ARTICLE

Within- and between-family genetic effects on educational achievement vary across countries and ages

Alexandra Starr[1,2,3,4]✉, Mirko Ruks[5,6], Alexandros Giannelis[7], Emily Willoughby[7], Oskar Pettersson[8], Charlotte K. L. Pahnke[9], Carlo Maj[10,11], Anastasia Andreas[12], Rafael Ahlskog[8], Louise Arseneault[13], Helen L. Fisher[13,14], Andreas J. Forstner[9,15], Christian Kandler[6,16], Matt McGue[7], Markus M. Nöthen[9], Sven Oskarsson[8], Frank M. Spinath[12], Scott Vrieze[7], Jasmin Wertz[17] and Sophie von Stumm[1]

Polygenic score (PGS) predictions of educational achievement are sizeable at the population level. Yet, population-level PGS predictions are environmentally confounded, due to gene-environment correlations, assortative mating, and population stratification. This confounding complicates the interpretation and application of PGS predictions of educational achievement. Here, we charted the variability of PGS predictions in $N = 8115$ dizygotic twins from UK, US, Swedish, and German samples aged 7 to 19 years. Population-level PGS predictions of educational achievement ranged from $\beta = 0.16$ to $\beta = 0.37$ across ages and countries. Discerning within- and between-family level estimates, we found that 10 to 65% of the population-level PGS predictions were due to environmental confounding, of which 29 to 100% were accounted for by family socioeconomic status. Variability in within-family and population-level PGS predictions was largely unsystematic across countries' school systems (multi-tiered vs. comprehensive) and children's ages. Therefore, interpretations regarding the sources of environmental confounding effects on educational achievement remain, at present, speculative.

Molecular Psychiatry; https://doi.org/10.1038/s41380-025-03342-0

INTRODUCTION

Whether children struggle or excel in school clusters within families due to genetic and environmental factors [1–4]. A growing body of research has shown that polygenic scores (PGS[1]), which are DNA-based indicators of children's genetic propensities to do well in school, account for about 15% of the variance in educational achievement in population-based samples [5–8]. Yet, population-level PGS predictions of educational achievement are potentially confounded by unmeasured environmental influences at the family level [7, 9]. This confounding, which cannot be disentangled in studies of unrelated individuals, severely complicates the interpretation and application of PGS in educational research.

Population-level PGS predictions are typically derived from samples of unrelated individuals (i.e., one individual per family) and comprise different variance components [6–8]. Variance that stems from between-family genetic effects is the same for all siblings in a family because it is estimated at the level of the family rather than at the individual level. By contrast, variance due to within-family genetic effects captures the genetic effects on a phenotype that differ between two or more biological siblings from a family, who share on average 50% of their segregating genes. Using data from dizygotic twin siblings, the population-level estimate can be obtained after accounting for the nested data structure (e.g., inclusion of a random effect of family; Fig. 1; Eq. 1). Further, it can be decomposed into genetic effects occurring between families and within families (Fig. 1; Eq. 2) [9–13]. The between-family estimate indexes the effect of the siblings' average PGS (i.e., the pair's mean PGS) on a phenotype that is confounded with shared family effects. Conversely, the within-family genetic effect captures the genetic effects on a phenotype that differ between two or more biological siblings in a family. Hence, within-family estimates reflect genetic effects net of shared family effects (i.e., the extent to which siblings' PGS differences predict their phenotypic differences within the same

[1]Polygenic scores are also referred to as genome-wide score, polygenic index (PGI), or polygenic risk score/genetic risk score (in the context of medical risk).

[1]Department of Education, University of York, York, UK. [2]Department of Biological Psychology, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands. [3]Research Institute LEARN!, Vrije Universiteit Amsterdam, Amsterdam, The Netherlands. [4]Amsterdam Public Health Research Institute, Amsterdam, The Netherlands. [5]Department of Sociology, Bielefeld University, Bielefeld, Germany. [6]Department of Psychology, University of Bielefeld, Bielefeld, Germany. [7]Department of Psychology, University of Minnesota Twin Cities, Minneapolis, MN, USA. [8]Department of Government, Uppsala University, Uppsala, Sweden. [9]Institute of Human Genetics, University of Bonn, School of Medicine & University Hospital Bonn, Bonn, Germany. [10]Centre for Human Genetics, University of Marburg, Marburg, Germany. [11]Institute for Genomic Statistics and Bioinformatics, University of Bonn, Bonn, Germany. [12]Department of Psychology, Saarland University, Saarbrücken, Germany. [13]Social, Genetic & Developmental Psychiatry Centre, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK. [14]ESRC Centre for Society and Mental Health, King's College London, London, UK. [15]Institute of Neuroscience and Medicine (INM-1), Research Center Jülich, Jülich, Germany. [16]Department of Psychology, University of Bremen, Bremen, Germany. [17]Department of Psychology, University of Edinburgh, Edinburgh, UK. ✉email: a.starr@vu.nl

Received: 28 April 2025 Revised: 8 October 2025 Accepted: 6 November 2025
Published online: 20 November 2025

(1) Population-level prediction:

$$Ph_{ij} \sim \beta(PGS_{ij}) + (1 \mid family\ ID) + \varepsilon_{ij}$$

(2) Discerning within- and between-family predictions:

$$Ph_{ij} \sim \beta_{Between}(\overline{PGS_j}) + \beta_{Within}(PGS_{ij} - \overline{PGS_j}) + (1 \mid family\ ID) + \varepsilon_{ij}$$

(3) Specifying environmental measures to explain discrepancy of within- and between-family predictions:

$$Ph_{ij} \sim \beta_{Between}(\overline{PGS_j}) + \beta_{Within}(PGS_{ij} - \overline{PGS_j}) + \beta_{Covariate}(ENV_j) + (1 \mid family\ ID) + \varepsilon_{ij}$$

Fig. 1 Estimating population-level, within- and between-family genetic effects in family data. *Ph* is the target phenotype, *i* refers to individuals clustered in sibling pairs (*j*), β is the regression estimate, *PGS* is the polygenic score, *family ID* uniquely identifies each sibling pair *j*, (1 | *family ID*) indicates a random intercept to account for the clustering of individuals in families, $\overline{PGS}$ is the sibling pair's PGS mean, *ENV* is the specified environmental measure, and ε is the residual term.

family). To the extent that population-level and between-family PGS predictions exceed within-family genetic effects, the discrepancy implies confounding due to environmental influences being intertwined with genetic effects [9–16].

