Correction

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Correction for "Genetic analysis of social-class mobility in five longitudinal studies," by Daniel W. Belsky, Benjamin W. Domingue, Robbee Wedow, Louise Arseneault, Jason D. Boardman, Avshalom Caspi, Dalton Conley, Jason M. Fletcher, Jeremy Freese, Pamela Herd, Terrie E. Moffitt, Richie Poulton, Kamil Sicinski, Jasmin Wertz, and Kathleen Mullan Harris, which was first published July 9, 2018; 10.1073/pnas.1801238115 (*Proc Natl Acad Sci USA* 115:E7275–E7284).

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Genetic analysis of social-class mobility in five longitudinal studies

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A summary genetic measure, called a "polygenic score," derived from a genome-wide association study (GWAS) of education can modestly predict a person's educational and economic success. This prediction could signal a biological mechanism: Education-linked genetics could encode characteristics that help people get ahead in life. Alternatively, prediction could reflect social history: People from well-off families might stay well-off for social reasons, and these families might also look alike genetically. A key test to distinguish biological mechanism from social history is if people with higher education polygenic scores tend to climb the social ladder beyond their parents' position. Upward mobility would indicate education-linked genetics encodes characteristics that foster success. We tested if education-linked polygenic scores predicted social mobility in >20,000 individuals in five longitudinal studies in the United States, Britain, and New Zealand. Participants with higher polygenic scores achieved more education and career success and accumulated more wealth. However, they also tended to come from better-off families. In the key test, participants with higher polygenic scores tended to be upwardly mobile compared with their parents. Moreover, in sibling-difference analysis, the sibling with the higher polygenic score was more upwardly mobile. Thus, education GWAS discoveries are not mere correlates of privilege; they influence social mobility within a life. Additional analyses revealed that a mother's polygenic score predicted her child's attainment over and above the child's own polygenic score, suggesting parents' genetics can also affect their children's attainment through environmental pathways. Education GWAS discoveries affect socioeconomic attainment through influence on individuals' family-of-origin environments and their social mobility.

genetics | social class | social mobility | sociogenomics | polygenic score

Genetics and social class may be thought of as fundamental causes of life outcomes because they are present at the beginning of human development; they are associated with a range of important life outcomes; they affect these outcomes through many different pathways; and their influences persist across cultures and over time (1–5). Twin studies and related designs establish that genetics influences education and social class (6, 7), but they are mostly silent on the nature of this influence (8, 9). Recently, genome-wide association studies (GWAS) have discovered molecular genetic associations with education (10–12), a determinant of social class (13). Observations that genetics discovered in GWAS of education are associated with social-class origins and with socioeconomic attainments (14–18) suggest these genetics and social class are connected. We considered three potential explanations for this connection.

One explanation for the connection between education-linked genetics and social class is that a person's education-linked genetics have causal effects on their attainments. A person's genetics cannot have a direct effect on their education. Instead, under this explanation, a person's genetics would influence their development of traits and behaviors that, in turn, contribute to their educational success (16, 19). For example, education-linked genetics could influence brain development in ways that affect behavior, leading to differences in achievement in school and beyond. If this explanation is right, education-linked genetics could help us understand molecular and behavioral mechanisms of social attainment, including gene–environment interplay in which genetics influences the environments and opportunities children encounter as they grow up.

Significance

Genome-wide association study (GWAS) discoveries about educational attainment have raised questions about the meaning of the genetics of success. These discoveries could offer clues about biological mechanisms or, because children inherit genetics and social class from parents, education-linked genetics could be spurious correlates of socially transmitted advantages. To distinguish between these hypotheses, we studied social mobility in five cohorts from three countries. We found that people with more education-linked genetics were more successful compared with parents and siblings. We also found mothers' education-linked genetics predicted their children's attainment over and above the children's own genetics, indicating an environmentally mediated genetic effect. Findings reject pure social-transmission explanations of education GWAS discoveries. Instead, genetics influences attainment directly through social mobility and indirectly through family environments.

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A second explanation for the connection between education-linked genetics and social class is that education-linked genetics carried by a person's relatives have causal effects on that person's attainments. Such effects could arise from genetic influences on parental characteristics that specifically affect their children, e.g., parental nurturance. They could also arise through genetic influences on attainment that affect children through processes of social transmission. Specifically, if genetics influence social attainment, children will inherit genetics that helped shape their parents' social class along with their parents' social class itself. In this way, genetics may influence a child's attainment through effects on the child's environment (20, 21). Such environmentally mediated genetic effects, in which parents' genetics affects the household environment in ways that influence their children's outcomes, are ruled out in genetic estimates from twin studies. However, they are not ruled out in GWAS (22, 23). Associations between a child's education-linked genetics and their attainment could thus reflect genetic influences on the child's traits and behaviors as well as effects of environments influenced by parents' genetics. If this explanation is right, it would direct attention toward features of the family environment as mechanisms linking DNA with social attainment.

A third explanation for the connection between education-linked genetics and social class is that it is spurious, e.g., because education-linked genetics is a correlate of a privileged social inheritance, i.e., having well-off ancestors. For example, social positions established long ago might be passed down across generations via socially transmitted inheritances, such as wealth transfers (24, 25). Because people tend to have children with mates from the same social class (26, 27), these historical differences in social position could carry a genetic signature. To the extent this genetic signature is not captured by principal components used to adjust for ancestry-related confounding in GWAS, it could be detected in GWAS but would have little to do with traits and behaviors that influence achievement. If this explanation is right, rather than providing clues to causal genetic processes identified in family-based genetic studies, education GWAS discoveries would be merely of genealogical interest.

One way to test the hypothesis that education-linked genetics influence social-class outcomes is to test if having more education-linked genetics predicts upward social mobility, defined as achieving more socioeconomic success relative to one's parents. By focusing on change in social position within a person's own lifetime, the analysis can separate the genetic and social legacy a child is born with from the influence of their genetics on future attainment. For example, if education-linked genetics mainly reflect a legacy of social privilege, then controlling for the socioeconomic status of a child's parents should reduce the association between the child's genetics and their future social attainment to zero. In contrast, if genetic associations with attainment persist even after controlling for parents' social class, this result would suggest that education-linked genetics influences social mobility.

