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Social and genetic associations with educational performance in a Scandinavian welfare state

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Recent research has suggested that across Western developed societies, the influence of genetics on educational outcomes is relatively constant. However, the degree to which family environment matters varies, such that countries with high levels of intergenerational mobility have weaker associations of family background. Research in this vein has relied on twin-based estimates, which involve variance decomposition, so direct assessment of the association of genes and environments is not possible. In the present study, we approach the question by directly measuring the impact of child genotype, parental genetic nurture, and parental realized education on educational achievement in primary and secondary school. We deploy data from a social democratic context (Norway) and contrast our findings with those derived from more liberal welfare state contexts. Results point to genetics only confounding the relationship between parent status and offspring achievement to a small degree. Genetic nurture associations are similar to those in other societies. We find no, or very small, gene–environment interactions and parent–child genotype interactions with respect to test scores. In sum, in a Scandinavian welfare state context, both genetic and environmental associations are of similar magnitude as in societies with less-robust efforts to mitigate the influence of family background.

educational achievement | social background | polygenic index | registry data | Norway

Significance

Children of highly educated parents perform better in school; this may be due to social environments or genes. Most studies do not distinguish between the two, meaning that genetic associations may confound environmental explanations (and vice versa). Those taking both factors into account have mostly focused on societies with relatively high degrees of inequality and low social mobility. We investigate these dynamics in a social democratic welfare state (Norway). Parental educational advantage is attenuated only to a small degree when accounting for genetics. There were no important interactions between children's genotypes and their parents' education or genotype. Genetic and social inheritance appear to wield two largely independent sources of influence on educational achievement, even in a robust, Scandinavian welfare state.

By and large, social science research has emphasized environmental explanations as to how family background influences educational outcomes. However, that the social influence of family background may be confounded by genetics has been known for the better part of the last century (1, 2). Indeed, a large amount of literature from behavioral genetics has shown that nearly every individual-level outcome of interest to social scientists or health scholars is heritable to some degree (3, 4). For example, twin studies of educational attainment show a considerable heritability, averaging around 40% (5, 6). Twin studies on educational achievement in adolescence show an even higher heritability, at 65%, and lower common environment influences than for adult educational attainment (7, 8). Thus, with two forms of transmission from parents to children, models that estimate one without considering the other are likely confounded.

Following the sequencing of the human genome there has been an increase in molecular genetic data sources. A small, but growing social science literature has emerged that directly links genomic measures to educational outcomes. In this approach, researchers estimate models that include both measures of parental educational attainment and a summative measure of genetic influences on education called a polygenic index (PGI; also called a polygenic score or genetic risk score) of both the parents and the offspring. Studies from the United States show that only one-sixth to one-third of the intergenerational correlation of educational attainment is due to shared genetics (9, 10), leaving a sizable social background component to intergenerational persistence in educational outcomes.

Much of this social science genetics research has considered educational attainment, and the literature on educational achievement in childhood and adolescence is even more sparse. Existing studies with genomic data have mostly supported the idea of two, parallel inheritance systems. Studies based on data from the United Kingdom, Germany, France, and Ireland find that both a PGI for educational attainment and the family's socioeconomic status influence educational achievement in adolescence and that they operate largely independent of each other (11, 12).

Complicating this picture of independent mechanisms, however, is what has been coined "genetic nurture." Including PGIs of children and parents in models simultaneously, studies have shown that parental genotypes may be associated with children's outcomes over and above children's own PGI (9, 10, 13, 14). This indicates that the parents' genes—or whatever environments parental genes are proxying—are associated with children's outcomes through environmental pathways. A recent review of genetic

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nurture studies concluded that most genetic nurture associations operate through parental phenotypes (i.e., parents' educational attainment) (15). Genetic nurture estimates are almost half as large as direct genetic associations, and maternal and paternal genetic nurture estimates are similar in magnitude. With two exceptions, all 12 reviewed studies used data from liberal welfare state countries.