Environmental confounding of genetic effects on educational achievement arise, first, because children's genes are correlated with their environments since parents pass on their genetics to their children and also create rearing environments that match their own genotypes (i.e., passive gene-environment correlation; rGE) [17, 18]. Second, effects of demography, including where people reside and who they choose to have children with (i.e., population stratification and assortative mating) further induce correlations between genetic and environmental effects. As a result, family-level genetic influences related to rGE, assortative mating, and population stratification imply the environmental confounding of population-level and between-family PGS predictions [19–21]. Environmentally confounded PGS predictions can distort studies that seek to identify the causes of children's differences in educational achievement. For example, population-level PGS predictions can bias interpretations of causal inference models (e.g., Mendelian randomisation) and of how individuals' genetic dispositions affect their response to environmental experiences (i.e., gene-environment interaction) [11, 22, 23]. If causal genetic effects are inferred from putatively direct, but environmentally confounded PGS predictions, the mechanisms that drive the gene-environment interplay will likely be misconstrued. As a consequence for research and application, effective targets, contexts, and timepoints for education interventions might be misidentified or missed altogether.

To identify the factors that drive family-level confounding effects due to rGE, environmental measures can be modelled as covariates (Fig. 1; Eq. 3) [9, 24]. One potential covariate is the family's socioeconomic status (SES). All 'environmental' factors, including SES, partly reflect heritable traits [25, 26]. Yet, accounting for measured covariates allows to contextualise the sources that underpin the environmental confounding of genetic effects. The extent to which such covariates account for the confounding effect is inferred from comparisons of within- and between-family PGS estimates. Comparisons between within-family and population-level estimates are not used, because population-level, within-family, and covariate effects cannot be estimated in the same model. The population-level PGS prediction of a trait is a weighted sum of the within- and between-family estimates (for details see Selzam et al. [9]). It can exceed or fall below the between-family estimate, depending on how much of the total phenotype's variance is attributable to the family versus the individual level – that is, how highly the co-twins in a family are correlated for the phenotype. If co-twins are strongly correlated, the population-level estimate will be higher, whereas it can approach the size of the within-family estimates if co-twins are essentially uncorrelated.

On average, population-level PGS predictions of education-related phenotypes (e.g., school grades, years spent in education, cognitive ability test scores) exceed predictions at the within-family level [7, 9–11, 13]. This discrepancy implies meaningful environmental confounding of genetic effects on education related to factors shared by children growing up in the same family (i.e., rGE), such as family SES, and effects of population-level dynamics (i.e., population stratification, assortative mating).

Previous studies reported that within-family PGS predictions of educational achievement were half those at the population level in a sample of UK adolescents (i.e., population-level $\beta = 0.42$ versus within-family $\beta = 0.21$) [9, 13]. Likewise, within-family PGS predictions of educational attainment in a sample of Dutch adults were attenuated compared to population-level predictions ($\beta = 0.09$ vs. $\beta = 0.20$). However, in the same study, population-level and within-family PGS predictions of educational achievement in Dutch children aged 12 years did not differ significantly [10]. Meta-analytic studies confirm that effect sizes of environmental confounding of genetic effects for educational outcomes range widely from zero (i.e., within-family equating population-level estimates) to accounting for up to 70% of the population-level PGS predictions, depending on the phenotype measure, cohort, and participant age [10, 11, 16]. The considerable variability of both the within-family and population-level genetic effects further complicates the interpretation and application of PGS in education research. Pinpointing when, how, and in relation to which contextual factors PGS predictions of educational achievement vary is a pre-requisite to improve our understanding of equity in teaching and learning. For instance, finding that environmental confounding of genetic effects is observable in some school systems but not others (e.g., multi-tiered vs. comprehensive schooling), or during certain developmental periods but not earlier or later, can help to inform efforts that seek to improve equity in education.

Our primary research goal here was to systematically chart the variability of population-level, within- and between-family genetic effects on educational achievement in family data from the UK, US, Sweden, and Germany across children's ages from 7 to 19 years. We tested three principal hypotheses.

First, we hypothesised (H1a) that PGS and family SES significantly predict educational achievement across countries at the population level, in line with previous research [27]. Because genetic influences on cognitive traits including educational achievement increase with age due to gene-environment transactions [5, 28–30], we also expected (H1b) population-level PGS predictions to increase in effect size as children grow older [5, 27]. School systems differ across countries in whether secondary education is comprehensive, as is the case in the UK, US, and in Sweden, or multi-tiered, as in Germany where students are allocated to secondary school tiers based on their primary school achievement [31]. Comprehensive versus multi-tiered secondary schooling likely intersects with the transmission of family background influences on education, because tiered school systems favour children from higher as compared to lower SES backgrounds to a greater extent than comprehensive school systems [32–37]. We therefore predicted (H1c) the effects of PGS and SES on educational achievement to be stronger in countries with multi-tiered systems and weaker in those with comprehensive school systems (i.e., stronger effects of family background in Germany, weaker ones in the UK, US, and Sweden) [36].

