Genetic Endowments, Educational Outcomes, and the Moderating Influence of School Quality*

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Abstract

Genetic endowments are fixed at conception and matter for the educational attainment of individuals. Do better schools mitigate or magnify the outcomes of this genetic lottery? We analyze the interplay of genetic endowments and school quality for educational attainment in the United States. Our results suggest that higher-quality schools are substitutes for genetic endowments: a 1 SD increase in school quality reduces the positive effect of a 1 SD increase in the relevant polygenic index (PGI) by 19%. This substitutability is underpinned by relative gains in health, language ability, patience, and risk aversion of low-PGI students.

JEL-Codes: I29; I21; J24
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1 INTRODUCTION

Education is a key determinant of life outcomes for individuals and societies as a whole (Acemoglu and Autor, 2011; Hanushek and Woessmann, 2008; Krueger and Lindahl, 2001). At the same time, a broad literature has shown that genetic endowments are crucial for educational success. For example, in heritability studies, genetic endowments account for 40% of the variation in years of education (Branigan et al., 2013; Lee et al., 2018). High heritability, however, does not imply that the impact of genes on education is immutable. On the contrary, growing evidence shows that the impact of genetic endowments on life outcomes varies with social environments like families, neighborhoods, and schools (Cesarini and Visscher, 2017; Koellinger and Harden, 2018). This observation raises important questions for researchers and policymakers: can schools moderate the link between genetic endowments and educational outcomes, and if yes, do they magnify or reduce educational inequalities based on genetic endowments?

Answers to these questions are essential to address concerns about educational inequality. Since genetic endowments are fixed at conception and remain constant over the life course of individuals, educational inequalities based on genetic endowments may be inconsistent with the widespread ambition of providing equal educational opportunities to all members of society (Alesina et al., 2018; Lergetporer et al., 2020).[1] In line with this policy goal, there is a long-standing literature in economics that inquires "the question of how well schools reduce the inequity of birth" (Coleman et al., 1966, p. 36). This literature has focused on inequalities by socioeconomic status (SES), race, and gender. However, detailed evidence of how well schools address inequality based on genetic endowments is scant due to the long-term unavailability of genetic data at the individual level.

In this paper, we address this gap. We use individual-level data on genetic endowments to study the interplay of genes and school quality in the production of educational attainment. That is, we investigate whether high-quality schools magnify or reduce educational inequality based on genetic endowments. Notably, the effect sign is unclear a priori since different branches of related literature suggest potential effects in both directions. On the one hand, behavioral geneticists have found that advantaged family environments and genetic endowments complement each other (Turkheimer et al., 2003; Woodley of Menie et al., 2021). Hence, if school environments worked similarly to family environments, high-quality schools would reinforce educational inequality based on genetic endowments. On the other hand, recent evidence on heterogeneity in school effects suggests that school quality and student SES are substitutes (Jackson et al., 2024). Hence, if disadvantages based on genetic endowments were sim-

[1] Different conceptions of equality of opportunity exist. Proponents of *luck egalitarianism* demand compensation for disadvantages from all factors beyond individual control, including genetic endowments (e.g., Roemer, 1998). Proponents of *fair equality of opportunity* demand compensation for disadvantages from all social circumstances but not genetic endowments (e.g., Rawls, 1971). In both cases, however, it is crucial to assess heterogeneous effects along the distribution of genetic predispositions to assess whether a given policy intervention moves society closer or further away from a fair outcome distribution. See also Arneson (2018) for an accessible summary discussion.

ilar to disadvantages based on students' SES, high-quality schools would reduce educational inequality based on genetic endowments.

To study the interplay of genetic endowments and school quality in the production of educational attainment, we use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a 5-wave panel study that follows a representative sample of US high school students from 1994/95 to the present. To measure genetic endowments, we leverage recent advances in molecular biology and use a polygenic index for educational attainment (PGI^{EA}, Becker et al., 2021; Dudbridge, 2013; Lee et al., 2018). PGI^{EA} is an individual measure of the genetic propensity to attain education.[2] It offers important advantages over traditional proxies for "innate ability," such as student test scores and IQ tests (Brinch and Galloway, 2012; Hanushek and Woessmann, 2008, 2012; Heckman et al., 2010). In particular, it is a fine-grained DNA-based measure fixed at conception that cannot be modified afterward. To measure the quality of school environments, we use information from headmaster surveys and construct school quality indicators based on the following observable characteristics: teacher experience, teacher turnover, teacher education, and class size. To the best of our knowledge, Add Health is the only (publicly available) data set that offers detailed information on schooling environments from survey and administrative sources for a reasonably sized genotyped sample.

For identification, we combine a between-family comparison with the control function approach suggested by Altonji and Mansfield (2018). We discuss the underlying identification assumptions in detail and test their satisfaction. A first identification challenge arises from the fact that the genetic endowments of children are a function of the genetic endowments of their parents. Therefore, our parameters of interest may be confounded by *genetic nurture effects*, i.e., genetic endowments of children may be correlated with other family characteristics that co-determine their educational attainment. In response, we show that the estimated effect of PGI^{EA} on educational attainment from the between-family design can be replicated in a smaller sibling sample, allowing us to control for genetic nurture by including family-fixed effects. This result suggests that any remaining confounding from genetic nurture effects is small and unlikely to overturn our main results. A second identification challenge arises from the fact that children sort into schools based on family background. Therefore, our parameters of interest may be confounded by *selection effects*, i.e., school quality may be correlated with other family characteristics that co-determine children's educational attainment. In response, we follow the control function approach suggested in Altonji and Mansfield (2018), i.e., we use group-level averages of observable characteristics to remove all cross-group variation that results from sorting into school catchment areas. We also calculate different summary statistics to quantify the potential magnitude of confounding by unobservables (Cinelli and Hazlett, 2020; Oster, 2019). The results suggest that any residual confounding from selection effects is small and unlikely

[2] In addition, PGI^{EA} is highly predictive of several life outcomes closely related to educational attainment. These outcomes include earnings, wealth and (non-)cognitive skills (Barth et al., 2020; Buser et al., 2024; Demange et al., 2021; Houmark et al., 2024; Lee et al., 2018; Muslimova et al., 2020; Papageorge and Thom, 2020).

to overturn our main results. Lastly, gene-environment interactions can only be identified if genetic endowments and the environmental variable of interest are uncorrelated. This prerequisite may be violated if children are sorted into schools based on their genetic endowments. In response, we show that we cannot reject the equality of PGI^{EA} distributions at different levels of school quality and that the correlation between PGI^{EA} and school quality is close to zero. These results speak against selection into schools based on genetic endowments. In summary, all our tests point to the satisfaction of crucial identification assumptions and provide support for the between-family design adopted in this paper.

Our results can be summarized as follows. First, genetic endowments and school quality are highly predictive of years of education: a one-standard-deviation increase in PGI^{EA} (school quality) increases educational attainment by ≈ 0.36 (0.12) years.[3] Second, genetic endowments and school quality are substitutes in the production of educational attainment: a one-standard-deviation increase in school quality reduces the positive association between educational attainment and PGI^{EA} from 0.36 to 0.29 years—a decrease of $\approx 19\%$. This result implies that improvements in school quality may reduce educational inequalities that follow from the genetic predispositions of individuals.

We perform various robustness checks. First, we conduct additional tests to inquire about the satisfaction of our main identification assumptions. To that end, we account for all possible interactions between our variables of interest and a broad set of family background characteristics (Feigenberg et al., forthcoming; Keller, 2014). Moreover, we use flexible specifications, including higher-order polynomials of PGI^{EA} and our measures for school quality, to test our functional form assumptions (Biroli et al., 2022). Additionally, we use a subsample of our data to estimate value-added models, including lagged test scores and GPAs, to check for sorting into schools. In all these robustness analyses, our main results remain unaffected. Second, we show that our measures for school quality do not pick up the effects of other school characteristics that may correlate with student outcomes. These characteristics comprise school-level policies such as the prevalence of ability grouping and retention as well as the demographic composition of teachers.

We also analyze mechanisms that underpin the substitutability of genetic endowments and school quality. First, we inquire whether our results are driven by parental responses to PGI^{EA} and school quality. We find that parents adjust their time investments and parenting styles as a function of their children's PGI^{EA}. However, we cannot detect differential effects depending on school quality.

Second, to uncover which types of skills drive our results, we analyze the associations of PGI^{EA} and school quality with a range of intermediate outcomes that are highly predictive of educational attainment. These intermediate outcomes include cognitive skills, economic

[3] These increases correspond to 16% (6%) of a standard deviation.

preferences, personality measures, and health. We find substitutability of genetic endowments and school quality for developing verbal intelligence, risk-aversion, patience, and subjective health. Hence, we consider these intermediate outcomes plausible transmission channels for the overall result regarding educational attainment. Furthermore, educational attainment summarizes information from various educational stages, where each stage requires a different mix of skills (Cunha et al., 2006, 2010). Therefore, we repeat our analysis by replacing years of education with binary variables indicating whether respondents have obtained a given educational degree. We find substitutability of genetic endowments and school quality for high school and college graduation, but there is no substitutability for post-graduate degrees. This pattern stands in notable contrast to the gene-environment interaction for family SES. Consistent with evidence from recent studies (Buser et al., 2024; Papageorge and Thom, 2020), we show that the interaction of genetic endowments and family SES switches its sign as individuals progress through the educational system and that there is a substantial complementarity for the attainment of post-graduate degrees. The contrast between these patterns indicates that gene-environment interplay varies across different sources of investments in children: relative to their high-PGI^{EA} peers, children with lower PGI^{EA} consistently gain more from attending a high-quality school than from having a high-SES family background.

Our study contributes to four strands of literature. First, we contribute to the literature on gene-environment interactions. Extensive literature shows that the association between genetic endowments and life outcomes varies with family SES (Figlio et al., 2017; Houmark et al., 2024; Papageorge and Thom, 2020; Ronda et al., 2022). However, evidence on gene-environment interactions in the school context is scant. Houmark et al. (2022) show that genetic gaps in achievement among Danish students increase across grades 2–8, particularly among children from low-SES families. In contrast to their study, we do not focus on the dynamics of genetic gradients across the educational biography of students but investigate whether school quality can moderate these gradients. Barcellos et al. (2021) use a regression discontinuity design to show relative gains in educational attainment for low-PGI^{EA} students after a compulsory schooling reform in the UK. In contrast to their paper, we do not focus on variation in the length of schooling but on variation in the quality of schools. Trejo et al. (2018) investigate whether the socioeconomic composition of schools moderates the strength of genetic gradients in educational and occupational attainment. Using data from Add Health and the Wisconsin Longitudinal Study, they find inconclusive evidence for gene-environment interplay. In contrast to their study, we do not focus on school contexts, e.g., the socioeconomic composition of schools, but on quality indicators that reflect school practices and policies: teacher experience, teacher turnover, teacher education, and class size.[4] This shift of focus also has methodological implications. The socioeconomic composition of schools directly reflects endogenous sorting into schools—potentially leading to gene-environment correlations. Since the independent distribution of genetic endowments and environmental variables is a prerequisite for identifying

[4]See also Jennings et al. (2015) and Raudenbush and Willms (1995) for the conceptual distinction between school context and school practice when estimating school effects.

their interaction, interpreting gene-environment interactions with school composition variables is not straightforward. To the contrary, we show that our measure of school quality is uncorrelated with PGI^{EA}, and we strengthen identification by using the control function approach of Altonji and Mansfield (2018) to account for cross-group variation that results from endogenous sorting into schools.

Second, we contribute to the literature on school quality. A broad literature demonstrates that school quality raises educational attainment, wages, and health and reduces crime (Beuermann and Jackson, 2022; Beuermann et al., 2022; Deming, 2011; Deming et al., 2014). Specifically, teacher quality, which is a core component of our school quality measure, has been shown to improve student outcomes in the short- and long-term (Chetty et al., 2014a,b; Jackson, 2019; Rivkin et al., 2005; Rockoff, 2004). However, which student groups benefit the most from school quality is less known. On the one hand, disadvantaged students may benefit more, as they have more room for improvement; on the other hand, they may benefit less, as they may not have the means to take advantage of better schools (Cunha et al., 2010). In line with the ambiguity of theoretical predictions, the empirical evidence is mixed. For example, Walters (2018) show that disadvantaged students benefit more from admission to charter schools in Boston, while Dustan et al. (2017) show that disadvantaged students benefit less from admission to elite public high schools in Mexico City. Many studies in this literature analyze samples of applicants to charter or elite schools, which are not representative of disadvantaged students in general. A notable exception is a study by Jackson et al. (2024), who use a sample of all schools and students in Chicago's public school district to demonstrate that disadvantaged students gain relatively more from higher-quality schools. In contrast to these previous studies, which have evaluated heterogeneity by socioeconomic status, we focus on genetic endowments as the source of disadvantage. The focus on genetic endowments is highly relevant since they are fixed at conception and cannot be influenced by individual choice. Therefore, they constitute a source of inequality perceived as unfair by many people in the United States and other Western societies (Almås et al., 2020). Furthermore, they have substantial explanatory power for educational attainment that is not captured by standard measures of disadvantage, like family SES.[5] Using a representative sample of schools from all over the United States, we show that students with lower values of PGI^{EA} gain relatively more from high-quality schools than their higher-PGI^{EA} peers.

