H1 FRONT MATTER

H2: Title

- Full Title: Polygenic score for education predicts adaptive development from preschool to midlife
- Short Title: A sociogenomic analysis of life attainments

H2:Authors

Daniel W Belsky[1,2*], Terrie E Moffitt[3-6] Richie Poulton[7], Avshalom Caspi[3-6]

H2:Affiliations

1. Department of Medicine, Duke University School of Medicine, Durham NC 27710.
2. Social Science Research Institute, Duke University, Durham NC 27708.
3. Department of Psychology & Neuroscience, Duke University, Durham NC 27708.
4. Department of Psychiatry & Behavioral Sciences, Duke University School of Medicine, Durham NC 27708.
5. Center for Genomic and Computational Biology, Duke University, Durham NC 27708.
6. Social, Genetic & Developmental Psychiatry Research Centre, Institute of Psychiatry, Kings College London, London SE58AF, United Kingdom.
7. Dunedin Multidisciplinary Health and Development Unit, Department of Psychology, University of Otago, Dunedin 9016, New Zealand.

* dbelsky@duke.edu, 2020 W Main St Suite 201, Durham NC 27708

H2:Abstract

A genome-wide association analysis (GWAS) of >100,000 individuals has identified molecular-genetic predictors of educational attainment. The meaning of this new GWAS finding is under debate and its ethical, legal, and social implications are unclear. We undertook an in-depth life-course investigation of the polygenic score derived from this educational-attainment GWAS, using the four-decade Dunedin Study of a birth cohort (N=918). Cohort children with higher polygenic scores tended to also come from higher social-class families, confirming that genes and environments are correlated. However, polygenic scores predicted outcomes over and above family social advantage, and even individuals born into lower social-class families showed significant upward mobility, if they had higher polygenic scores. The polygenic score predicted educational attainment (as in the original GWAS), but its predictions of other life outcomes did not depend on educational attainment, suggesting education-associated alleles influence more than responsiveness to education. In mediation analysis, the polygenic score operated through cognitive ability, but also through self-control and interpersonal skill. As young children, participants with higher polygenic scores tended to talk and read precociously, and as pupils they out-performed their peers. As adults, participants with higher polygenic scores were more likely to migrate abroad for work, became skilled in managing personal finances, and paired with better-off, better-educated mates. More education-associated alleles did not bring more life satisfaction. Measured polygenic influences were small, and are not translatable to genetic testing. Rather, the behaviors connecting DNA sequence with life outcomes may provide targets for interventions to promote population-wide positive development.

H1 MAIN TEXT

H2:Introduction

In 2013, scientists reported the first successful genome-wide association study (GWAS) of a social-science outcome, educational attainment *(1)*. Their analysis of millions of genetic variants in over 100,000 individuals hinted at the existence of a molecular map to success in schooling written in the alphabet of DNA. Rather than finding a "gene for education", this study revealed a genetic continuum: some individuals carry very few attainment-associated alleles, the bulk of the population carries some, and a few carry many. This continuum, measured as a "polygenic score" *(2)*, has since been shown to predict educational attainments in cohorts on three continents, and even differences in educational attainments between siblings in the same family *(3–7)*. Although the magnitudes of associations are small, findings have provoked controversy and concern about misuse. Controversy and concern arise partly from uncertainty about the extent to which genetic discoveries predict life outcomes beyond schooling. To provide an empirical foundation for productive public discussion of the new science of sociogenomics, this paper asks three sets of questions. Do genetic discoveries for educational attainments predict outcomes beyond schooling? If so, what are the developmental and behavioral pathways that connect DNA sequence differences with divergent life outcomes? And do psychological characteristics act as mediators of genetic associations? Although these questions may seem premature, it is important to ask them now, before technologies using genetics to predict social outcomes become possible.

These questions were addressed by examination of data prospectively collected from a population-representative birth cohort followed to midlife, the Dunedin Study (N=1,037) *(8)*. Across repeated in-person assessments, Study members were evaluated for developmental milestones in childhood; traits, behaviors, and aspirations through adolescence; and ultimately attainments and outcomes in adulthood (**Table 1**). Assessments were carried out at birth and ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and, most recently, 38 years, when 95% of the 1,007 study members still alive took part. Thus, findings illustrate genetic associations with life courses and life outcomes without bias from selective attrition due to illness or challenging life circumstances. Our analysis tested the scope, pathways, and psychological mechanisms of genetic influence on socioeconomic attainments across the first half of the life course.

H2: Results

Analysis included the 918 non-Maori Study members who provided DNA samples. Cohort members' genomes were scored according to published GWAS results for educational attainment (*(1)* see **Supplementary methods, Figure S1**; scores were standardized to have M=0, SD=1). The analysis proceeded in three parts. Part 1 examined divergent outcomes of high- and low-scoring children, first in education, and then in the acquisition of social and economic capital through midlife and the social mobility it reflected. Part 2 investigated how higher-scoring children came to grow apart from their lower-scoring peers. Analysis tested genetic differences in the timing of early-life milestones; in when children learned to read; in the decision to test for secondary education credentials and university enrollment, and performance on those tests; in geographic mobility in search of training and employment; and in selection of mates, formation of households, and forging of careers. Part 3 analyzed candidate psychological characteristics through which genetic influences on development and life outcomes might come about.

Part 1. What do discovered genetics of educational attainment mean for life outcomes beyond schooling? Analysis tested associations between Dunedin Study members' polygenic scores and their life attainments when they were aged 38 years, roughly the midpoint in the human lifespan. All analyses are adjusted for the first 10 principal components computed from genome-wide SNP data (**Supplementary methods** *(9, 10)*, **Table S1**) to adjust for potential population stratification, genome-wide patterning of allele frequency differences that might induce spurious correlations between the polygenic score and study outcomes. Unadjusted estimates are reported in the **Table S1.**

Do individuals with higher polygenic scores achieve higher degrees? In replication of the original discovery about the genetics of educational attainment, Dunedin cohort members with higher polygenic scores tended to go on to achieve higher degrees as compared to peers with lower scores (r=0.15, p<0.001, **Figure 1 Panel A**). This correlation between polygenic score and educational attainment was nearly identical to the estimate from the original report *(1)*. As in previous studies, the genetic effect was small in magnitude; e.g., having a polygenic score 1 standard deviation above the mean was associated with a 19% increase in likelihood of completing a university degree (RR=1.19, 95% CI [1.07-1.32]).

Do individuals with higher polygenic scores go on to achieve socioeconomic success beyond schooling? Adult socioeconomic attainments of Study members were measured using data from structured interviews about jobs, income, wealth, and financial difficulties, and by conducting administrative record searches of governmental and credit bureau databases. Factor analysis of these multiple measures was used to compute an Adult Attainment factor score (**Supplementary methods, Table S2, Figure S2**). By midlife, individuals with higher polygenic scores tended to be more socioeconomically successful: they held more prestigious occupations, earned higher incomes, accumulated more assets, reported fewer difficulties paying their expenses, relied less on social welfare benefits, and had higher credit scores ($r=0.13$, $p<0.001$ for the Adult Attainment factor, **Figure 1 Panel B**). Although it may seem unsurprising that a polygenic score that predicts educational attainment also continues to predict success during the years that follow after education, less than half of the genetic association was accounted for by higher educational attainments among individuals with higher polygenic scores; when we repeated our genetic analysis of the Adult Attainment factor including education as a covariate, the adjusted effect size was $r=0.07$ ($p=0.035$). (Genetic effect-sizes for the individual attainment measures and effect sizes after adjustment for educational attainment are shown in **Figure S3.**)

In sum, in the Dunedin cohort, individuals with higher polygenic scores tended to grow up to become more successful, not only in schooling, but in their economic and professional lives. This success depended only partly on their educational attainments.

Are children with higher polygenic scores more often born into socially advantaged families? Previous research estimates parent-offspring polygenic score correlations at ~$r=0.6$ *(6)*. Moreover, if a generation of individuals who achieve more occupational and economic success carry a certain genotype or set of genotypes, it stands to reason that their own children will inherit not only their genetics, but also their social success. To test this hypothesis of social stratification of genotypes, analysis compared polygenic scores for children whose parents occupied different social positions. Parents' socioeconomic status (SES) was measured from repeated assessments conducted when the cohort members were growing up, during their first 15 years of life (**Supplementary methods** *(11)*). Our findings point to a gene-environment correlation: the polygenic score for educational attainment was stratified by childhood SES such that children with higher polygenic scores tended to have grown up in higher-SES families while children with lower polygenic scores tended to have grown up in lower-SES families ($r=0.13$, $p<0.001$).

Are children with higher polygenic scores more likely to achieve upward social mobility?

Social mobility analysis tested whether the higher life attainments of children with higher polygenic scores were independent of their social origins. Social mobility analysis repeated the analysis of adult socioeconomic outcomes, this time adding a statistical control for the SES of a child's family during their first 15 years of life (**Supplementary methods**). Social mobility analysis considered three interrelated outcomes: the Study member's educational attainment, their SES measured as occupational prestige (in parallel to the status of their parents), and their score on the Adult Attainment factor. Children with higher polygenic scores tended to attain more regardless of whether when they began life in a family that was well-off or one that was socially-disadvantaged (more education, r=0.10, p=0.002; more prestigious occupations, r=0.11, p<0.001; higher Adult Attainment factor scores, r=0.11, p=0.002). **Figure 2** summarizes three findings from genetic analysis of intergenerational mobility. First, the staggered levels of the dashed horizontal lines showing mean socioeconomic attainment in each of the panels indicate substantial intergenerational continuity in attainment; how far the children were able to go in life was, to an extent, anchored by the socioeconomic level at which they started. Put another way, children born well-off rarely became poor and children born poor only rarely became well-off. Second, the box plots at the bottom of the panels show that polygenic scores were socially stratified; as noted above, children born into socially-disadvantaged families tended to have slightly below-average polygenic scores whereas children born into socially-advantaged families tended to have slightly above-average polygenic scores. Third, the parallel slopes of the red regression lines show that genes make independent, additive contributions to intergenerational mobility (see also **Figure S4**); in families with low, middle, and high social position, children with higher polygenic scores did better, on average. If they were born into socially disadvantaged families, they tended to achieve upward mobility. If they were born 'with a silver spoon,' they were more likely to hold on to their social inheritance.

Dunedin data confirmed that children with higher polygenic scores had grown up in families with more socioeconomic resources *(12)*. But the data also showed that even for children born into socially disadvantaged circumstances, higher polygenic scores predicted upward social mobility.

Part 2. How do children with higher polygenic scores grow apart from their peers? If children with higher polygenic scores do achieve higher levels of attainment in schooling and beyond, it is important to know how this comes about. The intermediate phenotypes that link DNA sequence with life

outcomes can provide clues about genetic mechanisms and can also suggest targets for interventions designed to improve children's outcomes *(13)*. The next analysis asked how children with higher polygenic scores grew apart from their peers beginning during the early school years and continuing through midlife.

Children with higher polygenic scores were more likely to say their first words at younger ages. When Study members were aged 3 years, their mothers were interviewed about how old they were when they achieved each of a series of developmental milestones. The milestones, ordered by the normative age at which they were reached, were smiling, walking, talking, feeding oneself, daytime potty training, communicating using sentences, and night time potty training (**Supplementary methods, Figure S4**). Study members with higher polygenic scores began talking earlier on average than peers with lower scores (Hazard Ratio (HR)=1.12, 95% CI [1.05-1.19], p<0.001) and were also somewhat quicker to begin communicating using sentences (HR=1.06 [1.00-1.13], p=0.052), although this difference was not statistically significant at the α=0.05 threshold. This accelerated development was restricted to verbal ability; study members with higher polygenic scores did not reach other developmental milestones ahead of peers.

Children with higher polygenic scores acquired reading skills at younger ages. Study members' reading skill was assessed with the Burt Reading Test *(14)* at each measurement age from 7-18 years. Growth-curve modeling found that already by age 7, children with higher polygenic scores were stronger readers (intercept b=2.79 SE (0.57), p<0.001) (**Supplementary methods**). Thereafter, these children improved their performance at a faster rate (linear slope b=0.25 (0.09), p=0.005) and reached their peak performance at an earlier age (quadratic slope b=-0.03 (0.01), p<0.001) (**Figure 3**). These results show that, on this educational fundamental, Study children with higher polygenic scores were often ahead of their peers already by the second grade and this gap in ability tended to expand through the middle-school years, although genetic differences were small.

Adolescents with higher polygenic scores had higher aspirations as high school students. When they were aged 15 years, Study members were asked about the highest level of education they planned to complete and also about the kind of job they hoped to have some day. At a critical developmental juncture when adolescents of this New Zealand birth cohort (1972-73) were making the choice to remain in school or to begin working, adolescents in the Dunedin cohort with higher polygenic scores aspired to higher educational attainments (r=0.15, p<0.001; for aspiration to a university degree, RR=1.24 [1.11-1.37]) and more prestigious occupations (r=0.12, p=0.001; for

aspiration to a high status "professional" occupation such as a doctor or engineer, RR=1.16 [1.06-1.27]).

Adolescents with higher polygenic scores tested at higher levels in high school. Students distinguish themselves academically by selecting into more competitive tracks and by their performance within those tracks *(15)*. At the time Dunedin Study members were in high school, New Zealand pupils sat for standardized exams in the 5th, 6th, and 7th forms (ages 15-17 years). For the 1972-73 birth cohort, the age-15 "Certificate" exam was required to earn a School-Leaving Certificate (the minimum secondary education credential at the time); the age-16 Sixth-Form Certificate was used for entry to various tertiary institutions; and the age 17 "Bursary" exam was the method through which the government allocated funds ("bursaries") to support living costs during university. Study members brought their official exam records to the research unit and their scores were recorded. Adolescents with higher polygenic scores were less likely to have left school without testing for a credential (RR=0.78 [0.66-0.93], p=0.006) and were more likely to advance to the next testing level at each age (ordered logit OR=1.32 [1.12-1.55], p=0.001). They also performed better on the tests (r=0.24 for the age-15 Certificate exam, p<0.001; r=0.19 for the age-16 Form-6 exam, p<0.001; r=0.19 for the Bursary exam, p=0.032). These findings show that adolescents with higher polygenic scores distinguished themselves from peers by more often competing at advanced academic levels and by outperforming peers on standardized tests.

Study members with higher polygenic scores were more likely to pursue occupational opportunities outside of New Zealand. Success in competitive professional environments sometimes depends on "going the extra mile." To test if Study members with higher polygenic scores did so literally, the next analysis tracked where Study members lived and worked from the time they were 21 years old through the end of follow-up using data from life history calendars *(16)* completed by the Study members at each adult assessment (**Supplementary methods**). Overseas work experience is common for New Zealanders, including Dunedin cohort members. By age 38, over a third of the Dunedin cohort (42%) had worked in a foreign country for a spell of at least 12 months. The most common destination for overseas work experience was Australia (about 41% of those who worked abroad did so in Australia but not elsewhere). Work experience in a foreign country beyond Australia has special significance in New Zealand and is known as "the Big OE" (for "Overseas Experience") *(17, 18)*. Study members with higher polygenic scores were more likely have an OE (RR=1.17 [1.05-1.32], p=0.007). Most New Zealanders who work abroad ultimately return home to raise their families. At the

time of the age-38 interviews, 18% of Study members lived and worked in Australia and an additional 7% lived and worked in another foreign country. Study members with higher polygenic scores were more likely to be among these migrants (RR=1.18 [1.05-1.32], p=0.005; as compared to those living in New Zealand, migrants to Australia had polygenic scores 0.19 [0.02-0.36] SDs higher, p=0.026, and those to other countries had polygenic scores 0.27 [0.02-0.51] SDs higher, p=0.032; **Figure 4**). These findings suggest that Study members with higher polygenic scores distinguished themselves in the labor force by more often pursuing job opportunities beyond New Zealand.

Study members with higher polygenic scores were more financially planful. At ages 32 and 38, friends and relatives who knew each Study member well reported about the Study member's ability to manage money (96% response rate). In addition, Study members were interviewed about financial building blocks (investments and retirement savings) and saving behaviors; scores on financial building blocks and savings behavior scales were averaged to calculate a Financial Planfulness score (**Supplementary Information**). Study members with higher polygenic scores were rated by their informants as having fewer difficulties managing their money (r=-0.08, p=0.013) and were more financially planful on average (r=0.09, p=0.008). These findings show that in addition to acquiring academic credentials and professional experience to command higher earnings, Study members with higher polygenic scores tended to be better managers of their financial resources.

Study members with higher polygenic scores selected partners with higher socioeconomic attainments. In addition to education, wages, and investments, so-called "marriage markets" contribute to a person's accumulation of social and financial resources *(19, 20)*. According to prior research, better-off men and women tend to pair with one another and this pattern of "homophilous" mating also occurs for the less well off *(21)*. By midlife, most Study members were in a serious relationship. Study members with higher polygenic scores were no more likely to be in a serious relationship than Study members with lower scores (RR=1.00 [0.98-1.03] p=0.776). Study members in serious relationships were interviewed about their partner's education and income. This partner information was available for 83% of the 918 Study members with genetic data (n=759). Information was used to classify partner socioeconomic status as low (31%), middle (49%), or high (20%) (**Supplementary Information**). Study members with higher polygenic scores tended to have higher-SES partners (r=0.09, p=0.011, **Figure 5**). These findings suggest that Study members with higher polygenic scores bolstered the socioeconomic advantages they accrued through their own educational and occupational attainments by partnering with socially advantaged mates.

Study members with higher polygenic scores were not more satisfied with their lives. A higher polygenic score predicted conventional indicators of success: educational achievement, occupational prestige, financial security, even securing a socioeconomically successful partner. Yet some conceptualizations of success extend beyond the realms of material and social attainment. We therefore tested if the polygenic score predicted Study members' self-rated satisfaction with life at age 38. It did not (r=0.04, p=0.189).

Genetic associations with pathways to socioeconomic success were not accounted for by study members' social origins. Because of evidence that Dunedin Study children's polygenic scores were associated with their families' socioeconomic circumstances (r=0.13, p<0.001), developmental pathway analyses were repeated with statistical adjustment for the SES of Study members' families when they were children. Genetic associations were largely independent of childhood SES. Complete results are included in **Table S3**.