Second, we hypothesised (H2a) that the effect of the PGS on educational achievement is lower within families compared to the population-level, in line with previous findings [9–11] and indicative of environmental confounding of genetic effects. We also predicted (H2b) these environmental confounding effects to be more pronounced in multi-tiered than in comprehensive

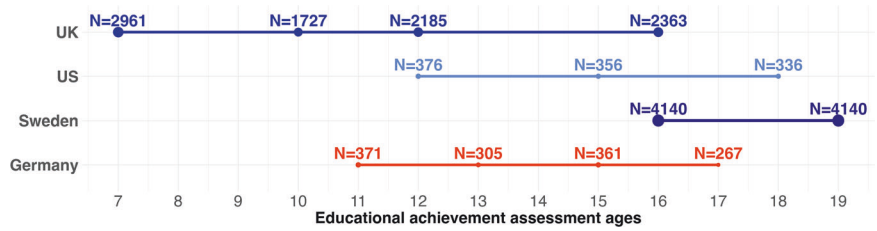

Fig. 2 Overview of samples, analysis sample sizes, and assessment ages. UK = Twins Early Development Study (TEDS) and Environmental Risk (E-Risk) Longitudinal Twin Study; US = Minnesota Twin Family Study (MTFS); Sweden = Swedish Twin Registry (STR); Germany = TwinLife; N = sample size (number of individuals with complete data included in multi-level analyses). Blue colour indicates comprehensive school systems; red colour indicates multi-tiered school systems. The samples from TEDS and E-Risk were combined to derive one comprehensive UK sample at the ages 7, 10, and 12 years; age 16 years data was only available from TEDS. In TEDS and E-Risk educational achievement was assessed via teacher reports, in the MTFS parents reported on children's grade point average (GPA), in TwinLife school grades were taken from annual report cards, and in the STR children's GPA was taken from the Statistics Sweden education register.

school systems [36]. We expected these country-level differences to emerge during the secondary school years, because primary schooling is comprehensive in all four countries [31].

Third, we hypothesised (H3a) that including family SES as a covariate partly accounts for environmental confounding of the PGS on educational achievement, in line with previous findings [9]. We expected (H3b) the role of SES to be stronger in multi-tiered (i.e., Germany) than in comprehensive school systems (i.e., UK, US, and Sweden) [38, 39]. While we expected findings to align with countries' respective school systems, we cannot rule out that results may vary across samples due to other factors, for example different welfare state regimes. In liberal regimes like the UK and US, high income inequality and weak resource redistribution might interfere with educational opportunities, by contrast to social-democratic regimes like Sweden that compensate to a greater extent for family background inequality [40]. Other sources of variation might include deviations in sampling, measurement, and methodology.

METHODS

Data

We capitalised on twin data from dizygotic (DZ) same-sex twins, drawn from the Twins Early Development Study (TEDS), the Environmental Risk (E-Risk) Longitudinal Twin Study, the Minnesota Twin Family Study (MTFS), the Swedish Twin Registry (STR), and TwinLife, five population-based, longitudinal twin family studies from the UK, US, Sweden, and Germany, respectively. Ethical approval was granted for all cohorts prior to data collection and informed consent was obtained from participants. Figure 2 provides an overview of the assessment ages and respective sample sizes in all cohorts. Further information on ethical approval and data availability are below. Details on the database, measures, and statistical analysis are included in the Supplementary Materials S1.

Measures

Educational achievement. In all samples, educational achievement was measured repeatedly over the course of primary and secondary education across the ages from 7 to 19 years. Data was available in the form of teacher- and self-reported grades (UK), grade point average (US), education register data (Sweden), or grades on report cards (Germany).

Polygenic scores for educational attainment. Twins' genotype data was collected in all samples and the educational attainment (EA) PGS for years spent in education was constructed using summary statistics of either EA3 [6] or EA4 (sample without 23&me) [7]. These two versions of the PGS have been shown to yield highly similar predictions due to large overlaps of the GWAS discovery samples from which the summary statistics to calculate the scores were derived [7]. The PGS were adjusted by the first 10 principal components to account for bias due to genetic ancestry, and were z-standardised (mean = 0, SD = 1). A family-level mean of the twins' PGS was computed to reflect the between-family PGS measure. Person-specific deviations from this mean reflect the within-family PGS measure (i.e., relative difference '$PGS_{mean} - PGS_{twin\ 1/2}$').

Socioeconomic status. In all samples, data was collected on mothers' and fathers' educational levels, their occupational status, and the family's household income. These indicators were combined to form a composite indicating SES at the family level (i.e., the same for both twins).

Twins' age and sex. In all samples, the twins' age was measured in (decimal) years at each time-point when data on educational achievement was collected. Age and twins' sex recorded at birth were included in the analyses as covariates.

Statistical analysis

The analysis plan for this study was preregistered: https://osf.io/jzp7c/?view_only=10fd9c9885844b519339de63c5a485fe.

Leveraging PGS predictions within families using genetic data from siblings (specifically DZ twins) reared together in the same family helps derive less biased genetic effects from PGS [12, 41, 42]. Because biological siblings are born to the same parents, they are matched on all family shared genetic and environmental effects; thus, biases due to assortative mating and population stratification are eliminated.

