Family Research (MCTFR) [a], USA	Willoughy et al.[29]	years of education completed	2,517	statistical control	EA3	5.5

[a]Participants in the MCTFR cohort were drawn from several longitudinal studies including the MTFS cohort, so in the meta-analysis they were considered as the same cohort.
[b]Educational outcomes consists of two broad categories, i.e., attainment and achievement. Years of schooling/education completed and highest obtained degree are categorized as educational attainment; the rest are categorized as educational achievement. More details of outcomes, including assessment time, are provided in Table S3.
[c]The largest sample size used to assess genetic nurture effects.
[d]GWASs (genome-wide association studies) used to derive the polygenic scores, including EA1 with N = 101,069,[19] EA2 with N = 293,723,[27] and EA3 with N = 1,131,881.[28]
[e]Quality score ranged from 0 (lowest) to 9 (highest) on methodological quality using an adjusted version of the Newcastle-Ottawa scale, criteria showed in subjects and methods and detailed scoring showed in Table S4.

Table 2. Three-level random effects models of genetic nurture and direct genetic effects on educational outcomes

	Genetic nurture effects	Direct genetic effects
k_{cohort}	8	8
$k_{estimate}$	22	16
β_{pooled}	0.08	0.17
$\beta_{95\%\ CI}$	0.07–0.09	0.13–0.20
$\beta_{robust\ CI}$[a]	0.06–0.10	0.12–0.21
$\sigma^2_{Level\ 2}$	$\chi^2 < 0.01$, $p = 0.5000$	$\chi^2 < 0.01$, $p = 0.5000$
$\sigma^2_{Level\ 3}$	$\chi^2 = 1.94$, $p = 0.0817$	$\chi^2 = 5.09$, $p = 0.0120$
$I^2_{Level\ 1}$	76.80%	17.67%
$I^2_{Level\ 2}$	<0.01%	<0.01%
$I^2_{Level\ 3}$	23.20%	82.33%
Publication bias	$Q = 6.12$, $p = 0.0134$	$Q = 0$, $p = 0.9976$

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 ($\sigma^2_{Level\ 2}$) and between-cohort variance ($\sigma^2_{Level\ 3}$) 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 was assessed by using precision (sampling variance) to predict the effect size.
[a]Robust confidence intervals were cluster-robust variance estimations; for details see supplemental note 7.1.

Genomic prediction of education: Evidence for genetic nurture and direct genetic effects

We observed a small effect of genetic nurture ($\beta_{genetic\ nurture} = 0.08$) on educational outcomes. Scaled with reference to two of the included studies, this could be translated to approximately 2 months of schooling[14,29] or 0.07 of GPA (4.0 scale)[31] gained in the United States for every standard deviation change in parental EA PGS(s). Our pooled estimate of direct genetic effects ($\beta_{direct\ genetic} = 0.17$) free from inflation due to genetic nurture corresponds to the lower bound of previous genomic predictions of educational outcomes within twin pairs (e.g., $\beta = 0.17$–27).[29,33] While we did observe substantial heterogeneity across cohorts in estimates of direct genetic effects, this may reflect differences in cohort characteristics (i.e., measurement of achievement or attainment) rather than actual heterogeneity in direct genetic effects between populations. Previous findings suggested that differential effects of the same variants across environments may reflect heterogeneity in phenotypic measurement or gene-environment interactions rather than true genetic heterogeneity.[28,34]

It is worth noting that our pooled estimate of genetic nurture represents the effects from an individual parent and should therefore be recalibrated to compare its relative size to the pooled estimate for direct genetic effects. With genetic nurture from both parents explaining potentially 1.28% ($2*\beta_{genetic\ nurture}^2$) of variance in offspring educational outcomes, the standardized effect size of genetic nurture from both parents can be estimated to be 0.11 (i.e., $\sqrt{1.28\%}$). As such, the ratio of genetic nurture effects originating in both parents and direct genetic effects originating in the offspring is about 0.65 (further information regarding this ratio is provided in supplemental note 7.2). This ratio corresponds well to the ratio of 0.63 derived from the relatedness disequilibrium regression (RDR) method, in which heritability is estimated by exploiting variation in relatedness due to random Mendelian segregation.[35] In addition to methods relying on genomic data of children and their biological parent(s), a few recent studies have implemented sibling[33] and adoption[36,37] designs to investigate genetic nurture effects. As evidence from these alternative designs accumulate, it will be key to examine the consistency of estimates across designs.[38]