Part 3. What personal characteristics help children with higher polygenic scores achieve social and economic success? The pattern of findings described above suggests that the genetics uncovered in GWAS of educational attainment contribute to certain underlying characteristics that influence not just educational success, but success in social and economic domains of life more broadly. Three possibilities were tested: higher cognitive ability, stronger non-cognitive skills, and overall better physical health.

Children with higher polygenic scores performed better on IQ tests and exhibited a more rapid pace of cognitive development during childhood. Study members completed cognitive assessments between ages 3 and 13 years (at age 3 they completed the Peabody Picture Vocabulary test *(22)*; at age 5 the Stanford-Binet test *(23)*; and thereafter at ages 7, 9, 11, and 13, the Wechsler Intelligence Scales for Children (WISC-R) *(24)*). Children with higher polygenic scores did not score significantly higher than their peers on the Peabody test at age 3 (r=0.05, p=0.133), but thereafter they showed an increasing cognitive advantage (r=0.13 for Binet IQ at age 5, r=0.13-0.19 for WISC-R IQ at ages 7-13, p<0.001 for all, **Figure 6 Panel A**).

This pattern of findings indicates genetic influence over the developmental process through which children accumulate cognitive abilities, a hypothesis suggested by previous twin research on intelligence *(25–27)*, but to our knowledge still untested in molecular data. To test hypotheses about polygenic influence on the course of cognitive development, data from repeated assessments of the

WISC-R were analyzed. Analysis focused on mental age scores, rather than IQ scores. This is because whereas IQ scores are age-corrected in order to make comparisons between a child and the population of children of the same chronological age (Sara's score is 66th percentile for her age), mental age scores express the child's level of performance as the chronological age for which his/her score is normative (although Sara is 10 years old, her mental age is 12). Mental age can be used to monitor each child's intra-individual development over time (e.g., a 10-year-old child with an unstandardized IQ score equal to the average unstandardized score for 12-year olds would have a mental age of 12) *(28)*. Growth-curve modeling tested if children with higher polygenic scores differed from peers in their cognitive development (**Supplementary methods**). Children with higher polygenic scores tended to have older mental ages at chronological age-7 baseline (intercept b=0.13 (0.04), p<0.001) and they exhibited a faster pace of cognitive development through age 13 years (slope b=0.05 (0.01), p<0.001, **Figure 6 Panel B**). Taken together, these effects mean that a child with a genetic score one standard deviation above the mean would, by the age of 13 years, accrue a roughly 6-month advantage in cognitive development relative to the population norm.

Children with higher polygenic scores had stronger non-cognitive skills. In addition to cognitive abilities, so-called non-cognitive skills influence individuals' attainments *(29)*. Genetic associations were tested with two non-cognitive skills, self-control and interpersonal skill.

As described previously *(30)*, dossiers of children's self-control skills were compiled from observational ratings, parent, and teacher reports between ages 3 and 11 years and self-reports at age 11 years. Children with higher polygenic scores tended to show better self-control skills across their first decade of life (r=0.10, p=0.001).

Children's interpersonal skill was measured from reports by trained research staff on behavioral observations of the Study members when they were ages 3, 5, 7, and 9 years *(31)*. At each age, children were given binary ratings if they impressed the staff as being friendly, confident, cooperative, and/or communicative. These ratings were used to form an Interpersonal Skill scale (**Supplementary methods**). Children with higher polygenic scores were rated as having better Interpersonal Skill (r=0.10, p=0.004).

Genetic associations with children's cognitive abilities and non-cognitive skills were independent of their social origins. Analysis of childhood psychological characteristics was repeated with statistical adjustment for the SES of the children's families. Genetic associations were independent of childhood SES. Complete results are included in the **Table S3**.

Cognitive abilities and non-cognitive skills mediated genetic influences on educational and socioeconomic attainments. Genetic associations with cognitive and non-cognitive skills suggest these characteristics could explain why children with higher polygenic scores went on to achieve higher educational and socioeconomic attainments. Mediation analysis tested if cognitive abilities and non-cognitive skills accounted for genetic associations with life attainments (**Supplementary methods, Figures S7 and S8, Table S4**). Cognitive ability, self-control, and interpersonal skill were all statistically significant mediators of genetic associations with educational and socioeconomic outcomes. Together, cognitive abilities and non-cognitive skills accounted for about 60% of the genetic association with educational attainment and about 47% of the genetic association with the Adult Attainment factor score ($p<0.001$ for both).

Children with higher polygenic scores were no healthier than their peers. Genetic associations with adult attainments might also result from general benefits to physical integrity that make individuals healthier as children, setting them up for success later in life *(32, 33)*. Dunedin Study children's health was measured from repeated clinical assessments of motor development, growth and obesity, cardiovascular and pulmonary functioning, and infections and injuries between ages 3 and 11 years (**Supplementary methods** *(34)*). Study members with higher polygenic scores were no healthier in childhood than their peers ($r=0.01$, $p=0.806$). Together with the abovementioned lack of association between the polygenic score and walking, feeding, and potty training, this suggests that GWAS of educational attainment have not identified a set of genetic influences on overall robust functioning of the body's physical systems.

As a second test of the physical robustness hypothesis, analysis considered the genetics of human height. Like education, human height is known to be related to socioeconomic attainments *(35–38)*. This analysis substituted a polygenic score derived from GWAS of human height for the education polygenic score in our analysis predicting life attainments. We used published results from large-scale GWAS of human height *(39)* to calculate height polygenic scores for Dunedin Study members. As expected, Study members' height polygenic scores were correlated with their measured stature ($r=0.54$, $p<0.001$). However, even though taller study members did tend to do better in life (for the Adult Attainment factor, $r=0.13$, $p=0.011$), we observed no association between the polygenic score for height and life attainments measured by the Adult Attainment factor ($r=0.00$, $p=0.952$).

H2: Discussion

This article describes how genetic discoveries made in genome-wide association study (GWAS) analysis of educational attainment (1) were related to the courses of human lives. We studied a population-representative birth cohort followed over 4 decades. Findings showed that genome-wide DNA-sequence differences identified from GWAS and summarized in a "polygenic score" were associated with basic processes of human social and economic success. Three points are important in interpreting the substance of these findings. First, genetic associations between the polygenic score and adult socioeconomic success were not fully accounted for by educational attainment. Second, although children's socio-economic origins were correlated with their polygenic scores, genetic associations with adult socioeconomic success, with the developmental and behavioral pathways to that success, and with the psychological characteristics we studied were mostly independent of children's socioeconomic origins. Third, across the board, effect sizes were small in magnitude.

The primary finding was that polygenic scores derived from GWAS of educational attainment predicted life outcomes well beyond schooling. Study members with higher polygenic scores were geographically mobile in search of professional opportunities; they built more successful careers; they secured higher social status mates; and they built stronger financial foundations for retirement. From childhood to midlife, Study members' genetic inheritance predicted their social mobility. Even among children born into socially disadvantaged homes, those with higher polygenic scores achieved more. Achievements of children with higher polygenic scores were enabled in part by a suite of psychological traits already evident from early life. Study members with higher polygenic scores talked earlier, did better on cognitive tests from age 5 years and showed a more rapid pace of cognitive development, and they developed better self-control and interpersonal skills. Collectively, these childhood psychological characteristics accounted for about half of the genetic association with social success in adulthood. Strikingly, the same genetic differences that predicted children's cognitive, emotional, and social functioning were not related to their attainment of non-verbal milestones or their physical health.

The substance of these findings is bolstered by evidence that GWAS discoveries for educational attainment are not genetic artifacts of a socially privileged class. Because children born into better off families are more likely to earn advanced degrees *(40)*, GWAS of educational attainment could have identified the genetics of better-off families rather than the genetics of a propensity to succeed. GWAS discoveries could be no more than markers of socially advantaged ancestry. Consistent with such a

possibility, both previous studies *(6, 7, 12)* and the current study found that children born into better off homes had higher polygenic scores. But two findings suggest that the genetic associations are non-spurious. First, studies that compare siblings within the same family (who share identical ancestries) find that the sibling with the higher polygenic score tends to complete more years of schooling *(7, 41)*. Second, our study shows that polygenic scores also influence changes in social position within a single generation, thereby suggesting a mechanism to explain the gene-environment correlation in which children of socially advantaged families tend to have higher polygenic scores.

We acknowledge limitations. Our study concerned a single, European-descent birth cohort in one country, New Zealand. The extent to which findings generalize to other birth cohorts growing up under other circumstances needs to be tested. Although New Zealand has levels of social inequality similar to the United States and Great Britain (after-tax Gini coefficient: NZ=0.33, UK= 0.34, US=0.37 *(42)*), international comparisons will prove informative. The measurement of the human genome we studied is necessarily preliminary. We studied a polygenic score based on the best available information about genetic correlates of educational success. But future GWAS with larger sample size are expected to yield a more precise set of genetic correlates. Replication checks with subsequent iterations of the polygenic score for education are needed because, although the assumption is that findings will strengthen as GWAS sample sizes grow, this is not a certainty. Follow-up of social and economic outcomes in our study is right censored, extending through the fourth decade of life, but not beyond. Extension of findings into longitudinal cohort studies of older adults is needed to clarify the extent of genetic associations into the second half of the life course. Finally, the set of outcomes, pathways, and traits we studied is not comprehensive. Studies of other samples with different measurement batteries are needed to expand our understanding of how genetic correlates of educational attainment relate to human life courses.

In light of these limitations, our study contributes to public and scientific conversation about genetic discoveries for educational attainment in five ways. First, GWAS discoveries for educational attainment are not about education only. They are discoveries about socioeconomic success more broadly (although perhaps not about satisfaction with life). Education accounted for under half of the relationship between genes and adult socioeconomic attainments, suggesting that the mechanisms of genetic influence are not limited to success in schooling and do not depend on it.

Second, the psychological mediators of genetic associations with socioeconomic success involve more than what IQ tests measure as intelligence. Multivariate twin research suggests that the

heritability of educational attainment reflects genetic influences on non-cognitive skills as well as intelligence *(43)*. We find molecular evidence to support this hypothesis; children's polygenic scores for educational attainment were correlated with their non-cognitive self-control and interpersonal skills as well as with their IQ scores. By working in a "top-down" way from an adult phenotype backward in development toward DNA sequence, these findings suggest behavioral mechanisms for genetic influences on educational attainment.

Third, children with higher polygenic scores grew apart from their peers along coherent developmental trajectories that began to form even before they entered school. Study members with higher polygenic scores began to talk at a younger age. Subsequently, they learned to read before many of their peers. This early success was followed by loftier academic aspirations and attainments extending into adulthood. These findings support the logic of interventions to promote early literacy, particularly those focusing on early language development *(44)*.

In addition, and more speculatively, the life-course analysis reported here also suggests that GWAS findings for educational attainment may provide a clue to the genetic roots of life-history differences in free-living humans. Unlike education, which is a relatively modern human experience, patterns of migration, mate selection, and resource acquisition and management are ancient human behaviors that plausibly bear the imprint of our species' evolutionary history. The finding that GWAS discoveries for education predict these ancient behaviors suggests a window into genetic regulation of humans' strategies to survive and reproduce. Our data cannot test if frequencies of education-associated genotypes reflect some Darwinian fitness strategy. Rather, the data suggest that individuals whose genomes carry more education-associated alleles are forging life histories that achieve success in the modern world and the pathways to this success include some that would be familiar to our ancestors.

Fourth, findings lend molecular weight to earlier twin-study observations that genes shape not just behavior, but environmental facts on the ground that contextualize and constrain behavioral choices *(45)*. The molecular realization of such gene-environment correlations creates opportunities for social theory and research. Results reported in this study suggest that by incorporating DNA sequence into studies of status attainment, migration, assortative mating, and financial behavior, social scientists may be able to frame novel "sociogenomic" research questions. For example, do public programs to build human capital (like improving teacher salaries or providing universal access to pre-kindergarten education) change the ways in which genes influence life attainments? If so, are the

returns greater for programs that magnify genetic influences or programs that reduce them? Do the genetics of educational attainment relate to social gradients in midlife health and aging? If so, how is this process shaped by health-care costs, quality, and access? As concerns about economic inequality increase, are genes linked with socioeconomic success becoming concentrated within social and geospatial elites? If so, is this process influenced by exogenous shocks such as natural disasters, policy shifts such as multinational trade and border agreements, or cultural changes in equality of opportunity?

Finally, findings shed light on the stakes of the public conversation that is now emerging about sociogenomic discoveries. The significance for the general public of new knowledge about how to measure and interpret DNA sequence is uncertain and hotly debated, even in the field of biomedicine, where clinical applications of genetic discoveries are already possible *(46–48)*. At present, genetic prediction of educational outcomes and life success in general is far from sensitive or specific enough to recommend any translational application. Although there is movement to improve the predictive power of polygenic scores through increased GWAS sample sizes and improved genomic measurements, a precision medicine-type approach to human capital development remains well out of reach. And yet, debate is already underway about the possibility for genetic testing to someday be used in forecasting human potential. Policy action may be needed to regulate the ethical use of genomic information in school admissions and tracking decisions, and such actions should be informed by realistic estimates of the magnitude of genetic effects.

H2: Materials and Methods

A more detailed description of study measures, design, and analysis is provided in the **Supplementary methods**.

Sample. Participants are members of the Dunedin Multidisciplinary Health and Development Study, which tracks the development of 1,037 individuals born in 1972–1973 in Dunedin, New Zealand.

Measuring polygenic scores. We used Illumina HumanOmni Express 12v1.1 BeadChip arrays (Illumina CA, USA) to assay common Single Nucleotide Polymorphism (SNP) variation in the genomes of our cohort members. We imputed additional SNPs using the impute2 software (version 2.3.1, https://mathgen.stats.ox.ac.uk/impute/impute_v2.html, *(49)*) and 1000 Genomes version 3 reference panel. We calculated polygenic scores according to the method described by Dudbridge *(50)* using the PRsice software (v1.22, http://prsice.info/, *(51)*). To account for potential population stratification, we

adjusted polygenic score analyses for the first ten principal components computed from the genome-wide SNP data using the EIGENSOFT smartPCA tool *(9, 10)*.

Measuring social-class origins. The socioeconomic statuses of Study members' families was averaged across repeated assessments of the higher of either parent's occupational statuses throughout the Study members' childhoods *(11)*.

Measuring Attainment. We measured educational attainment as the highest degree a Study member had completed through the time of the age-38 assessment. We measured attainment beyond education from Study members' reports of their income, assets, credit problems, and difficulties paying expenses when they were aged 38 years.

Measuring Pathways to Success. We measured the age at which Study members achieved early developmental milestones from interviews with their mothers when the Study members were aged 3 years. We measured reading ability from Burt Reading Test *(14)* scores at ages 7-18 years. We measured educational and socioeconomic aspirations from surveys completed by the Study members at age 15 years. We measured academic performance from scores on standardized tests taken at ages 15-18 years. We measured geographic mobility from Study member Life History Calendar *(16)* reports about place of work and residence from age 21-38 years. We measured financial planfulness from surveys of Study members' friends and relatives and structured interviews with the Study members themselves when they were ages 32 and 38 years. We measured the socioeconomic status of Study members' romantic partners from Study member reports on their partner's income and education in structured interviews conducted at age 38 years.

Measuring life satisfaction. When they were aged 38 years, Study members completed the 5-item Satisfaction with Life Scale *(52)* (e.g., In most ways my life is close to ideal, So far I have gotten the important things I want in life).

Measuring traits and abilities. We measured cognitive ability and cognitive development using the Peabody Picture Vocabulary Test *(22)* at age 3 years, the Stanford Binet IQ Test at age 5 years *(23)*, and the Wechsler Intelligence Scales for Children at ages 7-13 years *(24)*. We measured Study members' childhood self control skills from observational ratings of their lack of control (ages 3 and 5 years) and parent, teacher, and self-reports of impulsive aggression, hyperactivity, lack of persistence, inattention, and impulsivity (ages 5-11 years) *(30)*. We measured Study members' childhood interpersonal skill from reports made by trained research workers following standardized testing

sessions at ages 3-9 years. We measured childhood health from medical exams, anthropometry, lung function testing, and interviews with parents at assessments spanning birth to age 11 years.

Measuring Height. Study members' height at age 38 was measured to the nearest millimeter using a stadiometer (Harpenden; Holtain, Ltd.).