First, we predicted educational achievement at the population level from PGS for educational attainment [6, 7] and SES, respectively. Second, leveraging the sibling design, we tested if environmental confounding was present by comparing the population-level and within-family prediction estimates. Third, we specified SES as a family-level covariate to test the extent to which it accounted for environmental confounding of the PGS predictions of educational achievement (i.e., the discrepancy between within- and between-family estimates). Analyses were performed separately for each assessment age of educational achievement and each country due to restrictions regarding data sharing. As a result, formal significance testing of differences between estimates across samples and ages was limited. Here, we relied on three statistics. First, we compared the estimates' 95% confidence intervals (CIs). When these do not overlap, a statistically significant difference between estimates is likely at about $p = 0.01$ [43]. Second, to test the statistical difference between estimates in independent samples (i.e., comparisons between countries), we divided the difference between the standardised beta coefficients by the standard error of the difference [44, 45]. Third, in case of dependent samples as for the comparison of population-level and within-family estimates, we used Fisher's Z transformation [46, 47]. To account for multiple comparisons, the significance level was adjusted to $\alpha = 0.01$, and p-values below this threshold were considered statistically significant. All analyses were performed in R (version 4.4.0) [48], package 'lme4' [49]. Further details on the statistical analysis are included in the Supplementary Materials S1, with deviations from the preregistration and information on data and analysis code availability being listed in the Supplementary Material S2 and S3, respectively. Descriptive statistics and correlations are included in Tables S1-S8, with notes on all Supplementary Tables provided in Supplementary Materials S4.

RESULTS

Do predictions of children's educational achievement from PGS and family SES differ across ages and countries?

In line with our hypothesis (H1a) and previous findings, PGS and SES significantly predicted children's educational achievement at

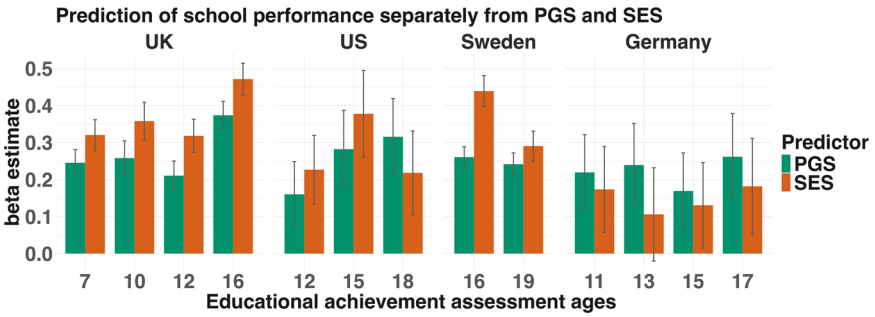

Fig. 3 Predictions of educational achievement from PGS and SES. PGS = Polygenic score for educational attainment; SES = Socioeconomic status. Sample sizes varied across countries and ages (see Fig. 2). Estimates are standardised betas from multi-level models (family ID as random effect). The error bars reflect 95% confidence intervals. In all models, twins' age and sex were included as covariates.

the population level, after controlling for age differences within assessment time-points and sex. However, the prediction strength differed considerably across countries and ages (Fig. 3; Tables S9-S12). In the UK and Sweden, SES was a stronger predictor of educational achievement than PGS across ages (SES: $\beta = 0.29$ - 0.47; PGS: $\beta = 0.21$ - 0.37); in the US, the same pattern emerged at ages 12 and 15 but not at age 18 years. In Germany, PGS tended to be stronger predictors than SES across ages (PGS: $\beta = 0.16$ - 0.26; SES: $\beta = 0.11$ - 0.18). However, these within-country differences in predictions from SES and PGS did not reach significance due to wide, overlapping 95% CIs.

A significant age trend in the hypothesised direction (H1b) was only observable in the UK, with predictions of educational achievement from the PGS increasing as children grew older ($\beta = 0.24$ at age 7 years to $\beta = 0.37$ at age 16 years). A similar, yet not significant age trend emerged in the US ($\beta = 0.16$ at age 12 years to $\beta = 0.31$ at age 18 years); no age trends for the PGS predictions emerged in the Swedish and German samples. SES predictions of educational achievement were largest at the ages of 15 to 16 years in the UK, US, and in Sweden ($\beta = 0.37$ - 0.47) – an age that coincides with the end of compulsory comprehensive education (UK) and transition to upper-secondary school (US, Sweden). In Germany, SES was overall less predictive of educational achievement compared to the other countries and even non-significant at age 13 ($\beta = 0.11$ - 0.18). Thus, contrary to our expectations (H1c), PGS and SES did not predict children's educational achievement more strongly in multi-tiered than in comprehensive school systems (i.e., Germany versus UK, US, and Sweden). In fact, the predictions from PGS and SES were in part significantly lower in Germany compared to those in the other three countries (see Table S14).

Does environmental confounding of genetic effects differ in magnitude across ages and countries?

We differentiated the PGS predictions into variance that occurs between families (level 2) and within families (level 1). We found that the within-family PGS predictions were attenuated compared to the population-level estimates, in line with our hypothesis (H2a; Fig. 4) and indicative of environmental confounding. However, the degree of attenuation differed considerably across countries: It was significant in the Swedish and the UK samples (except at age 10 years), but not in the US and Germany according to Fisher's Z difference tests. In the UK and in Sweden, within-family estimates of PGS were attenuated by 26-47% relative to the population level, suggesting that in both countries environmental confounding of genetic effects significantly influences children's educational achievement. The attenuation observed in the US (17-65%) did not reach significance. In Germany, the attenuation effect was least pronounced relative to all other countries (10-31%) and not significant. This finding rejects our hypothesis (H2b) of observing stronger environmental confounding of genetic effects in Germany's multi-tiered school system than in the comprehensive systems of the UK, US, and Sweden.