Third, our study contributes to the literature on human capital formation, which estimates the degrees of substitutability or complementarity between different inputs (Cunha and Heckman, 2008; Cunha et al., 2010). Most of this literature focuses on parental investments. It shows that the returns to such investments are usually highest during early childhood and that the largest returns accrue to individuals with low prior skills (Agostinelli and Wiswall, forthcoming). We contribute to this literature by focusing on the interaction of genetic endowments with high

[5]See Supplementary Figure S.1 where we show that PGI^{EA} is correlated with family SES but that PGI^{EA} distributions have significant overlap at different levels of family SES. This pattern suggests that genetic endowments are a distinct dimension of student advantage that cannot be captured by family SES alone.

school quality—an input factor more directly subject to policy intervention than parental investments.[6] Our finding that children with lower PGI^{EA} gain disproportionately more from attending a high-quality school than from having a high-SES family background is consistent with recent evidence documenting significant relative gains of disadvantaged students from attending high-quality schools in the United States (Cohodes et al., 2021; Jackson et al., 2024). It also has wider implications for policies to address inequalities in human capital formation. While parental investments tend to reinforce initial skill differences (e.g., Houmark et al., 2024), our results show that high-quality schools reduce them. This suggests that policies channeling monetary resources to schools rather than families may address human capital inequalities more broadly.

Lastly, our paper relates to the literature on educational inequality and intergenerational educational mobility (Blanden et al., 2023; Chetty et al., 2014c,d; Hanushek et al., 2021; Rossin-Slater and Wüst, 2020). This literature shows that educational inequality is highly persistent across generations, partly explained by the intra-family transmission of genetic endowments. Our results suggest that investments in school quality can alleviate intergenerational persistence in the United States.

The remainder of this paper is structured as follows. In section 2, we introduce the measurement of genetic endowments. In section 3, we detail our empirical strategy. After describing our data sources in section 4, we present results in section 5. Section 6 concludes the paper.

2 MEASURING GENETIC ENDOWMENT

The "First Law of Behavior Genetics" states that all human traits are heritable; that is, genetic endowments explain the expression of each trait, at least to some extent (Turkheimer, 2000). The empirical challenge is to identify specific sequences in the genome related to the traits of interest.[7] Recent advances in molecular genetics have enabled a novel method of genetic discovery: genome-wide association studies (GWAS). GWAS exploit the most common genetic variation between humans, so-called single-nucleotide polymorphisms (SNPs). SNPs occur when a single nucleotide—the basic building block of DNA molecules—differs at a specific position in the genome. Humans have around ten million SNPs. GWAS estimate separate linear regressions that relate the SNP of individual i at genome location j to an outcome of interest y:

$$y_i = \psi_j^y SNP_{ij} + \delta C_i + \varepsilon_i. \tag{1}$$

[6] Houmark et al. (2024) incorporate genetic variation into a skill formation model. However, they also focus on family investments and do not estimate gene-environment interactions with inputs from other sources.

[7] Human genetic information is stored in 23 chromosome pairs containing deoxyribonucleic acid (DNA) molecules. These chromosomes, in turn, contain 20,000 to 25,000 genes—specific DNA sequences that provide instructions for building proteins. More than 99% of the sequences are identical in all humans.

$SNP_{ij} \in \{0, 1, 2\}$ is a count variable and indicates the number of minor alleles that individual i possesses at location j. Minor alleles are the less frequent genetic variants within a population. As humans inherit one of each chromosome from each parent, they possess either zero, one, or two minor alleles at each location j. C_i is a vector of control variables to filter out spurious correlations due to non-biological differences across population groups. A particular SNP coefficient ψ_j^y is considered genome-wide significant if the null hypothesis of non-association is rejected at a level of $p < 5 \times 10^{-8}$ (Chanock et al., 2007). The p-value is set low to account for multiple hypothesis testing.

The association of single SNPs with y is small, but jointly they can explain a substantial share of observed outcome differences between individuals (Lee et al., 2018). In particular, the estimated SNP coefficients can be used to construct polygenic indexes (PGI). PGIs are scalar measures of an individual's genetic predisposition for an outcome y relative to the population. Formally, PGI_i^y are constructed as a linear aggregation of SNP_{ij} using the GWAS coefficients $\widehat{\psi}_j^y$ as weights:

$$PGI_i^y = \sum_j \widehat{\psi}_j^y SNP_{ij}. \tag{2}$$

PGIs are now available for a variety of outcomes. These include, for example, the body mass index and height (Yengo et al., 2018), attention deficit hyperactivity disorder (Demontis et al., 2019), major depressive disorder (Howard et al., 2019), intelligence (Savage et al., 2018), smoking (Liu et al., 2019), and sleep duration (Jansen et al., 2019). For our analysis, we rely on the PGI for educational attainment based on the GWAS of Lee et al. (2018), which we denote by PGI^{EA}. It is based on information from 1.1 million individuals and explains around 12.7% of the variance in educational attainment in the United States. PGI^{EA} is constructed including all SNP_{ij}, not just those reaching genome-wide significance. This is common practice to maximize the predictive power of PGIs. Furthermore, to avoid overfitting, the weights $\widehat{\psi}_j^y$ are estimated on a meta-analysis sample that excludes our main dataset Add Health (Wray et al., 2014). In turn, PGI^{EA} is constructed by applying the estimated $\widehat{\psi}_j^y$ to the SNP counts in the Add Health sample.

The interpretation of PGIs is not trivial. First, PGIs are not pure measures of biological influence but may correlate with environmental factors. For example, GWAS coefficients may capture population stratification across geographic regions (Abdellaoui et al., 2019). To address this concern, we follow standard practice in the literature and always control for the first 20 principal components of the genetic data in our empirical analysis. Furthermore, we replicate our estimates for genetic effects using within-sibling variation in PGIs, bolstering confidence that environmental influences do not confound our estimates. Second, PGIs are noisy measures of genetic endowments. For example, GWAS coefficients are estimated in finite samples, leading to measurement error in the PGI weights. Furthermore, the explanatory power of PGIs depends on the context of their application. If a PGI is applied in one context while the underly-

ing GWAS is estimated in another context, the predictive power of the PGI will be attenuated.[8] To address the concern of attenuation bias due to measurement error, we use the procedure of Becker et al. (2021) and provide robustness checks based on measurement-error-corrected estimates for the coefficients of PGI^{EA} and the gene-environment interaction. These analyses suggest that our conclusions about the relative strength of gene-environment interactions are not affected by measurement error in PGI^{EA}.

3 EMPIRICAL STRATEGY

3.1 Conceptual framework

In this section, we provide a conceptual framework to guide our analysis. We illustrate a stylized model of how genetic endowments and school quality influence children's educational attainment. This illustration builds on Houmark et al. (2024), who model skill formation as a function of genetic endowments and family investments. We differ from Houmark et al. (2024) in the following main aspects. First, we abstract from the effect of parental genetic endowments on skill formation. Second, we focus on adolescents during high school. Therefore, we include school quality in our analysis. Third, we focus on educational attainment instead of skills as the main outcome of interest. However, the model may be easily modified to multiple skill dimensions following Cunha and Heckman (2007), Cunha and Heckman (2008), and Cunha et al. (2010).

We consider a model in which children's educational attainment is a function of initial endowments and inputs received during high school. In particular, educational attainment Y of child i is determined by genetic endowments G, family investments during high school I^F, high school quality I^S, skills at the beginning of high school θ, and stochastic shocks ϵ:

$$Y_i = f(G_i, I_i^F, I_i^S, \theta_i, \epsilon_i).$$

Genetic endowments are determined at conception. Family investments may include health behaviors of parents, monetary investments such as buying books, and time investments such as helping with homework. They may also include parenting styles (Agostinelli et al., forthcoming). School quality may capture teachers' instructional skills, school policies to regulate behavior, or the existence of programs for college guidance and career mentoring.

We are especially interested in the interplay of genetic endowments G and high school quality I^S. Note that the genetic effects of interest are partly reflected in prior skills θ. To capture the

[8] For example, educational attainment in a country without compulsory schooling likely correlates with a different set of genetic endowments than in a country with high-quality compulsory schools. In our study, this concern is limited: we apply PGI^{EA} to a sample from the United States, while the underlying GWAS predominantly draws on samples from the United States and other industrialized countries with similar education systems (Lee et al., 2018).

overall genetic effect on educational attainment, we replace θ with $\theta^{\perp}$, which is the part of θ that is orthogonal to genetic endowments G. $\theta^{\perp}$ can be interpreted as a summary statistic for the full history of skill-relevant inputs from parents, schools, and stochastic shocks up to high school that are unrelated to children's genetic endowments:

$$Y_i = f(G_i, I_i^F, I_i^S, \theta_i^{\perp}, \epsilon_i). \tag{3}$$

The outlined technology suggests that genetic endowments G and school quality I^S could influence children's educational attainment via four channels: (i) directly, (ii) indirectly by affecting family investments I^F, (iii) indirectly by affecting each other, or (iv) indirectly by affecting prior skills $\theta^{\perp}$.

In this exposition, we will only focus on (i) and (ii) for the following reasons. First, under our definition of prior skills, $\partial\theta_i^{\perp}/\partial G_i = \partial\theta_i^{\perp}/\partial I_i^S = 0$, allowing us to abstract from indirect effects via these channels. Second, we assume $\partial I_i^S/\partial G_i = 0$, allowing us to abstract from indirect genetic effects through school quality. This assumption is substantiated by empirical evidence on the non-correlation of genetic endowments and school quality. We will discuss this evidence in detail in the empirical section of the paper (Figure 2, Supplementary Figure S.3, and Supplementary Table S.2). Similarly, high school quality cannot influence genetic endowments such that $\partial G_i/\partial I_i^S = 0$.

Based on our assumptions, the effects of genetic endowments and school quality on educational attainment can be decomposed into (i) and (ii), which we call *skill effects* and *investment effects*, respectively:[9]

$$\frac{\partial Y_i}{\partial G_i} = \underbrace{\frac{\partial f(\cdot)}{\partial G_i}}_{\text{Skill effect}} + \underbrace{\frac{dI_i^F}{dG_i} \times \frac{\partial f(\cdot)}{\partial I_i^F}}_{\text{Investment effect}}; \qquad \frac{\partial Y_i}{\partial I_i^S} = \underbrace{\frac{\partial f(\cdot)}{\partial I_i^S}}_{\text{Skill effect}} + \underbrace{\frac{dI_i^F}{dI_i^S} \times \frac{\partial f(\cdot)}{\partial I_i^F}}_{\text{Investment effect}}. \tag{4}$$

Furthermore, the effect of genetic endowments on educational attainment may vary with the quality of schools, and vice versa. This gene-environment interaction can again be decomposed

[9] The genetic *skill effect* differs from the *direct effect* in Houmark et al. (2024). Their *direct effect* only considers the contemporaneous effect of G over and above prior skills but not the cumulative effect of genetic endowments on skill formation in the past.

into a *skill effect* and an *investment effect*:

$$\frac{\partial^2 Y_i}{\partial G_i \partial I_i^S} = \underbrace{\frac{\partial^2 f(\cdot)}{\partial G_i \partial I_i^S}}_{\text{Skill effect (GxE)}} + \underbrace{\frac{dI_i^F}{dG_i} \times \frac{\partial^2 f(\cdot)}{\partial I_i^F \partial I_i^S} + \frac{dI_i^F}{dI_i^S} \times \frac{\partial^2 f(\cdot)}{\partial I_i^F \partial G_i} + \frac{d^2 I_i^F}{dG_i dI_i^S} \times \frac{\partial f(\cdot)}{\partial I_i^F} + \frac{dI_i^F}{dG_i} \times \frac{dI_i^F}{dI_i^S} \times \frac{\partial f^2(\cdot)}{\partial^2 I_i^F}}_{\text{Investment effect (GxE)}}. \tag{5}$$

Equation 5 shows that the sign of the gene-environment interaction is theoretically ambiguous for both the skill and the investment effects. We illustrate this ambiguity using several examples. The skill effect in the gene-environment interaction captures heterogeneous returns to school quality along the distribution of genetic predispositions. For example, children with a genetic predisposition for learning difficulty may benefit more strongly from high-quality teachers, or vice versa. The investment effect in the gene-environment interaction captures four components. First, the impact of genetic endowments on family investments (dI_i^F/dG_i) mediated through the interactive effect of family investments and school quality on educational attainment $(\partial^2 f(\cdot)/\partial I_i^F \partial I_i^S)$. For example, parents may invest more in high-PGI children, but their investment may have lower marginal returns if their child is in a high-quality school, or vice versa. Second, the impact of school quality on family investments (dI_i^F/dI_i^S) mediated through the interactive effect of family investments and genetic endowments on educational attainment $(\partial^2 f(\cdot)/\partial I_i^F \partial G_i)$. For example, parents may increase time investments to compensate their children for a low-quality school, and their investment may have higher marginal returns if their child has a high PGI, or vice versa. Third, the joint effect of genetic endowments and school quality on family investments $(d^2 I_i^F/dG_i dI_i^S)$ mediated through the impact of family investments on educational attainment $(\partial f(\cdot)/\partial I_i^F)$. For example, parents may increase their time investment especially when their children are facing both disadvantageous genetic endowments and a low-quality school, or vice versa. Lastly, there are potential nonlinear effects of family investments on children's educational attainment $(\partial f^2(\cdot)/\partial^2 I_i^F)$, from which we will however abstract in the following for the sake of parsimony.

3.2 Estimation approach

We construct an empirical analog of the education production function in equation 3 as follows. First, we obtain measures for its underlying components from the available data. For our baseline estimates, we use completed years of education as a summary measure of educational attainment. We measure children's genetic endowments using the polygenic score PGI^{EA}. Furthermore, we construct a summary index for the quality of high schools children attend, which we denote by Q^{S}. See also section 4 for a detailed description of these variables.

Second, drawing on these data inputs, we make the following functional form assumptions to estimate equation 3:

$$Y_i = \alpha_0 + \alpha_1 PGI_i^{EA} + \alpha_2 Q_i^S + \alpha_3 (Q_i^S \times PGI_i^{EA}) + \alpha_x \mathbf{X}_i + \xi_i, \tag{6}$$

where $\mathbf{X}$ denotes a vector of predetermined control variables that is necessary to estimate the parameters of interest. ξ is a composite error terms that capture idiosyncratic technology shocks and various non-linear effects of PGI^{EA} and Q^{S}.