If education-linked genetics influence social mobility, this raises the question of how one generation's genetics may affect their children's attainments. Direct transmission of genetics from parents to children is one path. However, a social–genetic effect in which parents' genetics influence their children's attainments through environmental pathways (28) is also possible. One way to test for such environmental transmission is to test if parental genetics predicts their children's attainments over and above the child's own genetics (22, 29).

We tested associations between education-linked genetics and social mobility in more than 20,000 individuals tracked over more than 1 million person-years of follow-up spanning birth through late life in five population-based longitudinal studies in the United States, the United Kingdom, and New Zealand (Fig. 1 and *SI Appendix*, 1.1–1.5 and Table S1). We measured education-linked genetics using the polygenic score method (30).
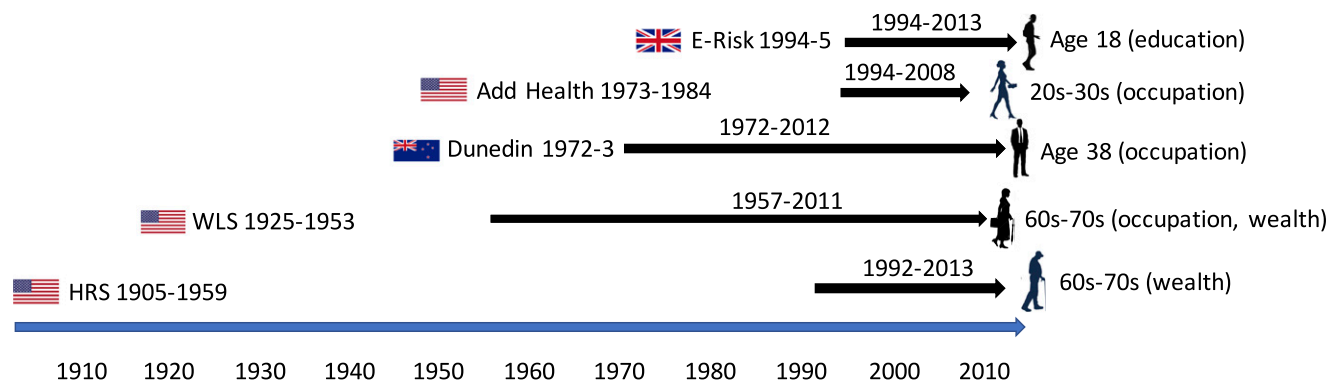
This method uses GWAS results as a scoring algorithm to compute a summary measure of genome-wide genetic influences on a phenotype. We measured social-class origins using data on parents' education, occupation, income, and financial difficulties. We analyzed social attainment in terms of education in adolescents, in terms of occupational attainment in young and midlife adults, and in terms of wealth in older adults. To test genetic associations with social attainment within a single lifetime, we first tested if participants' polygenic scores for education predicted their social attainments. We next tested if participants' polygenic scores were correlated with their social-class origins. This analysis tested for gene–environment correlations that could confound polygenic score associations with attainment. Finally, we tested polygenic score associations with social mobility by comparing the attainments of participants relative to their social-class origins. As a further analysis of mobility, we conducted sibling-difference analyses that tested if sibling differences in polygenic score predicted sibling differences in attainment. This analysis rules out confounding by any factors shared by siblings in a family that might not be captured in our measures of social-class origins. To test how parents' genetics might influence their children's attainments, we conducted mother–child social–genetic analysis. Specifically, we tested if mothers' polygenic scores predicted their children's attainments independent of the child's own polygenic score. This analysis tested if parents' genetics might influence their children's attainments through mechanisms of environmental transmission.

Results

Young People with Higher Polygenic Scores Were Upwardly Mobile in Their Education. We first analyzed educational mobility in a population-representative 1994–1995 birth cohort of twins in England and Wales followed to age 18 y: the Environmental Risk Longitudinal Twin (E-Risk) Study ($n = 1,860$ participants of European descent with genetic and education data) (*SI Appendix*, 1.1). Educational levels of E-Risk participants and their parents were measured by General Certificate of Education Examination (GCSE) level (none, 1, 2, or 3) and, for parents only, an additional category of 4 indicating completion of a university degree. Based on GWAS, we expected E-Risk participants with higher polygenic scores would achieve higher GCSE levels by age 18 y. They did (polygenic score–GCSE level $r = 0.27$, $P < 0.001$). We next tested if E-Risk participants with higher polygenic scores tended to have grown up in better-educated households. If so, this would indicate a gene–environment correlation that could confound associations between participants' polygenic scores and their educational attainment. E-Risk participants with higher polygenic scores did tend to grow up in better-educated households (participant polygenic score–parental education $r = 0.28$, $P < 0.001$). To test if this gene–environment correlation fully explained polygenic score associations with attainment, we conducted an educational-mobility analysis. We tested if E-Risk participants' polygenic scores continued to predict their educational attainment after controlling for their parents' education. Even after taking account of parents' education, E-Risk participants with higher polygenic scores tended to achieve upward educational mobility (parental-education adjusted $r = 0.16$, $P < 0.001$).

Sibling-difference analysis. As a second educational-mobility analysis, we conducted sibling-difference analysis. Because monozygotic twins are genetically identical, this analysis was restricted to dizygotic twins ($n = 388$ pairs; correlation of dizygotic twins' polygenic scores $r = 0.56$; association of polygenic scores with GCSE level in the dizygotic twin sample $r = 0.32$, $P < 0.001$). Sibling-difference analysis tested if the sibling with the higher polygenic score tended to achieve a higher GCSE level compared with the twin with a lower polygenic score. Sibling-difference analysis controls for any influences on education shared by siblings growing up in the same household, including any that may not be captured in measures of parental education. We conducted sibling-difference

Fig. 1. Cohorts included in analysis of social mobility. Sample sizes reflect participants of European descent with available genetic and attainment data.