Contrary to our expectations, the extent of environmental confounding did not vary systematically across ages. In the UK, the discrepancy of the population-level and within-family effect was greatest at the end of secondary education at age 16 years. Yet, similar trends were not observable in the other three samples, where the discrepancy was either stronger at younger ages or non-significant altogether (see Tables S9-S12 for full model results and Fisher's Z difference tests). To test whether the non-significant results in the US and Germany were due to small sample sizes and reduced statistical power, we conducted sensitivity analyses (not preregistered) in a random sample of $N = 350$ families drawn from the UK sample (N chosen for comparability with the US and German sample sizes). In this smaller UK sample, the differences between the within- and population-/between-family estimates were not significant, according to Fisher's Z difference tests. However, when considering the point estimates, the general pattern of results was largely comparable to the overall UK sample results (full model results in Supplementary Materials S5 and Table S13). This suggests that differences in the significance of environmental confounding of genetic effects between the samples may be due to sample size and statistical power issues.

Is the environmental confounding of genetic effects on educational achievement accounted for by SES?

We tested if family SES accounted for the environmental confounding effects by specifying it as a covariate (see Tables S9-S12 for full model results). Confirming our hypothesis (H3a), adding SES to the models reduced the discrepancy of the within- and between-family PGS predictions of educational achievement where this discrepancy was present (i.e., UK and Sweden). Figure 5 shows the between-family estimates before (dark red) and after including family SES (orange), as well as the within-family estimates (light blue; identical to those in Fig. 4, because SES does not comprise level 1 variance) in the UK and Sweden. Note that here within-family estimates are compared to between-family, rather than to population-level estimates [9]. After adding SES, the between-family estimates were substantially reduced and approached the within-family estimates. This effect was strongest in the UK at the ages 7 and 10 years, when controlling for SES accounted for 94 and 100% of the discrepancy between the within- and between-family PGS prediction, respectively. That is, environmental confounding of genetic effects on educational achievement could be almost entirely attributed to differences in family SES. At the ages 12 and 16 years, SES still explained about 64% of environmental confounding in the UK. In Sweden, family SES accounted for a smaller proportion of environmental confounding (about 45% at age 16 years, and 29% at age 19 years) compared to the UK.

Because the discrepancy of between- and within-family PGS predictions of educational achievement was not significant in the

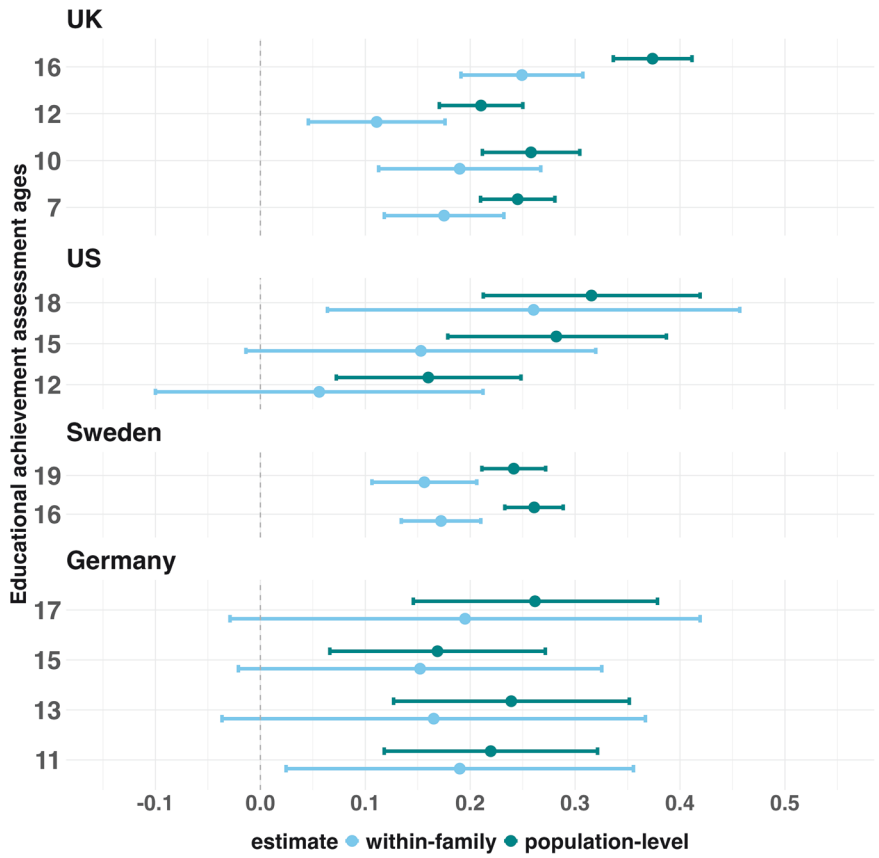

Fig. 4 PGS prediction of educational achievement at population level and within families. PGS = Polygenic score for educational attainment. Sample sizes varied across countries and ages (see Fig. 2). Estimates are standardised betas from multi-level models (family ID as random effect). The error bars reflect 95% confidence intervals. In all models, twins' age and sex were included as covariates.

US and in Germany, no environmental confounding emerged that could have been explained by SES. These findings contradicted our expectations (H3b) that family SES would have stronger effects on the between-family PGS predictions in Germany's multi-tiered school system than in the comprehensive systems in the UK, US, and Sweden.