Throughout the paper, we present multiple empirical tests to defend the underlying functional form assumptions and to show that ξ is orthogonal to our variables of interest. We will briefly discuss two examples in the following. First, the empirical specification of the skill formation model assumes that conditional on $\mathbf{X}$, prior skills $\theta^{\perp}$ and their interactions with genetic endowments and school quality can be excluded from the estimation model. We support this assumption by showing that our baseline effects replicate in a value-added model where we explicitly control for prior skills and their interactions with our variables of interest (Table 4). Second, we assume that $\text{PGI}^{\text{EA}}$and Q^{S} influence educational attainment linearly and through their interaction while abstracting from more complicated functional forms. We support this parsimonious model specification by showing that our results are robust to alternative functional forms, including higher-order polynomials and their interactions (Table 4).

If $\alpha_3 < 0$, genetic endowments and school quality are *substitutes* in the production of educational attainment, i.e., high-quality schools are more productive for individuals endowed with a relatively low PGI^{EA}. Reversely, if $\alpha_3 > 0$, genetic endowments and school quality are *complements* in the production of educational attainment, i.e., high-quality schools are more productive for individuals endowed with a relatively high PGI^{EA}.

Note that α_3 captures the composite of skill effects and investment effects. In section 5.4, we investigate the importance of investments effects. In particular, we measure family inputs by constructing indexes for parental time investments and (authoritarian) parenting styles (Agostinelli et al., forthcoming). We denote these indexes by Q^{F} and use them as the outcomes of interest:

$$Q_i^F = \gamma_0 + \gamma_1 PGI_i^{EA} + \gamma_2 Q_i^S + \gamma_3 (Q_i^S \times PGI_i^{EA}) + \gamma_x \mathbf{X}_i + \mu_i, \tag{7}$$

where γ_1, γ_2, and γ_3 estimate $\frac{dI_i^F}{dG_i}$, $\frac{dI_i^F}{dI_i^S}$ and $\frac{d^2 I_i^F}{dG_i dI_i^S}$ from equation 5. If $\gamma_1 = \gamma_2 = \gamma_3 = 0$, investments effects are unlikely to explain the gene-environment interaction α_3.

3.3 *Conditions for identification*

The gene-environment interaction α_3 is identified if the following conditions are met: (i) exogenous variation in PGI^{EA}, (ii) exogenous variation in Q^S, and (iii) absence of gene-environment correlations between PGI^{EA} and Q^S (Almond and Mazumder, 2013; Biroli et al., 2022; Johnson and Jackson, 2019; Nicoletti and Rabe, 2014). Furthermore, it needs to be the case that (iv) the interactions between $\mathbf{X}$ with PGI^{EA} and Q^S can be excluded from the model (Feigenberg et al., forthcoming; Keller, 2014). In the following, we discuss these conditions, potential threats to their satisfaction, and how we address these threats.

(i) Exogenous variation in PGI^{EA}. Genetic endowments are not exogenous to family characteristics as the genetic endowments of children are drawn from the genetic pool of their biological parents. Consequently, PGI^{EA} is a function of maternal and paternal genetic endowments that may correlate with prior family and school inputs. Hence, when estimating equation (6), α_1 and α_3 may be confounded by *genetic nurture effects* (Kong et al., 2018). In particular, a positive correlation may exist between advantageous genetic endowments and family environments in which children receive more investments. Genetic nurture can be controlled by estimating a sibling fixed effects model that relies on within-family variation in PGI^{EA} only (Houmark et al., 2024; Kweon et al., 2020; Selzam et al., 2019); in a non-transmitted genes design, in which both maternal and paternal genetic endowments are included in the control vector $\mathbf{X}$; or in an adoption design, in which offspring are biologically unrelated to their parents.[10] Note that all these designs are very data-demanding. For example, the sibling design requires a large sample of biological siblings with sequenced DNA data to construct PGI^{EA}. Therefore, it can only be applied to a limited set of existing data sets.

In this study, we estimate a between-family model using an extensive set of pre-determined family background characteristics to control for genetic nurture. This approach is standard in the literature and aims to approximate condition (i) while maximizing statistical power to estimate the gene-environment interaction α_3 (Domingue et al., 2020). We formally assess the residual potential for confounding through genetic nurture effects by comparing the estimates of the genetic base effect α_1 from the between-family model to a sibling fixed effects model that we estimate on a subset of our data ($N = 677$, Table 2). Reassuringly, we cannot reject the null hypothesis that they are equal ($p = 0.898$). This result suggests that after conditioning on $\mathbf{X}$, residual genetic nurture is close to zero and unlikely to overturn our main findings.

(ii) Exogenous variation in Q^S. School quality is not exogenous to family characteristics as parents choose schools for their children (Altonji et al., 2005; Beuermann et al., 2022; Rothstein, 2006). Consequently, Q^S is a function of family and child characteristics that may correlate

[10] See Demange et al. (2022) for a detailed comparison of all three approaches.

with prior family and school inputs. Hence, when estimating equation (6), α_2 and α_3 may be confounded by *selection effects* (Altonji et al., 2005; Altonji and Mansfield, 2018). In particular, there may be a positive correlation between school quality and family environments where children receive more investments. Sorting into schools can be controlled in quasi-experimental settings, e.g., by using variation from admission lotteries (Angrist et al., 2016; Cullen et al., 2006), or the geographic design of catchment areas (Laliberté, 2021). However, existing data sets that avail such variation do not contain sequenced DNA data needed to measure genetic endowments at the individual level.

In this study, we use an extensive set of pre-determined family background characteristics to account for selection into schools. Furthermore, we apply the control function approach proposed by Altonji and Mansfield (2018). In their paper, Altonji and Mansfield (2018) consider settings where individuals sort into treatments, e.g., when families move to neighborhoods based on school quality. They show that group-level averages of observable characteristics are correlated with unobservable characteristics. Based on this insight, they suggest that controlling for a limited number of group-level averages may remove all cross-group variation in observable and unobservable characteristics. Agrawal et al. (2019) extend this result to settings where the variable of interest is an interaction between group-level factors, such as Q^S, and observed individual characteristics, such as PGI^{EA}. We provide supporting evidence for their conjecture in our setting by showing that school-level averages for only five student characteristics remove all correlation between educational attainment and an extensive set of measures for family background, including parental education and wages (Table 2). In addition, we formally assess the sensitivity of our results to residual confounding by calculating summary statistics for selection on unobservables (Cinelli and Hazlett, 2020; Oster, 2019). Reassuringly, these summary statistics consistently point to a low potential for a selection on unobservables (Supplementary Figure S.2). These results suggest that after conditioning on $\mathbf{X}$, residual confounding due to selection into schools is low and unlikely to overturn our main findings. We further support this assertion in Appendix C. This appendix shows analytically and based on simulations that any residual confounding due to positive selection into schools will likely attenuate our estimate for the gene-environment interaction α_3 towards zero.

(iii) Absence of gene-environment correlation between PGI^{EA} and Q^S. In addition to conditions (i) and (ii), PGI^{EA} and Q^S have to be uncorrelated. A strong correlation between PGI^{EA} and Q^S would imply little variation in PGI^{EA} at different levels of Q^S and vice versa. As a consequence, there would be insufficient variation to identify $\alpha_1, \alpha_2,$ and α_3 separately from each other.

To verify that condition (iii) is satisfied, we present graphical evidence and formal tests to compare PGI^{EA} distributions at different levels of school quality. The distributions are largely overlapping—both unconditionally and conditional on $\mathbf{X}$ (Figure 2 and Supplementary Figure S.3). We note that this finding does not imply that parents choose schools randomly. In fact,

in Supplementary Table S.1, we show a positive correlation of PGI^{EA} with peer characteristics. However, consistent with existing literature, this pattern suggests that parents select schools based on geographical proximity and peer quality but not necessarily based on the school practices underpinning our school quality measure Q^S (Abdulkadiroğlu et al., 2020; Beuermann et al., 2022).

(iv) Irrelevance of interacted controls X. We identify the parameters of interest by conditioning on **X**. Feigenberg et al. (forthcoming) and Keller (2014) show that in such settings, heterogeneous treatment effects must be estimated in a model where one allows for all possible interactions between the variables of interest and the necessary control variables. This requirement can only be relaxed if these interactions do not belong in the model, i.e., if they are orthogonal to the interaction of interest or uncorrelated with the outcome of interest.

Given our relatively small sample, we present our baseline results without the full interaction of **X**. However, we perform robustness tests, including interaction terms of PGI^{EA}, Q^S, and all control variables, respectively (Table 4). Our results remain unchanged, suggesting that the interactions we collect in the composite error term ξ of equation 6 do not belong in the causal model and do not confound our parameters of interest.

In summary, an ideal design to estimate the gene-environment interaction α_3 would combine a sibling fixed effects model with experimental variation in school characteristics among children of the same biological parents. We are unaware of any data set that simultaneously includes genetic data at the individual level, a large set of siblings, and quasi-experimental within-family variation in school assignments. Therefore, we approximate the ideal-type conditions with the best data available. Within this setting, causal identification of $\alpha_1, \alpha_2,$ and α_3 relies on rather strong assumptions. We provide extensive empirical evidence that supports the satisfaction of these assumptions and the validity of our research design. Nevertheless, in the absence of clear-cut quasi-experimental variation, we choose to err on the side of caution and speak of *associations* instead *causal effects* in the remainder of the paper.

4 DATA

We use data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a 5-wave panel study focusing on the determinants of health-related behaviors and health outcomes. Add Health is a nationally representative sample of adolescents in grades 7–12 in 1994/95. Initial information (wave 1, $N = 20,745$) was collected from a stratified sample of 80 high schools across the United States and their associated feeder schools. In addition to in-depth adolescent interviews, questionnaires were administered to school representatives, parents, and roughly 90,000 students in the sampled schools. Follow-up in-home questionnaires

were collected in 1996 (wave 2, $N = 14,738$), 2001/02 (wave 3, $N = 15,179$), and 2008/09 (wave 4, $N = 15,701$). In the most recent wave (2016/18, $N = 12,300$), Add Health respondents are between 33 and 43 years old.

In the following, we describe our main variables of interest. Detailed descriptions of all variables used in our analysis are disclosed in Supplementary Material D.

Outcomes. We measure educational attainment Y by the total years of education after age 27. In each wave, respondents were asked about their highest level of education at the time of the interview. For each individual, we use the most recent information and transform education levels into years of education, following the mapping suggested by Domingue et al. (2015).[11]

To analyze the mechanisms behind our headline results, we additionally use a series of measures for family investments, (non-)cognitive skills, health, and academic degrees. First, as suggested in section 3, the estimated gene-environment interaction may be driven by parental responses to PGI^{EA} and Q^{S}. To test this hypothesis, we construct measures for parental time investments and (authoritarian) parenting styles following Agostinelli et al. (forthcoming). Second, measures for (non-)cognitive skills and health serve as proxy variables for skills after high school and allow us to analyze the dimensions of skill development that drive the main findings on educational attainment. We proxy cognitive skills using the Picture Vocabulary Test (PVT), a test of receptive hearing vocabulary that is a widely used measure of verbal ability and scholastic aptitude. We proxy non-cognitive skills by self-reported measures of general risk aversion and patience (Falk et al., 2018) and self-reported information on the Big Five personality traits (Almlund et al., 2011). Regarding health, we use quality-adjusted life years (QALY), which we derive from self-assessed health measures, as well as a summary index of diagnosed health conditions. Second, academic degrees allow us to investigate at which educational stage our results emerge. We focus on whether respondents finished high school, obtained a 2-year or 4-year college degree, or completed a post-graduate degree.

Genetic endowments. Add Health obtained saliva samples from consenting participants in wave 4. After quality control procedures, genotyped data is available for 9,974 individuals and 609,130 SNPs. Add Health uses this data to calculate different PGIs using summary statistics from existing GWAS. We use a PGI for educational attainment, referred to as PGI^{EA}, that is

[11] Numeric values in parentheses: eighth grade or less (8), some high school (10), high school graduate (12), GED (12), some vocational/technical training (13), some community college (14), some college (14), completed vocational/technical training (14), associate or junior college degree (14), completed college (16), some graduate school (17), completed a master's degree (18), some post-baccalaureate professional education (18), some graduate training beyond a master's degree (19), completed post-baccalaureate professional education (19), completed a doctoral degree (20).

based on the GWAS by Lee et al. (2018).[12]

Lee et al. (2018) perform a meta-analysis of 71 quality-controlled cohort-level GWAS. Their meta-analysis produced association statistics for around 10 million SNPs, of which 1,271 reached genome-wide significance. Genes near these genome-wide significant SNPs are relevant for the central nervous system, and many of them encode proteins that carry out neurophysiological functions such as neurotransmitter secretion or synaptic plasticity. They are relevant for brain development processes before and after birth.

PGI^{EA} is highly predictive of educational attainment and has been widely used in existing studies. Lee et al. (2018) suggest that PGI^{EA} is a better predictor for years of education than household income. Including the score in a regression of years of education on a set of controls yields an incremental R^2 of 0.127 in the Add Health sample. PGI^{EA} has been used to study the formation of early childhood skills (Belsky et al., 2016; Houmark et al., 2024), educational attainment (Domingue et al., 2015), earnings (Papageorge and Thom, 2020), wealth accumulation (Barth et al., 2020), and social mobility (Belsky et al., 2018).

School quality. In waves 1 and 2, Add Health administered detailed questionnaires to headteachers of Add Health schools. The schools are also linked to the Common Core of Data (CCD) and the Private School Survey (PSS). We use these sources to construct an indicator for Q^S using a principal component analysis that includes the following school-level inputs (component weights in parenthesis): (i) the share of teachers with a master's degree ($+0.63$), (ii) the share of teachers with school-specific tenure of more than five years ($+0.62$), (iii) the share of new teachers in the current school year (-0.36), and (iv) the average class size (-0.31). As an alternative to the principal component analysis, we also construct an indicator for Q^S using the linear aggregation method proposed by Anderson (2008) and Kling et al. (2007).