H2: References and Notes

1. C. a Rietveld, S. E. Medland, J. Derringer, J. Yang, T. Esko, N. W. Martin, H.-J. Westra, K. Shakhbazov, A. Abdellaoui, A. Agrawal, E. Albrecht, B. Z. Alizadeh, N. Amin, J. Barnard, S. E. Baumeister, K. S. Benke, L. F. Bielak, J. a Boatman, P. a Boyle, G. Davies, C. de Leeuw, N. Eklund, D. S. Evans, R. Ferhmann, K. Fischer, C. Gieger, H. kon K. Gjessing, S. Hägg, J. R. Harris, C. Hayward, C. Holzapfel, C. a Ibrahim-Verbaas, E. Ingelsson, B. Jacobsson, P. K. Joshi, A. Jugessur, M. Kaakinen, S. Kanoni, J. Karjalainen, I. Kolcic, K. Kristiansson, Z. Kutalik, J. Lahti, S. H. Lee, P. Lin, P. a Lind, Y. Liu, K. Lohman, M. Loitfelder, G. McMahon, P. M. Vidal, O. Meirelles, L. Milani, R. Myhre, M.-L. Nuotio, C. J. Oldmeadow, K. E. Petrovic, W. J. Peyrot, O. Polasek, L. Quaye, E. Reinmaa, J. P. Rice, T. S. Rizzi, H. Schmidt, R. Schmidt, A. V. Smith, J. a Smith, T. Tanaka, A. Terracciano, M. J. H. M. van der Loos, V. Vitart, H. Völzke, J. Wellmann, L. Yu, W. Zhao, J. Allik, J. R. Attia, S. Bandinelli, F. Bastardot, J. Beauchamp, D. a Bennett, K. Berger, L. J. Bierut, D. I. Boomsma, U. Bültmann, H. Campbell, C. F. Chabris, L. Cherkas, M. K. Chung, F. Cucca, M. de Andrade, P. L. De Jager, J.-E. De Neve, I. J. Deary, G. V. Dedoussis, P. Deloukas, M. Dimitriou, G. Eiríksdóttir, M. F. Elderson, J. G. Eriksson, D. M. Evans, J. D. Faul, L. Ferrucci, M. E. Garcia, H. Grönberg, V. Guðnason, P. Hall, J. M. Harris, T. B. Harris, N. D. Hastie, A. C. Heath, D. G. Hernandez, W. Hoffmann, A. Hofman, R. Holle, E. G. Holliday, J.-J. Hottenga, W. G. Iacono, T. Illig, M.-R. Järvelin, M. Kähönen, J. Kaprio, R. M. Kirkpatrick, M. Kowgier, A. Latvala, L. J. Launer, D. a Lawlor, T. Lehtimäki, J. Li, P. Lichtenstein, P. Lichtner, D. C. Liewald, P. a Madden, P. K. E. Magnusson, T. E. Mäkinen, M. Masala, M. McGue, A. Metspalu, A. Mielck, M. B. Miller, G. W. Montgomery, S. Mukherjee, D. R. Nyholt, B. a Oostra, L. J. Palmer, A. Palotie, B. W. J. H. Penninx, M. Perola, P. a Peyser, M. Preisig, K. Räikkönen, O. T. Raitakari, A. Realo, S. M. Ring, S. Ripatti, F. Rivadeneira, I. Rudan, A. Rustichini, V. Salomaa, A.-P. Sarin, D. Schlessinger, R. J. Scott, H. Snieder, B. St Pourcain, J. M. Starr, J. H. Sul, I. Surakka, R. Svento, A. Teumer, H. Tiemeier, F. J. a van Rooij, D. R. Van Wagoner, E. Vartiainen, J. Viikari, P. Vollenweider, J. M. Vonk, G. Waeber, D. R. Weir, H.-E. Wichmann, E. Widen, G. Willemsen, J. F. Wilson, A. F. Wright, D. Conley, G. Davey-Smith, L. Franke, P. J. F. Groenen, A. Hofman, M. Johannesson, S. L. R. Kardia, R. F. Krueger, D. Laibson, N. G. Martin, M. N. Meyer, D. Posthuma, a R. Thurik, N. J. Timpson, A. G. Uitterlinden, C. M. van Duijn, P. M. Visscher, D. J. Benjamin, D. Cesarini, P. D. Koellinger, GWAS of 126,559 individuals identifies genetic variants associated with educational attainment., *Science* **340**, 1467–71 (2013).

2. C. F. Chabris, J. J. Lee, D. Cesarini, D. J. Benjamin, D. I. Laibson, The Fourth Law of Behavior Genetics, *Curr. Dir. Psychol. Sci.* **24**, 304–312 (2015).

3. C. A. Rietveld, T. Esko, G. Davies, T. H. Pers, P. Turley, B. Benyamin, C. F. Chabris, V. Emilsson, A. D. Johnson, J. J. Lee, C. de Leeuw, R. E. Marioni, S. E. Medland, M. B. Miller, O. Rostapshova, S. J. van der Lee, A. A. E. Vinkhuyzen, N. Amin, D. Conley, J. Derringer, C. M. van Duijn, R. Fehrmann, L. Franke, E. L. Glaeser, N. K. Hansell, C. Hayward, W. G. Iacono, C. Ibrahim-Verbaas, V. Jaddoe, J. Karjalainen, D. Laibson, P. Lichtenstein, D. C. Liewald, P. K. E. Magnusson, N. G. Martin, M. McGue, G. McMahon, N. L. Pedersen, S. Pinker, D. J. Porteous, D. Posthuma, F. Rivadeneira, B. H. Smith, J. M. Starr, H. Tiemeier, N. J. Timpson, M. Trzaskowski, A. G. Uitterlinden, F. C. Verhulst, M. E. Ward, M. J. Wright, G. Davey Smith, I. J. Deary, M. Johannesson, R. Plomin, P. M. Visscher, D. J. Benjamin, D. Cesarini, P. D. Koellinger, Common genetic variants associated with cognitive performance identified using the proxy-phenotype method, *Proc. Natl. Acad. Sci. U. S. A.* **111**, 13790–13794 (2014).

4. M. E. Ward, G. McMahon, B. St Pourcain, D. M. Evans, C. A. Rietveld, D. J. Benjamin, P. D. Koellinger, D. Cesarini, Social Science Genetic Association Consortium, G. Davey Smith, N. J. Timpson, Genetic variation associated with differential educational attainment in adults has anticipated associations with school performance in children, *PloS One* **9**, e100248 (2014).

5. E. L. de Zeeuw, C. E. M. van Beijsterveldt, T. J. Glasner, M. Bartels, E. A. Ehli, G. E. Davies, J. J. Hudziak, Social Science Genetic Association Consortium, C. A. Rietveld, M. M. Groen-Blokhuis, J. J. Hottenga, E. J. C. de Geus, D. I.

Boomsma, Polygenic scores associated with educational attainment in adults predict educational achievement and ADHD symptoms in children, *Am. J. Med. Genet. Part B Neuropsychiatr. Genet. Off. Publ. Int. Soc. Psychiatr. Genet.* **165B**, 510–520 (2014).

6. D. Conley, B. Domingue, D. Cesarini, C. T. Dawes, C. A. Rietveld, J. Boardman, Is the Effect of Parental Education on Offspring Biased or Moderated by Genotype?, *Sociol. Sci.* **2**, 82–105 (2015).

7. B. W. Domingue, D. W. Belsky, D. Conley, K. M. Harris, J. D. Boardman, Polygenic Influence on Educational Attainment, *AERA Open* **1**, 2332858415599972 (2015).

8. R. Poulton, T. E. Moffitt, P. A. Silva, The Dunedin Multidisciplinary Health and Development Study: overview of the first 40 years, with an eye to the future, *Soc. Psychiatry Psychiatr. Epidemiol.* **50**, 679–693 (2015).

9. A. L. Price, N. J. Patterson, R. M. Plenge, M. E. Weinblatt, N. A. Shadick, D. Reich, Principal components analysis corrects for stratification in genome-wide association studies, *Nat. Genet.* **38**, 904–909 (2006).

10. N. Patterson, A. L. Price, D. Reich, Population structure and eigenanalysis, *PLoS Genet.* **2**, e190 (2006).

11. R. Poulton, A. Caspi, B. J. Milne, W. M. Thomson, A. Taylor, M. R. Sears, T. E. Moffitt, Association between children's experience of socioeconomic disadvantage and adult health: a life-course study., *Lancet* **360**, 1640–1645 (2002).

12. E. Krapohl, R. Plomin, Genetic link between family socioeconomic status and children's educational achievement estimated from genome-wide SNPs, *Mol. Psychiatry* (2015), doi:10.1038/mp.2015.2.

13. D. W. Belsky, T. E. Moffitt, A. Caspi, Genetics in population health science: strategies and opportunities, *Am. J. Public Health* **103 Suppl 1**, S73–83 (2013).

14. Scottish Council for Research in Education, The Burt Word Reading Test (1976).

15. K. L. Alexander, M. Cook, E. L. McDill, Curriculum Tracking and Educational Stratification: Some Further Evidence, *Am. Sociol. Rev.* **43**, 47–66 (1978).

16. A. Caspi, T. E. Moffitt, A. Thornton, D. Freedman, J. W. Amell, H. Harrington, J. Smeijers, P. A. Silva, The life history calendar: A research and clinical assessment method for collecting retrospective event-history data, *Int. J. Methods Psychiatr. Res.* **6**, 101–114 (1996).

17. Overseas experience*Wikipedia Free Encycl.* (2014) (available at http://en.wikipedia.org/w/index.php?title=Overseas_experience&oldid=633298303).

18. B. J. Milne, R. Poulton, A. Caspi, T. E. Moffitt, Brain drain or OE? Characteristics of young New Zealanders who leave, *N. Z. Med. J.* **114**, 450–453 (2001).

19. R. Breen, L. Salazar, Educational Assortative Mating and Earnings Inequality in the United States, *Am. J. Sociol.* **117**, 808–843 (2011).

20. M. Nick, P. R. Walsh, Building the Family Nest: Premarital Investments, Marriage Markets, and Spousal Allocations, *Rev. Econ. Stud.* **74**, 507–535 (2007).

21. C. R. Schwartz, Trends and Variation in Assortative Mating: Causes and Consequences, *Annu. Rev. Sociol.* **39**, 451–470 (2013).

22. L. Dunn, *The Peabody Picture Vocabulary Test* (American Guidance Service, Minneapolis, 1965).

23. L. M. Terman, M. A. Merrill, *Stanford-Binet Intelligence Scale: Manual for the third revision, Form L-M* (Houghton Mifflin, Oxford, England, 1960).

24. D. Wechsler, *Wechsler Intelligence Scale for Children* (Harcourt Assessment, San Antonio, TX, 4th (UK Version)., 2003).

25. R. Plomin, Genetics: How intelligence changes with age, *Nature* **482**, 165–166 (2012).

26. I. J. Deary, J. Yang, G. Davies, S. E. Harris, A. Tenesa, D. Liewald, M. Luciano, L. M. Lopez, A. J. Gow, J. Corley, P. Redmond, H. C. Fox, S. J. Rowe, P. Haggarty, G. McNeill, M. E. Goddard, D. J. Porteous, L. J. Whalley, J. M. Starr, P. M. Visscher, Genetic contributions to stability and change in intelligence from childhood to old age, *Nature* **482**, 212–215 (2012).

27. M. Trzaskowski, J. Yang, P. M. Visscher, R. Plomin, DNA evidence for strong genetic stability and increasing heritability of intelligence from age 7 to 12, *Mol. Psychiatry* **19**, 380–384 (2014).

28. Lezak, DM, Howieson, DB, Loring, DW, Hannay, HJ, Fischer, JS, *Neuropsychological Assessment* (Oxford University Press, ed. 4, 2004).

29. J. J. Heckman, Skill formation and the economics of investing in disadvantaged children, *Science* **312**, 1900–1902 (2006).

30. T. E. Moffitt, L. Arseneault, D. Belsky, N. Dickson, R. J. Hancox, H. Harrington, R. Houts, R. Poulton, B. W. Roberts, S. Ross, M. R. Sears, W. M. Thomson, A. Caspi, A gradient of childhood self-control predicts health, wealth, and public safety, *Proc. Natl. Acad. Sci.* **108**, 2693–8 (2011).

31. A. Caspi, B. Henry, R. O. McGee, T. E. Moffitt, P. A. Silva, Temperamental origins of child and adolescent behavior problems: from age 3 to age 15, *Child Dev.* **66**, 55–68 (1995).

32. A. Case, A. Fertig, C. Paxson, The lasting impact of childhood health and circumstance, *J. Health Econ.* **24**, 365–389 (2005).

33. J. Currie, *Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development* (National Bureau of Economic Research, 2008; http://www.nber.org/papers/w13987).

34. D. W. Belsky, A. Caspi, S. Israel, J. A. Blumenthal, R. Poulton, T. E. Moffitt, Cardiorespiratory fitness and cognitive function in midlife: Neuroprotection or neuroselection?, *Ann. Neurol.* **77**, 607–617 (2015).

35. T. A. Judge, D. M. Cable, The Effect of Physical Height on Workplace Success and Income: Preliminary Test of a Theoretical Model, *J. Appl. Psychol.* **89**, 428–441 (2004).

36. P. Lundborg, P. Nystedt, D.-O. Rooth, Height and Earnings: The Role of Cognitive and Noncognitive Skills, *J. Hum. Resour.* **49**, 141–166 (2014).

37. E. Lindqvist, Height and Leadership, *Rev. Econ. Stat.* **94**, 1191–1196 (2011).

38. A. Case, C. Paxson, Stature and status: Height, ability, and labor market outcomes, *J. Polit. Econ.* **116**, 499–532 (2008).

39. A. R. Wood, T. Esko, J. Yang, S. Vedantam, T. H. Pers, S. Gustafsson, A. Y. Chu, K. Estrada, J. 'an Luan, Z. Kutalik, N. Amin, M. L. Buchkovich, D. C. Croteau-Chonka, F. R. Day, Y. Duan, T. Fall, R. Fehrmann, T. Ferreira, A. U. Jackson, J. Karjalainen, K. S. Lo, A. E. Locke, R. Mägi, E. Mihailov, E. Porcu, J. C. Randall, A. Scherag, A. A. E. Vinkhuyzen, H.-J. Westra, T. W. Winkler, T. Workalemahu, J. H. Zhao, D. Absher, E. Albrecht, D. Anderson, J. Baron, M. Beekman, A. Demirkan, G. B. Ehret, B. Feenstra, M. F. Feitosa, K. Fischer, R. M. Fraser, A. Goel, J. Gong, A. E. Justice, S. Kanoni, M. E. Kleber, K. Kristiansson, U. Lim, V. Lotay, J. C. Lui, M. Mangino, I. Mateo Leach, C. Medina-Gomez, M. A. Nalls, D. R. Nyholt, C. D. Palmer, D. Pasko, S. Pechlivanis, I. Prokopenko, J. S. Ried, S. Ripke, D. Shungin, A. Stancáková, R. J. Strawbridge, Y. J. Sung, T. Tanaka, A. Teumer, S. Trompet, S. W. van der Laan, J. van Setten, J. V. Van Vliet-Ostaptchouk, Z. Wang, L. Yengo, W. Zhang, U. Afzal, J. Arnlöv, G. M. Arscott, S. Bandinelli, A. Barrett, C. Bellis, A. J. Bennett, C. Berne, M. Blüher, J. L. Bolton, Y. Böttcher, H. A. Boyd, M. Bruinenberg, B. M. Buckley, S. Buyske, I. H. Caspersen, P. S. Chines, R. Clarke, S. Claudi-Boehm, M. Cooper, E. W. Daw, P. A. De Jong, J. Deelen, G. Delgado, J. C. Denny, R. Dhonukshe-Rutten, M. Dimitriou, A. S. F. Doney, M. Dörr, N. Eklund, E. Eury, L. Folkersen, M. E. Garcia, F. Geller, V. Giedraitis, A. S. Go, H. Grallert, T. B. Grammer, J. Gräßler, H. Grönberg, L. C. P. G. M. de Groot, C. J. Groves, J. Haessler, P. Hall, T. Haller, G. Hallmans, A. Hannemann, C. A. Hartman, M. Hassinen, C. Hayward, N. L. Heard-Costa, Q. Helmer, G. Hemani, A. K. Henders, H. L. Hillege, M. A. Hlatky, W. Hoffmann, P. Hoffmann, O. Holmen, J. J. Houwing-Duistermaat, T. Illig, A. Isaacs, A. L. James, J. Jeff, B. Johansen, Å. Johansson, J. Jolley, T. Juliusdottir, J. Junttila, A. N. Kho, L. Kinnunen, N. Klopp, T. Kocher, W. Kratzer, P. Lichtner, L. Lind, J. Lindström, S. Lobbens, M. Lorentzon, Y. Lu, V. Lyssenko, P. K. E.