DISCUSSION

This study is the first to combine both a multi-national and developmental perspective to chart the variability of PGS predictions for educational achievement, derived from genome-wide association studies for educational attainment [6, 7]. We used a within-family sibling design to parse population-level, within- and between family genetic effects on educational achievement across UK, US, Swedish, and German samples aged 7 to 19 years. We observed sizeable variability in three ways. First, population-level PGS predictions of educational achievement were positive and significant across countries and ages, but they varied in effect size. Predictions from family SES for educational achievement varied across countries and ages to a similar degree, but this variability did not align with the variability of the PGS predictions. Second, the magnitude of environmental confounding of genetic effects on educational achievement differed, accounting for 10 to 65% of the population-level PGS prediction across countries and ages. Third, the extent to which family SES accounted for this environmental confounding on educational achievement ranged from 29 to 100% across countries and ages. We could not attribute these three variability findings to systematic patterns, such as age trends. Also, they did not confirm our hypotheses about the contextual factors that likely affect PGS predictions, in particular country-level differences in secondary education (i.e., multi-tiered versus comprehensive schooling). We discuss our findings in the context of previous research.

Variability of within-family and population-level genetic effects on educational achievement

Our results confirmed that PGS predictions are on average weaker within families compared to the population-level estimates, in line with recent findings [10, 11]. That said, our results contradicted previous research showing that genetic effects are consistent across cognitive and educational phenotypes, ages of assessment, and samples' countries of origin [9, 11, 16]. The discrepancy between our current and earlier findings may be partly due to analysing different education phenotypes: We focused here on educational achievement, but others studied educational attainment [11, 16]. Educational achievement refers to individuals' performance in an educational setting (e.g., exam grades, teacher-reported performance), while educational attainment pertains to the level of education that a person acquires (e.g., school leaving certificate, university degree). Educational achievement and attainment have different aetiologies [50, 51] and might be differently affected by family-level environmental confounding, even though both constructs are correlated phenotypically and genetically [8]. Still, the mechanisms through which genetics affect educational achievement in primary and secondary school may not be the same as those that pertain to attainment in adulthood.

No systematic pattern of environmental confounding of genetic effects across countries' secondary school systems

The country-level institutional factor of multi-tiered versus comprehensive secondary schooling could not explain the

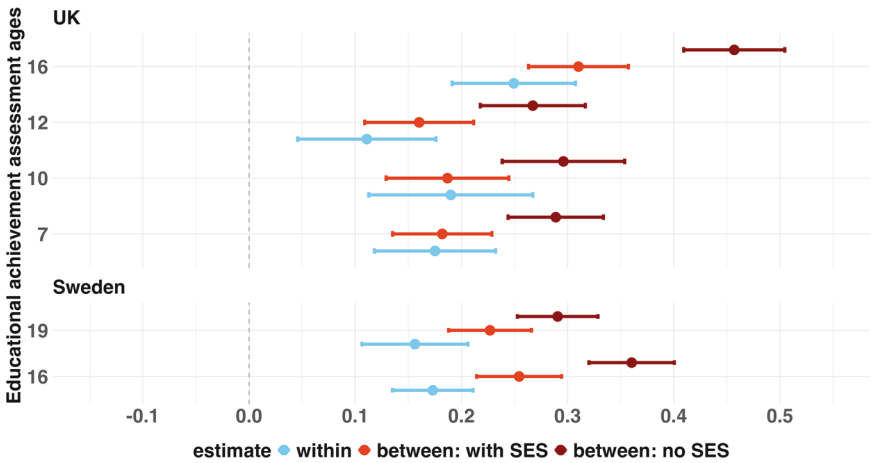

Fig. 5 Within- and between family PGS predictions of educational achievement in the UK and Sweden with and without SES as covariate. PGS = Polygenic score for educational attainment, SES = Socioeconomic status. Sample sizes varied across countries and ages (see Fig. 2). Estimates are standardised betas from multi-level models (family ID as random effect). The error bars reflect 95% confidence intervals. In all models, twins' age and sex were included as covariates.

observed variability in environmental confounding of genetic effects on educational achievement. Significant within-family attenuation of PGS predictions was evident in Sweden and in the UK (both comprehensive), but not in the US and Germany (comprehensive and multi-tiered). Other country-level differences, such as the extent of social and economic inequality [40, 52, 53], may help explaining the variability in environmental confounding of genetic effects on educational achievement. While the UK and US are liberal welfare states where disparities in wealth, welfare provision, and access to education are high, Sweden and Germany feature social-democratic and conservative welfare state regimes, respectively, which are characterized by less income inequality and greater intergenerational mobility [54–57]. Thus, to which extent access to socioeconomic resources relates to educational outcomes might intersect with the emergence of environmental confounding of genetic effects on educational achievement. We caution, however, that these and similar attributions do not fully align with our findings and are post-hoc and speculative at present. Alternatively, it is possible that other country-level differences in school environments not studied here, such as the level of teachers' qualifications or classroom sizes [58, 59], which also relate to family background inequalities [60], drive the variability of environmental confounding. To elucidate these mechanisms, studies are necessary that include more detailed information on school environments and sample populations from additional countries to achieve sufficient statistical power.

Similar to our results for Germany, previous findings based on a Dutch sample also reported negligible environmental confounding of genetic effects on educational achievement in children at age 12 years [10]. While both the Netherlands and Germany feature multi-tiered secondary school systems, it should be noted that tracking only starts after age 12 in the Dutch system, whereas it starts at age 10 in Germany. Thus, additional data would be required to test whether environmental confounding of genetic effects arise at later stages when children are enrolled in multi-tiered secondary schools in the Netherlands, or if they remain absent throughout children's secondary school years, as our results suggest for Germany.