For our baseline indicator of Q^S, we only focus on school inputs for which there is strong evidence in the existing literature on how they enter the education production function. Bernal et al. (2016), Clotfelter et al. (2010), and Jacob et al. (2018) demonstrate that academic credentials, which we proxy by the share of teachers with a master's degree, are positively associated with teacher effectiveness. Papay and Kraft (2015) and Rockoff (2004) show that teaching experience, which we proxy by the share of teachers with more than five years of tenure, correlates with teacher performance and student achievement. Hanushek et al. (2016), Hill and Jones (2018), Hwang et al. (2021), and Ronfeldt et al. (2013) demonstrate that a high teacher turnover, which we proxy by the share of new teachers, impairs teaching quality and student achievement. Finally, the literature on class size reductions (Angrist and Lavy, 1999; Angrist et al.,

[12] Lee et al. (2018) construct PGI^{EA} for two prediction cohorts, Add Health and the Health and Retirement Study (HRS). PGI^{EA} is based on results from the meta-analysis in which these two cohorts were excluded from the discovery sample. PGI^{EA} was generated from HapMap3 SNPs using the software LDpred—a Bayesian method that weights each SNP by the posterior mean of its conditional effect given other SNPs.

2019; Bernal et al., 2016; Chetty et al., 2011; Fredriksson et al., 2013; Krueger, 1999; Leuven and Løkken, 2020) finds either positive or zero associations with student achievement. In a robustness check, we expand Q^S by including information on other school policies (retention, ability grouping, and school sanctions) and a private school indicator. Our results remain unaffected. However, as we do not have independent exogenous variation for each component of Q^S, our school quality measure may also pick up unobserved factors that correlate with the components of Q^S. This correlation is not a threat to identification but matters for interpretation. In particular, we caution against overinterpreting the importance of the specific components of Q^S for the results.

Control variables. Add Health provides detailed information on the environments to which respondents were exposed during childhood and in school. We approximate the identification prerequisites discussed in section 3 by choosing a vector $\mathbf{X}$ that includes an extensive set of predetermined variables for child and family characteristics as well as school-level averages of observable characteristics to implement the control function approach by Altonji and Mansfield (2018).

We control for child characteristics by including child age at the time of the survey (in months), biological sex, the interaction of child age with biological sex, and an indicator for firstborns. Furthermore, we follow standard practice in the literature and account for population stratification in genetic endowments by including the first 20 principal components of the full matrix of genetic data.

We control for family background characteristics by including maternal and paternal education (in years), the family's religious affiliation (Christian/non-Christian), parental birthplace (US/non-US), and maternal age at birth (in years). Furthermore, we include the mean and standard deviation of potential wages for mothers and fathers across children ages 0–14.[13] Furthermore, all estimations include a vector of state-fixed effects. We focus on predetermined variables fixed before the observation period to avoid smearing through "bad controls" (Angrist and Pischke, 2009).

To further account for selection into schools, we include a control function based on group characteristics that are observable at the school level. The in-school questionnaires in wave 1 of Add Health collect information about approximately $90,000$ students in Add Health schools. We use these data to construct five variables for the control function: the share of white peers, the share of peers with single mothers, the average years of education of peers' mothers, the share of female peers, and the share of peers with a migration background. All school-level

[13]Note that Add Health contains information on actual income. However, actual income may be a "bad control" as it reflects parental responses to both PGI^{EA} and Q^S. Therefore, we follow the procedure of Shenhav (2021) and combine data from the 1970 Census and the March Current Population Survey (1975–2000) to construct potential wages for gender/education/census region/race/ethnicity cells and match these potential wages to parents of children aged $a = 1, ..., 14$.

averages are calculated while excluding the respondent's cohort to avoid mechanical relationships.[14]

Analysis sample. We apply the following sample selection criteria. First, we restrict our sample to genotyped respondents of European descent. This restriction is common practice in the literature because GWAS are predominantly conducted on samples of European ancestry. Different ancestry groups are characterized by different allele frequencies and linkage disequilibrium structures. Therefore, PGIs derived from a GWAS on a particular population group have less predictive power in other population groups (Martin et al., 2017; Ware et al., 2017). For example, it has been demonstrated that PGI^{EA} has a much lower predictive power for the educational attainment of African Americans (Lee et al., 2018), potentially leading to biased estimates of the genetic base effects and gene-environment interactions.

Second, our main sample comprises individuals who attended an Add Health high school or an associated feeder school in wave 1. We focus on high school quality only. Therefore, we assign students who attended a feeder school in wave 1 the school quality measure of the high school to which they were scheduled to transfer. For a subset of individuals from the feeder schools, we do not have information about whether they transferred to the designated Add Health school.[15] In a robustness check presented in Supplementary Table S.3, we drop these individuals from the sample. In another robustness check, we additionally exclude respondents for whom we do not know whether they graduated from an Add Health high school. We note that neither of these alternative sample restrictions overturns our main conclusions.

Third, we drop all observations with missing information in Y, PGI^{EA}, Q^{S}, and $\mathbf{X}$ by list-wise deletion.

Applying these restrictions, we obtain a sample of $4,036$ individuals from 72 high schools across the United States, for which we provide summary statistics in Table 1. 54% are female, and the average age measured at wave 1 is $\approx$ 16 years (192 months). Our sample's average educational attainment after age 27 is 14.7 years, which exceeds the average educational attainment in the parental generation by $\approx$ 1.1 years. 96% graduate from high school, which is unsurprising given that our sample is restricted to individuals of European descent

[14]The resulting school-level averages contain information from cohorts that follow a respondent at a particular school. This may raise concerns about a bad control problem since selection patterns in subsequent cohorts could reflect student outcomes in contemporaneous cohorts. In Supplementary Table S.2, we use a subsample of our data to compare estimates based on the leave-cohort-out specification and an alternative where we leave out all subsequent cohorts that follow a respondent. Note that this sample is restricted by excluding the oldest Add Health cohort (i.e., those respondents who are in grade 12 at wave 1) since we do not have information on their predecessors. Results are very similar in both control function specifications. This result suggests that the potential bias from including subsequent cohorts is negligible, and we present our headline results on the larger sample.

[15]In the subsample of feeder school students for whom high school graduation transcripts are available, 67% have indeed graduated from their designated Add Health high school. Since students can drop out or change schools before graduation, we consider this a lower bound for having ever attended the designated Add Health high school.

TABLE 1 – Summary Statistics

	N=4,034; High Schools=72			
	Mean	SD	Min	Max
Educational Attainment				
Years of Education	14.68	2.27	8.00	20.00
High School Degree	0.96	0.20	0.00	1.00
2-year College Degree	0.50	0.50	0.00	1.00
4-year College Degree	0.39	0.49	0.00	1.00
Post-Graduate Degree	0.14	0.35	0.00	1.00
Child and Family Characteristics				
PGI^{EA}	0.00	1.00	-4.18	3.40
Female	0.54	0.50	0.00	1.00
Firstborn	0.48	0.50	0.00	1.00
Age in Months (Wave 1)	192.41	19.62	144.00	256.00
Maternal Age at Birth	25.33	4.83	16.00	46.08
Christian	0.82	0.38	0.00	1.00
Education Mother (in Years)	13.54	2.48	0.00	19.00
Education Father (in Years)	13.56	2.68	0.00	19.00
Foreign-born Mother	0.03	0.17	0.00	1.00
Foreign-born Father	0.03	0.16	0.00	1.00
Potential Wage/Hour Mother	12.57	1.39	9.40	14.27
Potential Wage/Hour Father	15.40	1.32	11.14	17.11
School Quality Indicators				
Q^S	0.00	1.00	-2.79	1.83
Teacher w/ MA (%)	51.20	24.11	0.00	95.00
Experienced Teacher (%)	66.65	23.43	0.00	98.00
New Teacher (%)	7.88	7.28	0.00	47.00
Class Size	24.40	4.50	12.00	38.00

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows summary statistics for the core analysis sample. The sample is restricted to genotyped individuals who (i) are of European descent, and (ii) attended an Add Health high school or an associated feeder school in wave 1. Observations with missing information in any of the displayed variables are dropped by list-wise deletion.

who attended an Add Health high school or an associated feeder school in wave 1. The 2-year college completion rate equals $\approx$ 50%.

To assess sample representativeness, we compare our analysis sample to the 1974–1983 birth cohorts of non-Hispanic Whites in the American Community Survey (ACS) and the Current Population Survey (CPS) (Supplementary Table S.4). This comparison shows a slight over-representation of females and children of young mothers in our sample. Otherwise, our sample is comparable to the corresponding groups in the ACS and CPS. In robustness analyses, we re-weight our sample to match the ACS and CPS in terms of gender composition, parents' educational attainment, and the age of mothers at birth. Our results remain unaffected (Supplementary Table S.3).

5 RESULTS

We present our results in four steps. In section 5.1, we discuss the association of educational attainment, genetic endowments, and school quality in light of the identifying assumptions discussed in section 3. In section 5.2, we present our estimates for the gene-environment interaction α_3. After robustness analyses in section 5.3, we conclude with an analysis of mechanisms in section 5.4. In all analyses, we standardize PGI^{EA}, Q^{S}, and the variables in $\mathbf{X}$ so that they have a mean of zero ($\mu = 0$) and a standard deviation of one ($\sigma = 1$).

5.1 The association of educational attainment with genetic endowments and school quality

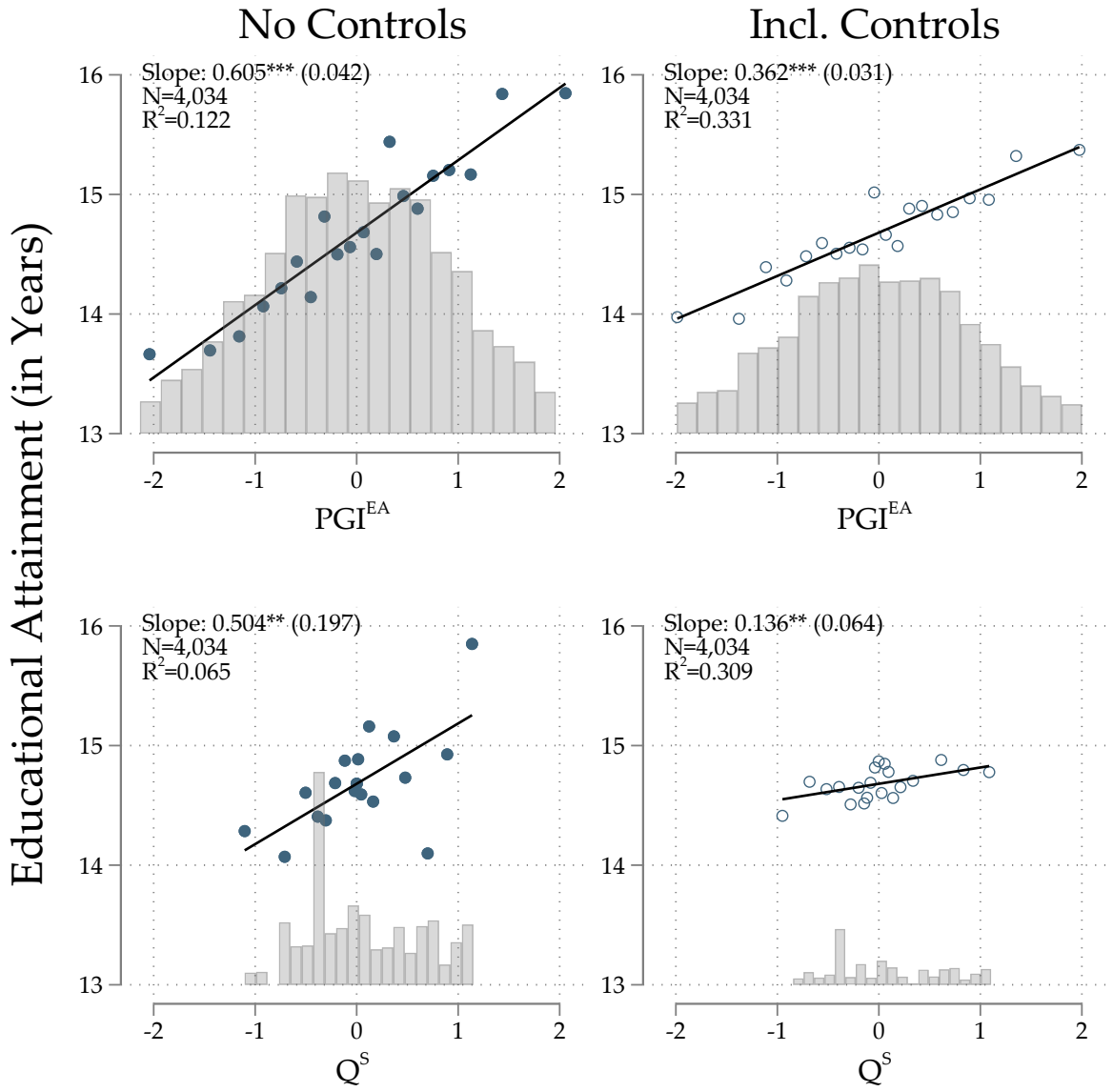
Figure 1 displays the association of educational attainment with our measures for genetic endowments PGI^{EA} and school quality Q^{S}. In the left column, we show raw correlations that do not account for the control variables $\mathbf{X}$. We show associations conditional on $\mathbf{X}$ in the right column.

First, PGI^{EA} is highly predictive of educational attainment. Without controls, an increase in PGI^{EA} by one standard deviation (1 SD) is associated with increased educational attainment of 0.605 years. This association does not have a causal interpretation as genetic nurture effects may confound it. When we control for pre-determined child and family characteristics and the control function, a 1 SD increase in PGI^{EA} is associated with an increase in educational attainment of 0.362 years. The attenuation of the association of PGI^{EA} and educational attainment is consistent with sibling studies showing that genetic nurture effects account for 40–50% of the raw association between PGI^{EA} and educational attainment (Kweon et al., 2020; Muslimova et al., 2020; Ronda et al., 2022; Selzam et al., 2019).