Compared to social democratic welfare states, liberal welfare state countries, such as the United States and the United Kingdom, are characterized by high levels of social inequality and lower rates of social mobility. Twin-based studies have found that the heritability of educational attainment rises with intergenerational mobility cross-nationally (16), as well as over birth cohorts in the same country, with the introduction of extensive social policies (17). However, these twin-based estimates may be confounded by differences in genetic assortative mating across contexts and differences in the degree to which the equal environments assumption (EEA) holds. While the EEA is difficult to test directly absent misclassified twins (18), (genetic) assortative mating has indeed been shown to vary across time and place (19–21).

Our approach of directly measuring child and parental genotypes is less affected by assortative mating patterns that may differ across societies or birth cohorts. Moreover, we do not need to make any assumptions about the covariance between genetic and environmental similarity within kinships (i.e., EEA).

Our context, Norway, is a country with a compulsory and free universal school system with very few students attending private schools and low variation in school quality (22). Social and economic policies have explicitly targeted transmission of intergenerational inequalities, seeking to limit the influence of family background on children's outcomes and opportunities. One important social policy is government-subsidized child care. Over 90% of children ages 1 to 5 are enrolled in child care in Norway (23), with standardized curriculums intended to homogenize the quality of education. Furthermore, welfare transfers are both universal and progressive, meaning that even as child care is heavily subsidized for all, low-income families may receive additional funds to provide for their children. In many liberal welfare states, areas with high-income parents have the highest educational expenditures (24). In Norway, by contrast, schools in areas with many low-income families receive more resources than those with many high-income families (25). Given these societal features, there may be a more limited role for parents to influence their children in educational achievement in Norway than in liberal welfare states.

Against this backdrop, the present study offers several contributions to the literature. We estimate genetic and social intergenerational associations with educational achievement using one of the largest genetic-trio datasets with measured genotypes of both parents and offspring available: the Norwegian Mother, Father, and Child Cohort Study (MoBa) (26). We use a recent iteration of a genome-wide association study (GWAS) of educational attainment based on 1.1 million people (27) to create a polygenic index for education for each family member. We link this to administrative register data and study social and genetic associations with children's educational performance. Given the age of the focal offspring, our analysis considers children's achievement scores on standardized tests in reading and mathematics, taken in the fifth, eighth, and ninth grades.

Our main analysis consists of four components. First, we establish the extent to which associations between parents' education and children's educational achievement (i.e., their test scores) are confounded by the children's own genotypes. Based on the comparative research finding that genetic influences are higher in societies with higher social security nets, we expect the degree of genetic confounding in Norway to be larger than what is found in more market-oriented societies, such as the United States.

Second, we estimate associations of genetic nurture: that is, associations of parental genotypes with educational achievement over and above children's own genotype. Based on the expectation that welfare-state policies are successful in limiting parents' possibilities to influence their children's educational outcomes, we expect genetic nurture associations to be smaller in Norway compared to in liberal welfare states. Moreover, we estimate models where we include both measured parental educational attainment and parental genomes, to investigate whether genetic nurture mechanisms operate chiefly through parents' realized educational attainment. In supplementary analyses, we also provide results separately for girls and boys and parental sex (i.e., sex-specific child–parent dyads). Although sex differences have been considered with respect to parental influences (i.e., maternal vs. paternal genetic nurture), the sex of the child has been far less studied.

Third, we examine any interactions between child genotype and parents' education, often referred to as the Scarr–Rowe hypothesis (28). According to the Scarr–Rowe hypothesis, the realization of children's genetic predispositions is that they are stronger in resource-rich home environments (29–31). In other words, if the Scarr–Rowe hypothesis is correct, there should be a positive interaction between parents' educational attainments and child genotype. In Norway, however, we anticipate that such differences are muted by the influence of social policies. For example, subsidized childcare and welfare benefits may attenuate resource-rich parents' relative advantages in nurturing children's genetic dispositions.

Fourth, we investigate interactions between parent's PGI and child's PGI as has been found for the US context (9). Such interactions would be evidence of synergistic associations between the genetically shaped traits and interests of parents and children. For example, genetically learning-oriented children would, under this hypothesis, learn more if they also have learning-oriented parents. This interaction may be termed "social epistasis," and we expect the same social policies that may break the association between genetic nurture and child educational achievement to be active here, meaning that the interactions found in other contexts may not be observed in Norway.