Magnusson, A. Mahajan, M. Maillard, W. L. McArdle, C. A. McKenzie, S. McLachlan, P. J. McLaren, C. Menni, S. Merger, L. Milani, A. Moayyeri, K. L. Monda, M. A. Morken, G. Müller, M. Müller-Nurasyid, A. W. Musk, N. Narisu, M. Nauck, I. M. Nolte, M. M. Nöthen, L. Oozageer, S. Pilz, N. W. Rayner, F. Renstrom, N. R. Robertson, L. M. Rose, R. Roussel, S. Sanna, H. Scharnagl, S. Scholtens, F. R. Schumacher, H. Schunkert, R. A. Scott, J. Sehmi, T. Seufferlein, J. Shi, K. Silventoinen, J. H. Smit, A. V. Smith, J. Smolonska, A. V. Stanton, K. Stirrups, D. J. Stott, H. M. Stringham, J. Sundström, M. A. Swertz, A.-C. Syvänen, B. O. Tayo, G. Thorleifsson, J. P. Tyrer, S. van Dijk, N. M. van Schoor, N. van der Velde, D. van Heemst, F. V. A. van Oort, S. H. Vermeulen, N. Verweij, J. M. Vonk, L. L. Waite, M. Waldenberger, R. Wennauer, L. R. Wilkens, C. Willenborg, T. Wilsgaard, M. K. Wojczynski, A. Wong, A. F. Wright, Q. Zhang, D. Arveiler, S. J. L. Bakker, J. Beilby, R. N. Bergman, S. Bergmann, R. Biffar, J. Blangero, D. I. Boomsma, S. R. Bornstein, P. Bovet, P. Brambilla, M. J. Brown, H. Campbell, M. J. Caulfield, A. Chakravarti, R. Collins, F. S. Collins, D. C. Crawford, L. A. Cupples, J. Danesh, U. de Faire, H. M. den Ruijter, R. Erbel, J. Erdmann, J. G. Eriksson, M. Farrall, E. Ferrannini, J. Ferrières, I. Ford, N. G. Forouhi, T. Forrester, R. T. Gansevoort, P. V. Gejman, C. Gieger, A. Golay, O. Gottesman, V. Gudnason, U. Gyllensten, D. W. Haas, A. S. Hall, T. B. Harris, A. T. Hattersley, A. C. Heath, C. Hengstenberg, A. A. Hicks, L. A. Hindorff, A. D. Hingorani, A. Hofman, G. K. Hovingh, S. E. Humphries, S. C. Hunt, E. Hypponen, K. B. Jacobs, M.-R. Jarvelin, P. Jousilahti, A. M. Jula, J. Kaprio, J. J. P. Kastelein, M. Kayser, F. Kee, S. M. Keinanen-Kiukaanniemi, L. A. Kiemeney, J. S. Kooner, C. Kooperberg, S. Koskinen, P. Kovacs, A. T. Kraja, M. Kumari, J. Kuusisto, T. A. Lakka, C. Langenberg, L. Le Marchand, T. Lehtimäki, S. Lupoli, P. A. F. Madden, S. Männistö, P. Manunta, A. Marette, T. C. Matise, B. McKnight, T. Meitinger, F. L. Moll, G. W. Montgomery, A. D. Morris, A. P. Morris, J. C. Murray, M. Nelis, C. Ohlsson, A. J. Oldehinkel, K. K. Ong, W. H. Ouwehand, G. Pasterkamp, A. Peters, P. P. Pramstaller, J. F. Price, L. Qi, O. T. Raitakari, T. Rankinen, D. C. Rao, T. K. Rice, M. Ritchie, I. Rudan, V. Salomaa, N. J. Samani, J. Saramies, M. A. Sarzynski, P. E. H. Schwarz, S. Sebert, P. Sever, A. R. Shuldiner, J. Sinisalo, V. Steinthorsdottir, R. P. Stolk, J.-C. Tardif, A. Tönjes, A. Tremblay, E. Tremoli, J. Virtamo, M.-C. Vohl, Electronic Medical Records and Genomics (eMEMERGEGE) Consortium, MIGen Consortium, PAGEGE Consortium, LifeLines Cohort Study, P. Amouyel, F. W. Asselbergs, T. L. Assimes, M. Bochud, B. O. Boehm, E. Boerwinkle, E. P. Bottinger, C. Bouchard, S. Cauchi, J. C. Chambers, S. J. Chanock, R. S. Cooper, P. I. W. de Bakker, G. Dedoussis, L. Ferrucci, P. W. Franks, P. Froguel, L. C. Groop, C. A. Haiman, A. Hamsten, M. G. Hayes, J. Hui, D. J. Hunter, K. Hveem, J. W. Jukema, R. C. Kaplan, M. Kivimaki, D. Kuh, M. Laakso, Y. Liu, N. G. Martin, W. März, M. Melbye, S. Moebus, P. B. Munroe, I. Njølstad, B. A. Oostra, C. N. A. Palmer, N. L. Pedersen, M. Perola, L. Pérusse, U. Peters, J. E. Powell, C. Power, T. Quertermous, R. Rauramaa, E. Reinmaa, P. M. Ridker, F. Rivadeneira, J. I. Rotter, T. E. Saaristo, D. Saleheen, D. Schlessinger, P. E. Slagboom, H. Snieder, T. D. Spector, K. Strauch, M. Stumvoll, J. Tuomilehto, M. Uusitupa, P. van der Harst, H. Völzke, M. Walker, N. J. Wareham, H. Watkins, H.-E. Wichmann, J. F. Wilson, P. Zanen, P. Deloukas, I. M. Heid, C. M. Lindgren, K. L. Mohlke, E. K. Speliotes, U. Thorsteinsdottir, I. Barroso, C. S. Fox, K. E. North, D. P. Strachan, J. S. Beckmann, S. I. Berndt, M. Boehnke, I. B. Borecki, M. I. McCarthy, A. Metspalu, K. Stefansson, A. G. Uitterlinden, C. M. van Duijn, L. Franke, C. J. Willer, A. L. Price, G. Lettre, R. J. F. Loos, M. N. Weedon, E. Ingelsson, J. R. O'Connell, G. R. Abecasis, D. I. Chasman, M. E. Goddard, P. M. Visscher, J. N. Hirschhorn, T. M. Frayling, Defining the role of common variation in the genomic and biological architecture of adult human height, *Nat. Genet.* **46**, 1173–1186 (2014).

40. R. Breen, J. O. Jonsson, Inequality of Opportunity in Comparative Perspective: Recent Research on Educational Attainment and Social Mobility, *Annu. Rev. Sociol.* **31**, 223–243 (2005).

41. C. A. Rietveld, D. Conley, N. Eriksson, T. Esko, S. E. Medland, A. A. E. Vinkhuyzen, J. Yang, J. D. Boardman, C. F. Chabris, C. T. Dawes, B. W. Domingue, D. A. Hinds, M. Johannesson, A. K. Kiefer, D. Laibson, P. K. E. Magnusson, J. L. Mountain, S. Oskarsson, O. Rostapshova, A. Teumer, J. Y. Tung, P. M. Visscher, D. J. Benjamin, D. Cesarini, P. D. Koellinger, Social Science Genetics Association Consortium, Replicability and robustness of genome-wide-association studies for behavioral traits, *Psychol. Sci.* **25**, 1975–1986 (2014).

42. List of countries by income equality*Wikipedia Free Encycl.* (2015) (available at https://en.wikipedia.org/w/index.php?title=List_of_countries_by_income_equality&oldid=686250962).

43. E. Krapohl, K. Rimfeld, N. G. Shakeshaft, M. Trzaskowski, A. McMillan, J.-B. Pingault, K. Asbury, N. Harlaar, Y. Kovas, P. S. Dale, R. Plomin, The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence, *Proc. Natl. Acad. Sci. U. S. A.* **111**, 15273–15278 (2014).

44. M. Talbot, The talking cure*New Yorker* (2015) (available at http://www.newyorker.com/magazine/2015/01/12/talking-cure).

45. R. Plomin, C. S. Bergeman, The nature of nurture: Genetic influence on "environmental" measures, *Behav. Brain Sci.* **14**, 414–427 (1991).

46. M. J. Khoury, J. P. Evans, A public health perspective on a national precision medicine cohort: balancing long-term knowledge generation with early health benefit, *JAMA* **313**, 2117–2118 (2015).

47. E. S. Lander, Cutting the Gordian helix--regulating genomic testing in the era of precision medicine, *N. Engl. J. Med.* **372**, 1185–1186 (2015).

48. N. J. Roberts, J. T. Vogelstein, G. Parmigiani, K. W. Kinzler, B. Vogelstein, V. E. Velculescu, The predictive capacity of personal genome sequencing., *Sci. Transl. Med.* **4**, 133ra58 (2012).

49. B. N. Howie, P. Donnelly, J. Marchini, A flexible and accurate genotype imputation method for the next generation of genome-wide association studies, *Plos Genet.* **5**, e1000529 (2009).

50. F. Dudbridge, Power and predictive accuracy of polygenic risk scores., *PLoS Genet.* **9**, e1003348 (2013).

51. J. Euesden, C. M. Lewis, P. F. O'Reilly, PRSice: Polygenic Risk Score software, *Bioinforma. Oxf. Engl.* **31**, 1466–1468 (2015).

52. W. Pavot, E. Diener, Review of the Satisfaction With Life Scale, *Psychol. Assess.* **5**, 164–172 (1993).

53. T. E. Moffitt, A. Caspi, A. R. Harkness, P. A. Silva, The natural history of change in intellectual performance: who changes? How much? Is it meaningful?, *J. Child Psychol. Psychiatry* **34**, 455–506 (1993).

54. B. J. Milne, U. Byun, A. Lee, *New Zealand socio-economic index 2006.* (Statistics New Zealand, Wellington, NZ, 2013).

55. S. Israel, A. Caspi, D. W. Belsky, H. Harrington, S. Hogan, R. Houts, S. Ramrakha, S. Sanders, R. Poulton, T. E. Moffitt, Credit scores, cardiovascular disease risk, and human capital, *Proc. Natl. Acad. Sci.* , 201409794 (2014).

56. S. J. Goldman-Mellor, A. Caspi, H. Harrington, S. Hogan, S. Nada-Raja, R. Poulton, T. E. Moffitt, Suicide attempt in young people: a signal for long-term health care and social needs, *JAMA Psychiatry* **71**, 119–127 (2014).

57. M. Rutter, A. Caspi, D. Fergusson, L. J. Horwood, R. Goodman, B. Maughan, T. E. Moffitt, H. Meltzer, J. Carroll, Sex differences in developmental reading disability: new findings from 4 epidemiological studies, *JAMA* **291**, 2007–2012 (2004).

58. P. A. Silva, *4000 Otago Teenagers: A Preliminary Report from the Pathways to Employment Project* (The Dunedin Multidisciplinary Health and Development Research Unit, Dunedin, NZ, 1987).

59. L. K. Muthen, B. O. Muthen, *MPLUS* (L.K. Muthen & B.O. Muthen, Los Angeles, 1998).

60. J. D. Singer, J. B. Willett, *Applied Longitudinal Data Analysis* (Oxford University Press, New York, 2003).

61. W. B. Elley, I. C. Irving, A socio-economic index for New Zealand based on levels of education and income from the 1966 census, *N. Z. J. Educ. Stud.* **7**, 153–167 (1972).

62. T. E. Moffitt, A. Caspi, H. Harrington, B. J. Milne, Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years, *Dev. Psychopathol.* **14**, 179–207 (2002).

63. A. Furnham, M.-P. Goletto-Tankel, Understanding Savings, Pensions and Life Assurance in 16-21-Year-Olds, *Hum. Relat.* **55**, 603–628 (2002).

64. Statistics New Zealand, *New Zealand Income Survey: June 2014 quarter* (Statistics New Zealand; http://www.stats.govt.nz/browse_for_stats/income-and-work/Income/NZIncomeSurvey_HOTPJun14qtr/Tables.aspx).

65. N. Bayley, *The Bayley Scale of Infant Development* (Psychological Corp, New York, NY, 1969).

66. D. McCarthy, *Scales of Children's Abilities.* (Psychological Corp, New York, NY, 1972).

67. D. Arnheim, S. W. Sinclair, *The Clumsy Child* (VC Mosby Co, St Louis Mo, 1974).

68. M. Cannon, A. Caspi, T. E. Moffitt, H. Harrington, A. Taylor, R. M. Murray, R. Poulton, Evidence for early-childhood, pan-developmental impairment specific to schizophreniform disorder - Results from a longitudinal birth cohort, *Arch. Gen. Psychiatry* **59**, 449–456 (2002).

69. D. W. Belsky, T. E. Moffitt, R. Houts, G. G. Bennett, A. K. Biddle, J. A. Blumenthal, J. P. Evans, H. Harrington, K. Sugden, B. Williams, R. Poulton, A. Caspi, Polygenic Risk, Rapid Childhood Growth, and the Development of Obesity: Evidence from a 4-Decade Longitudinal Study, *Arch. Pediatr. Adolesc. Med.* **166**, 515–521 (2012).

70. S. Williams, R. Poulton, Birth Size, Growth, and Blood Pressure between the Ages of 7 and 26 Years: Failure to Support the Fetal Origins Hypothesis, *Am. J. Epidemiol.* **155**, 849–852 (2002).

71. M. R. Sears, J. M. Greene, A. R. Willan, E. M. Wiecek, D. R. Taylor, E. M. Flannery, J. O. Cowan, G. P. Herbison, P. a Silva, R. Poulton, A longitudinal, population-based, cohort study of childhood asthma followed to adulthood., *N. Engl. J. Med.* **349**, 1414–22 (2003).

72. R. M. Baron, D. A. Kenny, The Moderator Mediator Variable Distinction in Social Psychological-Research - Conceptual, Strategic, and Statistical Considerations, *J. Pers. Soc. Psychol.* **51**, 1173–1182 (1986).

73. K. J. Preacher, A. F. Hayes, Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models, *Behav Res Methods* **40**, 879–91 (2008).

74. K. J. Preacher, K. Kelley, Effect size measures for mediation models: Quantitative strategies for communicating indirect effects, *Psychol. Methods* **16**, 93–115 (2011).

75. D. P. Mackinnon, J. H. Dwyer, Estimating mediated effects in prevention studies, *Eval. Rev.* **17**, 144–158 (1993).

H2:Acknowledgments

Acknowledgements. H3 General We thank the Dunedin Study members, their parents, teachers, partners, and peer informants, and Study founder Phil Silva. **H3 Funding** The Dunedin Multidisciplinary Health and Development Research Unit is supported by the New Zealand Health Research Council and New Zealand Ministry of Business, Innovation and Employment (MBIE). This research received support from US-National Institute of Aging grants R01AG032282, R01AG048895, and 1R01AG049789, UK Medical Research Council grant MR/K00381X, and UK ESRC grant ES/M010309/1. Additional support was provided by the Jacobs Foundation. DWB received support from the NIA through P30AG028716. DWB and BD received support from the NICHD through R21HD078031. **H3 Author Contributions** DWB, TEM and AC conceived the research and wrote the paper; TEM, AC, HLH, SH, SR, and RP collected the data; DLC, KS, and BSW prepared the genotype data; DWB, TEM, DC, HLH, RH, and AC analyzed the data; all authors reviewed drafts, provided critical feedback, and approved the final manuscript. **H3 Competing Interests** The authors report no conflict of interest. The study protocol was approved by the institutional ethical review boards of the participating universities. Study members gave informed consent before participating. **H3 Data and materials availability** The Otago University Ethics Committee provided ethical approval for the Dunedin Study. Participants gave written consent before data were collected. Dunedin Study data are available to researchers on application. A managed-access process ensures that approval is granted to research that comes under the terms of participant consent and privacy (see Supplementary methods for data-sharing details).

H2: Figures and Tables

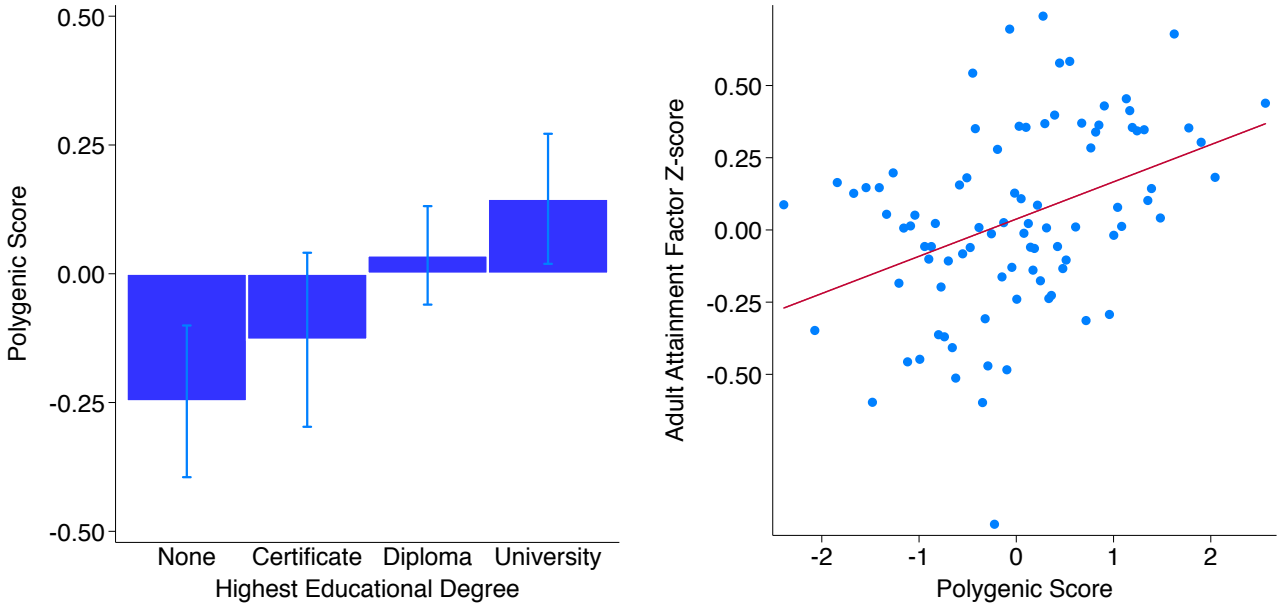


Figure 1. Individuals with higher polygenic scores earned higher degrees and achieved more financial success. Panel A shows the genetic association with educational attainment by graphing mean polygenic score values and 95% confidence intervals for each stratum of educational attainment defined by the highest degree completed: None (15% of cohort), School Leaving Certificate (15%), High School Diploma (42%), University Degree (29%). There was a stepwise increase in average genetic score from cohort members with no credential to those with a university degree. **Panel B** shows the genetic association with a factor score of adult socioeconomic attainments (occupational prestige, income, assets, credit problems, difficulties paying expenses, social welfare benefit use, and credit score). The figure is a binned scatterplot. Each plotted point represents mean X and Y coordinates for a "bin" of 10 Study members.

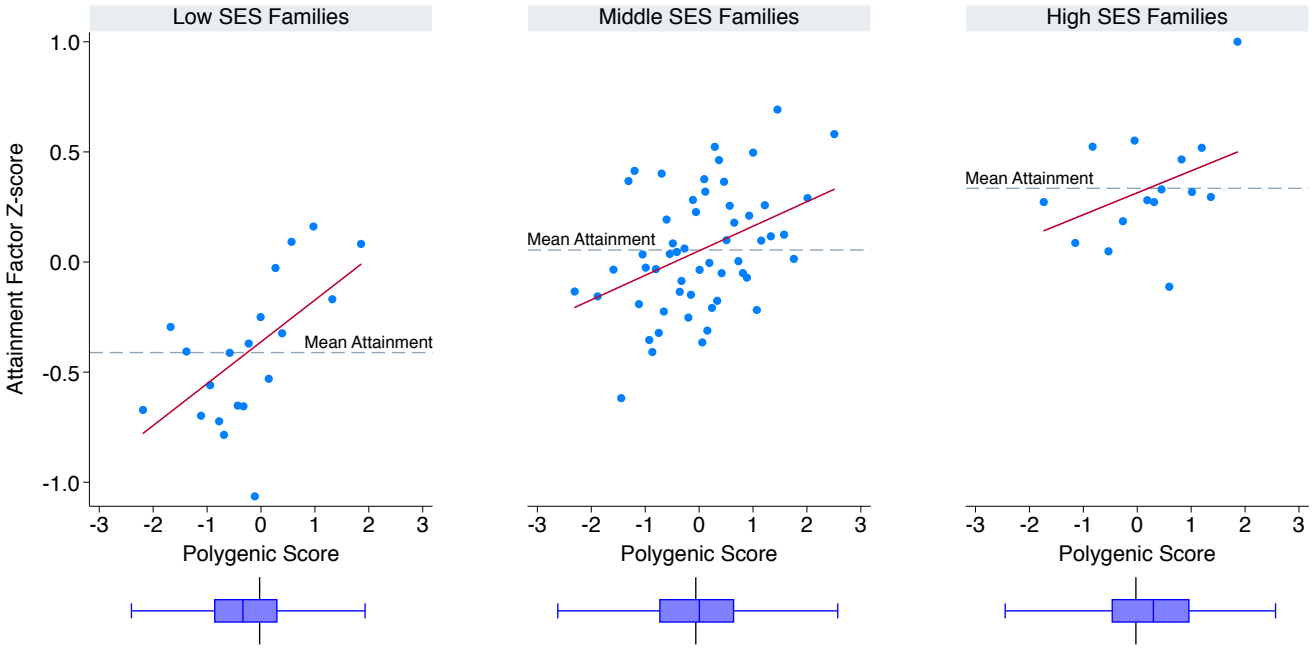


Figure 2. Polygenic scores were socially stratified, but children with higher scores were more likely to succeed no matter their social origin. The figure shows binned scatterplots of the genetic association with the Adult Attainment factor for children born in low, middle, and high socioeconomic-status (SES) families. Each plotted point represents mean X and Y coordinates for a "bin" of about 10 Study members (total n=175 for low SES families; 570 for middle SES families; 152 for high SES families). The solid red line graphs the association in the raw data. The dashed blue line shows the subgroup mean level of attainment. The distribution of polygenic scores within each subgroup is shown in the boxplots at the bottom of the figure. The black vertical line beneath the box plots shows the cohort mean polygenic score.