It is worth noting that this meta-analysis can only detect genetic nurture and direct genetic effects to the extent that PGS capture heritability in educational outcomes. To date, PGSs based on the most accurate GWASs still only capture a fraction of the corresponding heritabilities.[39,40] RDR findings provided a "ceiling" for potential gains from increasing the predictive accuracy of PGSs.[35] Our estimate of genetic nurture based on PGSs explained 1.28% (supplemental note 7.2) of variance in offspring educational outcomes (versus 6.6% for RDR), while direct genetic effects based on PGSs explained 2.89% of variance in educational outcomes (calculated as $\beta_{direct\ genetic}^2$) (versus 17% for RDR).

While missing heritability may lead to underestimates of the true extent of genetic nurture, assortative mating and population stratification may have inflated our genetic nurture effects.[9] Bias resulting from assortative mating has been found to be small in magnitude,[8] although its exact magnitude remains unclear.[9] Population stratification was controlled for by using principal component analysis in most studies included in the meta-analysis but residual population stratification may still exist. Emerging methods should, in the future, better account for these potential sources of bias by capitalizing further on family-based designs.[41–43]

Genomic prediction of education: Sources of heterogeneity

There are several explanations for observing genetic nurture effects of similar magnitude in mothers and

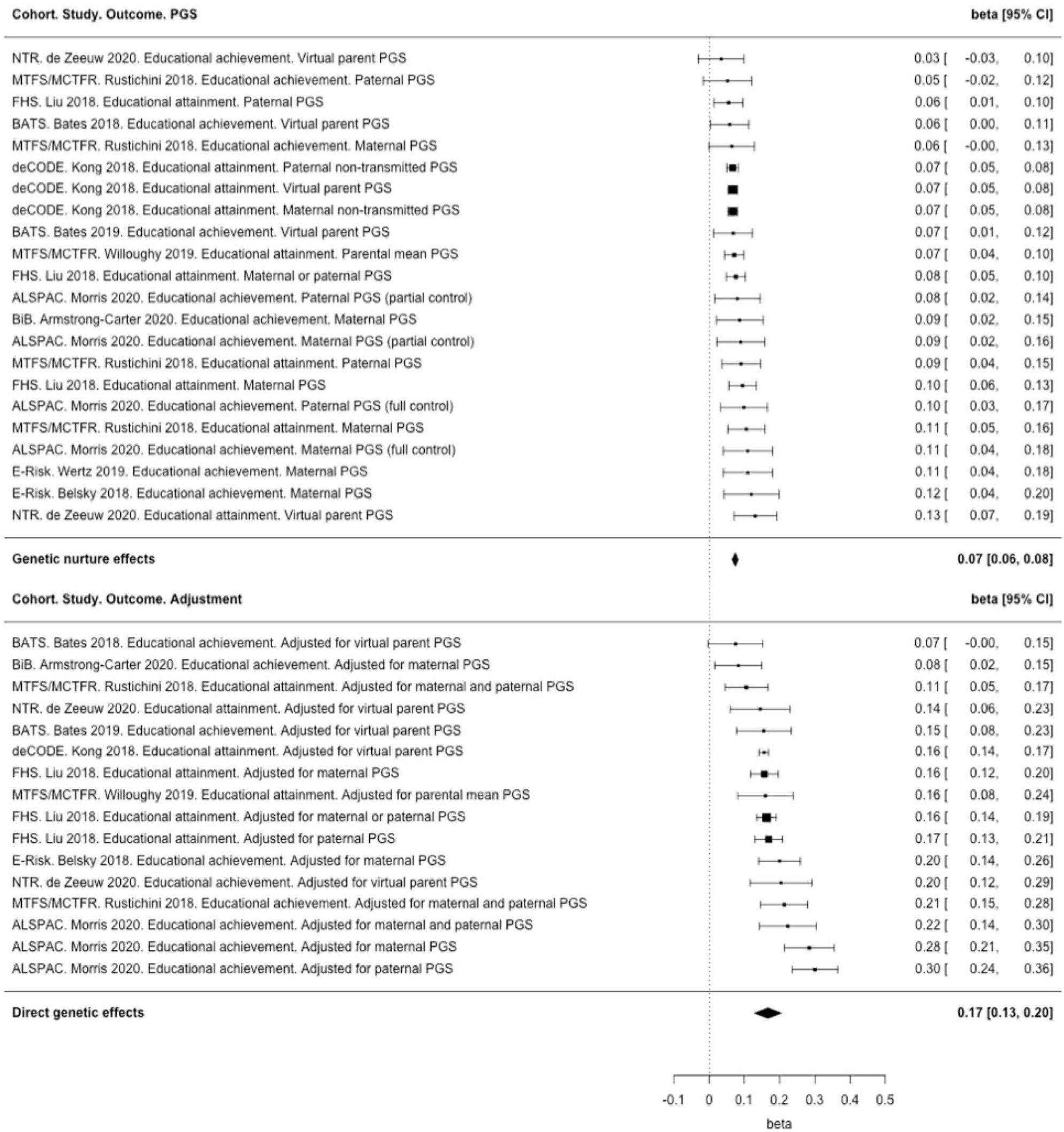

Figure 2. Forest plot of multilevel random effects model for genetic nurture effects and direct genetic effects on educational outcomes

Effect sizes were standardized beta coefficients, which represent how many standard deviations of change in educational outcome occur per standard deviation of change in EA PGS.