Study	Social Origins	Social Attainments
E-Risk N=1,860 Born 1994-5 England & Wales	Parental education in 2000, when participants were aged 5 years.	Educational attainment in 2013, when participants were aged 18-19 years
Add Health N=5,526 Born 1973-84 USA	Composite index of parental education, parental occupation, household income, and household receipt of public assistance measured in 1994-5, when participants were in middle school and high school.	Socioeconomic index score (34, 35) based on occupation in 2007-8, when participants were in their late 20s and early 30s.
Dunedin N=831 Born 1972-3 Dunedin, NZ	Socioeconomic index score (69) based on parents' occupations measured at repeated intervals through the Study member's 15th birthday (1987-8) (39).	Socioeconomic index score (38) based on occupations in 2010-12, when participants were aged 38 years.
WLS N=7,111 Born 1925-53 Wisconsin, USA	Composite index of parents' education, father's occupation, and household income in 1957, when the focal WLS respondents graduated from high school.	Socioeconomic index score (34) based on occupations 1972-2005, when most participants were in their 30s-60s, and household wealth, measured in 2005-2011, when most participants were in their 60s-70s.
HRS N=8,546 Born 1905-59 USA	Composite index of parental education, father's occupation, household economic problems, and perceived family socioeconomic status in childhood (70).	Household wealth measured through 2013, when the average Study member was aged 70.

analysis using family-level fixed-effects regression. We denominated siblings' polygenic scores and educational attainments in sample-wide SD units so that the sibling-difference effect size (b) could be compared with the full-sample effect size (r). Consistent with the mobility hypothesis, the sibling with the higher polygenic score tended to achieve a higher GCSE level by age 18 y (sibling-difference $b = 0.13$ $P = 0.012$), indicating a genetic association with educational mobility.

Social–genetic analysis of maternal genetic effects. In the E-Risk cohort, the polygenic score associations with educational attainment were attenuated by about half after statistical control for parental education and in sibling-difference analysis. This attenuation suggests environmental confounding of polygenic score associations with educational attainment. We hypothesized this environmental confounding could arise from parental genetic effects. Specifically, parents' education-linked genetics could contribute to shaping the environments that influence their children's educational attainment. To test this hypothesis, we conducted a social–genetic analysis, which is a test of effects of one individual's genotype on another individual's phenotype (14, 31, 32). We conducted social–genetic analysis using data on E-Risk participants and their mothers ($n = 1,574$ E-Risk participants and their mothers in 804 families in which maternal and child genetic data and child education were available). If a mother's genetics predicts her child's education independent of that child's own genetics, it would suggest that there is environmental mediation of maternal genetic effects, supporting our hypothesis. If a mother's genetics do not predict her child's education independent of the child's own genetics, it would suggest that maternal genetic effects are transmitted exclusively via genetic transmission.

As expected, mothers with higher polygenic scores had higher levels of education ($r = 0.29$, $P < 0.001$). Mothers' polygenic scores also predicted their children's educational attainment ($r = 0.23$, $P < 0.001$). Also as expected, polygenic scores of mothers and children were correlated ($r = 0.49$, $P < 0.001$). However, even when we controlled for a child's own polygenic score, having a mother with a higher polygenic score predicted the child's achieving higher educational attainment (adjusted $r = 0.12$, $P < 0.001$). This social–genetic effect suggests environmental mediation of maternal genetic effects on children's educational attainment. Such environmental mediation offers one explanation for why education-linked genetics are correlated with environments that contribute to educational attainment.

Replication in the Add Health Study. We next tested polygenic score associations with educational mobility among participants of European descent in the United States-based National Longitudinal Study of Adolescent to Adult Health (hereafter, the "Add Health Study") ($n = 5,526$ with genetic and attainment data) (*SI Appendix*, 1.2). The Add Health Study first enrolled participants in 1994–1995, when they were in secondary school. Parents' education was measured at that time. Participants reported their own educational attainment in 2007–2008, when they were in their late 20s and early 30s. Education was reported in terms of the highest degree completed and was converted to a numeric value of years following the procedure used in the original GWAS of education (12). Consistent with the E-Risk analysis, Add Health participants with higher polygenic scores completed more schooling by young adulthood follow-up ($r = 0.28$, $P < 0.001$). Also consistent with E-Risk analysis, participants with higher polygenic scores tended to have grown up with better-educated parents, indicating a gene–environment correlation ($r = 0.28$, $P < 0.001$). We tested polygenic score associations with educational mobility by repeating polygenic-score analysis of educational attainment, this time including a control for parents' education. Even after parents' education was taken into account, Add Health participants with higher polygenic scores tended to achieve upward educational mobility (adjusted $r = 0.22$, $P < 0.001$).

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The Add Health cohort included siblings (*n* = 352 pairs; correlation of siblings' polygenic scores *r* = 0.54; association of polygenic scores with educational attainment in the sibling sample *r* = 0.30, *P* < 0.001). We conducted sibling-difference analysis using family-level fixed-effects regression in parallel with the E-Risk analysis. Compared with the sibling with a lower polygenic score, the sibling with the higher polygenic score tended to achieve a higher level of educational attainment (sibling-difference *b* = 0.15 *P* = 0.020).

We conducted additional replication analyses of polygenic score associations with educational mobility in a New Zealand birth cohort followed to midlife (the Dunedin Multidisciplinary Health and Development Study, hereafter the "Dunedin Study") (*SI Appendix*, 1.3) and in a US older adult cohort (the Health and Retirement Study, hereafter "HRS") (*SI Appendix*, 1.5). Results were similar to the Add Health Study. Effect sizes for the analysis of educational attainment are reported in Figs. 2 and 3 and *SI Appendix*, Tables S2 and S3.

Young and Midlife Adults with Higher Polygenic Scores Were Upwardly Mobile in Their Occupational Attainment. Beyond education, a second important constituent of social class is occupational attainment (33). We first analyzed social mobility in terms of occupational attainment in the Add Health Study (*n* = 5,526 participants of European descent with genetic and attainment data). Add Health participants reported their occupations in 2007–2008, when they were in their late 20s and early 30s. We scored these occupations using Hauser and Warren Occupational Income and Occupational Education scales to create a composite reflecting average income and educational levels of job-holders in US Census data (*SI Appendix*, 1.2) (34, 35). Add Health participants with higher polygenic scores achieved higher levels of occupational attainment by young adulthood (*r* = 0.20, *P* < 0.001). This was true even after accounting for differences in their educational qualifications (adjusted *r* = 0.06, *P* < 0.001).

To create measures for the test of social mobility, we computed social-origin scores for Add Health participants based on their parents' education and occupation, their household income, and household receipt of social welfare benefits measured when the participants were teenagers (*SI Appendix*, 1.2). Add Health participants with higher polygenic scores tended to have grown up with better social origins (*r* = 0.29, *P* < 0.001), a gene–environment correlation. Nevertheless, the test of social mobility showed that, independent of their social origins, Add Health participants with higher polygenic scores tended to achieve higher levels of occupational attainment, indicating upward social mobility (social-origins adjusted *r* = 0.15, *P* < 0.001) (Figs. 2 and 4*A*). Social mobility is sometimes expressed in terms of change in rank within a population distribution. Expressed in terms of this metric, Add Health participants with polygenic scores one SD above the mean showed an average improvement of over three percentiles in the ranking of attained social position relative to their parents. Social-mobility transition matrices (36) showing rank mobility within low, middle, and high polygenic score groups are in *SI Appendix*, Fig. S1.