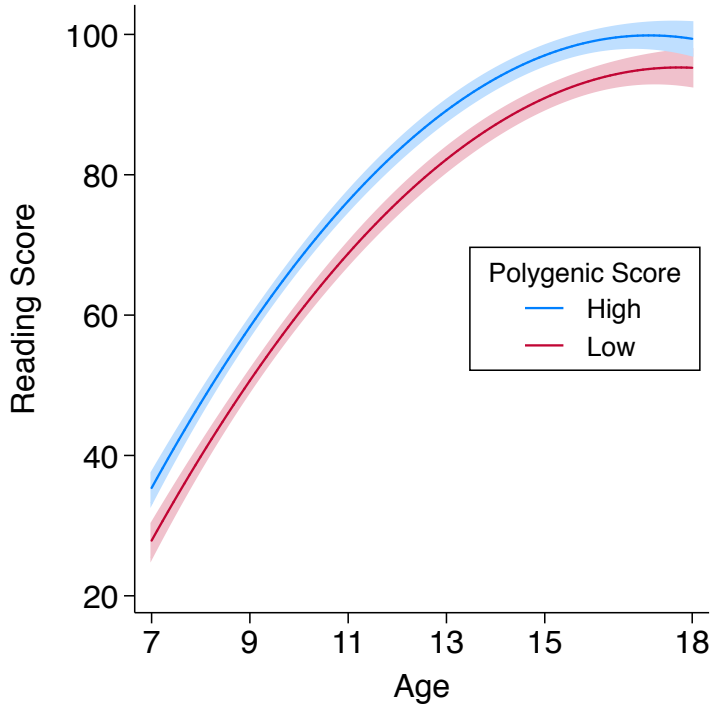


Figure 3. Children with higher polygenic scores acquired reading skills more rapidly. The figure graphs trajectories of reading skill development as measured by the Burt Reading Test *(14)* for children with high polygenic scores (1 SD or more above the mean, blue line, n=159) and for children with low polygenic scores (1 SD or more below the mean, red line, n=147). Shaded areas show 95% confidence intervals. Growth model analysis (**Supplementary methods**) showed that each SD increase in a child's polygenic score predicted a 2.79 SE (0.57) point advantage in reading score at age 7 baseline ($p<0.001$), an increase in linear slope of 0.25 (0.09) points per year ($p=0.005$), and decrease in quadratic slope of 0.03 (0.01) points per year ($p<0.001$).

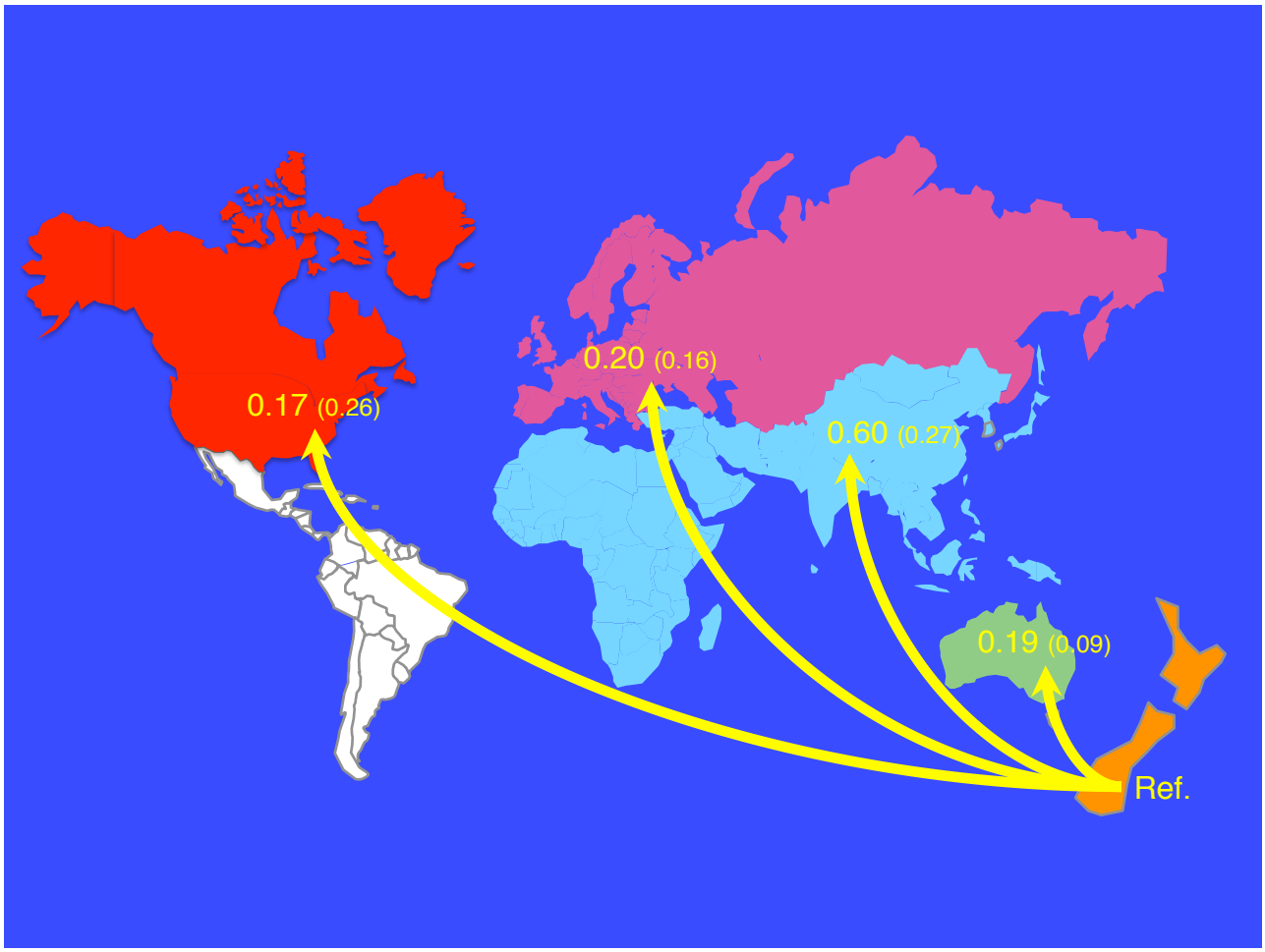


Figure 4. Study members with higher polygenic scores were more likely to migrate out of New Zealand. Migrants were identified as Study members who had lived and worked abroad for a minimum of 12 months since age 21 years and who were still living abroad at the time of the age-38 assessment. Study members with higher polygenic scores were more likely to be in this group RR=1.18 ([1.05-1.32], p=0.005). The figure shows the average difference in polygenic score (in SD units, relative to Study members who remained in or returned to New Zealand) and the standard error of this estimate (in parentheses) for individuals who migrated to North America (n=14), Europe (n=41), Asia and Africa (n=13), and Australia (n=162).

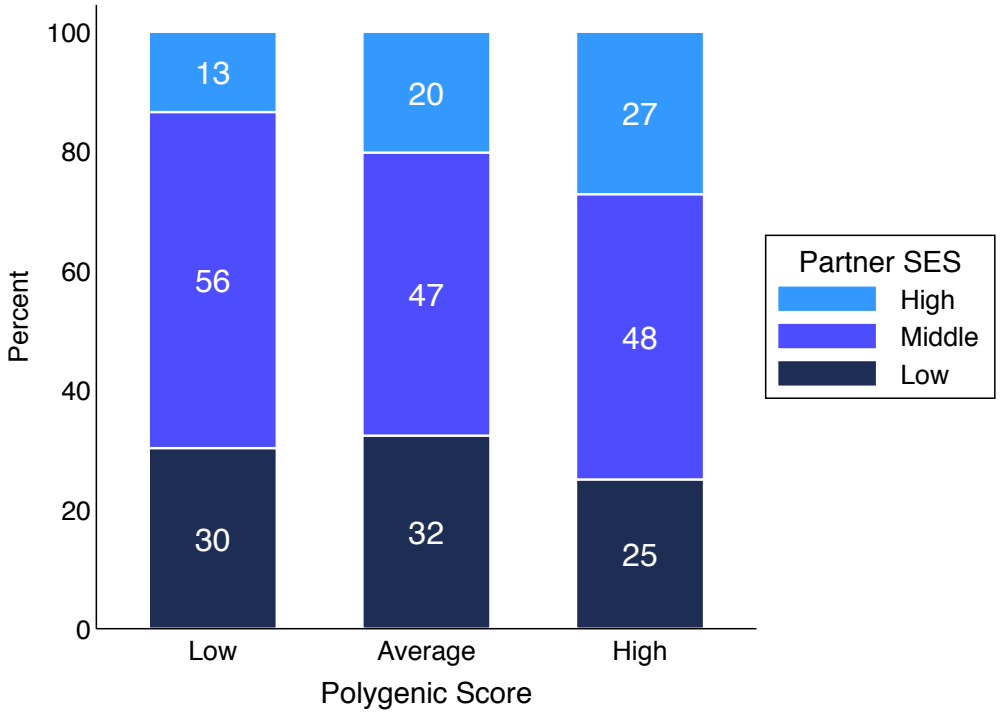


Figure 5. As adults, Study members with higher polygenic scores selected higher socioeconomic-status (SES) mates. The figure shows the distribution of partner SES among partnered Study members with low polygenic scores (1 SD or more below the mean, n=119), average polygenic scores (within 1 SD of the mean, n=504), and high polygenic scores (1 SD or more above the mean, n=136). Partner's SES was defined according to whether they had completed a university degree and whether their income was above the national sex-specific median. A score of 2 (high) meant that the partner had a university education and an above-median income; a score of 1 (middle) meant the partner met one of these criteria; a score of zero (low) meant the partner met neither criterion. White numbers inside the bars show percentages of the polygenic score subgroups.

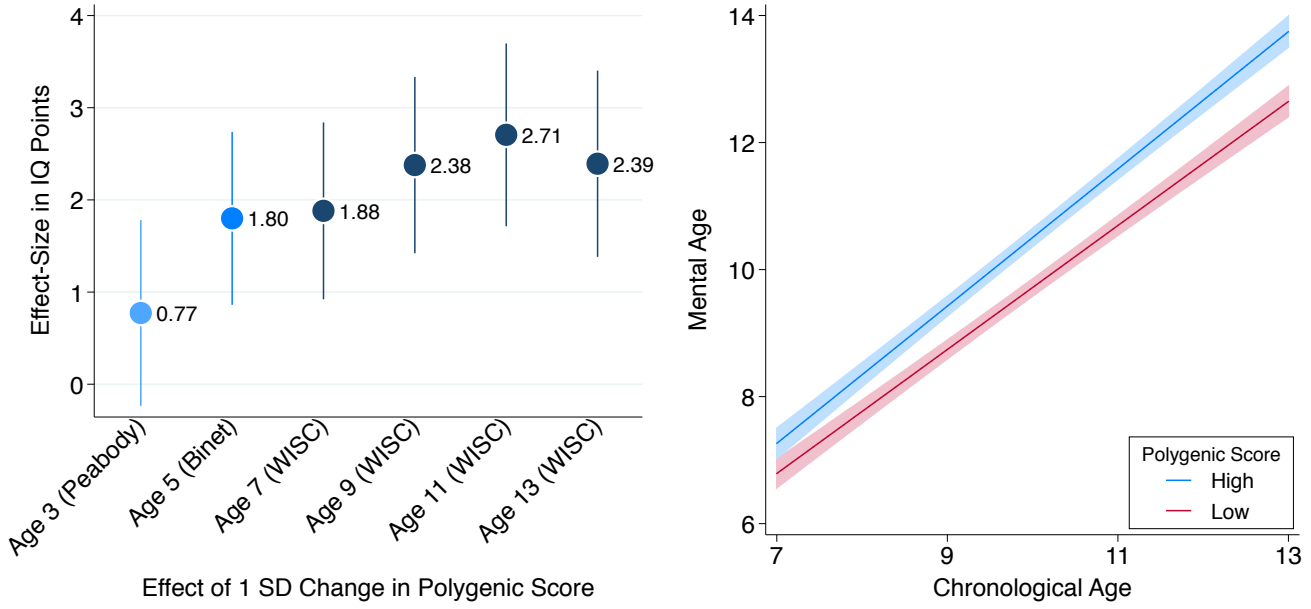


Figure 6. Children with higher polygenic scores scored higher on IQ tests, and differences grew larger over the course of childhood development. Panel A shows genetic associations with cognitive ability at ages 3, 5, 7, 9, 11, and 13 years. Plotted points and 95% CI bars show effect sizes for a 1 SD difference in polygenic score in standardized IQ points (1/15 of one SD). Cognitive ability was measured with the Peabody Picture Vocabulary test *(22)* at age 3, Stanford-Binet IQ test *(23)* at age 5, and Wechsler Intelligence Scale for Children (WISC-R) at ages 7-13. Genetic associations with cognitive ability increased in magnitude across childhood. Panel B shows genetic associations with cognitive development from age 7 to 13 years. Mental age was measured with the WISC-R at ages 7, 9, 11, and 13 years *(53)*. Slopes of cognitive development and 95% CIs are graphed for children with high polygenic scores (1 SD or more above the mean, blue line, n=159) and children with low polygenic scores (1 SD or more below the mean, red line, n=147). Children with higher polygenic scores had advanced mental age at chronological-age-7 baseline and exhibited more rapid cognitive development through age 13 years.

Table 1. Tracking the development of socioeconomic success

Phenotype	Measure	Data Source	Ages
Success in Schooling	Highest Degree	Structured interview	15-38
Success Beyond Schooling	Adult Attainment factor	Occupation (prestige score based on NZ Census data), income, assets, credit problems scale, difficulty paying expenses scale, days of social welfare benefit use (NZ Social Welfare Administration), credit score (VEDA credit bureau) *(30, 54–56)*	38
	Social Mobility	Childhood social class based on parental occupation *(11)*, adult attainment measured using each of, education, occupation, and the Adult Attainment factor	Birth-15, 38
Pathways to Success	Developmental Milestones	Interviews with mothers	3
	Burt Reading Test	Testing by trained research worker *(14, 57)*	7-18
	Aspirations	Questionnaire *(58)*	15
	Standardized testing	NZ Ministry of Education test record form	18
	Geographic mobility	Life history calendar interview *(16)*	21-38
	Financial planfulness	Structured interview and informant reports *(30)*	32-38
	Mate selection	Structured interview in which Study members reported their relationship status and, for those in a serious relationship, partner's highest educational degree and income.	38
Skills & Abilities	Cognitive ability	Peabody Picture Vocabulary Test, Stanford-Binet IQ Test, Wechsler Intelligence Scale for Children *(22–24)*	3-13
	Self-control skills	Staff observations, parent and teacher reports and interviewes with Study members *(30)*	3-11
	Interpersonal skill	Staff observations *(31)*	3-9
	Physical health	Medical exams, anthropometry, lung function testing, clinical interviews with parents *(34)*	3-11

H2: Supplementary Materials

Materials and Methods

Sample Description. Participants are members of the Dunedin Study, a longitudinal investigation of health and behavior in a complete birth cohort. Study members (N=1,037; 91% of eligible births; 52% male) were all individuals born between April 1972 and March 1973 in Dunedin, New Zealand (NZ), who were eligible based on residence in the province and who participated in the first assessment at age 3. The cohort represents the full range of socioeconomic status on NZ's South Island and matches the NZ National Health and Nutrition Survey on key health indicators (e.g., BMI, smoking, GP visits) *(8)*. The cohort is primarily white; fewer than 7% self-identify as having non-Caucasian ancestry, matching the South Island *(8)*. Assessments were carried out at birth and ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, and, most recently, 38 years, when 95% of the 1,007 study members still alive took part. At each assessment, each study member is brought to the research unit for a full day of interviews and examinations. The Otago Ethics Committee approved each phase of the study and informed consent was obtained from all study members.

Genotyping and Imputation. We used Illumina HumanOmni Express 12v1.1 BeadChip arrays (Illumina CA, USA) to assay common Single Nucleotide Polymorphism (SNP) variation in the genomes of our cohort members. We imputed additional SNPs using the impute2 software (version 2.3.1, https://mathgen.stats.ox.ac.uk/impute/impute_v2.html, *(49)*) and 1000 Genomes version 3 reference panel. Imputation was conducted on autosomal SNPs appearing in dbSNP (v140) that were called in >98% of the Dunedin Study samples. Invariant SNPs were excluded. Pre-phasing and imputation were conducted using a 50M base-pair sliding window. The resulting genotype database included genotyped SNPs and SNPs imputed with 90% probability of a specific genotype among the non-Maori members of the Dunedin cohort (n=918) and in Hardy-Weinberg equilibrium (p>0.01 for all).