Results

We begin by considering the magnitude of genetic confounding in the association between parents' education and children's test scores. To this end, we estimate three models of the association between parents' education, child PGI, and test scores. By comparing results across these models, we can assess the importance of genetic confounding. Fig. 1 shows coefficients from these and two additional regression models, with 95% confidence intervals. Complete results from all regression models are included in the *SI Appendix*.

Model 1 shows that Child PGI is strongly associated with test scores. The estimated coefficient for the child's PGI is around 0.25 for both math and reading scores, which means that a 1 SD increase in the PGI results in 0.25 SD increase in the test score. The child's PGI is the strongest individual predictor of test performance.

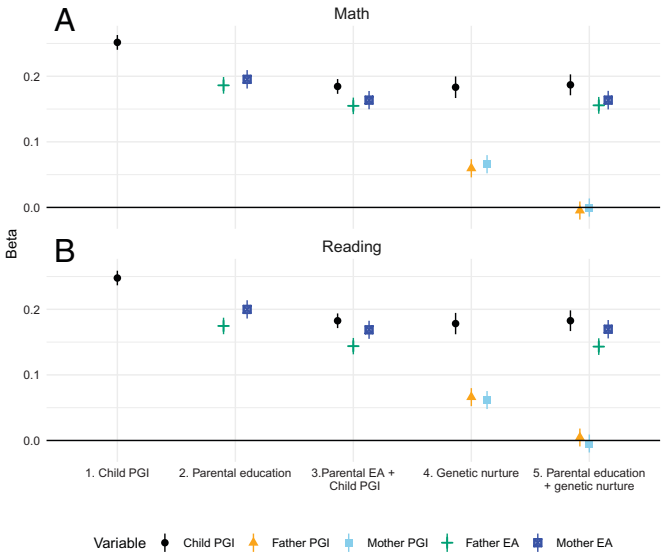

Fig. 1. Coefficients of Polygenic Indices (PGIs) and parental education in five models of (*A*) math test scores and (*B*) reading test scores. Bars indicate 95% confidence intervals. Model 1 includes child PGI. Model 2 includes parental education. Model 3 includes child PGI and parental education. Model 4 includes both child and parents' PGIs. Model 5 includes all PGIs and parental education. All models include child's sex and cohort, 10 principal components, and genotype batch.

Model 2, "Parents' education," shows the coefficients for parents' education in a model with test scores where no other variables are included. It shows, in correspondence with the extant literature, that parents' educations are associated with child test scores. The results are quite similar across math and reading, and the importance of mothers and fathers also seem to be similar in magnitude. We note moreover, that although child PGI is the strongest individual predictor on educational achievement, the sum of the two coefficients for parents' education is larger than the coefficient of the child PGI.

In model 3, there are relatively minor changes in the coefficients for parents' education once the child PGI for education is controlled, contrary to our hypothesis that in the Norwegian context, a larger portion of the relationship would represent genetic transmission. A fairly small fraction of the zero-order association of parents' education on test scores is confounded by child genetics, between 14% and 18%. This degree of confounding matches what was found in the US context for educational attainment (when not adjusting for measurement error in the PGI) (9). Another study from the United States found confounding to be around 20% (10). The latter uses the same GWAS for educational attainment as we do (27), while the former used an earlier iteration of the educational attainment GWAS (32).

Next, we probed the existence of genetic nurture associations (model 4). These are associations of parents' genotypes above and beyond the direct transmission of DNA from parents to children at conception. In this step, we first modeled both the child's own PGI and parents' PGIs to assess the magnitude of any genetic nurture associations with test scores. We did find genetic nurture associations with children's educational achievement. The coefficients for the mother's and father's PGIs were statistically significant and of a measurable, but not large, magnitude. Both coefficients were estimated at below 0.1 SD, and these associations are thus markedly smaller than the coefficient for the child's PGI. A 1 SD increase in one parent's PGI is associated with approximately a 0.06 SD increase in test performance. Again, this pattern is similar across the two tests.