Occupational structures and opportunities for upward mobility vary from country to country and across historical time and social context (37). To test if the occupational-attainment social-mobility finding was robust to variation in setting, we conducted replication analyses in two place-based cohort studies, the Dunedin Study and the Wisconsin Longitudinal Study (WLS). We also conducted sibling-difference analysis in the Add Health Study and WLS sibling samples.

Replication in the Dunedin Study. We tested replication of polygenic score associations with social mobility in a 1972–1973 New Zealand birth cohort followed to age 38 y from the Dunedin Study (*n* = 831 participants of European descent with genetic and attainment data). We measured participants' occupational

attainment through age 38 y by scoring their jobs using the New Zealand Socioeconomic Index (NZSEI) (16, 38), a measure similar to the Hauser and Warren scales analyzed in the Add Health Study and WLS. We measured participants' social origins by scoring the jobs of participants' parents when the participants were children (*SI Appendix*, 1.3) (39). As in the Add Health Study, participants in the Dunedin Study with higher polygenic scores achieved higher levels of occupational attainment by age 38 y (*r* = 0.26, *P* < 0.001). This was true even after accounting for differences in their educational qualifications (adjusted *r* = 0.11, *P* < 0.001). We also observed evidence of gene–environment correlation. Children with higher polygenic scores tended to grow up in better social origins (*r* = 0.15, *P* < 0.001). The test of social mobility showed that, independent of their social origins, children in the Dunedin Study with higher polygenic scores tended to achieve upward social mobility (social-origins adjusted *r* = 0.21, *P* < 0.001) (Figs. 2 and 4*B*). Expressed in terms of social-position percentile-rank change among participants in the Dunedin Study, having a one SD higher polygenic score was associated with a six-percentile-rank improvement in attained social position relative to their parents. Social-mobility transition matrices showing rank mobility within low, middle, and high polygenic score groups are in *SI Appendix*, Fig. S1.
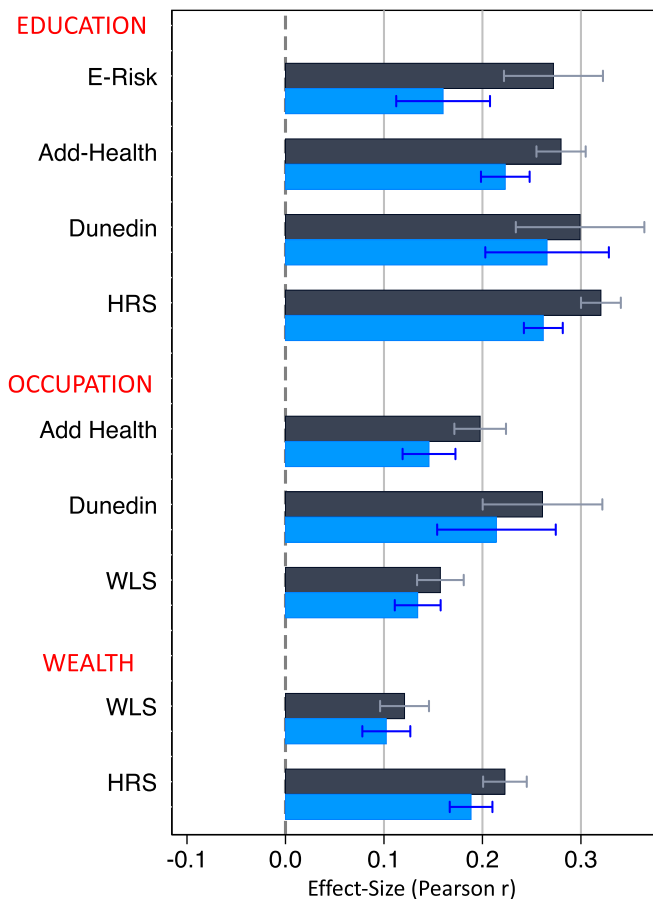

Fig. 2. Effect-size estimates for education polygenic score associations with social attainment and mobility in five cohorts. The figure graphs effect-size estimates (Pearson's *r*) for education polygenic score associations with social attainment and mobility. Polygenic score associations with attainment are graphed in navy blue. Polygenic score associations with social mobility are graphed in light blue. Error bars show 95% CIs for effect-size estimates. Mobility effect sizes were estimated from analysis of attainment with covariate adjustment for social origins. Model details are in *SI Appendix*, 1.7. Result details are in *SI Appendix*, Table S2.
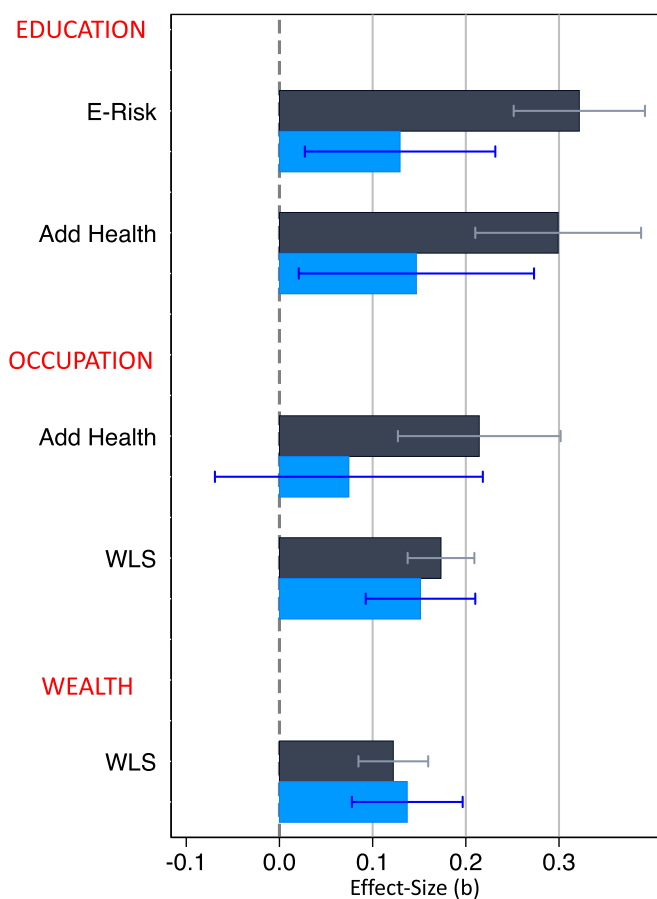
Fig. 3. Sibling-difference effect-size estimates for education polygenic score associations with social attainment and mobility in three cohorts with sibling data. The figure graphs effect-size estimates (comparable to the Pearson's r reported for full-sample analysis) for education polygenic score associations with social attainment and mobility from analyses of siblings in the E-Risk, Add Health, and WLS cohorts. Polygenic score associations with attainment in the samples of siblings are graphed in navy blue. Polygenic score associations estimated in sibling-difference models are graphed in light blue. Error bars show 95% CIs for effect-size estimates. Sibling-difference effect sizes were estimated from family fixed-effects regression models. Model details are in *SI Appendix*, 1.7. Results details are in *SI Appendix*, Table S3.