Is $\mathbf{X}$ sufficient to control for genetic nurture effects? We test whether there remains confounding due to genetic nurture by comparing estimates of the between-family model with a sibling fixed effects model that we estimate on a subsample of our data ($N = 677$). The within-family comparison allows us to control for genetic nurture effects perfectly. Therefore, a substantial divergence of between- and within-family estimates would suggest residual genetic nurture not picked up by $\mathbf{X}$. Table 2 shows that the between-family estimate yields a point estimate of 0.415 after controlling for $\mathbf{X}$. The within-family comparison yields a point estimate of 0.432. Both point estimates are significant at the 1%-level and very close to each other. We cannot reject the null hypothesis of their equality at conventional levels of statistical significance ($p = 0.898$). This result suggests that the potential for confounding through genetic nurture is low after conditioning on $\mathbf{X}$, lending credence to our research design.

Second, Q^{S} is highly predictive of educational attainment. Without controls, a 1 SD increase in Q^{S} is associated with an increase in educational attainment of 0.504 years. This association

FIGURE 1 – Association of Educational Attainment with PGI^{EA} and Q^S



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows the correlation of completed years of education with PGI^{EA} and Q^S, respectively. We bin scatterplots using 20 quantiles of the variable of interest. Gray bars indicate density distributions of the (residualized) variable of interest. Black lines are fitted from linear regressions of educational attainment on the variable of interest. In the left-column, we only control for state fixed effects. In the right column, we introduce the full set of control variables. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors are clustered at the school level.

does not have a causal interpretation due to sorting into schools. When we control for predetermined child and family characteristics and the control function, a 1 SD increase in Q^S is associated with an increase in educational attainment of 0.136 years. This decrease reflects pos-

TABLE 2 – Testing for Genetic Nurture and Selection into Schools

	Years of Education: Between- vs. Within-Family		Predicted Years of Education: w/o vs. w/ Control Function	
	(1)	(2)	(3)	(4)
PGI^{EA}	0.415*** (0.085)	0.432*** (0.141)	–	–
Q^S	–	–	0.264** (0.129)	0.014 (0.050)
Difference in coefficients	-0.017 (0.129) [-0.269, 0.236]		0.250** (0.115) [0.023, 0.476]	
Child Controls	✓	✓	×	×
Family Controls	✓	✓	×	×
Control Function	✓	✓	×	✓
Sibling Fixed Effect	×	✓	×	×
N	677	677	4,034	4,034
R^2	0.420	0.795	0.084	0.184
Outcome Mean	14.722	14.722	14.681	14.681
Outcome SD	2.277	2.277	1.163	1.163

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the associations of PGI^{EA} and Q^S with years of education (left panel) and predicted years of education (right panel). The left panel shows estimates in the sibling sample: Column (1) displays results from a between-family comparison. Column (2) displays results from a within-family comparison. The right panel shows estimates in the core analysis sample: Column (3) displays results without any controls. Column (4) displays results including the control function variables. Predicted education is calculated from a regression of completed years of education on all *Child Controls* and *Family Controls*. We test for the equality of coefficients in columns (1) and (2) as well as (3) and (4), respectively. Next to point estimates, we present standard errors (in parentheses), and 95% confidence intervals (in brackets). *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

itive selection into schools—a pattern thoroughly documented in the literature for the United States (Deming et al., 2014; Rothstein, 2006). Nevertheless, even when accounting for selection, the association of Q^S and educational attainment remains strong and positive. This result is consistent with prior literature showing positive effects of high school quality on students' educational success (Angrist et al., 2019; Deming, 2014; Hanushek and Rivkin, 2010; Jackson et al., 2020).

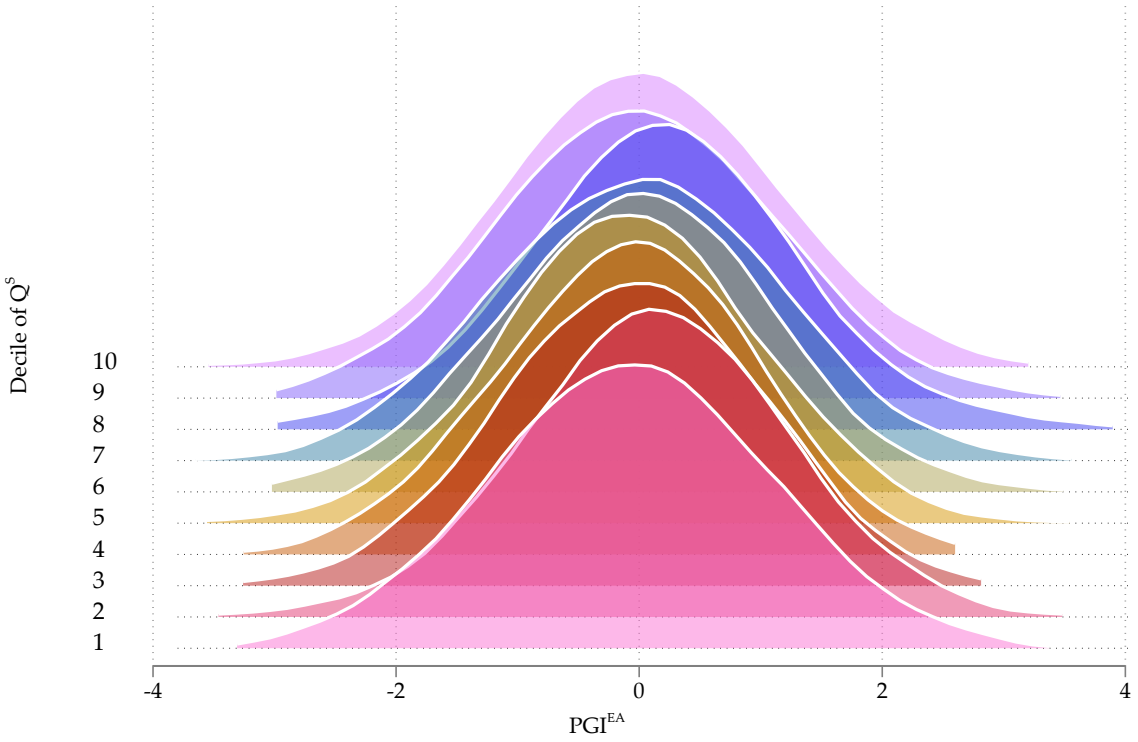
Is $\mathbf{X}$ sufficient to control for selection into schools? To control for sorting into schools, we follow the control function approach suggested in Altonji and Mansfield (2018) and include school-level averages of observed individual characteristics into $\mathbf{X}$. We test the effectiveness of this control function approach as follows: First, we replace educational attainment with pre-

dicted educational attainment. In particular, we predict years of education using the full set of pre-determined child and family characteristics included in **X**. These characteristics are highly predictive of years of education ($R^2 = 0.297$) and other measures of educational attainment and skills (Supplementary Figure S.4). Second, we assess the correlation between predicted educational attainment and Q^S before and after conditioning on the variables of the control function. If the control function approach is effective, any positive correlation between predicted educational attainment and Q^S should be attenuated to zero after conditioning on the variables of the control function. Table 2 shows that the unconditional association between predicted educational attainment and Q^S is 0.264, indicating significant positive selection into schools. Conditional on the control function, this correlation shrinks to 0.014 and becomes statistically insignificant. This result suggests that the control function approach is effective in controlling for selection into schools. Therefore, conditional on **X**, residual sorting into schools will likely be low, lending further credence to our research design. In Supplementary Table S.5, we show that this conclusion holds for all components that underpin Q^S.

Alternatively, we can assess potential confounding through remaining selection effects by assuming that changes in the coefficient of Q^S due to the introduction of **X** provide information about the extent of confounding due to unobservables (Altonji et al., 2005; Cinelli and Hazlett, 2020; Oster, 2019). We follow Cinelli and Hazlett (2020) and assess what association unobserved confounders would need to have with both the variable of interest (Q^S) and the outcome of interest (educational attainment) to change our conclusions. In Supplementary Figure S.2, we show that Q^S would remain positive and statistically significant at the 5%-level even if the partial R^2 of unobserved confounders with Q^S and educational attainment were more than ten times higher than the corresponding partial R^2 of paternal education with these variables. Given the decisive role of parental education in school choices and its strong predictive power for educational outcomes of children (Burgess et al., 2014; Hofflinger et al., 2020), these results lend further confidence that our results are genuine and not a mere reflection of selection into schools based on family background.

In addition to genetic nurture and selection effects, a high correlation between PGI^{EA} and Q^S would pose another threat to the identification of the gene-environment interaction. Such a correlation could arise if children were sorted into schools based on their genetic endowments. Figure 2 shows that this concern does not apply in our setting. In this figure, we plot unconditional PGI^{EA} distributions by decile of Q^S. Visual inspection suggests that PGI^{EA} distributions are almost congruent to each other. This conclusion also holds after residualizing PGI^{EA} and Q^S from **X** (Supplementary Figure S.3). More formally, we compute two-sample Kolmogorov-Smirnov tests for the equality of PGI^{EA} distributions within the deciles of Q^S after residualizing both variables from **X**. Out of 45 pairwise comparisons, only 3 differences are significant at the 10% level—a result consistent with chance. Similarly, we show that the correlation of PGI^{EA} and Q^S is close to zero with and without controls **X** (Supplementary Table S.1). This evidence leads us to conclude that PGI^{EA} and Q^S are likely uncorrelated and that we can

FIGURE 2 – Distribution of PGI^{EA} by Q^S



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows unconditional PGI^{EA} distribution by deciles of Q^S. Density distributions are smoothed using the Epanechnikov kernel function with a bandwidth of 0.5.

identify the gene-environment interaction of interest independently of the base effects.[16]

5.2 The interplay of genetic endowments and school quality

Table 3 shows our baseline estimates for the interaction of genetic endowments and school quality. In all regressions, we include the vector **X** to control for genetic nurture and selection into schools. As noted previously, **X** comprises an extensive set of pre-determined child and family characteristics as well as the control function using school-level averages of observed individual characteristics (see section 4).

The point estimates in column (1) replicate the findings from Figure 1 and show a strong and positive association of PGI^{EA} and Q^S with educational attainment. A 1 SD increase in PGI^{EA} (Q^S) increases educational attainment by ≈ 0.36 (≈ 0.12) years. The coefficient of the interaction $PGI^{EA} \times Q^S$ is our estimate of α_3. The negative interaction coefficient indicates

[16] In Supplementary Table S.1, we show that PGI^{EA} is correlated with the socioeconomic composition of schools but not with Q^S. All gene-environment correlations vanish once we account for sorting into schools by conditioning on **X**.

TABLE 3 – Association of Years of Education with PGI^{EA} and School Environments

	Overall		Decomposition of Q^S			
Outcome: Years of Education	PCA (1)	Anderson (2008) (2)	(3)	(4)	(5)	(6)
PGI^{EA}	0.361*** (0.028)	0.361*** (0.029)	0.360*** (0.027)	0.362*** (0.029)	0.362*** (0.030)	0.362*** (0.030)
Q^S	0.124** (0.057)	0.098** (0.048)	–	–	–	–
$PGI^{EA} \times Q^S$	-0.068*** (0.026)	-0.064** (0.028)	–	–	–	–
Teacher w/ MA	–	–	0.166** (0.071)	–	–	–
$PGI^{EA} \times$ Teacher w/ MA	–	–	-0.072*** (0.026)	–	–	–
Exp. Teacher	–	–	–	0.069 (0.059)	–	–
$PGI^{EA} \times$ Exp. Teacher	–	–	–	-0.045* (0.026)	–	–
New Teacher	–	–	–	–	-0.020 (0.047)	–
$PGI^{EA} \times$ New Teacher	–	–	–	–	0.038 (0.029)	–
Class Size	–	–	–	–	–	-0.008 (0.044)
$PGI^{EA} \times$ Class Size	–	–	–	–	–	-0.004 (0.032)
Child Controls	✓	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓	✓
N	4,034	4,034	4,034	4,034	4,034	4,034
R^2	0.333	0.333	0.334	0.332	0.332	0.331
Outcome Mean	14.681	14.681	14.681	14.681	14.681	14.681
Outcome SD	2.268	2.268	2.268	2.268	2.268	2.268

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

that genetic endowments and school quality are *substitutes* in the production of educational attainment: a 1 SD increase in school quality reduces the positive association of educational

attainment with PGI^{EA} by $\approx 19\%$ (= 0.068/0.361).[17]

In column (2), we replicate the analysis for an alternative way of constructing the school quality indicator. Instead of using weights from principal component analysis, we aggregate dimensions using the method of Anderson (2008) and Kling et al. (2007). Our results are not sensitive to this alternation: a 1 SD increase in school quality reduces the positive association of educational attainment with PGI^{EA} by $\approx 18\%$ (= 0.064/0.361).

In columns (3)-(6), we decompose the overall index Q^S into its underlying components. Columns (3) and (4) show that teacher education and school-specific tenure have positive average effects on educational attainment. These results are consistent with previous literature showing that teacher education and teaching experience positively affect student learning (Hill and Jones, 2018; Hwang et al., 2021; Jackson and Bruegmann, 2009; Rockoff, 2004). Furthermore, there is evidence for substitutability, i.e., the positive impact of more educated and more experienced teachers is more substantial for students in the lower parts of the PGI^{EA} distribution. On the contrary, Columns (5) and (6) show that the percentage of new teachers and class size neither increase average educational attainment nor is there evidence for heterogeneity across the PGI^{EA} distribution. Overall, the decomposition suggests that the results for the index Q^S are mostly driven by teachers' education levels and school-specific tenure or, given the lack of independent exogenous variation of the respective components, by factors correlated with those.