Polygenic Scoring. We calculated polygenic scores according to the method described by Dudbridge *(50)* using the PRsice software (v1.22, http://prsice.info/, *(51)*).To calculate the polygenic score for educational attainment, we matched genotypes from our data with GWAS results for educational attainment reported by the Social Science Genetic Association Consortium *(1)* and used the approximately 2.3 million matched genotypes to 'score' each of our Study members' genetic predisposition to educational attainment. For each genotype, we counted the number of education-associated alleles (0, 1, or 2) and multiplied this count by the effect-size estimated in the original GWAS. (Most genotypes had effect sizes very near zero.) We then summed weighted counts across all genotypes to calculate each Study member's score. Scores ranged from -30.51-73.77 and were normally distributed in the Dunedin birth cohort (M=17.73, SD=17.94). We standardized scores to have M=0, SD=1 for analysis (**Supplementary Figure 1**). Based on the original GWAS results, Study members with polygenic scores greater than zero would be expected to complete more years of schooling and Study members with polygenic scores below zero would be expected to complete fewer years of

schooling. We used this same method to calculate polygenic scores for height, this time using the results from the GIANT Consortium's most-recent GWAS of height *(39)*.

Principal Components Analysis of Genome-wide SNP data. Polygenic score values may be influenced by subtle differences in ancestry, even among individuals in a European-descent cohort such as ours. To account for ancestry-related genome-wide patterns of allele-frequency differences, we conducted a principal components analysis of our genome-wide SNP database using the EIGENSOFT smartPCA tool *(9, 10)*. We extracted the first ten principal components from the genome-wide SNP data (EIGENSOFT's default). The first principal component explained ~2% of the variance in the education polygenic score. Other principal components explained <1% of variance. Together, the 10 principal components explained 3% of the variance in the education polygenic score.

To correct for any potential population stratification, association analyses were conducted with statistical adjustment for the first 10 principal components estimated from the genome-wide SNP data. Analysis results without this adjustment are reported in **Supplemental Table 2**.

Parents' Socioeconomic Status (SES). The socioeconomic statuses of cohort members' families were measured using a 6-point scale that assessed parents' occupational statuses, defined based on average income and educational levels derived from the New Zealand Census. Parents' occupational statuses were assessed when Study members were born and again at subsequent assessments up to age-15 years. The highest occupational status of either parent was averaged across the childhood assessments *(11)*.

Educational Attainment. We measured educational attainment as the highest degree a Study member had completed through the time of the age-38 assessment. For the 1972-73 birth cohort we studied, compulsory education ended at age 15 years, at which point students could elect to sit for a School Leaving Certificate exam. 15% of our sample obtained no educational credential. 15% obtained the School Leaving Certificate but did not progress further. 42% earned a full high school diploma. 29% completed a university degree.

Adult Attainment. Study members reported their income, assets, credit problems, and difficulties paying expenses to trained Study staff during structured in-person interviews *(30)*.

Occupational prestige. We measured Study members' occupational prestige from self-reported occupation according to the New Zealand Socioeconomic Index (NZSEI-06), a 6-point scale that assessed self-reported occupational status and allocates each occupation to 1 of 6 categories (1 = unskilled laborer, 6 = professional) *(54)*. Homemakers and those not working were pro-rated based on their occupation at the previous interview (when they were aged 32 years). The mean occupational prestige score in the cohort was 3.77 (SD=1.44).

Income. Following the New Zealand Census, Study members were asked to list their sources of income and given the choice of 13 different income categories to report their total pre-tax annual income from all sources in their own currency. For Study members living outside of New Zealand,

income was converted from local currency to NZD. For the cohort, mean income was NZD 62,434 (SD=44,013).

Assets. Study members were asked to estimate the value of each of a series of assets (savings, property, vehicles, homes, etc.) in local currency. For Study members living outside of New Zealand, income was converted from local currency to NZD. For the cohort, mean assets were NZD 603,042 (SD=946,575).

Difficulty paying expenses. Study members were asked about difficulties paying for each of food and necessities, housing, household bills, entertainment, holidays, property upkeep, family obligations, physician visits, and medication costs. They were also asked if they lived paycheck to paycheck, if they had needed to borrow money from family and friends, and if they had needed to take money out of a savings or retirement account to make ends meet. The count of positive response formed the Difficulty Paying Expenses scale (M=5.06, SD=5.76).

Social welfare benefit use. We measured the length of time that Study members drew on government welfare benefits by conducting record linkage with the New Zealand Ministry of Social Development *(56)*. Data on welfare benefit receipt were available from 1 January 1993, with this date marking the beginning of reliable electronic data capture in New Zealand, allowing us to measure duration of benefit use from ages 21-38 years. We obtained information about incident spells and monthly duration of the following New Zealand government benefits: Unemployed Benefit, Invalids Benefit, Sickness and Emergency Benefits, Domestic Purposes Benefit-Sole Parent and Emergency Maintenance Allowance, Training Benefit, Emergency Benefit (for those who do not usually meet entitlement conditions). Only one benefit can be received at any given time. The mean number of months of benefit receipt among cohort members was 23 (SD=43).

Credit problems. Study members were asked about each of a series of credit problems (Have you been turned down for a credit card? Have you defaulted on a credit card payment? Have you missed a bill, mortgage, or loan payment? Have you sold an asset to pay a bill? Have you sold any of your belongings to a pawnbroker? Have you been declared bankrupt? Have you had a house foreclosed on or sold at mortgagee auction by the bank? Have you had something repossessed? (like a car, T.V., or furniture?)) The count of positive response formed the Credit Problems Scale (M=0.43, SD=0.89).

Credit scores. Credit scores were acquired at the age-38 assessment phase from the Veda Company *(55)*. The Veda credit score algorithm is proprietary. Scores are based on 5-year histories of consumer credit activity and include the following factors: the number and types of credit applications and inquiries, age of credit file, residential stability, adverse information such as payment defaults and judgments, and the existence of any current or prior insolvency information. Factors such as race, national origin, marital status, occupation, salary, employment history, medical or academic records are not included in Veda scoring. The mean VEDA score among cohort members was 678 (SD=166).

Adult Attainment Factor. To calculate the Adult Attainment Factor, we conducted a confirmatory factor analysis (CFA) in MPlus v7.3 *(59)*. We categorized severely skewed variables (occupational prestige, credit problems, value of assets, personal income, benefit days) and treated these variables as ordinal in the CFA; VEDA credit scores were divided by 100 (model convergence is

facilitated when all items are scaled similarly). Data for 6 or more of the 7 attainment measures were available for 97% of the cohort. Missing data was imputed using Full Information Maximum Likelihood. The model fit well: χ^2 (N = 971, df = 14) = 130.080, p = 0.00; RMSEA = 0.092 (90% CI: 0.078, 0.107); CFI = 0.933, TLI = 0.900. Standardized factor loadings (95% CI) are presented in **Supplementary Table 3**. Individual factor scores were output and used in subsequent analyses. The factor score was standardized to have mean=0 SD=1 for analysis (**Supplementary Figure 2**). **Supplementary Figure 3** shows effect-sizes for associations between the polygenic score and the attainment factor and each of its components. The figure shows effect-sizes before and after adjustment for educational attainment.

Developmental Milestones. When Study members were aged 3 years, their mothers were interviewed about the age at which their child had reached each of a series of developmental milestones. Mothers reported the age at which their child first smiled, when the child began to walk, defined as taking 6 steps, when the child began feeding himself/herself with a spoon without requiring assistance, when the child began to talk, defined as using 6 words appropriately, when the child began to potty train during the day, defined as staying dry all day 6 out of 7 days per week, when the child began to communicate using sentences, and when the child began to potty train at night, defined as staying dry all night 3 out of 4 nights. **Supplementary Figure 4** shows survival curves illustrating when Dunedin Study members reached each of these milestones.

Reading. We measured the development of reading skills using repeated assessments of the Burt Reading Test *(14)*. At ages 7, 9, 11, 13, 15, and 18 years, children were tested according to a standard protocol by a trained staff member. We used multilevel longitudinal growth models *(60)* to analyze children's development of reading skills. We set the model intercept at the age-7 baseline measurement. Because Burt scores show a curvilinear development trajectory (**Supplementary Figure 5**), we modeled both linear and quadratic slopes. The intercept captured the cohort mean Burt score at age 7 (b=30.50). The linear slope term captured average annual change in reading score across the age 7-18 interval (b=12.50). The quadratic slope term captured deceleration of change, that is, the convexity of the trajectory across childhood (b=-0.60). All model terms were statistically significant (p<0.001). We tested genetic influence on growth by modeling intercept and slope terms of the growth curve as functions of the polygenic score and covariates. Polygenic score coefficients measure the effect of a 1-SD difference in polygenic score on reading at age 7 (intercept), on the linear change per year in reading score from age 7-18 (linear slope), and on the deceleration of that change with increasing age (quadratic slope).

Aspirations. When they were aged 15 years, Study members completed a questionnaire about their educational and occupational aspirations *(58)*. They indicated how far they wanted to go in school and what type of occupation they hoped to hold as an adult. Occupational responses were coded according to the Elley and Irving occupational prestige scale *(61)*.

Standardized Testing. In New Zealand, at the time Dunedin Study members were in high school, standardized exams were administered during 5th, 6th, and 7th forms (ages 15-17 years). For the 1972-73 birth cohort, the age-15 "Certificate" exam was required to earn a School-Leaving Certificate (the minimum secondary education credential at the time); the age-16 Sixth-Form Certificate was used for entry to various tertiary institutions; the age 17 "Bursary" exam was the method through which the government allocated funds ("bursaries") to support room and board costs during university. Study members brought their official exam records to the research unit and their scores were recorded.

Geographic Mobility. We measured geographic mobility from Study members' reports about their place of residence and work, recorded to monthly resolution, during Life History Calendar interviews at ages 26, 32, and 38 years *(16)*.

Financial Planfulness. We measured Study members' financial planfulness from informant reports about their ability to manage money and from interviews with the Study members themselves about financial building blocks and savings behavior.

Money Management. At the age 32 and 38 assessments, we mailed a brief questionnaire to people nominated by the Study member as knowing him/her well (informants included friends, partners, and family members). Full details of the Dunedin Study informant rating system are provided elsewhere *(62)*. Information from informants was available for 96% of Study members. Informants rated the Study member on two items ("poor money manager," "lacks enough money to make ends meet") using a 3-point scale (0=not a problem, 1=bit of a problem, 2=yes, a problem). Scale scores were averaged across ages 32 and 38 to calculate the Money Management Difficulties index (M=0.67, SD=0.84).

Financial Planfulness. At the age-32 and -38 assessments, Study members were interviewed about financial building blocks and about their savings behavior. They were asked if they had investments such as stocks or business investments, and if they had a retirement plan. We counted the number of these building blocks across the two measurement ages to create a 0-4 Financial Building Blocks scale (M=2.24, SD=1.27). Study members' attitudes toward saving and saving behaviors were assessed with seven questions: "Is saving for the future important to you?", "Do you save money to buy expensive items by putting money away and not touching it?", "Do you make regular savings into a special bank account?", "Do you think that saving money makes people more independent?", "Were you encouraged to save money as a child?", "Are you often puzzled by where your money goes?", "Do you think it is important to live within your budget?" *(63)*. Scale scores were averaged across ages 32 and 38 to form the final Saving Behavior scale (M=4.11, SD=1.09). We computed the final Financial Planfulness index by standardizing the Financial Building Blocks and Savings Behavior scales and averaging.

Mate Selection. At the age-38 assessment, Study members were interviewed about their romantic relationships. Most Study members (89%) reported being in a serious relationship. These Study members were further asked about the highest educational degree their partner had completed and

what their income was. We used these data to classify partners according to whether they had completed a university degree and if their income was above the national median for their sex. Reports of partner income for Study members living outside of New Zealand were converted from local currency to NZD. National age-specific median incomes were queried from Statistics New Zealand *(64)* to form cut points. We then classified partners as low, middle, and high SES according to whether they met none (31%), one (49%), or both (20%) of these criteria.

Life satisfaction. When they were aged 38 years, Study members completed the 5-item Satisfaction with Life Scale *(52)* (e.g., In most ways my life is close to ideal, So far I have gotten the important things I want in life). The scale was converted to a Z-score, mean=0, SD=1.

Cognitive Ability. We measured children's cognitive ability from intelligence tests administered by trained psychometrists at ages 3, 5, 7, 9, 11, and 13 years. At age 3, children completed the Peabody Picture Vocabulary Test *(22)*. At age 5, children completed the Stanford-Binet IQ test *(23)*. At ages 7-13, children completed the Wechsler Intelligence Test for Children (WISC-R) *(24)*.

Cognitive Development. We measured children's cognitive development from repeated assessments of mental age made with the Wechsler Intelligence Scale for Children (WISC-R) *(24)* at ages 7, 9, 11, and 13. Mental age scores express the child's level of performance as the chronological age for which his/her score is normative. (For example, although Sara is 10 years old, her mental age is 12.) Mental age can be used to monitor each child's intra-individual development over time *(28)*. (For example, a 10-year-old child with an IQ score equal to the average score for 12-year olds would have a mental age of 12.) We used multilevel longitudinal growth models *(60)* to analyze children's cognitive development, i.e. the "growth" of their mental age. The model intercept captured the cohort mean mental age at chronological age 7 years (b=7). The linear slope term captured average annual change in mental age (b=1). Model terms were statistically significant ($p<0.001$). We tested genetic influence on growth by modeling intercept and slope terms of the growth curve as functions of the polygenic score and covariates. Polygenic score coefficients measure the effect of a 1-SD difference in polygenic score on mental age at chronological age 7 (intercept), and on the linear change per year in mental age from chronological age 7-13 (linear slope).

Self-Control Skills. Children's self-control during their first decade of life was measured using a multioccasion/multi-informant strategy, as previously described *(30)*. Briefly, the composite score includes nine measures: observational ratings of children's lack of control (at 3 and 5 years of age), parent and teacher reports of impulsive aggression, hyperactivity, lack of persistence, inattention, and impulsivity (at 5, 7, 9, and 11 years of age), and self-reports at age 11 years.

Interpersonal Skill. We measured children's interpersonal skill from reports made by trained research workers following standardized testing sessions when the children were aged 3, 5, 7, and 9 years *(31)*. At each age, research workers gave children binary ratings for being friendly (rated as "very friendly" or

"extremely friendly"), confident (rated as "more than usual confidence" or "very self-confident"), cooperative (rated as "reasonably cooperative" or "accepts directions more easily"), and communicative (rated as "readily answers questions, may elaborate" or "answers freely"). Children were given a score ranging 0-100 based on the percent of items endorsed by the research workers (M=52, SD=16).

Childhood Physical Health. As described previously *(34)*, we measured childhood health from medical exams, anthropometry, lung function testing, and clinical interviews with parents at assessments spanning birth to age 11 years. Motor development was assessed at ages 3, 5, 7, and 9 using the Bailey Motor Scales (age 3) *(65)*, McCarthy Motor Scales *(66)* (age 5) and Basic Motor Ability Test *(67)* (ages 7 and 9) *(68)*. Children's overall health at ages 3, 5, 7, 9, and 11 years was rated by two Unit staff members based on review of birth records and assessment dossiers including clinical assessments and reports of infections, diseases, injuries, hospitalizations, and other health problems collected from children's mothers during standardized interviews. Ratings were made on a five-point scale (inter-rater agreement=0.85). Body mass index was calculated from height and weight measurements taken at ages 5, 7, 9, and 11 years. In addition, tricep and subscapular skinfold thicknesses were measured at ages 7 and 9 years by trained anthropometrists *(69)*. (For calculation of the overall measure, tricep and subscapular skinfold thicknesses were averaged to create a single score.) Systolic and diastolic blood pressure were measured at ages 7, 9, and 11 years using a London School of Hygiene and Tropical Medicine blind mercury sphygmomanometer (Cinetronics Ltd., Mildenhall, United Kingdom) *(70)*. Fixed expiratory volume in one second (FEV_1) and the ratio of FEV_1 to forced vital capacity (FVC) were measured at ages 9 and 11 using a Godart water spirometer *(71)*. To calculate the childhood health measure, assessments were standardized to have mean=0 SD=1 within age and sex specific groups. Cross-age scores for each measure were then computed by averaging standardized scores across measurement ages. The final childhood health score was calculated by taking the natural log of the average score across all measures, resulting in a normally distributed childhood health index.

Mediation Analysis. For each potential mediator (cognitive ability, self-control skills, interpersonal skill), we tested associations between the polygenic score and the mediator; we tested associations between the mediator and the educational attainment and Adult Attainment Factor score outcomes; and we tested the association between polygenic score and each outcome, including the mediator as a covariate. We used the system of equations described by Baron and Kenny *(72)* and the methods described by Preacher et al. *(73, 74)* to calculate total, direct, and indirect effects, and to estimate the proportion of the genetic effect mediated by each of the mediators (**Supplementary Figure 7**). We also fitted a multiple mediator model in which all three mediators were included as covariates in the final regression. Results are reported in **Supplementary Table 5**. **Supplementary Figure 8** shows results for multiple mediator analyses of attainment (left side) and pathways to success measures (right side).

Data Sharing. The Dunedin Study has not sought informed consent for unrestricted data sharing because data from the Dunedin study have historically been deemed by the Duke and Otago Institutional Review Boards (IRBs) as being in a high-risk category that precludes making the data set available for unrestricted, unsupervised open-access data sharing. Consent documents for the study used over the past 40 years have informed each study member that "…all the information obtained by the researchers at the Dunedin Multidisciplinary Health and Development Research Unit will be treated as STRICTLY CONFIDENTIAL to members of the research team," and "Only approved Dunedin Study researchers will have access to your data." These consent documents were last signed by Study members at the age-38 assessment, which ended in 2012. This means that the Dunedin Study participants have not at this point given their informed consent for unrestricted data sharing, and therefore data deriving from their participation cannot be made available for unrestricted use.