Replication in the WLS. We next tested the replication of polygenic score associations with social mobility in a cohort of 1957 high-school graduates and their siblings, the WLS (*n* = 7,111 participants of European descent with genetic and attainment data) (40). We measured occupational attainment in this cohort through 2005, by which time most WLS members were in their 60s. We scored occupations using the same procedure as in the Add Health Study (*SI Appendix*, 1.4). We computed social-origin scores based on parents' education, father's occupation, and household income in 1957, the year most WLS participants graduated from high school (*SI Appendix*, 1.4). As in the Add Health Study, WLS participants with higher polygenic scores tended to achieve higher levels of occupational attainment (*r* = 0.16, *P* < 0.001). This association was explained mostly by differences in education (adjusted *r* = 0.03, *P* = 0.014). We also observed evidence of gene–environment correlation. Children with higher polygenic scores tended to grow up with better social origins (*r* = 0.12, *P* < 0.001). The test of social mobility showed that, independent of their social origins, children with higher polygenic scores tended to achieve upward social mobility (social-origins adjusted *r* = 0.13, *P* < 0.001) (Figs. 2 and 4*C*).

Expressed in terms of social-position percentile-rank change, WLS participants with polygenic scores one SD above the mean showed an average improvement of four percentiles in the ranking of attained social position relative to their parents. Social mobility-transition matrices showing rank mobility within low, middle, and high polygenic score groups are in *SI Appendix*, Fig. S1.

Effect sizes for occupational attainment analysis are reported in *SI Appendix*, Table S2. Effect sizes for social-origins analysis are in *SI Appendix*, Table S4. Estimates for percentile-rank mobility analysis are in *SI Appendix*, Table S5.

Sibling-difference analysis. As a final test, we conducted sibling-difference analysis using family-level fixed-effects regression. We denominated siblings' polygenic scores and occupational attainments in sample-wide SD units so that sibling-difference effect sizes (*b*) could be compared with full-sample effect sizes (*r*). In the Add Health Study (*n* = 352 Add Health pairs; correlation of siblings' polygenic scores *r* = 0.54; association of polygenic scores with occupational attainment in the sibling sample *r* = 0.21, *P* < 0.001), the sibling with the higher polygenic score tended to achieve higher occupational attainment compared with the sibling with a lower polygenic score, but this association was not statistically significant (sibling-difference *b* = 0.07, *P* = 0.298). In WLS (*n* = 1,779 WLS pairs; correlation of siblings' polygenic scores *r* = 0.52; association of polygenic scores with occupational attainment in the sibling sample *r* = 0.17, *P* < 0.001), the sibling with the higher polygenic score tended to achieve higher occupational attainment compared with the sibling with a lower polygenic score, and the association was statistically significant (sibling-difference *b* = 0.15, *P* < 0.001). Sibling-difference effect sizes are shown in Fig. 3 and are reported in *SI Appendix*, Table S3.

Note: We did not analyze occupation in the E-Risk cohort because many of the 18-y-old participants had not yet entered the labor market. We did not analyze occupation in the HRS cohort because many of these participants had already exited the labor market.

Older Adults with Higher Polygenic Scores Were Upwardly Mobile in Their Accumulation of Wealth. We analyzed mobility in terms of wealth accumulation among older adults. Wealth data were measured from structured interviews in the WLS (*n* = 7,007 participants of European descent with genetic data) and the HRS (*n* = 8,533 participants of European descent with genetic data) (*SI Appendix*, 1.4 and 1.5) (41, 42). WLS and HRS participants with higher polygenic scores accumulated more wealth across their lives (WLS *r* = 0.12, *P* < 0.001; HRS *r* = 0.22, *P* < 0.001). This was true even after accounting for differences in their educational attainment (adjusted WLS *r* = 0.06, *P* < 0.001; adjusted HRS *r* = 0.11, *P* < 0.001).

WLS Analysis. We analyzed social mobility in terms of wealth in the WLS using the social-origins measure described in the previous section. As described above, there was a gene–environment correlation in which participants with higher polygenic scores grew up with better social origins (*r* = 0.12, *P* < 0.001). Nevertheless, the test of social mobility showed that, independent of their social origins, WLS participants with higher polygenic scores were upwardly mobile in terms of wealth accumulation (*r* = 0.10, *P* < 0.001) (Fig. 2). Expressed in terms of social-position percentile-rank change, WLS participants with polygenic scores one SD above the mean showed an average improvement of four percentiles in the ranking of attained wealth position relative to their social origins.