Taken at face value, these associations would mean that parents' with high PGIs for education shape their children's rearing environments in ways that in turn contribute to their children's improved learning and ultimately higher test score results. Contrary to our expectations, the size of the genetic nurture associations we discovered match those found in liberal welfare state contexts (15), as does the size of the direct genetic association. The pooled estimate across the studies included in a recent metaanalysis for genetic nurture (combining maternal and paternal associations) is 0.11, of very similar magnitude to our 0.12 (combining maternal and paternal PGI in our figure). Therefore, genetic nurture does not seem to be of lower magnitude in a social democratic context, such as Norway. The overall direct association of children's own genes is estimated to be 0.17 based on data predominantly from liberal welfare states (15). This is of the same magnitude as our estimates of 0.18 for both reading and math, which indicates that genetic influences are of similar magnitude regardless of welfare state regime.

In model 5, we added parents' realized education to the model with genetic nurture to assess whether genetic nurture associations are mediated by parents' observed educational attainment and whether associations between parents' educations and children's test scores remain once the genetic profile of child, mother, and father all are adjusted for. Parents' education is of course a mediator in the causal path from parents' genetics to children's achievement, and thus in principle breaks the causal interpretation of the estimates of parents' PGI. The genetic nurture associations between parents' PGIs and child test scores are washed out once parents' educations are included. Strong associations between parents' education and test scores remain, when PGIs for all three family members are included. This is in line with findings from several studies that genetic nurture associations are largely explained by observed parental education (15).

Genetic nurture mechanisms may vary by parent and child sex. A previous finding was that mothers' genetic nurture were more important than fathers' genetic nurture for health-related phenotypes, but not educational attainment (13). We examined the importance of four types of genetic nurturing in parent–child dyads: mothers of boys, mothers of girls, fathers of boys, and fathers of girls (*SI Appendix*, Table S2). All nurture coefficients are stronger for girls than for boys, but these differences were not statistically significant. We found only one significant coefficient across the four dyads: fathers' nurture associations with math were slightly weaker for boys than for girls. Overall, the sex composition of the dyad does not seem to matter much for nurture associations with educational achievement.

In Fig. 2, we estimate the strength of any interactions between child genotype and parental educational attainment (black points), and child genotype and parents' own genotype (yellow point). For math there are no gene–environment interactions of the Scarr–Rowe variety (parental education–child genotype). For reading there is a borderline significant interaction effect between child PGI and maternal educational attainment, but in the opposite direction of the expectations from the Scarr–Rowe hypothesis. Having a mother with higher education is a relative disadvantage if the child has a high PGI. The effect size is very small. For children whose mothers' educational attainments are 1 SD above the mean, the coefficient for child's own PGI is 10% lower (−0.02) than for children whose mothers have average educational attainment. Nonetheless, our results indicate that there appear to be no Scarr–Rowe-type interactions in the Norwegian context, contrary to some (33), but not all (9, 34),

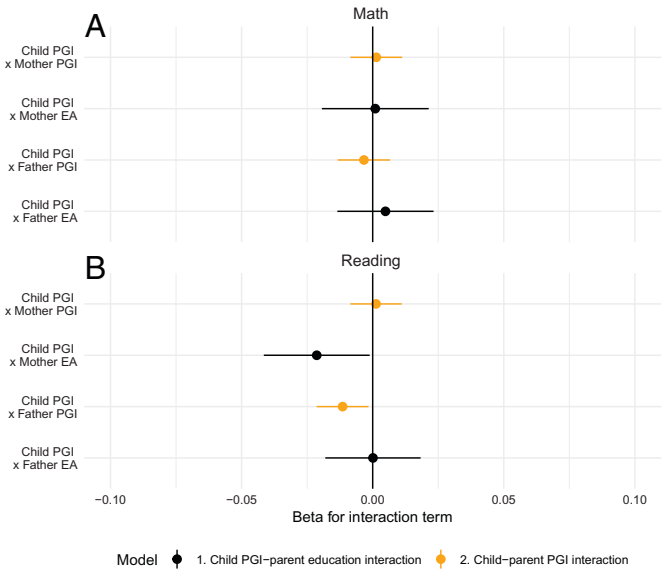

Fig. 2. Coefficients for interactions between child PGI and parents' PGI and educational attainments in models of (*A*) math and (*B*) reading test scores. Bars indicate 95% confidence intervals. Model 1 shows interaction estimates for parents' education and child's PGI. Model 2 shows interaction estimates for parents' PGI and child's PGI. All models include controls for child sex, child cohort, 10 principal components, and genotype batch.

molecular genetic-based evidence from the United States and United Kingdom.