fathers. First, it is possible that both parents are equally important in shaping the environment that, in turn, influences their offspring's educational outcomes. However, our findings do not preclude the possibility that parents may influence child educational outcomes through different mechanisms (e.g., via distal factors like increased family income or by proximal factors like reading to the child). Behavioral studies have shown that the relationship between parental involvement and children's educational achievement was equally strong for fathers and mothers.[44,45] In light of this and our findings, a renewed emphasis on the role of fathers is needed and, whenever possible, fathers should be included in research and intervention efforts. Research should also examine genetic nurture effects in alternative family arrangements (e.g., single-parent families) and in families with varying levels of parental involvement. In the presence of genuine nurturing effects, we would expect genetic nurture effects

Table 3. Moderator analysis: sources of heterogeneity in MREM of genetic nurture effects and direct genetic effects

		Genetic nurture effects					Direct genetic effects				
Moderator	Subgroup	k_{cohort}	$k_{estimate}$	β_{pooled}	$\beta_{95\%\ CI}$	$p_{moderator}$	k_{cohort}	$k_{estimate}$	β_{pooled}	$\beta_{95\%\ CI}$	$p_{moderator}$
Parental PGS[a]	Maternal	6	9	0.08	0.07–0.10	.6680	4	4	0.17	0.12–0.23	.4885
	Paternal	4	6	0.07	0.06–0.09	–	2	2	0.20	0.13–0.27	–
	mixed parental	5	7	0.08	0.06–0.09	–	6	10	0.16	0.12–0.20	–
Design[b]	virtual parent	3	7	0.07	0.06–0.08	.0443	3	5	0.15	0.08–0.21	.5039
	partial statistical control	5	9	0.09	0.07–0.10	–	5	8	0.18	0.13–0.24	–
	full statistical control	2	6	0.09	0.06–0.11	–	2	3	0.15	0.08–0.22	–
Outcome[c]	educational attainment	4	10	0.09	0.07–0.11	.3079	4	7	0.14	0.08–0.19	.0466
	educational achievement	6	12	0.07	0.05–0.10	–	6	9	0.19	0.14–0.24	–
GWAS[d]	EA3	6	15	0.09	0.08–0.11	.0066	6	11	0.18	0.14–0.23	.1784
	EA2	3	7	0.07	0.06–0.08	–	3	5	0.14	0.08–0.20	–
Methodological quality[e]	NOS score	8	22	−0.02	−0.03–0.00	.0072	8	16	0.01	−0.07–0.08	.8692
Sample size[f]	effective N	8	22	0.00	0.00–0.00	.0225	8	16	0.00	−0.01–0.00	.7390
Attrition in cohort[g]	attrition rate	8	22	−0.01	−0.05–0.03	.7046	8	16	0.03	−0.07–0.13	.5466
Parental education/ family SES[h]	unadjusted	8	22	0.07	0.07–0.08	<.0001	8	16	0.17	0.13–0.20	.0098
	adjusted	5	18	0.02	0.01–0.03	–	3	11	0.14	0.10–0.18	–