HRS Analysis. To develop measures for a test of social mobility in the HRS, we computed social-origin scores for HRS participants based on their parents' education and occupation, an index of household economic problems measured from retrospective reports made by participants, and participant ratings of their
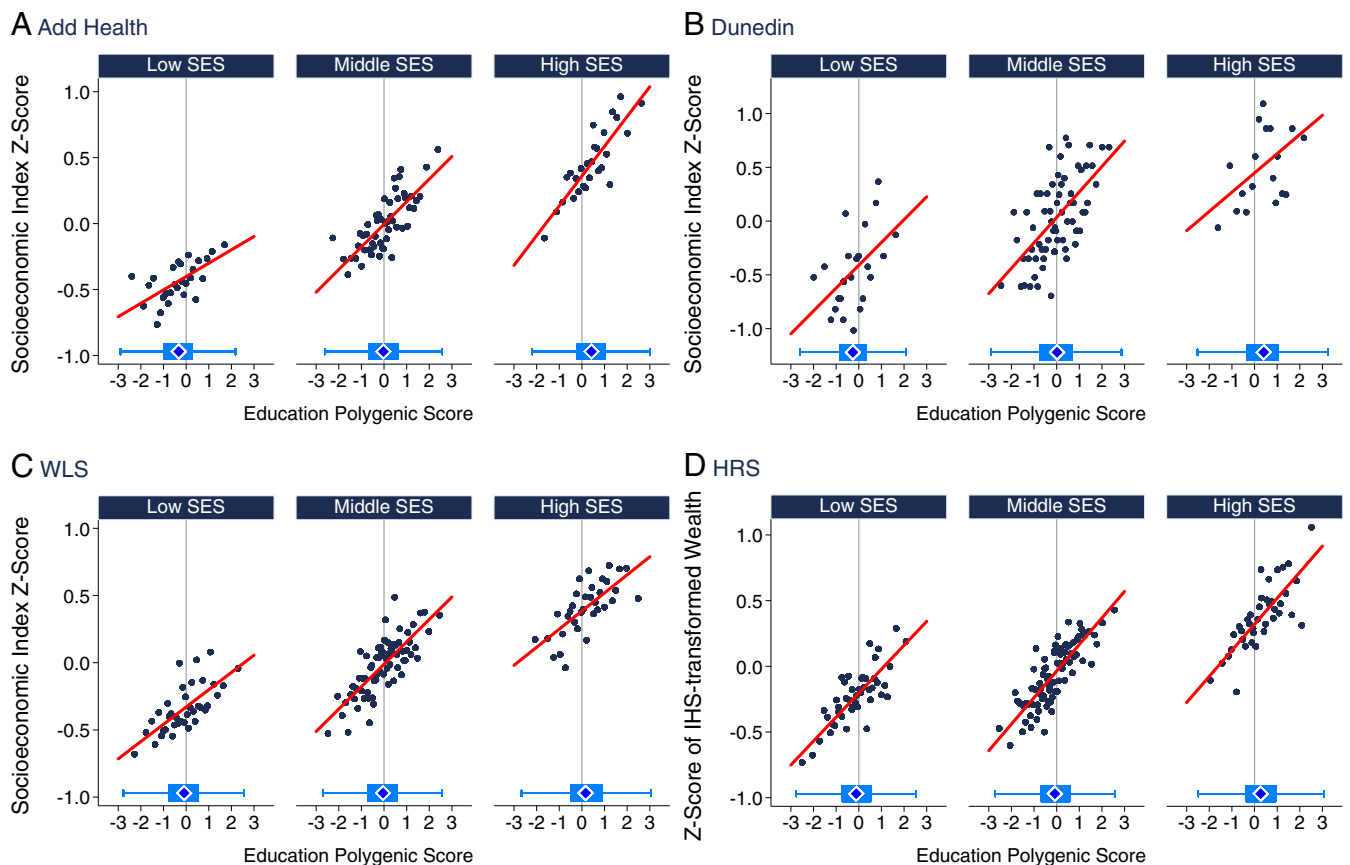
Fig. 4. Education polygenic score associations with social attainment for Add Health Study, WLS, Dunedin Study, and HRS participants with low-, middle-, and high-socioeconomic status (SES) social origins. The figure plots polygenic score associations with socioeconomic attainment for Add Health Study (*A*), Dunedin Study (*B*), WLS (*C*), and HRS (*D*) participants who grew up in low-, middle-, and high-SES households. For the figure, low- middle-, and high-SES households were defined as the bottom quartile, middle 50%, and top quartile of the social origins score distributions for the Add Health Study, WLS, and HRS. For the Dunedin Study, low SES was defined as a childhood NZSEI of two or lower (20% of the sample), middle SES was defined as childhood NZSEI of three to four (63% of the sample), and high SES was defined as childhood NZSEI of five or six (17% of the sample). Attainment is graphed in terms of socioeconomic index scores for the Add Health Study, Dunedin Study, and WLS and in terms of household wealth in the HRS. Add Health Study and WLS socioeconomic index scores were calculated from Hauser and Warren (34) occupational income and occupational education scores. Dunedin Study socioeconomic index scores were calculated similarly, according to the Statistics New Zealand NZSEI (38). HRS household wealth was measured from structured interviews about assets. All measures were z-transformed to have mean = 0, SD = 1 for analysis. The individual graphs show binned scatterplots in which each plotted point reflects average *x* and *y* coordinates for a bin of 50 participants for the Add Health Study, WLS, and HRS and for a bin of 10 participants for the Dunedin Study. The red regression lines are plotted from the raw data. The box-and-whisker plots at the bottom of the graphs show the distribution of the education polygenic score for each childhood SES category. The blue diamond in the middle of the box shows the median; the box shows the interquartile range; and the whiskers show upper and lower bounds defined by the 25th percentile minus 1.5× the interquartile range and the 75th percentile plus 1.5× the interquartile range, respectively. The vertical line intersecting the *x* axis shows the cohort average polygenic score. The figure illustrates three findings observed consistently across cohorts: (*i*) participants who grew up in higher-SES households tended to have higher socioeconomic attainment independent of their genetics compared with peers who grew up in lower-SES households; (*ii*) participants' polygenic scores were correlated with their social origins such that those who grew up in higher-SES households tended to have higher polygenic scores compared with peers who grew up in lower-SES households; (*iii*) participants with higher polygenic scores tended to achieve higher levels of attainment across strata of social origins, including those born into low-SES families.

families' social positions when they were children (*SI Appendix,* 1.5). HRS participants with higher polygenic scores tended to have grown up with better social origins ($r = 0.17$, $P < 0.001$), a gene–environment correlation. Nevertheless, the test of social mobility showed that, independent of their social origins, HRS participants with higher polygenic scores were upwardly mobile in terms of wealth accumulation (social-origins adjusted $r = 0.19$, $P < 0.001$) (Figs. 2 and 4*D*). Expressed in terms of social-position percentile-rank change, HRS participants with polygenic scores one SD above the mean showed an average improvement of more than five percentiles in the ranking of attained wealth position relative to their social origins.