Finally, we tested for interactions between parents' genetics and their child's genetics, what may be termed social epistasis. A previous study found a positive interaction between child PGI and mother PGI (9). In the same vein as the gene–environmental interactions, there were no genotype–genotype interactions in math. In reading, there was a borderline significant interaction between child genotype and paternal genotype. Again, the interaction is in the opposite direction of what one would expect. Having a father with a high PGI is a relative disadvantage for the child's test score in reading if the child also has a high PGI. However, the substantive importance of this coefficient is also marginal. For children whose father's PGI is 1 SD above the mean, a 1 SD increase in the child's PGI results in a decrease of 0.01. We found no interaction between mother PGI and child PGI.

Discussion

Associations between parents' and children's educational outcomes may arise through both genetic and environmental pathways. Prior research using twin-based approaches has advanced the claim that the strength of genetic associations is relatively constant across societies but that the degree to which family background (i.e., environmental influences of the family context) matters depends on the policy landscape (16, 17). A clear limitation of twin-based methods is the lack of measured genotypes. Here, we made use of a large dataset with genotypes from a social democratic welfare state, Norway, and compared our results to studies with genotype data from liberal welfare states.

Child PGI is the strongest individual predictor of educational achievement in Norway. Genetic confounding of the parent–child association of parental education, however, was small. Only 15% of the zero-order, intergenerational correlation in educational outcomes could be explained by the child's PGI for educational attainment. Contrary to our expectations, the degree of confounding is the same in Norway as was reported in studies on educational attainment (albeit a related but different phenotype) from the United States (9, 10). Also much to our surprise, genetic nurture associations in Norway (0.06) were almost exactly the same as average associations (0.07) in a recent metaanalysis of educational attainment and educational achievement based on data from mostly liberal welfare states (15). Like in other studies (15), we find that genetic nurture seems to operate through realized parental educational attainment.

Educational outcomes are among the few traits with a substantial shared environmental component in twin studies (3). Our results support evidence from twin studies that do not find variation in shared environmental components between countries (5–7, 35). Even though Scandinavian countries have among the highest economic mobility rates in the world, educational mobility is considerably lower than income-mobility (36). For educational outcomes, one should perhaps not be surprised that parental educational attainment—and thus genetic nurture, which operates through parental educational attainment—influences children's educational attainments regardless of country policies.

Gender has also been shown to influence genetic associations (37). We found miniscule differences in genetic nurture associations by parent and child sex. We are not aware of other studies using sex-specific parent–child dyads in investigating genetic nurture associations. The lack of such differences in our sample, born in the early 2000s, may be the result of the continuing trend toward gender equality, in which Norway and other Nordic countries have been at the vanguard (38).

If welfare state policies influence genetic associations, they would work through intermediate institutions, such as schools, families, and neighborhoods. We found that there are gene–environment interactions counter to what is postulated in the Scarr–Rowe hypothesis; having parents with higher education is a relative disadvantage if children have a high PGI. The same holds for social epistasis. Our findings are in line with a metaanalysis of twin studies showing that in countries with broader welfare policies, Scarr–Rowe interactions are nonexisting or reversed, contrary to what has been found in the United States (39). US-based studies have found both Scarr–Rowe and (positive) social epistasis interactions using molecular genetic data (9, 33). Our study indicates that Norwegian social policies, like subsidized child care and very low between-school quality differences, may mute—and perhaps counteract—such interactions.