No systematic developmental age trends in environmental confounding of genetic effects

Environmental confounding of genetic effects on educational achievement was inconsistent across children's ages in our analyses. Prior reports suggested that population-level PGS predictions of cognitive and educational outcomes increase as children grow older [5]. As a result, environmental confounding would be likely to strengthen because gene-environment transactions amplify (population-level) genetic predictions across ages [29, 30]. Yet, the reverse pattern is also plausible: Children's educational achievement differences might be particularly affected by environmental confounding during the earlier years of schooling when environmental contributions to phenotypic variation are stronger [3, 11]. Our results support neither of these hypotheses as no systematic age trends in environmental confounding of genetic effects on educational achievement emerged. Thus, we cannot pinpoint developmental periods during the primary and secondary school years that are likely to maximize the effectiveness of efforts that aim to reduce family background inequality and improve children's educational achievement. Future research is needed to explore if the absence of systematic developmental patterns also applies to other samples than those we included here.

SES explains environmental confounding of genetic effects to varying degrees

We observed significant environmental confounding in the UK (except at age 10 years) and in Sweden. In both countries, family SES accounted for more than a quarter and up to all of these environmental confounding effects, reflecting that population-level PGS predictions capture genetic effects on educational achievement that are due to children's rearing environments. Parents' education, occupation, and income likely reflect, albeit coarsely, the quality of children's home learning environments and access to education opportunities [61]. The extent to which SES accounted for environmental confounding of genetic effects on educational achievement was not much related to countries' school systems (i.e., multi-tiered vs. comprehensive). That said, the attenuation of PGS predictions within families occurred in those countries where family SES predictions of educational achievement were stronger. Variability of the PGS prediction effect size at the population level is likely to stem from processes involving transactions and interactions between genetic and environmental factors, for example when parents' ability to provide rearing environments that support their children's educational achievement (i.e., transaction) vary depending on their access to socioeconomic resources (i.e., interaction) [61].

Family SES explained a larger share of the environmental confounding of genetic effects on educational achievement in the UK than in Sweden. For one, this difference may be due to higher levels of educational inequality in the UK compared to Sweden

[40, 55]. For the other, the relation between SES and population phenomena, such as assortative mating and population stratification, is assumed to be stronger in the UK, which likely confounds population-level PGS predictions of educational achievement [12, 20]. Prior studies have shown that in the UK, families of high SES and thus, high genetic propensities for education, often live in affluent regions with high-quality schools [62–64]. This alignment between environmental and genetic advantages results in part from assortative mating, which contributes to the geographic clustering of alleles associated with an increased or decreased potential for educational success [19, 21, 65]. Therefore, adjusting for family SES in UK samples might 'sweep out' a greater proportion of environmental confounding in the PGS prediction of educational achievement than in data from other countries, including Sweden. Alternatively, SES might account for less variance in the discrepancy between population-level and within-family estimates in Sweden than in the UK because of dynastic effects. Recent studies identified complex population and dynastic effects in Sweden and Norway that extend beyond the nuclear family (i.e., including effects of grandparents' or other family members' genotypes [66, 67]). Dynastic effects may include socioeconomic stratification of environments that occur across multiple generations and are associated with parents' genetics. If dynastic effects are at play, behaviours at the extended family level and social circumstances that became correlated with the wider family's genetic predisposition would contribute to children's educational achievement [67]. In this case, our study's SES measure based solely on parents' education, occupation, and income might not adequately reflect the complexity of environmental processes across extended families and generations. Because studies that compare assortative mating, population stratification, and dynastic effects across countries are not available, we can only speculate about their respective effects on our findings.

Limitations and future research

Despite the strengths of this research, including the use of genetic data of twin siblings across four countries, repeated measures of educational achievement, and a state-of-the-art modelling approach, it is not without weaknesses.

First, our samples differed substantially in size and statistical power to detect effects (Germany: $N = 267$-371; UK: $N = 1727$-2961; US: $N = 336$-376; Sweden: $N = 4140$ individuals). Results from our sensitivity analyses demonstrated that the German and US samples were likely underpowered for estimates to reach significance, yet the results pattern might reflect valid effects. To address this point conclusively larger samples are required, which are currently not available.

Second, explaining the variability in PGS predictions that we observed here was difficult. Sample-specific differences, such as the measurement of family SES and educational achievement, genetic data processing and PGS calculation, and wider country-level factors related to educational opportunity may have confounded our results.

Third, the generalisability of our findings to the wider populations of the samples' respective countries is limited, because highly-educated families are likely overrepresented due to attrition that affects all longitudinal studies [68]. For instance, variance restriction as a result of non-random drop-out could have downwardly biased SES predictions, which might explain the weak SES predictions of educational achievement at ages 13 and 15 years in Germany. Attrition issues do not apply to the Swedish data, which was sourced from national register data and thus, does not suffer from loss-to-follow-up bias. Another issue concerning the generalisability is that all samples came from WEIRD countries with predominantly European genetic ancestry. Future research needs to explore the variability in environmental confounding of genetic effects on educational achievement across more diverse populations and genetic ancestry backgrounds.

Lastly, we point out that SES or similar putatively 'environmental' factors should not be understood as purely environmental covariates because these measures are partly heritable themselves [25, 26]. Adjusting our estimates for family SES might therefore be seen as overcorrecting, even when parsing PGS predictions from potentially confounding effects of family-level factors is currently of high interest in genetically-informed research [9, 24, 41].

CONCLUSION

Population-level PGS predictions of educational achievement capture valid genetic effects as well as environmental confounding. Such environmental confounding of genetic predictions indicates that associations between children's educational achievement and their genetic propensities also reflect effects of their social environment at the family level, in part due to gene-environment correlations. We showed here that the magnitude of population-level, within- and between-family genetic effects on educational achievement varied considerably across four countries (UK, US, Sweden, and Germany) and children's ages (7 through 19 years). We could not attribute this variability in environmental confounding of genetic effects to school systems as a country-level institutional factor or children's age. We speculate that other systematic factors might explain the variability and contribute to the transmission of family background inequality in education. Future research might build on our findings to identify such factors by leveraging large genetically informative family samples from diverse backgrounds. Such analyses may reveal aspects of children's learning environment that are proximal and malleable and thus, suitable targets for efforts to improve children's educational achievement.