Sibling-difference analysis. We repeated our test of social mobility using sibling-difference analysis. We denominated siblings' polygenic scores and wealth in sample-wide SD units so that sibling-

difference effect sizes (*b*) could be compared with full-sample effect sizes (*r*). Among WLS siblings ($n = 1,778$ pairs with wealth data; correlation of siblings' polygenic scores $r = 0.52$; association of polygenic scores with wealth in the sibling sample $r = 0.12$, $P < 0.001$), the sibling with the higher polygenic score tended to accumulate more wealth (sibling-difference $b = 0.14$, $P < 0.001$). Sibling-difference effect sizes are shown in Fig. 3 and are reported in *SI Appendix,* Table S3.

Genetic Associations with Socioeconomic Attainments Varied Slightly Depending on Social Origins but Not in the Same Direction Across Cohorts. Some studies of twins and families suggest genetic influences on attainment may vary across the context of childhood social origins (43–45). We therefore tested if the magnitude of associations between participants' education polygenic scores and their socioeconomic attainments varied depending on their social

origins, a hypothetical gene–environment interaction (*SI Appendix,* 2.1). We observed some evidence of gene–environment interaction in the Add Health cohort (*P* value for interaction <0.001); in the Add Health Study, genetic effects on outcome were larger for children born into higher socioeconomic status families. In contrast, interaction-effect estimates were not statistically significant in the other cohorts (*SI Appendix,* Table S6).

Discussion

We tested if genetics discovered in GWAS of educational attainment were related to socioeconomic mobility across the life course in five cohorts from the United States, Britain, and New Zealand. Across these studies, there were three consistent findings. First, education-linked genetics were related to social attainment: Children with higher education polygenic scores tended to complete more years of schooling, build more successful occupational careers, and accumulate more wealth. Second, there was a gene–environment correlation: Children with higher polygenic scores tended to grow up in socioeconomically better-off homes. Third, education-linked genetics were related to social mobility: Regardless of where they started in life, children with higher polygenic scores tended to move up the social ladder in terms of education, occupation, and wealth, even compared with siblings in their own families.

These findings clarify how education-linked genetics and social class are connected. First, the findings argue against the explanation that the connection is spurious. The finding that participants' education-linked genetics predicted change in their social position within their own lives, replicated across five cohorts in three countries, argues against the explanation that education-linked genetics are simply a correlate of a privileged social inheritance that escaped ancestry controls in GWAS. Instead, findings support the explanation that education-linked genetics are connected to social class because they influences attainment: Participants' education-linked genetics predicted their social mobility, and differences in education-linked genetics between siblings predicted differences between siblings in life-course attainments.

Second, the findings suggest that education-linked genetics may be connected to social class in part because education-linked genetics carried by a person's relatives can influence that person's own attainment. Genetic associations with attainments were attenuated when models accounted for participants' social origins. This finding suggests that genetic associations with social attainment could arise, in part, from gene–environment correlations between participants' education-linked genetics and environments related to participants' social origins. Such gene–environment correlations could reflect effects of parents' genetics on family environments, which parents subsequently give to their children along with genotypes (5, 21). Our social–genetic analysis of pairs of mothers and children found mothers' polygenic scores predicted their children's educational attainment independent of the children's own polygenic scores. This finding is consistent with the hypothesis that parents' education-linked genetics contribute to shaping the environments that influence their children's subsequent attainment.

We acknowledge limitations. First, our genetic measurement is imprecise. The education polygenic score explains only a fraction of the estimated total genetic influence on education (10). Our effect sizes are thus attenuated by substantial measurement error in the polygenic score. This bias toward the null makes our analysis a conservative estimate of genetic associations with social mobility. To provide an estimate of the extent of this bias, effect-size estimates corrected for measurement error using a recently proposed method (46, 47) are reported in *SI Appendix,* Table S7. The problem is more severe in analysis of non-Europeans (*SI Appendix,* 2.3). With larger GWAS sample sizes, new GWAS in non-European populations, and identifica-

tion of which specific genetic variants are causal, these limitations will be partly mitigated (48, 49).

Second, analyses do not completely exclude potential bias due to population stratification (50), the nonrandom patterning of genotypes across different ancestries. We used the best available methods to account for confounding by ancestry-related genetic differences that could be correlated with social attainment. We focused analyses on relatively genetically homogenous samples of individuals of European descent and further applied covariate adjustment for genetic principal components (*SI Appendix,* 1.6). Even so, it is possible that unmeasured population stratification could influence results. Sibling-difference analysis does exclude population stratification as a confounder (15, 51), establishing a floor for effect-size magnitudes.

Third, the genetics of socioeconomic attainment and mobility may vary slightly across different birth cohorts, presumably reflecting changes in the social context of attainment (52). This could cause incomplete genetic correlation between mothers and their children and may introduce confounding into our social–genetic mother-child analysis. Fourth, we lack complete genetic information on the parents of the people whose lives we studied. In the E-Risk cohort, in which we analyzed maternal genetic data, fathers did not give DNA. Thus, we cannot fully isolate genetic from environmental mechanisms of intergenerational transmission. However, our designs do allow certain conclusions about the direct genetic effects of an individual's own DNA on their attainment and about the socially transmitted genetic effects of a parent's DNA on their child's attainment. Sibling-difference analysis, which controls for the genetics of both parents, can test for direct genetic effects. Our sibling-difference analysis establishes a floor for the size of direct genetic effects and rules out purely social transmission as an explanation for the associations between children's education-linked genetics and their attainment. Social–genetic analysis in which the child's education is regressed on the polygenic scores of one parent and the child can test for socially transmitted genetic effects. Assuming the parent's and child's polygenic scores are measured with the same error, the effects of genetics transmitted from parent to child are captured by the child's polygenic score; i.e., the child's polygenic score acts as a control for the direct genetic effect. The residual association between the parent's polygenic score and the child's attainment can be interpreted as a socially transmitted parental genetic effect. Although we cannot rule out differences in measurement error between polygenic scores of mothers and their children, our social–genetic analysis provides some evidence for socially transmitted maternal genetic effects on children's educational attainment, consistent with a recent analysis that included genetic information from both parents (22, 53).