Our results, like many molecular-based studies, deviate from what twin-based studies find in the magnitude of social and genetic associations. The discrepancy between twin and molecular genetic studies are likely to be methodological in nature. The predictive accuracy of the PGI depends on the heritability of the trait, where higher heritability yields more accurate PGIs. The estimated twin-heritability of educational achievement is higher than for educational attainment. We have used a GWAS from educational attainment to study educational achievement in adolescence, as there is no extant GWAS for educational achievement. This may lead to downward bias in both the direct and the indirect genetic associations we try to characterize; however, it is not clear whether such attenuation would alter the relative importance of direct and indirect associations.

Another potential problem for the predictability of the PGI is different genetic architectures of educational outcomes across different environments (32). We have used weights from a GWAS of educational attainment based on genomes of adults from other countries and birth cohorts to create PGIs in Norwegian children born mostly in the 2000s. If associations

between single-nucleotide polymorphisms (SNPs) and educational outcomes vary across contexts and cohorts, the predictive accuracy of our PGI is downwardly biased. Indeed, recent evidence suggests that genetic associations with adult educational attainment in Norway may have changed over generations (40).

Finally, all current PGIs only capture a fraction of the total genetic variation in the relevant outcomes. There are approaches to address measurement error, such as genetic instrumental variables (41), which rely on a number of assumptions. Yet, in assessing the relative impact of direct and indirect associations across contexts, there is little reason to suspect that the less-accurate predictive power of PGIs when used to estimate models should lead to systematic bias by cohort or place, and reducing classic measurement error does not solve the problem if there were systematic errors.

Notwithstanding such problems with PGIs deployed across contexts, this measured genotypic approach offers some strengths compared to twin studies. Twin-based cross-national comparisons rest on the assumption that gene–environment covariance and genetic assortative mating both remain constant across context. In the present study, in contrast, we compared estimates of direct genetic associations, of parental genetic nurture and of parental measured education to those obtained in other contexts. These estimates provide a necessary complement to twin-based estimates. The approach of estimating direct genetic, genetic nurture, and phenotypic associations on educational achievement should perhaps allow for a better test of whether the social and policy context matters for the relative influence of these factors. Likewise, while there is both phenotypic and genotypic assortative mating in our sample (20), direct genetic associations estimated in a within-family model should be less confounded by such deviations from random mating, in contrast to the heritability estimated in a twin model.

Our study concerns early adolescence. The interplay of genetics and social background may well change over the life course, as children increasingly operate independently of their parents. In early adolescence there is a lot of direct monitoring by parents, and parents may try to assist their children with schoolwork and this may spill over into the standardized test results. Once children age they will to a larger degree have to rely on their own talents, and not parents' active assistance. It is conceivable that the associations with social background we observe may fade at higher ages. Another limitation of our study concerns Scarr–Rowe interactions. Children who participate in MoBa are performing better than the average child on national test scores (*SI Appendix*, Table S1). Our results may lack common support in the data to investigate Scarr–Rowe interactions as they are described in societies with higher inequality. Our data, and indeed Norway in general, have very few children growing up in what could be described as deeply deprived conditions.

To conclude, our results outline a world that appears to be simple, linear, and mostly independent: parents' own education affects children's educational performance. Although the association of the child's own genetically anchored dispositions is the strongest individual predictor, the combined estimates of the mother's and the father's education is much higher. Some twin studies have found that the heritability of educational outcomes is higher when societies provide more equal opportunities (16, 17). Other comparative studies of the heritability of educational outcomes find no differences between social democratic welfare state countries and liberal welfare state countries (5–7, 35). Our results not only confirm that direct genetic associations are likely to be fairly constant across societies, but they also raise the possibility that social associations of family background do not systematically vary across policy contexts when models are estimated that are robust to differences in assortative mating and gene–environment covariance.

Even in a universal welfare state with relatively low levels of inequality there are still two systems of inheritance, one genetic and one social. The genetic confounding of intergenerational transmission of educational achievement from parents to children is low. Social science researchers can continue to emphasize environmental explanations without fearing that their interpretations are fully confounded.