Our data-sharing policy provides for researchers outside the Study to access data used in a published paper by becoming "honorary" staff members of the Dunedin Unit, so they can access the data via collaboration (policy on the Dunedin Study website [http://dunedinstudy.otago.ac.nz]). Applicant investigators are invited to submit a concept paper describing the data analysis project they wish to carry out.

Access requirements in a nutshell. Proposed data-analysis projects from qualified scientists must have a concept paper describing the purpose of data access, IRB approval at the applicants' university, and provision for secure data access. We offer secure access on the Duke and Otago campuses.

All scripts and analysis files for Dunedin Study published papers are available.

Our data-sharing policy was last approved in 2015 by NIA as part of a review of Dunedin Study competing-renewal funding.

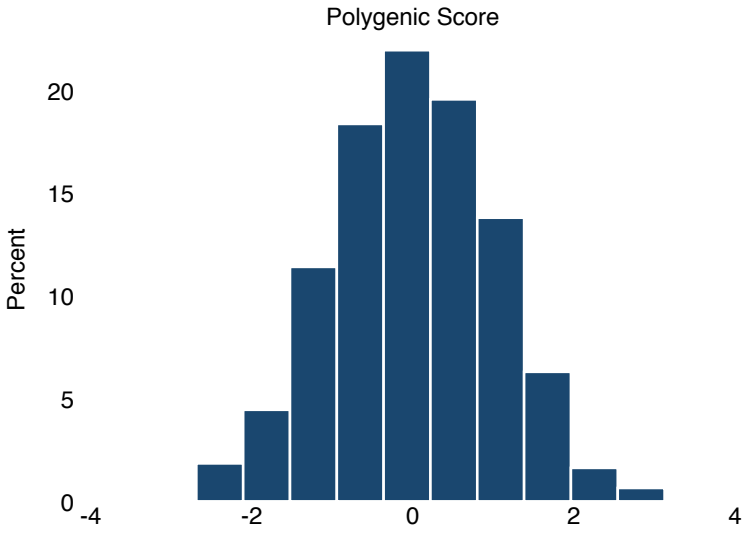


Figure S1. Distribution of the polygenic score for educational attainment in the Dunedin cohort. The x-axis of the figure shows polygenic score z-scores (one unit corresponds to one standard deviation).

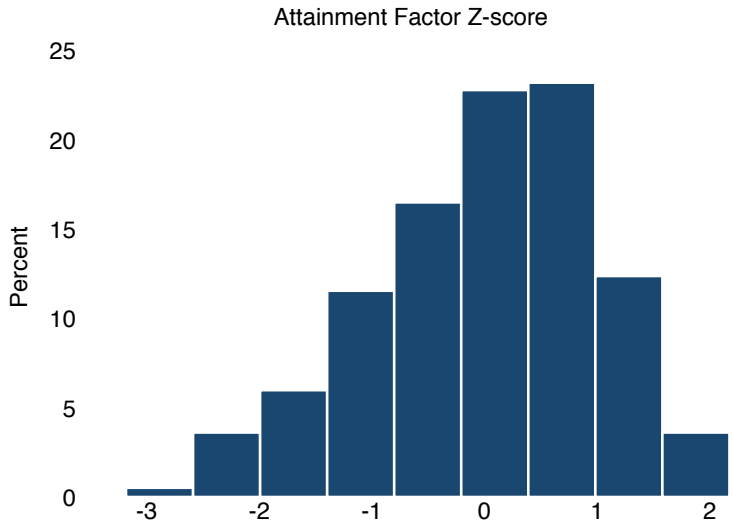
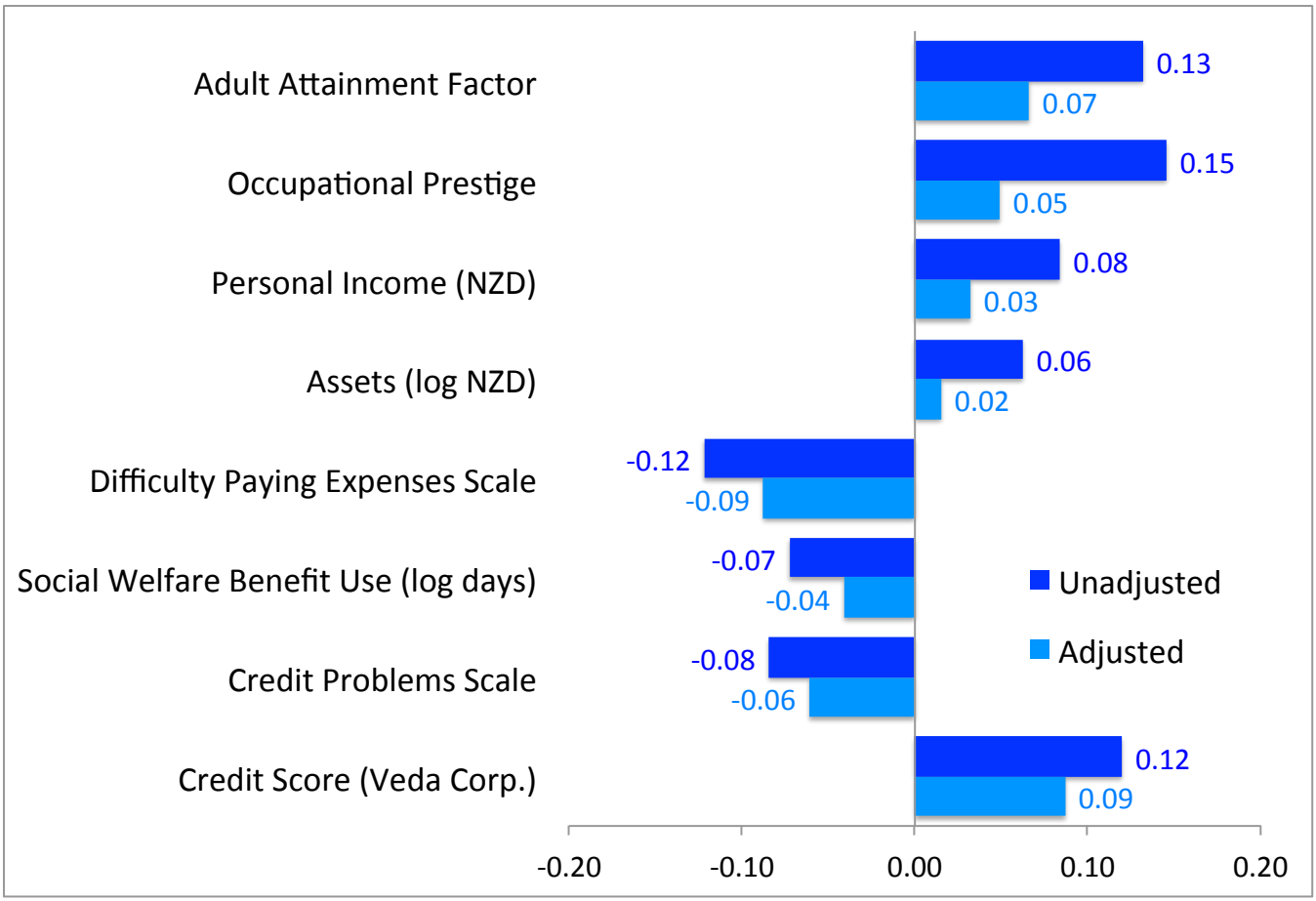


Figure S2. Distribution of the Adult Attainment Factor score in the Dunedin cohort. The x-axis of the figure shows Attainment Factor z-scores (one unit corresponds to one standard deviation).



	Unadjusted Estimate			Estimate Adjusted for Educational Attainment			
	r	(SE)	p-value	r	SE	p-value	N
Adult Attainment Factor	0.13	(0.03)	1.18E-04	0.07	(0.03)	0.035	901
Occupational Prestige	0.15	(0.03)	7.30E-06	0.05	(0.03)	0.089	866
Personal Income (NZD)	0.08	(0.03)	0.006	0.03	(0.03)	0.283	882
Assets (log NZD)	0.06	(0.03)	0.059	0.02	(0.03)	0.644	889
Difficulty Paying Expenses Scale	-0.12	(0.03)	3.97E-04	-0.09	(0.03)	0.009	887
Social Welfare Benefit Use (log days)	-0.07	(0.03)	0.034	-0.04	(0.03)	0.226	897
Credit Problems Scale	-0.08	(0.03)	0.010	-0.06	(0.03)	0.062	887
Credit Score (Veda Corp.)	0.12	(0.04)	0.001	0.09	(0.04)	0.014	803

Figure S3. Effect-size estimates for genetic associations with the adult attainment measures before and after adjustment for educational attainment. Effect-size estimates are standardized regression coefficients (equivalent to Pearson's r). All models included sex and the first ten principal components estimated from the genome-wide SNP data as covariates. Unadjusted estimates are shown with dark blue bars. Estimates adjusted for educational attainment are shown with light blue bars. Adjusting for educational attainment reduced genetic effect sizes by 25-70%.

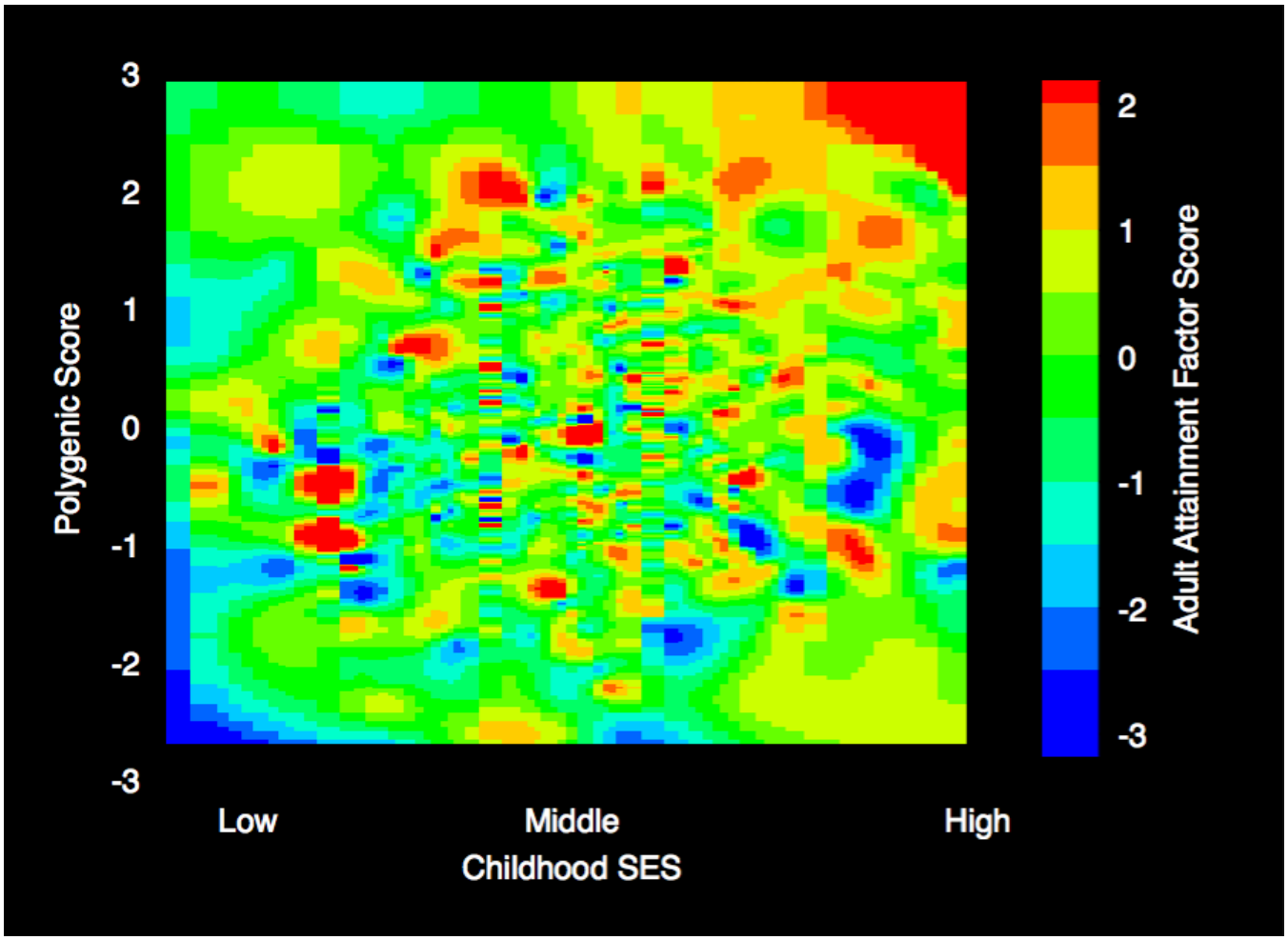


Figure S4. Genetic and social inheritance combine to influence life attainments. The heat map shows variation in adult attainment (low to high attainment scaled from blue to red on the color axis) across the distributions of social inheritance (x-axis) and polygenic scores (y-axis). The clustering of blue toward the bottom left and of red toward the upper right illustrates and additive combination of genetic and social inheritance influencing life attainments.

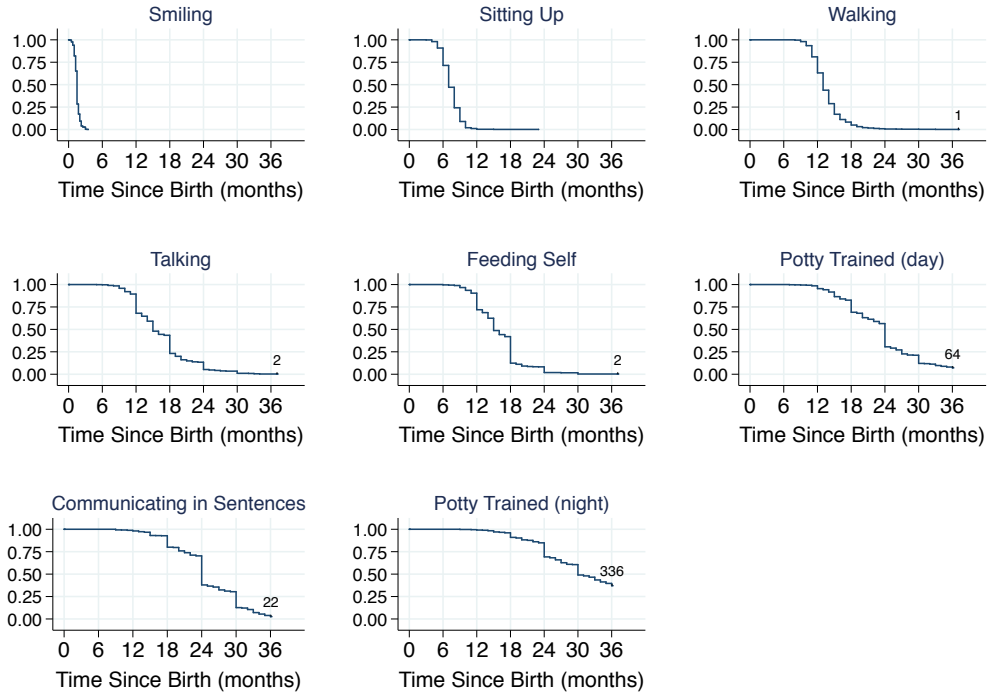


Figure S5. Survival curves illustrating when Dunedin Study members reached each of a series of developmental milestones.

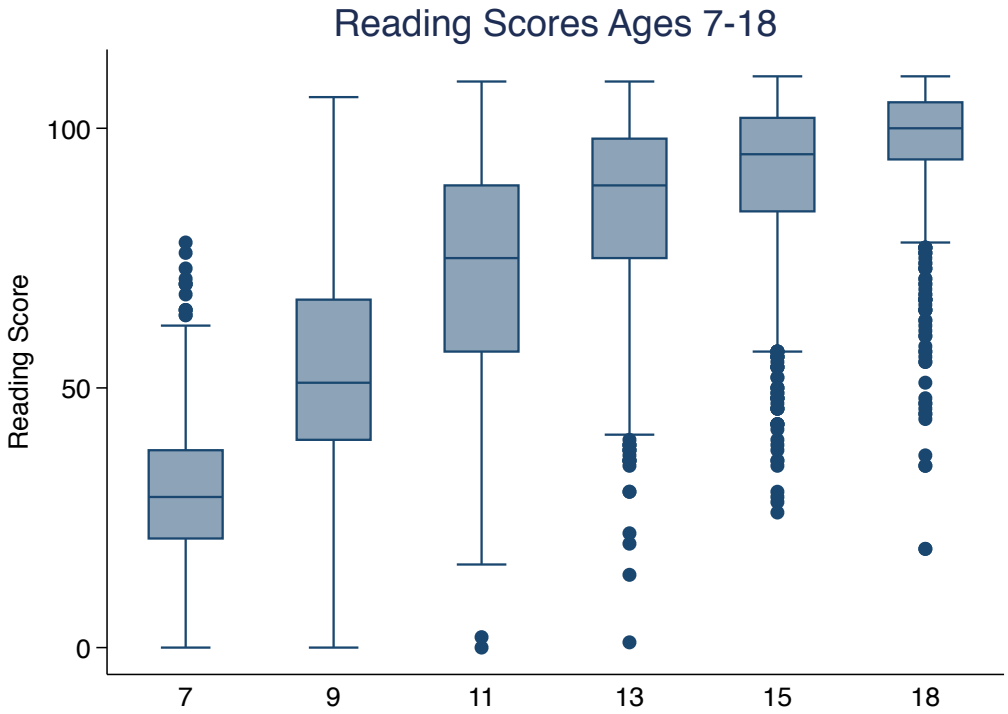


Figure S6. Development of reading skill from age 7 to 18 years in the Dunedin Cohort. The box plots show distributions of Burt Reading Test scores in the Dunedin cohort when Study members were ages 7, 9, 11, 13, 15, and 18 years.