Against the background of these limitations, our analysis suggests three take-home messages. The first take-home message is that genetics research should incorporate information about social origins. For genetics, our findings suggest that estimates of genetic associations with socioeconomic achievement reflect direct genetic effects as well as the effects of social inheritance correlated with genetics. Future genetic studies of social attainment can refine inferences about direct genetic effects by including measures of social origins in their study designs. The same is true for genetic studies of other phenotypes, because childhood socioeconomic circumstances are implicated in the etiology of many different traits and health conditions (54–56). Such analysis will help clarify interpretation of studies that analyzed GWAS data and found evidence of genetic overlap between educational attainment and several biomedical phenotypes (57, 58). The advent of national biobanks and other large genetic datasets is increasing the power of GWAS to map genetic risks. Research to investigate how much of the genetic risk measured from GWAS discoveries arises within a single generation and how much accrues from social inheritance correlated with genetics across successive generations is needed.

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The second take-home message is that social science research should incorporate information about genetic inheritance. For the social sciences, our findings provide molecular evidence across birth cohorts and countries of genetic influence on social attainment and social mobility. This evidence supports theory in the social sciences that frames genetics as one mechanism among several through which social position is transmitted across generations (9, 20, 21, 59). These theories imply that genetic factors can confound estimates of social environmental effects. However, because genetics have been difficult to measure, studies addressing these theories have had to estimate genetic contributions to attainment indirectly, while other social science research has simply ignored the problem. Now, genetically informed theories of social attainment and mobility can be revisited, tested, and elaborated using molecular genetic data available in an ever-growing array of genetically informed social surveys and longitudinal cohort studies.

Beyond theory, integration of measured genetic inheritance into research on social mobility can add value in at least three ways. First, genetic controls can improve the precision of estimates of environmental effects (11, 14), e.g., of how features of parents' social circumstances shape children's development. Second, genetic measurements can provide a starting point for developmental investigations of pathways to social mobility (16, 60), e.g., to identify skills and behaviors that can serve as targets for environmental interventions to lift children out of poverty. Third, genetic measurements can be used to study gene–environment interplay; e.g., how policies and programs may strengthen or weaken genetic and nongenetic mechanisms of intergenerational transmission (61). In our analysis, modeling effects of social origins attenuated genetic-effect sizes by 10–50%, depending on the outcome and cohort. This variation is consistent with evidence that genetic influences on individual differences may vary across cultures and cohorts and across stages of the life course (62, 63). Research is needed to understand how molecular genetic effects on socioeconomic attainment may operate differently across environmental, historical, or economic contexts and the extent to which they may wax or wane across adult development.

The third take-home message is that genetic analysis of social mobility can inform programs and policies that change children's environments as a way to promote positive development. The genetics we studied are related to socioeconomic attainment and mobility partly through channels that are policy-malleable. Personal characteristics linked with the attainment-related genetics we studied involve early-emerging cognitive and noncognitive skills, including learning to talk and read, act planfully, delay gratification, and get along with others (10, 16). These skills represent intervention targets in their own right, for example by policies and programs that safeguard perinatal development and provide enriching, stable family and educational environments (64). A significant contribution of our study is that the nongenetic social and material resources children inherit from their parents represent a further mechanism linking genetics and attainment over the life course. Policies and programs cannot change children's genes, but they can help give them more of the resources that children who inherit more education-linked genetics tend to grow up with. Our findings suggest that such interventions could help close the gap. The next step is to find out precisely what those resources are.

Conclusion

A long-term goal of our sociogenomic research is to use genetics to reveal novel environmental intervention approaches to mitigating socioeconomic disadvantage. The analysis reported here takes one step toward enabling a study design to accomplish this. We found that measured genetics related to patterns of social attainment and mobility, partly through direct influences on individuals and partly

through predicting the environments in which they grew up. Specifically, parents' genetics influence the environments that give children their start in life, while children's own genetics influence their social mobility across adult life. As we learn more about how genetics discovered in GWAS of education influence processes of human development that generate and maintain wealth and poverty, we can identify specific environments that shape those processes. Ultimately, this research approach can suggest interventions that change children's environments to promote positive development across the life-course.

Methods

Detailed descriptions of data and measures are included in *SI Appendix*, 1.1–1.5; analysis is described in *SI Appendix*, 1.6 and 1.7.

Data Sources. Data were used from five studies: the E-Risk Study, the Add Health Study, the Dunedin Study, the WLS, and the US HRS.

Polygenic Scoring. We computed polygenic scores for participants in the E-Risk Study, Add Health Study, Dunedin Study, WLS, and HRS based on all SNPs analyzed in the most recent Social Science Genetic Association Consortium (SSGAC) GWAS of educational attainment (12). No statistical significance threshold was applied to select SNPs for inclusion in polygenic score analysis. For the E-Risk Study and the Dunedin Study, polygenic scores were computed following the method described by Dudbridge (30) using the PRSice software (65). For the Add Health Study, WLS, and HRS, polygenic scores were computed by the SSGAC using the LD Pred software (66). The Add Health Study, WLS, and HRS data were included in the SSGAC GWAS of educational attainment. For each of these datasets, polygenic scores were computed using summary statistics from GWAS meta-analyses from which the target dataset for polygenic scoring was excluded. Within each dataset, we regressed SSGAC-computed polygenic scores on the first 10 principal components estimated from the genome-wide SNP data (67) and calculated residual values. Finally, we standardized these residual values to have mean = 0, SD = 1 within each dataset to form the final versions of the polygenic scores used for analysis.

Socioeconomic Origins and Attainments. Participants' social origins and socioeconomic attainments were measured from available data, with the aim of deriving measurements to compare social origins with attainments. Measurements are described briefly in Fig. 1.

Other Measures. We measured educational attainment as GCSE level (0, 1, 2, or 3) in the E-Risk Study, as years of schooling in the Add Health Study, WLS, and HRS, and as a four-category variable coding the highest degree attained, as described previously (16), in the Dunedin Study.

Analysis. We tested associations using linear regression models. For cohorts of mixed birth years (Add Health Study, HRS, and WLS), we included dummy variables for years of birth. For cohorts sampled from schools (Add Health Study and WLS), we included dummy variables for school in analyses of genetic associations with attainment and mobility. School dummies were not included in the analysis of gene–environment correlation with social origins. For cohorts including siblings or spouses (Add Health Study, HRS, and WLS), we clustered SEs at the family level. We conducted sibling-difference analyses using family fixed-effects regression (68–70). For analysis, we denominated polygenic scores and outcome variables in units of sample-specific SDs. We refer to effect sizes denominated in this metric as "r" to reflect their parallel interpretation with Pearson correlations. We used this same standardization for sibling-difference analysis so that effect sizes can be compared between full-sample and sibling-difference models.

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