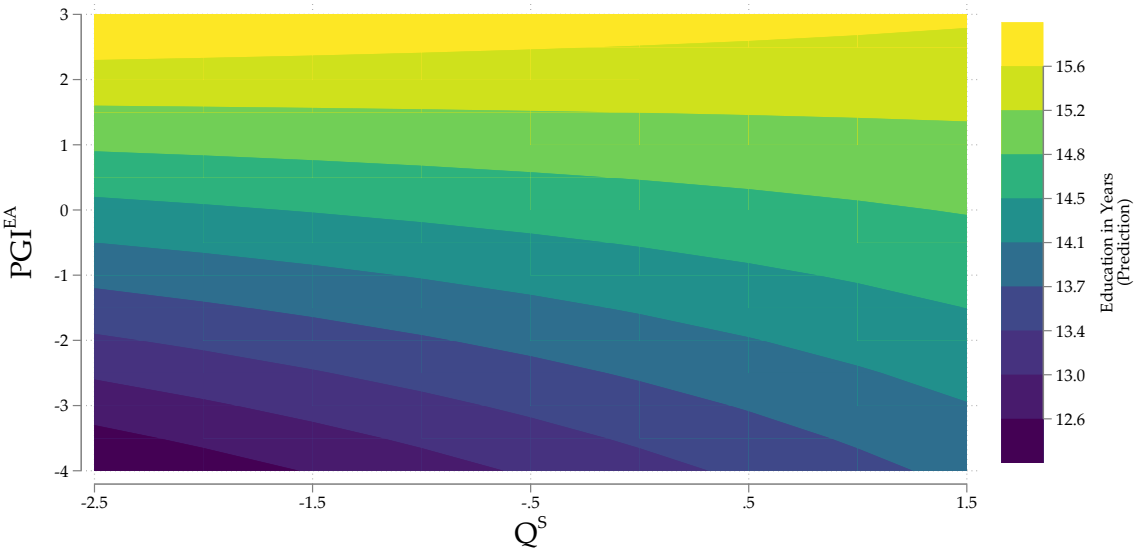
In principle, the negative gene-environment interaction shown in Table 3 could be due to low-PGI^{EA} students benefiting or high-PGI^{EA} students losing from high-quality schools. In Figure 3, we provide evidence for the former but not for the latter. This figure shows predictive margins of years of education for different combinations of PGI^{EA} and Q^S while controlling for $\mathbf{X}$. Moving horizontally from left to right at a given PGI^{EA} level, predicted education increases sharply in the lower parts of the PGI^{EA} distribution. On the contrary, predicted education remains unchanged regardless of school quality in the upper ranges of the PGI^{EA} distribution. This pattern is encouraging as it suggests that investments in school quality mitigate inequalities based on genetic predispositions without compromising the attainment of genetically advantaged students.[18]

It is interesting to contrast this substitutability result with existing evidence on the interaction of genetic endowments and parental SES. In this literature, researchers tend to find (weakly) positive gene-environment interactions between PGI^{EA} and parental SES. This implies that, in

[17] In Supplementary Table S.6, we show the changes in coefficients as we sequentially introduce the control variables. In line with expectations, the gene-environment interaction becomes more negative and significant as we expand the set of control variables, thus reducing confounding through genetic nurture and selection in schools. See Supplementary Material C for a discussion of the underlying econometric rationale.

[18] One might suspect that ceiling effects in educational attainment drive this pattern. To check this potential explanation, we replace educational attainment with the PVT, i.e., an outcome not artificially censored from above. The pattern is the same (Supplementary Figure S.7).

FIGURE 3 – Association of Years of Education with PGI^{EA} by Q^S



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows predictions of completed years of education by PGI^{EA} and Q^S cell. Predictions are calculated using the model estimated in column (1) of Table 3 while allowing for non-linear effects over the range of -4.0(0.5)3.0 of PGI^{EA} and -2.5(0.5)1.5 of Q^S.

the family context, genetically advantaged children benefit more from environments associated with higher investment into children.[19] On the contrary, we find that the sign of interplay between genetic endowments and high-investment environments reverses in the school context, i.e., children endowed with a relatively low PGI^{EA} benefit more from environments associated with higher investments.

5.3 Robustness analysis

We check the robustness of our results in two steps. First, we conduct additional specification checks to verify the satisfaction of the identifying assumptions outlined in section 3. Second, we investigate whether our school quality measure picks up other school characteristics that may predict student outcomes. We also provide a summary of various additional robustness checks which we collect in the Supplementary Material.

Identifying assumptions. In Table 4, we conduct specification checks in order to verify the satisfaction of the identifying assumptions outlined in section 3. For ease of comparison, we replicate our baseline estimates in column (1) of the table.

[19]See, for example, Ronda et al. (2022) and Turkheimer et al. (2003) for positive interactions between children's genetic endowments and parental SES, and Figlio et al. (2017) for a null finding. We are not aware of any studies finding negative interaction effects.

TABLE 4 – Association of Years of Education with PGI^{EA} and School Environments

	Baseline	Interacted controls	Non-linearities	Subsample w/ lagged ability measures		
Outcome: Years of Education	(1)	(2)	(3)	(4)	(5)	(6)
PGI^{EA}	0.361***	0.353***	0.389***	0.333***	0.334***	0.337***
	(0.028)	(0.029)	(0.036)	(0.050)	(0.048)	(0.048)
Q^S	0.124**	0.073	0.139**	0.252*	0.254*	0.255*
	(0.057)	(0.059)	(0.066)	(0.144)	(0.132)	(0.133)
$PGI^{EA} \times Q^S$	-0.068***	-0.072**	-0.075***	-0.088*	-0.086**	-0.089**
	(0.026)	(0.030)	(0.026)	(0.047)	(0.042)	(0.042)
PVT	–	–	–	–	0.223***	0.225***
					(0.047)	(0.047)
$PVT \times Q^S$	–	–	–	–	–	-0.003
						(0.045)
GPA Science	–	–	–	–	0.381***	0.383***
					(0.060)	(0.060)
GPA Science $\times Q^S$	–	–	–	–	–	0.069
						(0.059)
GPA Math	–	–	–	–	0.283***	0.281***
					(0.075)	(0.074)
GPA Math $\times Q^S$	–	–	–	–	–	-0.004
						(0.080)
Child Controls	✓	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓	✓
All interactions (Q^S, PGI^{EA}, $\mathbf{X}_i(a)$)	×	✓	×	×	×	×
2^{nd} Polynomial (Q^S, PGI^{EA})	×	×	✓	×	×	×
N	4,034	4,034	4,034	1,039	1,039	1,039
R^2	0.333	0.345	0.334	0.437	0.510	0.511
Outcome Mean	14.681	14.681	14.681	14.520	14.520	14.520
Outcome SD	2.268	2.268	2.268	2.309	2.309	2.309

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education. In column (2), we control for all possible interactions between PGI^{EA}, Q^S, *Child Controls*, *Family Controls*, and the *Control Function*. In column (3), we control for second-order polynomials of PGI^{EA} and Q^S and allow for all possible interactions of both indicators. In column (4), we replicate our baseline specification on a sample of students for which we observe ability measures before they enter high school. In column (5) and (6), we control for lagged ability measures and their interaction with Q^S. All lagged ability measures are residualized by regressing them on PGI^{EA}, *Child Controls*, *Family Controls*, and the *Control Function*. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

In column (2), we test whether our estimates are confounded by the fact that we do not estimate a fully interacted model (see also our discussion in section 3). In particular, Feigenberg et al. (forthcoming) argue that the estimation of heterogeneous treatment effects may be susceptible to omitted variable bias if the estimation model does not account for the heterogeneous impact of control variables on the outcome of interest.[20] For illustration, consider the following example: Jackson et al. (2024) show that school quality especially improves the educational outcomes of low-SES students, i.e., $Cov(Y, SES \times Q^S) \neq 0$. If in addition $Cov(PGI^{EA} \times Q^S, SES \times Q^S) \neq 0$, the estimates of the gene-environment interaction of interest would be susceptible to an omitted variable bias if we were to exclude $SES \times Q^S$ from our estimation model. To address this concern, Feigenberg et al. (forthcoming) suggest assessing whether the interactions of $\mathbf{X}$ and the treatment variables of interest belong in the estimation model. We follow this suggestion and extend our estimation model by interacting both PGI^{EA} and Q^S with the full set of controls $\mathbf{X}$. In doing so, we allow for the possibility that family socioeconomic status and student characteristics interact with genetic endowments and school quality. In this model, the interpretation for the base coefficients of PGI^{EA} and Q^S changes because the estimated coefficients now reflect effects for the subgroup of individuals that sit at the mean of all control variables in $\mathbf{X}$.[21] This change in interpretation explains the lower coefficient for Q^S compared to the baseline model. Importantly, however, our estimate for the interaction of genetic endowments and school quality remains unaffected, suggesting that our baseline estimates are not confounded by omitting the interactions of the variables of interest and $\mathbf{X}$.

In column (3), we test for non-linearities in the associations of both PGI^{EA} and Q^S with educational attainment. In estimation equation 6, we have restricted the functional form to include base effects and the two-way interaction of the variables of interest. However, it could be the case that the positive effect of genetic endowments on educational attainment is mainly clustered in the tails of the PGI^{EA} distribution or that the impact of Q^S on educational attainment is concave. Then, our model would be misspecified, and the estimate of the gene-environment interaction biased (Biroli et al., 2022). To address this concern, we include second-order polynomials of PGI^{EA} and Q^S into the model and allow for all possible interactions between the variables. Our results are unaffected by this expansion and support the parsimonious model specification of our baseline.

In columns (4)–(6), we re-run our analyses on a subsample of students for whom we observe ability measures before they enter high school. These students were in grade 8 and lower during wave 1 of Add Health. In this subsample, we can use the lagged PVT and GPAs for Math and Science as additional control variables to account for sorting into schools. Since we compare the outcomes of students with similar abilities before entering high school, this analysis is reminiscent of a value-added analysis (Jackson et al., 2024). In column (4), we replicate our

[20] See Domingue et al. (2020) and Keller (2014) for similar arguments in the context of gene-environment interactions.

[21] Effects are estimated at the mean of the control variables since we standardize all variables in $\mathbf{X}$ to have a mean of zero and a standard deviation of one.

baseline model in this subsample. The substantial decrease in sample size inflates our standard errors and the coefficient of Q^S. However, our conclusions about the substitutability of PGI^{EA} and Q^S remain unaffected. In column (5), we introduce lagged ability measures as additional controls. Note that lagged ability partly reflects the influence of PGI^{EA}. Therefore, we construct measures of $\theta^{\perp}$ by residualizing all lagged ability measures from PGI^{EA} and $\mathbf{X}$. These residualized lagged ability measures are highly predictive of school outcomes—their inclusion increases the R^2 from 0.437 (column 4) to 0.510 (column 5). However, the coefficients of Q^S and the associated gene-environment interaction remain unaffected. In column (6), we illustrate that this result also holds when accounting for potential interactions between lagged ability and Q^S. The results of these analyses give credence to our assumption that the pre-determined controls in $\mathbf{X}$ capture sorting into schools and that we can collect lagged ability measures in the error term of equation 6 without confounding our parameters of interest.

Overall, these results further support our functional form assumptions and that our results are not confounded by genetic nurture and sorting into schools.

Measurement of school quality. In Table 5, we check the robustness of our school quality measure by investigating whether Q^S picks up variation from other school characteristics that may correlate with student outcomes. We note that the existence of such correlations would not invalidate our identification strategy. However, they would alter our conclusions regarding which aspects of school environments likely drive our results. In general, we caution against overinterpreting the importance of the specific components of Q^S due to the lack of independent variation in different school environments. For ease of comparison, we replicate our baseline estimates in column (1) of the table.

In columns (2)–(4), we sequentially introduce indicators for other school policies and their interaction with PGI^{EA}. Indicators for other school policies include the average share of retained students across grades 9-11 (column 2), a binary indicator of whether schools group students by English ability (column 3), and an index for the strictness of school sanctions (column 4).[22] Comparing columns (2)–(4) to our baseline estimate, we see that all coefficients of interest are robust to the inclusion of these indicators.[23]

In column (5), we account for potential quality differences between private and public schools

[22]The strictness index is based on headmaster questionnaires. Headmasters were asked about the school's policy in the following domains of behavior: cheating, fighting with or injuring another student, alcohol or drug possession, drinking alcohol or using illegal drugs, smoking, verbally or physically abusing a teacher, and stealing school property. Possible measures are (i) no action, (ii) verbal warning, (iii) minor action, (iv) in-school suspension, (v) out-of-school suspension, and (vi) expulsion. Following Anderson (2008) and Kling et al. (2007) we standardize each response dimension to $\mu = 0$ and $\sigma = 1$ and aggregate them linearly to obtain the strictness index. See Supplementary Material D for details.

[23]Alternatively, we could use these school characteristics in our measure Q^S by including them in the PCA that extracts the school quality factor. Supplementary Table S.7 shows that such expanded indicators yield similar results to our baseline indicators for Q^S.

TABLE 5 – Robustness to Additional School Characteristics

	Baseline	+ School Policies			+ Private School	+ Teacher Composition		+ School FE
Outcome: Years of Education	(1)	Retention Policy (2)	Ability Groups (3)	Strict. Index (4)	(5)	White Teacher (6)	Female Teacher (7)	(8)
PGI^{EA}	0.361*** (0.028)	0.366*** (0.029)	0.361*** (0.028)	0.362*** (0.029)	0.362*** (0.029)	0.361*** (0.028)	0.360*** (0.028)	0.350*** (0.029)
Q^S	0.124** (0.057)	0.116** (0.053)	0.127** (0.058)	0.135** (0.060)	0.144** (0.056)	0.121** (0.055)	0.135** (0.062)	–
$PGI^{EA} \times Q^S$	-0.068*** (0.026)	-0.066*** (0.025)	-0.068*** (0.025)	-0.064** (0.027)	-0.076*** (0.026)	-0.064** (0.026)	-0.065** (0.026)	-0.064** (0.027)
School Characteristic	–	-0.103* (0.060)	0.049 (0.034)	0.062* (0.036)	0.101** (0.043)	-0.013 (0.074)	-0.022 (0.048)	–
$PGI^{EA} \times$ School Characteristic	–	0.034 (0.030)	-0.016 (0.030)	0.019 (0.024)	-0.049** (0.023)	-0.029 (0.033)	0.045 (0.034)	–
Child Controls	✓	✓	✓	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓	✓	✓	✓
N	4,034	3,969	4,034	4,034	4,034	4,034	4,034	4,034
R^2	0.333	0.334	0.333	0.334	0.334	0.333	0.334	0.343

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education. We control for additional school characteristics and their interaction with PGI^{EA}. The relevant school characteristics are indicated in the column header. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

(Altonji et al., 2005). Including a binary indicator for private schools and its interaction with PGI^{EA} does not affect our main results.