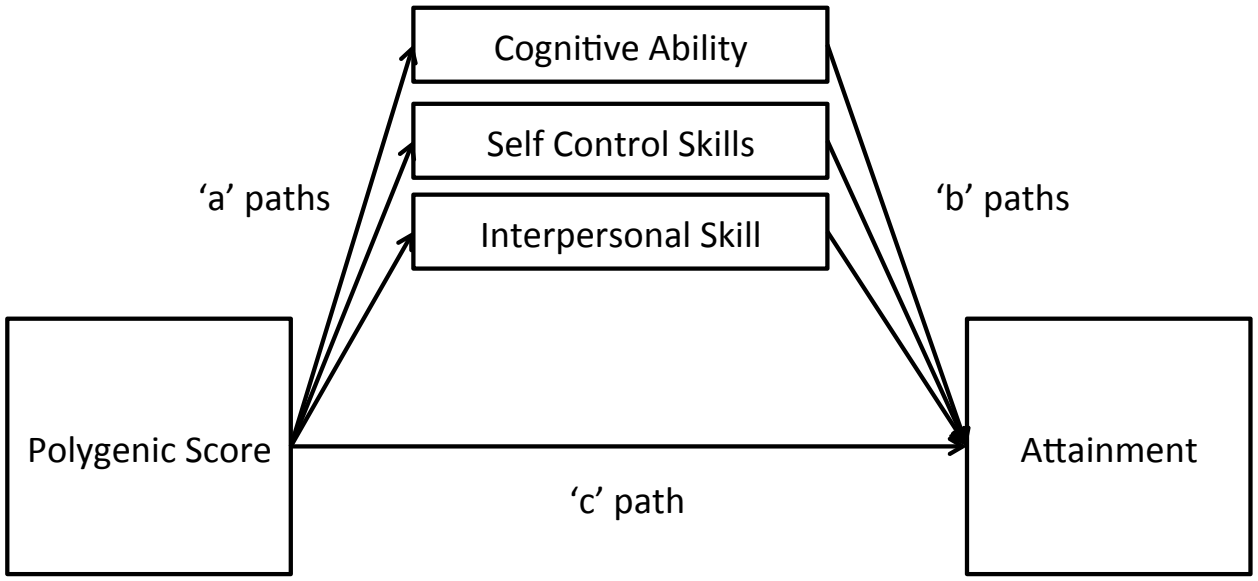


Figure S7. Path diagram of mediation analysis. The path diagram is a graphical representation of the mediation analysis. We analyzed two attainment outcomes, educational attainment and adult socioeconomic attainment (measured as the adult attainment factor score). In addition to the multiple mediator model depicted below, we also conducted single-mediator analyses in which each candidate mediator was analyzed on its own (see Supplementary Table 3). Indirect effects were estimated as the products of 'a' and 'b' paths. Direct effects were estimated as the 'c' paths.

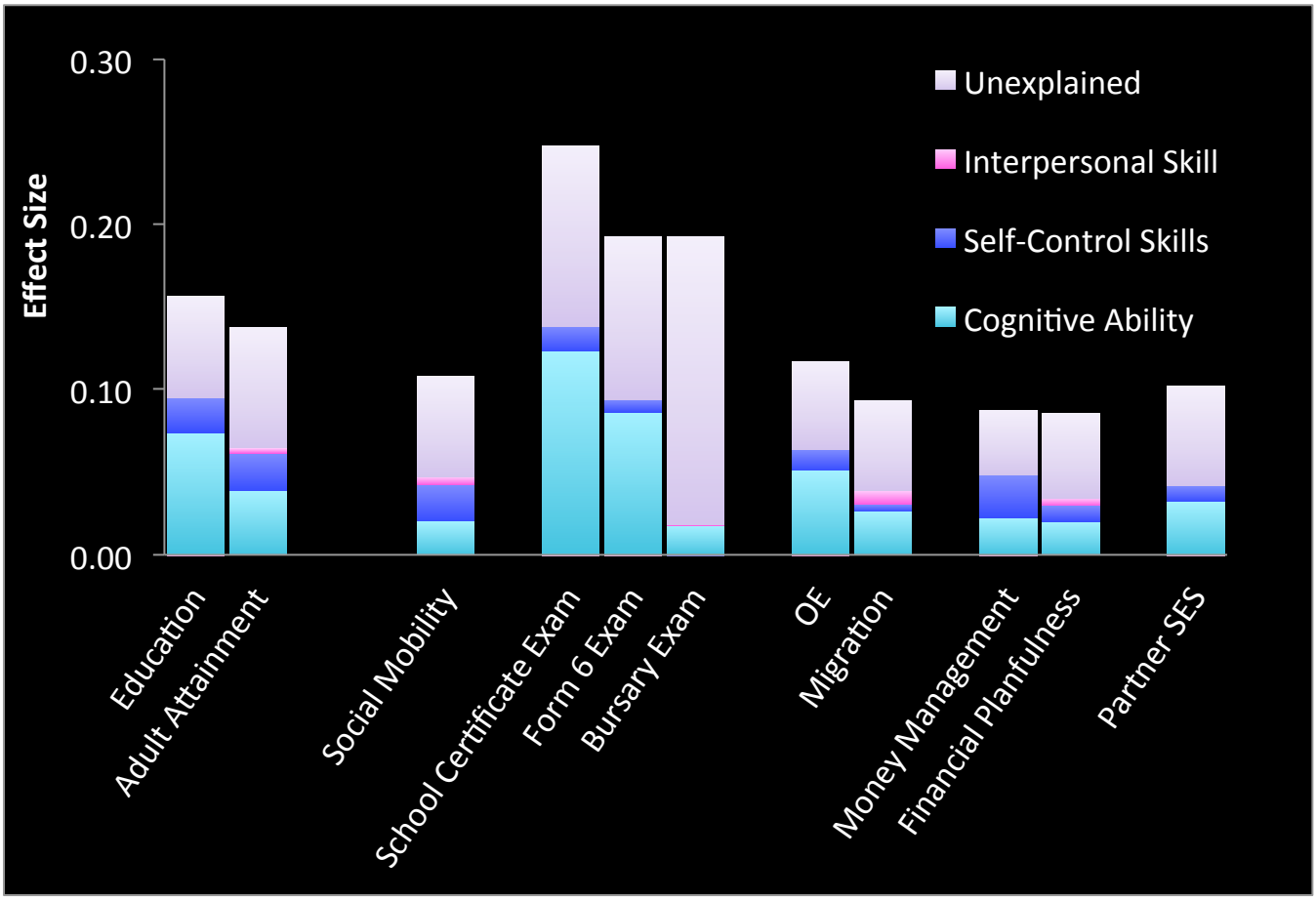


Figure S8. Mediation of genetic associations with adult attainments and pathways to success by cognitive ability, self-control skills, and interpersonal skill. The figure graphs effect estimates from multiple-mediator models of genetic associations with attainments and pathways to success. Bar height gives the total effect estimate. Colored segments of bars show the indirect effects of cognitive ability (light blue), self-control skills (dark blue), and interpersonal skill (pink), and the portion of the total effect not explained by these mediators (lavender). Estimates for dichotomous dependent variables (OE, Migration) were derived using the method described by Mackinnon and Dwyer *(75)*.

Table S1. Effect-size estimates for genetic associations with adult attainments, pathways to success, and abilities and skills: Models without adjustment for principal components estimated from the genome-wide SNP data and models with adjustment for principal components. Effect-size estimates are standardized coefficients (denoted as 'r') from linear regressions, hazard ratios (denoted as 'HR') from Cox models, relative risks (denoted as 'RR') from from Poisson models, odds ratios (denoted 'OR') from ordered logistic models, and unstandardized coefficients (denoted as 'b') from mixed-effects growth models. All models included sex as a covariate. Models under the heading "Adjusted for Principal Components" were additionally adjusted for the first ten principal components estimated from the genome-wide SNP data. Stars next to coefficients indicate p-values *** <0.001, ** <0.01, *<0.05. 95% Confidence intervals are provided for relative risks and odds ratios. Confidence intervals that do not include 1 are statistically significant at the α=0.05 level. Confidence intervals that include 1 are denoted with gray text.

		Undadjusted		Adjusted for Principal Components	
Educational Attainment	r	0.14 ***		0.15 ***	
Adult Attainment	r	0.15 ***		0.13 ***	
Pathways to Success					
Milestones					
Smiling	HR	0.99	[0.94-1.05]	1.00	[0.95-1.06]
Sitting Up	HR	1.00	[0.94-1.05]	1.00	[0.94-1.06]
Walking	HR	1.01	[0.95-1.06]	1.01	[0.95-1.06]
Talking	HR	1.11	[1.05-1.18]	1.12	[1.05-1.19]
Feeding Self	HR	0.98	[0.93-1.04]	0.98	[0.92-1.04]
Potty Training (day)	HR	1.03	[0.97-1.09]	1.02	[0.96-1.09]
Potty Training (night)	HR	0.95	[0.88-1.02]	0.95	[0.88-1.03]
Communicating in Sentences	HR	1.06	[1.00-1.13]	1.06	[1.00-1.12]
Reading					
Reading: Intercept (age 7)	b	2.69 ***		2.79 ***	
Reading: Linear Slope	b	0.25 *		0.25 *	
Reading: Quadratic Slope	b	-0.03 **		-0.03 **	
Aspirations					
Educational Aspirations	r	0.15 ***		0.15 ***	
Aspiration to University Degree	RR	1.23	[1.11-1.36]	1.24	[1.11-1.37]
SES Aspiration	r	0.12 ***		0.12 ***	
Aspiration to Professional Occupation	RR	1.15	[1.05-1.25]	1.16	[1.06-1.27]
Standardized Testing					
No Educational Certification	RR	0.80	[0.68-0.94]	0.78	[0.66-0.93]
Testing Level	OR	1.33	[1.17-1.52]	1.36	[1.18-1.56]
School Certificate Exam Score	r	0.24 ***		0.24 ***	
Form 6 Exam Score	r	0.21 ***		0.19 ***	
Bursary Exam Score	r	0.19 *		0.19 *	
Geographic Mobility					
OE	RR	1.18	[1.05-1.32]	1.17	[1.05-1.32]
Migration	RR	1.18	[1.05-1.32]	1.18	[1.05-1.32]
Financial Planfulness					
Financial Problems	r	-0.09 **		-0.08 *	
Financial Planfulness	r	0.10 **		0.09 **	
Mating					
Partner SES	r	0.09 *		0.09 *	
Life Satisfaction	r	0.04		0.04	
Abilities and Skills					
Cognitive Ability					
Peabody IQ	r	0.06		0.05	
Stanford-Binet IQ	r	0.15 ***		0.13 ***	
WISC-R IQ (age 7)	r	0.14 ***		0.13 ***	
WISC-R IQ (age 9)	r	0.18 ***		0.16 ***	
WISC-R IQ (age 11)	r	0.18 ***		0.18 ***	
WISC-R IQ (age 13)	r	0.16 ***		0.16 ***	
Cognitive Development					
Mental Age: Intercept (age 7)	b	0.14 ***		0.13 ***	
Mental Age: Linear Slope	b	0.05 ***		0.05 ***	
Non-Cognitive Skills					
Self-Control Skills	r	0.11 ***		0.10 **	
Interpersonal Skill	r	0.11 **		0.10 **	
Physical Health	r	-0.01		0.01	

Table S2. Standardized factor loadings for adult attainment indicators.

Measure	Categorization	Loading	95% CI
Occupational Prestige	6 Categories	0.48	[0.42, 0.55]
Personal Income	10 Categories	0.51	[0.46, 0.57]
Value of Assets	10 Categories	0.71	[0.66, 0.75]
Difficulty Paying Expenses		-0.60	[-0.66, -0.55]
Benefit Days	8 Categories	-0.66	[-0.71, -0.61]
Credit Problems	7 Categories	-0.66	[-0.73, -0.60]
Credit Score (VEDA)	÷ 100	0.53	[0.47, 0.59]

Table S3. Effect-size estimates for genetic associations with adult attainments, pathways to success, and abilities and skills. Effect-size estimates are standardized coefficients (denoted as 'r') from linear regressions, hazard ratios (denoted as 'HR') from Cox models, relative risks (denoted as 'RR') from Poisson models, odds ratios (denoted 'OR') from ordered logistic models, and unstandardized coefficients (denoted as 'b') from mixed-effects growth models. All models included sex and the first ten principal components estimated from the genome-wide SNP data as covariates. Models under the heading "Adjusted for Childhood SES" were additionally adjusted for childhood SES *(11)*. Stars next to coefficients indicate p-values *** <0.001, ** <0.01, *<0.05. 95% Confidence intervals are provided for relative risks and odds ratios. Confidence intervals that do not include 1 are statistically significant at the α=0.05 level. Confidence intervals that include 1 are denoted with gray text.

		Undadjusted	Adjusted for Childhood SES
Educational Attainment	r	0.15 ***	0.10 **
Adult Attainment	r	0.13 ***	0.11 **
Pathways to Success			
Milestones			
Smiling	HR	1.00 [0.95-1.06]	1.00 [0.94-1.05]
Sitting Up	HR	1.00 [0.94-1.06]	0.99 [0.94-1.06]
Walking	HR	1.01 [0.95-1.06]	1.01 [0.95-1.06]
Talking	HR	1.12 [1.05-1.19]	1.11 [1.05-1.18]
Feeding Self	HR	0.98 [0.92-1.04]	0.97 [0.92-1.03]
Potty Training (day)	HR	1.02 [0.96-1.09]	1.03 [0.96-1.09]
Potty Training (night)	HR	0.95 [0.88-1.03]	0.96 [0.88-1.04]
Communicating in Sentences	HR	1.06 [1.00-1.12]	1.04 [0.98-1.11]
Reading			
Reading: Intercept (age 7)	b	2.79 ***	2.27 ***
Reading: Linear Slope	b	0.25 *	0.15
Reading: Quadratic Slope	b	-0.03 **	-0.02 *
Aspirations			
Educational Aspirations	r	0.15 ***	0.12 ***
Aspiration to University Degree	RR	1.24 [1.11-1.37]	1.18 [1.06-1.32]
SES Aspiration	r	0.12 ***	0.10 **
Aspiration to Professional Occupation	RR	1.16 [1.06-1.27]	1.13 [1.03-1.24]
Standardized Testing			
No Educational Certification	RR	0.78 [0.66-0.93]	0.86 [0.72-1.02]
Testing Level	OR	1.36 [1.18-1.56]	1.23 [1.06-1.42]
School Certificate Exam Score	r	0.24 ***	0.19 ***
Form 6 Exam Score	r	0.19 ***	0.16 ***
Bursary Exam Score	r	0.19 *	0.18 *
Geographic Mobility			
OE	RR	1.17 [1.05-1.32]	1.13 [1.00-1.27]
Migration	RR	1.18 [1.05-1.32]	1.17 [1.05-1.32]
Financial Planfulness			
Financial Problems	r	-0.08 *	-0.06
Financial Planfulness	r	0.09 **	0.07 *
Mating			
Partner SES	r	0.09 *	0.07
Life Satisfaction	r	0.04	0.03
Abilities and Skills			
Cognitive Ability			
Peabody IQ	r	0.05	0.02
Stanford-Binet IQ	r	0.13 ***	0.09 **
WISC-R IQ (age 7)	r	0.13 ***	0.08 *
WISC-R IQ (age 9)	r	0.16 ***	0.11 ***
WISC-R IQ (age 11)	r	0.18 ***	0.13 ***
WISC-R IQ (age 13)	r	0.16 ***	0.11 ***
Cognitive Development			
Mental Age: Intercept (age 7)	b	0.13 ***	0.09 *
Mental Age: Linear Slope	b	0.05 ***	0.03 **
Non-Cognitive Skills			
Self-Control Skills	r	0.10 **	0.07 *
Interpersonal Skill	r	0.10 **	0.09 *
Physical Health	r	0.01	0.02

Table S4. Mediation analysis results. The table shows standardized estimates of total, direct, and indirect effects from mediation models. 95% Confidence intervals are percentile based, estimated from 500 bootstrap repetitions.

	Educational Attainment				Adult Attainment Factor			
	Est.	SE	p-value	95% CI	Est.	SE	p-value	95% CI
Multiple Mediator Model (Cognitive Ability, Self-Control Skills, Social Skills)								
Total Effect	0.15	(0.03)	<0.001	[0.09-0.22]	0.14	(0.04)	<0.001	[0.07-0.20]
Direct Effect	0.06	(0.03)	0.037	[0.00-0.12]	0.07	(0.03)	0.025	[0.00-0.14]
Total Indirect Effect	0.09	(0.02)	<0.001	[0.06-0.13]	0.06	(0.01)	<0.001	[0.04-0.09]
% Mediation	60%				47%			
Individual Mediator Models								
IQ								
Total Effect	0.15	(0.03)	<0.001	[0.09-0.22]	0.14	(0.03)	<0.001	[0.07-0.20]
Direct Effect	0.07	(0.03)	0.022	[0.01-0.13]	0.08	(0.03)	0.010	[0.02-0.14]
Indirect Effect	0.09	(0.02)	<0.001	[0.05-0.12]	0.06	(0.01)	<0.001	[0.03-0.08]
% Mediation	57%				41%			
Self-Control Skills								
Total Effect	0.15	(0.03)	<0.001	[0.09-0.22]	0.14	(0.03)	<0.001	[0.07-0.20]
Direct Effect	0.11	(0.03)	<0.001	[0.05-0.18]	0.10	(0.03)	0.001	[0.04-0.16]
Indirect Effect	0.04	(0.01)	0.002	[0.02-0.07]	0.03	(0.01)	0.004	[0.01-0.06]
% Mediation	27%				24%			
Interpersonal Skill								
Total Effect	0.15	(0.03)	<0.001	[0.08-0.21]	0.14	(0.03)	<0.001	[0.07-0.20]
Direct Effect	0.14	(0.03)	<0.001	[0.06-0.20]	0.13	(0.03)	<0.001	[0.06-0.19]
Indirect Effect	0.02	(0.01)	0.011	[0.01-0.03]	0.01	(0.01)	0.021	[0.00-0.02]
% Mediation	10%				9%			