Materials and Methods

Data. We used data from the Norwegian MoBa and register data from several Norwegian national administrative registration systems. MoBa is a population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health (26). Participants were recruited from all over Norway from 1999 to 2008. The women consented to participation in 41% of the pregnancies. The cohort now includes 114,500 children, 95,200 mothers, and 75,200 fathers. The current study is based on version 12 of the quality-assured data files, which includes around 98,000 genotyped individuals. The establishment of MoBa and initial data collection were based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics. The MoBa cohort is now based on regulations related to the Norwegian Health Registry Act. We obtained genetic data on the children and their parents through MoBa Genetics v1.0, an infrastructure for genomic data in MoBa. Blood samples were obtained from both parents during pregnancy and from mothers and children (umbilical cord) at birth. MoBa is likely a high functioning sample, meaning they experience fewer social problems and health symptoms compared to the prevalence in the population and this may in turn limit generalizability of results (42).

The register data stem from several different administrative registers (43). Basic demographic data and kinship links are taken from the Norwegian central population register, and both information on parents' education and children's results on national standardized tests are taken from the National Educational Database (NuDB). The linkage between MoBa and register sources was done using the Norwegian national ID number system, with minimal loss of information. The register data generally suffer no attrition and little, if any, error in registration (43).

Measures, Models, and Final Sample.

Polygenic indices. We constructed PGIs based on the results from the third GWAS for educational attainment (32). From the available SNP data, PGIs were calculated following best practice (44), using the PRSice software (45). After removing SNPs in linkage disequilibrium by clumping, we use information from all available SNPs when calculating the indices (i.e., a *P*-value threshold of 1.00). Documentation on the quality control and filtering of SNPs for creating the PGIs is included in *SI Appendix*. During this process, ∼5% of the genotyped samples were excluded. Finally, the indices were *z*-standardized separately for each role (mothers, fathers, children).

Parent's education at age 30. The data on parents' educational attainments cover the years 1970 and 1980 to 2018. We chose the measure that was closest in time to when the parents were aged 30. Originally coded on the NUS2000-standard, a taxonomy of educational programs similar to ISCED (46), the levels were converted to the expected number of years needed to achieve that level and then *z*-standardized.

National standardized tests. As of 2007, all students in Norway take nationwide standardized tests in mathematics, reading comprehension, and English (Directorate for Education 2010). All three tests are administered in fifth, eighth, and ninth grades, while English is administered in fifth and eighth grade only. The youngest children in our sample have not passed through all three grades, which implies that the number of children with valid observations vary over these test outcomes. Children's scores for math and reading tests were averaged across grades to reduce measurement error. We found no systematic trend over grades in test scores. Scores were *z*-standardized the scores within each test and year combination for the full population before we linked it to the MoBa sample, so that our outcome variables measure where in the distribution of scores the student places within his/her own cohort on a specific test.

Models. We estimated models of educational achievement with the child's PGI, parents' PGIs, and parents' educational attainments as predictors using ordinary least squares. We included the child's sex and birth cohort, the first 10 principal components of the SNP genotype dataset, and categorical variables for genotyping batch in all the models. We also estimated separate models for boys and girls in a supplementary analysis.

Final sample. Our sample is delineated in several ways. The final sample consists of participant families from MoBa who meet the following criteria. First, both parents must have consented to participate in MoBa and must all have been genotyped. Second, the child and both parents must have a valid PGI. Third, the child must have a valid result on at least one of the set of eight standardized test scores we studied. The final sample includes 26,518 complete child–mother–father trios.

Data Access and Replication. This work was approved by the Norwegian National Center for Research Data, the Regional Committee on Medical Ethics, data owners, and Statistics Norway. Norwegian privacy regulations limit our ability to share our register data, and the consent given by the MoBa participants does not open for storage of data from MoBa on an individual level in repositories or journals. Individual researchers may apply to obtain permissions and subsequently access the data. Researchers who want access to MoBa datasets for replication should submit an application to datatilgang@fhi.no. Access to datasets requires approval from The Regional Committee for Medical and Health Research Ethics in Norway and agreements with the above mentioned institutions. In *SI Appendix*, we provide a description of the procedure used to clean the genomic data and calculate the PGIs, the code used to link all the data sources together and analyze the data.

Data Availability. Administrative register and genetic data must be applied for to data owners by researchers and cannot be shared.

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