In columns (6)–(7), we address the concern that our index for school quality may correlate with the demographic composition of teachers at a school. This possibility arises if teachers sort into particular schools based on student characteristics (Jackson, 2009a). To this end, we sequentially introduce teacher demographics indicators and their interaction with PGI^{EA}. Indicators for teacher demographics include the share of white teachers (column 6), and the share of female teachers (column 7). Our results remain robust to the inclusion of these variables.

In column (8), we re-estimate the coefficients of interest while accounting for unobserved differences across schools by introducing school fixed effects. In this specification, we cannot estimate the effect of Q^S on educational attainment. However, it is reassuring that the estimates

for PGI^{EA} and the gene-environment interaction are very close to our baseline estimates.[24]

Overall, these results suggest that our estimates of α_2 and α_3 do not pick up the effects of other school characteristics that may be correlated with student outcomes.

Further robustness checks. In the Supplementary Material, we show the results of additional robustness analyses. First, we check whether outlier schools drive our results. Therefore, we re-run our analysis 72 times, excluding one school from the sample per iteration. In each iteration, the results are very close to our baseline estimates (Supplementary Figure S.5). Second, we check whether ceiling effects in educational attainment drive our findings. To this end, we re-run our analysis, artificially censoring educational attainment step-wise from above. If ceiling effects drove our results, we would expect the gene-environment interaction to increase across parts of the censoring interval. However, this is not the case. Instead, the corresponding coefficient decreases monotonically (Supplementary Figure S.6). Supplementary Figure S.7 supports the absence of ceiling effects. In this figure, we replicate Figure 3 while replacing educational attainment with the PVT, i.e., an educational outcome that is not artificially censored from above. The figure replicates the data pattern of Figure 3 very well, reinforcing our conclusion that relative gains of low-PGI^{EA} students drive our results. Third, we check whether we pick up the relevant genetic variation. In particular, we sequentially control for six alternative PGIs that target phenotypes other than educational attainment and their interaction with Q^S. While some PGIs have predictive power over and above PGI^{EA}, our estimates for PGI^{EA}, Q^S and the gene-environment interaction of interest remain unaffected (Supplementary Table S.8). Fourth, we re-estimate our baseline model while applying the correction method of Becker et al. (2021) to account for measurement error and resulting attenuation bias in PGI^{EA} and the gene-environment interaction. As expected, the measurement-error-corrected estimates for PGI^{EA} and the gene-environment interaction are substantially higher than in our baseline estimates. However, they increase in roughly equal proportions. As a result, our conclusions about the relative strength of the substitutability of PGI^{EA} and Q^S remain unaffected. In the measurement-error-corrected version, a 1 SD increase in school quality reduces the positive association of educational attainment with PGI^{EA} by $\approx$ 15–16% (Supplementary Table S.9). Lastly, we run a placebo test as an alternative way of inference. That is, we re-estimate our baseline model while permuting the values of Q^S 10,000 times and keeping constant all other variables. In Supplementary Figure S.9, we show that the resulting distribution of t-statistics for the interaction of PGI^{EA} and Q^S is well-behaved and that roughly 1%, 5%, and 10% of the placebo regressions yield t-statistics at the corresponding critical values. Slightly less than 1% of the placebo regressions yield a t-statistic more extreme than the one from our baseline estimates, suggesting that our results are not driven by chance.

In summary, this battery of additional checks further supports the conclusion that our main

[24] Note that the variables of the control function can be estimated in the school fixed effects model since they are constructed as leave-cohort-out averages.

findings are genuine.

5.4 Mechanisms

In this section, we analyze the mechanisms underlying the substitutability of genetic endowments and school quality.

Family investments. In section 3, we considered a model where genetic endowments, family investments, and school inputs determine the skills of children. Since parents may adjust their investments in children in response to PGI^{EA} and Q^S, family investments could be one mechanism to rationalize the gene-environment interaction detected in our baseline estimates.

To investigate the importance of family investments, we construct two indexes. First, we consider an index for parental time investments that captures the joint activities of parents and children. Second, we consider an index for an authoritarian parenting style that captures how strongly parents interfere with their children's choices.[25] In turn, we use each of these indexes as outcomes when estimating the family investment function (equation 7). Table 6 summarizes the results.

In columns (1)–(4), we estimate the parameters γ_1, γ_2, and γ_3 of equation 7. The evidence for the importance of investment effects in explaining our results is mixed. On the one hand, we find that parents adjust their time investments and parenting styles in response to the genetic endowments of their children. They invest more time in high-PGI children—a finding consistent with Houmark et al. (2024). Furthermore, they adopt less authoritarian parenting styles with high-PGI children—a finding that is consistent with Agostinelli et al. (forthcoming) who show that parents become less authoritarian if their children are of higher ability. These findings also hold in a sibling sample where we additionally control for the sibling's PGI^{EA}.[26] Therefore, the impact of genetic endowments on family investments (mediated through the interactive effect of family investments and school quality on educational attainment) may be partly driving our estimates of the gene-environment interaction α_3. On the other hand, the

[25] For parental time investments, we collect information on a series of activities that the child has engaged in with either their mother or father over the past four weeks. For both parents, these activities include shopping, playing sports, going to church, talking about dates, going to the movies and similar events, talking about personal problems and school work, working together on school work, and talking about other things than school. For parenting styles, we follow Agostinelli et al. (forthcoming) and collect information on how strongly parents interfere with the choices of their children, e.g., choosing their friends, at what time to be home at night, what to wear, and at what time to go to bed. Following Anderson (2008) and Kling et al. (2007) we standardize the questions underpinning each index to $\mu = 0$ and $\sigma = 1$ and aggregate them linearly to obtain an overall index. See Supplementary Material D for details.

[26] This result is in contrast to Sanz-de-Galdeano and Terskaya (forthcoming) who also use AddHealth and find no significant effects of PGI^{EA} on parental time investment once the siblings' PGI^{EA} is taken into account. We note that our analysis differs in several dimensions. Among others, we do not restrict the sample to firstborn children and use a more comprehensive index of parental time investments.

TABLE 6 – Association of Parental Investments and Parenting Styles with PGI^{EA} and Q^S

	Parental Investment		Parenting Style	
	(1)	(2)	(3)	(4)
PGI^{EA}	0.065***	0.088**	-0.068***	-0.084*
	(0.015)	(0.041)	(0.015)	(0.043)
Q^S	-0.027	-0.036	-0.006	-0.012
	(0.017)	(0.030)	(0.027)	(0.050)
$PGI^{EA} \times Q^S$	-0.013	0.041	-0.022	-0.050
	(0.015)	(0.029)	(0.015)	(0.054)
Child Controls	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓
Control Function	✓	✓	✓	✓
Sibling PGI^{EA}	×	✓	×	✓
N	4,034	677	4,034	677
R^2	0.084	0.119	0.159	0.146
Outcome Mean	0.000	0.000	0.000	0.000
Outcome SD	1.000	1.000	1.000	1.000

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with indexes of parental time investments. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

estimates for Q^S and the interaction of PGI^{EA} and Q^S are close to zero and insignificant in all regressions. This suggests that parents do not (differentially) adjust their investments in response to the quality of schooling environments and that the associated investment effects are unlikely to drive our estimates of α_3. Nevertheless, we interpret these results as suggestive since the precision of the estimates does not allow us to rule out meaningful parental investment responses.

Intermediate skills. Children's skills at the end of high school are plausible candidates to explain the results on educational attainment. The multidimensional skills influencing educational attainment comprise a broad set of (non-)cognitive skills and health (Almlund et al., 2011; Heckman and Mosso, 2014). Furthermore, current literature shows that each of these skill dimensions is shaped, in part, by genetic influence (Buser et al., 2024; Demange et al., 2021, 2022).

We evaluate these potential channels by analyzing the associations of PGI^{EA} and Q^S with in-

termediate outcomes. Regarding cognitive skills, we use the Picture Vocabulary Test (PVT) to measure verbal intelligence. Furthermore, we focus on personality and preferences as two distinct conceptualizations of non-cognitive skills (Becker et al., 2012; Humphries and Kosse, 2017). In particular, we use measures for self-reported risk aversion, patience, and Big Five personality traits. Regarding health outcomes, we focus on subjective health, measured by quality-adjusted life years (QALY), and objective health, measured by an index that comprises information on whether the respondent is obese, has first-stage hypertension, or has high cholesterol. All measures were collected in waves 3 and 4 of Add Health after respondents had left high school but potentially before they had completed their highest level of education (see Supplementary Material D for details).[27]

Existing literature shows cognitive skills, risk aversion, patience, and health are strong predictors of educational attainment (Burks et al., 2015; Castillo et al., 2018a,b; Jackson, 2009b). Furthermore, openness and emotional stability—the opposite of neuroticism—are positively associated with educational attainment (Becker et al., 2012; Buser et al., forthcoming).[28] Based on this evidence, one would expect positive associations of both PGI^{EA} and Q^S with these intermediate outcomes. The sign of the gene-environment interaction is unclear a priori. However, given the substitutability of PGI^{EA} and Q^S in the production of educational attainment, we expect similar substitutability patterns for a subset of these intermediate outcomes.

Table 7 summarizes the results. In column (1) of Panel (a), we focus on the PVT as a measure of cognitive skills. As expected, our results show positive associations of both PGI^{EA} and Q^S with the PVT. A 1 SD increase in PGI^{EA} (Q^S) is associated with a 0.174 SD (0.113 SD) increase in the PVT. Furthermore, both factors are substitutes for each other. A 1 SD increase in school quality reduces the positive association of PVT and PGI^{EA} by $\approx 29\%$ $(= 0.050/0.174)$.

In columns (2)–(3) of Panel (a), we focus on economic preferences. As expected, we find positive associations of PGI^{EA} and Q^S with risk aversion and patience. 1 SD increases in PGI^{EA} and Q^S are associated with increases in risk aversion by 0.04 SD and 0.07 SD, respectively. The corresponding increases in patience are 0.08 SD and 0.07 SD. Furthermore, PGI^{EA} and Q^S are substitutes for each other. A 1 SD increase in Q^S reduces the positive associations of risk aversion and patience with PGI^{EA} by $\approx 103\%$ $(= 0.041/0.040)$ and $\approx 33\%$ $(= 0.029/0.086)$, respectively.

In columns (4)–(5) of Panel (a), we focus on health outcomes. Our results show a positive association of PGI^{EA} with subjective and objective health. A 1 SD increase in PGI^{EA} increases

[27] We note that interpreting these measures as intermediate outcomes is not straightforward as many respondents reach their highest level of education before waves 3 and 4. Therefore, skills measured in waves 3 and 4 may result from educational attainment, not vice versa. To assuage this concern, we present additional analyses in Supplementary Table S.10, where we estimate the gene-environment interaction for similar skills measured during high school. While point estimates are smaller and standard errors are larger, the findings are qualitatively similar. This finding supports our interpretation that the considered skill measures may mediate the interplay of genetic endowments and school quality in the production of educational attainment.

[28] We replicate these findings in our data by showing that each intermediate outcome is highly predictive for educational attainment conditional on our set of controls $\mathbf{X}$. See Supplementary Figure S.9.

TABLE 7 – Association of Skill Measures with PGI^{EA} by Q^S

Panel (a)	Cognitive	Preferences		Health	
	PVT (1)	Risk (2)	Patience (3)	Subjective (4)	Objective (5)
PGI^{EA}	0.174***	0.040**	0.086***	0.077***	0.042***
	(0.014)	(0.017)	(0.017)	(0.016)	(0.016)
Q^S	0.113**	0.071**	0.074*	0.034	-0.004
	(0.044)	(0.030)	(0.041)	(0.038)	(0.023)
$PGI^{EA} \times Q^S$	-0.050***	-0.041***	-0.029**	-0.038***	-0.001
	(0.011)	(0.016)	(0.012)	(0.013)	(0.017)
Child Controls	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓
N	3,380	3,465	3,465	3,471	4,034
R^2	0.225	0.111	0.103	0.078	0.053

Panel (b)	Personality				
	Open-ness (1)	Conscient-iousness (2)	Extra-version (3)	Agree-ableness (4)	Neuro-ticism (5)
PGI^{EA}	0.075***	-0.007	-0.021	0.042**	-0.087***
	(0.014)	(0.015)	(0.019)	(0.017)	(0.019)
Q^S	0.011	0.044	-0.068***	0.039	-0.043*
	(0.025)	(0.033)	(0.025)	(0.030)	(0.023)
$PGI^{EA} \times Q^S$	-0.011	-0.006	-0.018	-0.000	0.019
	(0.011)	(0.014)	(0.023)	(0.014)	(0.019)
Child Controls	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓
N	3,996	4,028	4,027	4,026	4,022
R^2	0.101	0.046	0.035	0.139	0.090

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with cognitive skills, preferences, health, and personality. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

subjective (objective) health by 0.077 SD (0.042 SD). Furthermore, the negative coefficient on the interaction of PGI^{EA} and Q^S suggests that this increase is particularly pronounced for low-PGI^{EA} students: a 1 SD increase in school quality reduces the positive association of subjective

health with PGI^{EA} by $\approx 49\%$ $(= 0.038/0.077)$.