Ethical approval

All studies were conducted in accordance with ethical standards and received approval from the relevant institutional review boards or ethics committees. For the Twins Early Development Study (TEDS), ethical approval and consent procedures were granted by the King's College London Ethics Committee (ref: PNM/09/10-104). The Environmental Risk (E-Risk) Longitudinal Twin Study was approved by the Joint South London and Maudsley and the Institute of Psychiatry Research Ethics Committee (NRES 1997/122); parents provided informed consent and twins gave assent. The Minnesota Twin Family Study (MTFS) received ethical approval from the University of Minnesota Institutional Review Board, with informed consent obtained from adult participants (including consent for their underage children) and assent from children. The Swedish Twin Registry (STR) study was approved by the Swedish Ethical Review Authority (reference 2017/083) and by the Steering Committee of the Swedish Twin Registry. Finally, TwinLife was approved by the Ethics Committee of the German Psychological Society (DGPs; protocol number RR 11.2009), and the molecular genetic analyses received additional approval from the Ethics Committee of the Medical Faculty of the University of Bonn (No. 113/18); informed consent (including parental consent for minors) was obtained during in-person interviews.

DATA AVAILABILITY

The datasets used for this study are not openly available due to privacy and ethical requirements, yet data access can be requested for research purposes from the respective data owners. TEDS data are available upon request (https://www.teds.ac.uk/researchers/teds-data-access-policy). Details on measurement and sample characteristics are available in the TEDS data dictionary (https://www.teds.ac.uk/datadictionary/home.htm). E-Risk data are available upon request (https://www.eriskstudy.com/data-access/). To access the MTFS data, external researchers can request to collaborate with MTFS researchers [69]. The Swedish

sample comprises use individual-level register data provided by Statistics Sweden, combined with data from the STR, which is administered by the Steering Committee of the Swedish Twin Registry. To access the data, researchers they must obtain approval from the Swedish Ethical Review Authority and from the Steering Committee of the Swedish Twin Registry. STR data are available upon request (https://ki.se/en/research/swedish-twin-registry-for-researchers). In this study, TwinLife data release 7.1.0 was used (https://doi.org/10.4232/1.14186). Data are available upon request (https://search.gesis.org/research_data/ZA6701). Information on measurement and sample characteristics are available via the TwinLife data documentation website (https://www.twin-life.de/documentation/downloads).

CODE AVAILABILITY

No custom code was used for the analyses in this study. Data analyses were conducted in R (version 4.4.0) [48] and RStudio (version 2024.04.1) [70]. The analysis code can be obtained here: https://osf.io/jzp7c/?view_only=10fd9c9885844b519339de63c5a485fe.

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ACKNOWLEDGEMENTS

We thank the participating families and many contributors to the Twins Early Development Study (TEDS), the Minnesota Twin Family Study, the Swedish Twin Registry (STR), and TwinLife. We are grateful to the Environmental Risk (E-Risk) Longitudinal Twin Study mothers and fathers, the twins, and the twins' teachers for their participation. Our thanks to Professors Terrie Moffitt and Avshalom Caspi, the founders of the E-Risk Study, and to the E-Risk team for their dedication, hard work and insights. The views expressed are those of the authors and not necessarily those of the ESRC or King's College London.

AUTHOR CONTRIBUTIONS

Conceptualization: AS and SvS, Methodology: AS and MR, Formal analysis: AS, MR, AG, OP, and CKLP, Resources: RA, LA, HLF, AJF, CK, MM, MMN, SO, FMS, SV, JW, and SvS, Data curation: AS, MR, AG, EW, OP, CKLP, CM, and AA, Writing – Original draft: AS and SvS, Writing – Review & Editing: All authors, Visualization: AS, Supervision: RA, LA, HLF, AJF, CK, MM, MMN, SO, FMS, SV, JW, and SvS, Funding acquisition: RA, LA, HLF, AJF, CK, MM, MMN, SO, FMS, SV, and SvS, All authors approved the final version of the manuscript.

FUNDING

TEDS is supported by the UK Medical Research Council (MR/V012878/1 and previously MR/M021475/1), with additional support from the US National Institutes of Health (AG046938). The Environmental Risk (E-Risk) Study is funded by grants from the UK Medical Research Council (MRC) [G1002190; MR/X010791/1]. Additional support for E-Risk was provided by the US National Institute of Child Health and Human Development [HD077482] and the Jacobs Foundation. The Minnesota Twin Family Study was supported in part through grants from the National Institutes of Health R01 DA 042755, R01HG011035, R01 DA 054087, R01 DA 044283. The Swedish Twin Registry (STR) is managed by the Karolinska Institute and receives additional funding through the Swedish Research Council (2017-00641). Additional funding for STR comes from the Ragnar Söderberg Foundation (E9/11), and the Swedish Research Council (421-2013-1061). TwinLife is funded by grants from the German Research Foundation (DFG; project number: 220286500 and 428902522). SvS held fellowships from the Jacobs Foundation (2022-2027) and the Paris Institute of Advanced Study (2023-2024) during the writing of this manuscript. HLF was supported by the UK ESRC Centre for Society and Mental Health at King's College London [ES/S012567/1]. The position of CP is funded by the DFG (funding number 428902552). OP, RA, and SO were supported by the Swedish Research Council (2019-00244; 2023-01343).

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41380-025-03342-0.

Correspondence and requests for materials should be addressed to Alexandra Starr.

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