For moderators marked with footnotes a–d and h, 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.
For moderators marked with footnotes e–g, the moderator was treated as a continuous variable.
[a]Parental genotype used to calculate polygenic score (PGS) as a categorical moderator with three categories: maternal (PGS derived from maternal genotype), paternal (PGS derived from paternal genotype), and mixed parental (PGS derived from mixed information from mothers and fathers, such as PGS from maternal or paternal genotype, PGS from the average of maternal and paternal genotype).
[b]Study design applied as a categorical moderator with three categories: virtual parent (using non-transmitted PGS to predict offspring EA), partial statistical control (using PGS of one parent to predict offspring educational outcomes while controlling for child's PGS), and full statistical control (using PGS of one parent to predict offspring educational outcomes while controlling for child's and the other parent's PGS).
[c]Type of the outcome assessed as a dichotomized moderator (educational attainment [the highest level of education completed, e.g., year of schooling], educational achievement [performance at school, e.g., high school grades]).
[d]GWASs used to compute PGS as a dichotomized moderator: EA3[28] (N = 1,131,881) and EA2[27] (N = 293,723). One study used EA1[19] (N = 101,069) but only reported estimates adjusted for parental education level, and thus was not included in the main meta-analysis but was included in the moderator analysis (moderator h).
[e]Quality score assessed by the adapted NOS (see details in Table S3), reflecting the methodological rigor of the study, as a continuous moderator.
[f]Number of participants to compute the estimate, reflecting the effective sample size, as a continuous moderator.
[g]Attrition in the cohort due to selective genotyping or outcome availability, reflecting the cohort representativeness, as a continuous moderator.
[h]Family-level adjustment as a binary moderator (0, unadjusted estimates; 1, adjusted estimates [estimates adjusted for parental education level or family socioeconomic status]).

on educational outcomes to vary accordingly (e.g., be stronger for the most involved parent), which could help shed further light on environmental factors mediating genetic nurture effects. Second, genetic nurture may operate through the broad family-level environments shared by both parents (e.g., neighborhood). Future investigations are required to identify such environmental mediators. A new genomic variance decomposition method[46] makes it possible to estimate the total variance explained by maternal versus paternal indirect genetic effects (not limited to PGS), and the covariance between maternal and paternal effects. This opens up opportunities to understand intrafamilial mechanisms in more depth. Third, spurious genetic nurture effects can arise from residual population stratification, in the absence of cultural transmission. This may help to explain why intergenerational twin studies, which are not affected by population stratification, report very weak or no evidence for cultural transmission. For example, twin studies found very little evidence of cultural transmission for intelligence,[47,48] reading performance,[49,50] and educational attainment.[51] Alternatively, it is possible that the polygenic score for education captures genuine genetic nurture effects reflecting a multiplicity of small environmentally mediated effects via a large range of intermediate variables within or outside the home. In which case, we would expect intergenerational twin studies to find only weak effects for any particular phenotype. We discuss additional sources of heterogeneity in genetic nurture in supplemental note 5.

Notably, accounting for observed measures of parental education or family SES decreased the effect of genetic nurture by three quarters. This suggests that a substantial amount of genetic nurture effects may be attributed to environmental pathways directly related to parental education, occupation, and income. It echoes the evidence that children's educational outcomes are influenced by the availability of resources in their family, indicated either by socioeconomic background or the education of their parents.[5,52,53] Future investigations should explore specific family-level pathways through which genetic nurture operates to inform compensatory interventions (e.g., financial support versus schooling access). Importantly, the finding that broad family-level social economic characteristics largely explain genetic nurture effects does not preclude the importance of proximal factors such as parenting in the chain of factors leading to educational outcomes.

Implications

Our study highlights that the environment created by parents relates to their offspring's educational outcomes independent of genetic transmission. Although the magnitude of this genetic nurture effect is small based on conventional metrics,[54] it is likely to be an underestimate given that PGSs capture only a fraction of heritability in educational outcomes—and thus will likely increase as the explanatory power of PGS increases. Understanding the specific environmental pathways through which genetic nurture operates may help to design better compensatory interventions to break the intergenerational cycle of educational underachievement. Such interventions could target environmental pathways by either targeting distal risk factors for educational outcomes (e.g., parental education, income distribution, equal access to good quality schooling) or more proximal pathways (rearing environment such as parenting). Nevertheless, it is important to note that how well children do in school does depend to a substantial degree on the genetic lottery (i.e., inheriting more genetic variants associated with educational success), a finding that policy makers often overlook[55] or arguably misinterpret.[56] At a broader level, our findings provide strong evidence that differences in education are consistently influenced by both endogenous sources of educational inequalities (e.g., one's own genetics) and exogenous sources of inequalities including genetic nurture effects originating in parents and mediated partially through broad-level family characteristics like SES. All these endogenous and exogenous sources of educational inequalities are largely beyond a child's responsibility/control and each may therefore further motivate compensatory interventions.

Limitations

First, we cannot completely rule out bias from unmeasured assortative mating, residual population stratification, and sibling genetic nurture,[41,42] which may inflate genetic nurture effects. Second, all included studies were conducted in a few developed Western countries. The similarities in populations and social contexts may lead to an overestimation of the homogeneity of genetic nurture effects. Third, all included studies were based on European ancestry populations and thus have a profound Eurocentric bias. The generalizability of our estimates to non-European population is unclear as genomic measures are not necessarily accurate across populations.[57] For example, the PGS constructed from EA3, which was conducted in white Europeans, captures 10.6% of the variation of educational attainment in white Americans but only about 1.6% of the variation among African Americans.[28] Fourth, differential measurement error in outcomes may affect genetic (nurture) effect sizes. Comparison between different outcome types (e.g., educational attainment versus achievement) should therefore be interpreted with caution.

Conclusions

This meta-analysis demonstrates that the genetics of parents influence their children's educational outcomes through the rearing environments that parents provide. This "genetic nurture" effect is largely consistent across studies and is similar for mothers and fathers. Genetic nurture effects originating in both parents are about two thirds of the size of direct genetic effects originating in the offspring due to genetic transmission. The effect of genetic nurture on child educational outcomes is largely explained by observed parental education and socioeconomic status. Further research is required to explore other downstream environmental pathways through which genetic nurture affects the intergenerational cycle of educational achievement.

Data and code availability

The dataset generated during this study can be retrieved by using the search strategy and term reported in supplemental notes 2 and 3.1. All included estimates are reported in detail in Table S4. The code supporting the current study is available on the Open Science Framework (https://osf.io/gau5y/).

Supplemental information

Supplemental information can be found online at https://doi.org/10.1016/j.ajhg.2021.07.010.

Acknowledgments

B.W. and J.-B.P. are funded by a Nuffield Foundation project (EDO/43939); J.R.B. is funded by a Wellcome Trust Sir Henry Wellcome fellowship (215917/Z/19/Z); T.S. is funded by a Wellcome Trust Sir Henry Wellcome fellowship (218641/Z/19/Z). D.B. is funded by the Economic and Social Research Council (ES/M001660/1) and Medical Research Council (MR/V002147/1). F.D. is a consultant for University College London funded by the Nuffield Foundation project.

Declaration of interests

The authors declare no competing interests.

Received: April 15, 2021
Accepted: July 21, 2021
Published: August 19, 2021

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