In Panel (b), we focus on personality traits. We find positive associations of PGI^{EA} with openness and agreeableness and a negative association of PGI^{EA} with neuroticism. Q^S is associated with decreases in extraversion and neuroticism. However, we find no evidence of an interaction between PGI^{EA} and Q^S in the production of personality traits.

To summarize, we find negative gene-environment interactions between genetic endowments and school quality in the production of cognitive skills, risk aversion, patience, and subjective health. Given their predictive power for educational attainment, these intermediate outcomes are plausible transmission channels for the substitutability of genetic endowments and school quality in the production of educational attainment.

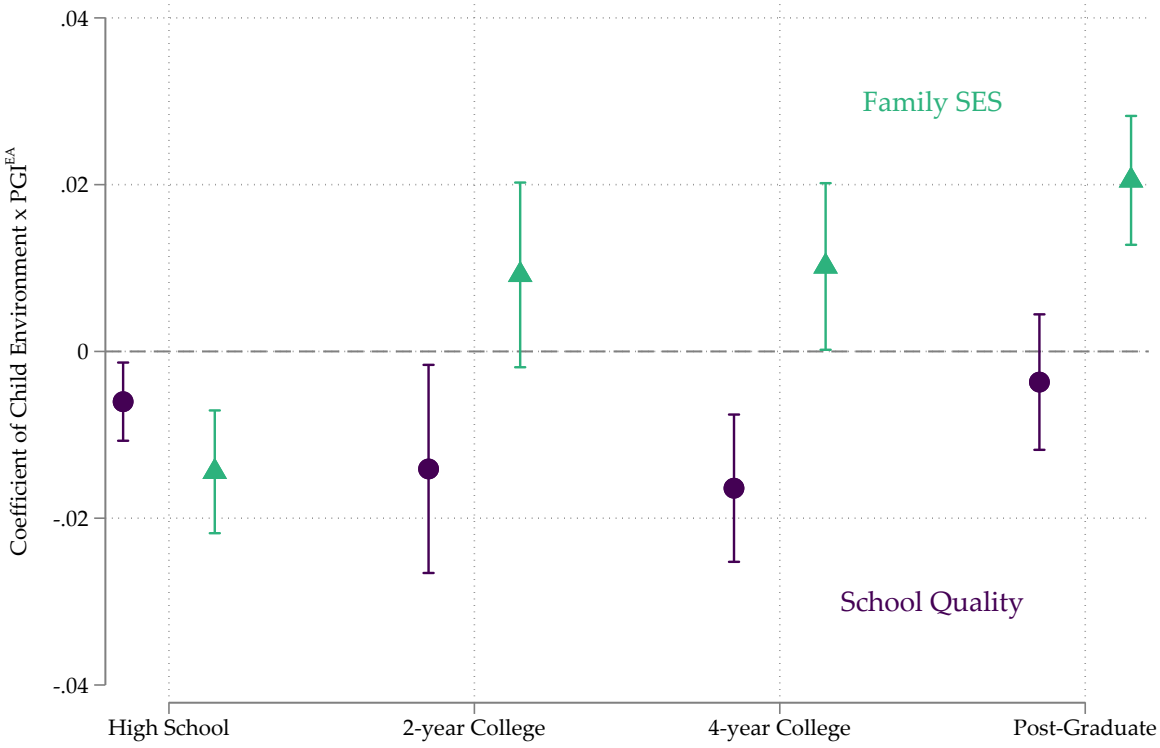
Educational degrees. Total years of education summarizes information from various educational stages, each requiring a different mix of skills (Cunha et al., 2006, 2010). Therefore, we repeat our analysis by replacing total years of education with binary variables for whether respondents obtained (i) at least a high school degree or GED, (ii) a 2-year college degree, (iii) a 4-year college degree, or (iv) a post-graduate degree.

Figure 4 presents the resulting point estimates for the gene-environment interaction and the associated confidence bands. The circular series suggests that the substitutability of school quality and genetic endowments follows a U-shaped pattern throughout the educational life cycle. For students with low PGI^{EA}, there is a slight decrease in the probability of dropping out of high school if they attend high-quality schools, followed by increases in substitutability for 2-year and 4-year college degrees. The substitutability of high-quality schools and genetic endowments levels off at the post-graduate level. This pattern is consistent with the following interpretation: high school graduation is a relatively "inclusive" educational outcome accessible to most, including low-PGI^{EA} students in low-quality schools. Evidence of this effect is the high school graduation rate of 96% in our sample (Table 1). In contrast, post-graduate education is a relatively "exclusive" educational outcome more accessible to students with high PGI^{EA} *and* who attend high-quality schools. In both cases, high-quality schools have limited scope to make a difference for low-PGI^{EA} students. College education, however, takes a middle ground between these two polar outcomes and therefore offers scope for low PGI^{EA} to be offset by school quality and vice versa. We interpret this pattern as suggestive since the confidence bands are too wide to statistically distinguish between the point estimates for different educational degrees.

The triangular series shows that the gene-environment interaction with family SES increases over the educational life-cycle of individuals.[29] This pattern replicates recent evidence from

[29] In particular, we use the "social origins score" from Belsky et al. (2018) measured in wave 1. Results for alternative measures of family SES, such as family income or potential wages of either parent, yield similar results.

FIGURE 4 – Association of Education Degrees with PGI^{EA} by Q^S and Family SES



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows point estimates and 90% confidence bands for the interaction of PGI^{EA} with Q^S , the interaction of PGI^{EA} with an indicator for family SES, and their association with education degrees. For each outcome, coefficients are estimated jointly following the specification of equation (6). *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Standard errors are clustered at the school level.

the United States and Sweden (Buser et al., 2024; Papageorge and Thom, 2020) and is consistent with the idea that endowments and investments may be substitutes at the early stages of childhood but that their complementarity increases over the life-cycle (Heckman and Mosso, 2014).

More generally, the contrast between the circular and the triangular series shows that the interplay of advantageous genetic endowments and conducive environments varies across different sources of investments in children. Regarding their outcomes after high school and relative to their high-PGI^{EA} peers, children with lower values of PGI^{EA} consistently gain more from attending a high-quality school than from a high-SES background. There are different potential explanations for this pattern. First, schools may allocate resources differently than parents. For example, Houmark et al. (2024) show that parents magnify skill inequality by investing more in children with higher genetic endowments. Hence, our results are consistent with a model where schools allocate resources more equally than families. Second, schools may provide dif-

ferent types of investments than families. For example, investments in schools tend to happen in the context of larger groups, whereas investments at home tend to be focused on individuals or smaller sibling groups. Hence, our results are consistent with a model where, relative to their high-PGI^{EA} peers, low-PGI^{EA} students respond more positively to the type of investments they receive in schools than to the type of investments they receive at home. While we cannot distinguish among these explanations, our results are generally consistent with recent evidence documenting significant relative gains of disadvantaged students from attending high-quality schools in the United States (Cohodes et al., 2021; Jackson et al., 2024).

6 CONCLUSION

The question of how natural endowments and environmental factors determine life outcomes has a long history of inquiry in philosophy and science (Darwin, 1859; Descartes, 1641; Lamarck, 1838; Locke, 1690). The assumption that life outcomes are the result of genetic and environmental factors initially led to the so-called "nature versus nurture" debate. However, current research has moved beyond this simplistic dichotomy and recognizes that individual life outcomes are the result of a complex interplay between nature and nurture. This insight highlights that the importance of genetic endowments for life outcomes is not immutable. Instead, it opens a path for policy interventions that shape the relevant environment.

In this paper, we contribute to this research agenda by studying the interplay of genetic endowments and school quality. Using recent advances in molecular genetics, we link an individual-level index of genetic predispositions for educational success with measures of school quality. In turn, we investigate whether the importance of genetic endowments varies with the quality of high schools.

Our findings suggest that investments in the quality of schools can mitigate the genetic gradient in educational attainment. Furthermore, we show that gains in language skills, risk aversion, patience, and subjective health mediate higher gains in educational attainment for students with lower genetic endowments.

The use of genetics in education research has an ugly history. Therefore, many people are wary of the emergence of genetic markers in this context, especially when these markers are used for genetic screening (Martschenko et al., 2019). We emphasize that our results do neither presuppose nor endorse the use of genetic screening for educational interventions. Instead, our results suggest that universal policy reform that increases the quality of schools for *all students* may provide an essential step to level the playing field regardless of a student's draw in the genetic lottery.

DATA AVAILABILITY

Code and data for replicating the tables and figures in this article can be found in Arold et al. (2025) in the Harvard Dataverse (https://doi.org/10.7910/DVN/2SQLDG). The replication package contains a README file detailing the data sources, computational requirements, and structure of the replication code.

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Genetic Endowments, Educational Outcomes, and the Mediating Influence of School Quality

Benjamin W. Arold, Paul Hufe & Marc Stöckli

Online Appendix

A SUPPLEMENTARY TABLES

TABLE S.1 – Gene-Environment Correlations

Outcome: PGI^{EA}	(1)	(2)	(3)	(4)	(5)	(6)
Q^S	0.027 (0.027)	0.025 (0.043)	–	–	–	–
Peer GPA (English)	–	–	0.075** (0.033)	0.028 (0.019)	–	–
Peer GPA (Math)	–	–	–	–	0.097*** (0.031)	0.021 (0.022)
Child Controls	×	✓	×	✓	×	✓
Family Controls	×	✓	×	✓	×	✓
Control Function	×	✓	×	✓	×	✓
N	4,034	4,034	4,034	4,034	4,034	4,034
R^2	0.001	0.090	0.006	0.090	0.009	0.090

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows gene-environment correlations between PGI^{EA} and various school characteristics. Peer GPAs are standardized in grade times state cells and calculated as leave-cohort-out school averages. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.2 – Robustness to Specification of Control Function

	Baseline	Excluding Grade 12 in Wave 1	
Outcome: Years of Education	(1)	Leave-cohort-out (2)	Leave-all-following-cohorts-out (3)
PGI^{EA}	0.361***	0.362***	0.362***
	(0.028)	(0.031)	(0.031)
Q^S	0.124**	0.140**	0.125**
	(0.057)	(0.060)	(0.064)
$PGI^{EA} \times Q^S$	-0.068***	-0.084***	-0.083***
	(0.026)	(0.031)	(0.031)
Child Controls	✓	✓	✓
Family Controls	✓	✓	✓
Control Function	✓	✓	✓
N	4,034	3,436	3,436
R^2	0.333	0.340	0.340

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education using different specifications of the control function. In column (2) we calculate school averages for the control function based on the original leave-cohort-out specification. In column (3) we calculate school averages for the control function while leaving out all subsequent cohorts that follow a respondent's cohort. Note that such school averages cannot be calculated for students in Grade 12 at Wave 1. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.3 – Robustness to Sample Composition

	Baseline	Alternative Sample Composition		
Outcome: Years of Education	(1)	Re-Weighted (2)	Excl. (Potential) Movers before High School (3)	Excl. (Potential) Movers during High School (4)
PGI^{EA}	0.361***	0.347***	0.353***	0.343***
	(0.028)	(0.031)	(0.035)	(0.040)
Q^S	0.124**	0.115*	0.159**	0.101
	(0.057)	(0.060)	(0.063)	(0.078)
$PGI^{EA} \times Q^S$	-0.068***	-0.061**	-0.086***	-0.086**
	(0.026)	(0.027)	(0.032)	(0.038)
Child Controls	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓
Control Function	✓	✓	✓	✓
N	4,034	3,968	2,962	2,439
R^2	0.333	0.313	0.350	0.344

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education. In column (2) we re-weight our analysis sample to match ACS and CPS with respect to gender composition, educational attainment of parents, and the age of mothers at birth—see also Supplementary Table S.4. In column (3) we exclude respondents that attended feeder schools in wave 1 and for whom we do not have information on subsequent high schools. In column (4) we additionally exclude respondents for whom we do not have information on whether they graduated from an Add Health high school. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.4 – Sample Representativeness

	Population (Cohorts 1974-1983)		Analysis Sample	
	All	Non-Hispanic White	Unweighted	Re-Weighted
Gender				
Male	0.498	0.503	0.462	0.503
Female	0.502	0.497	0.538	0.497
Education Mother				
$\leq$ High School	0.536	0.489	0.507	0.489
$>$ High School; $<$ College Degree	0.281	0.302	0.218	0.301
$\geq$ College Degree	0.183	0.209	0.275	0.210
Education Father				
$\leq$ High School	0.472	0.425	0.514	0.425
$>$ High School; $<$ College Degree	0.255	0.271	0.187	0.271
$\geq$ College Degree	0.273	0.304	0.298	0.303
Age Mother at Birth				
$<$ 25 Years	0.353	0.330	0.503	0.330
$\geq$ 25 Years	0.647	0.670	0.497	0.670
Parental Income				
$<$ \$50,000	0.557	0.491	0.536	0.511
$\geq$ \$50,000; $<$ \$100,000	0.352	0.403	0.386	0.405
$\geq$ \$100,000	0.091	0.106	0.078	0.083
Education Respondent				
$\leq$ High School	0.301	0.225	0.197	0.181
$>$ High School; $<$ College Degree	0.327	0.344	0.408	0.406
$\geq$ College Degree	0.372	0.431	0.396	0.413

Data: National Longitudinal Study of Adolescent to Adult Health, American Community Survey (ACS), Current Population Survey (CPS).
Note: Own calculations. This table shows summary statistics of the core analysis sample in comparison to other population samples. It shows respondents' characteristics for the following samples: (i) the US population from birth cohorts 1974–1983, (ii) the Non-Hispanic White US population from birth cohorts 1974–1983, (iii) the core estimation sample, and (iv) the core estimation sample re-weighted to match (ii) with respect to *Gender*, *Education Mother*, *Education Father*, and *Age Mother at Birth*. Population data on *Gender* and *Education Respondent* from IPUMS ACS 2019 (Ruggles et al., 2020). Population data on *Education Mother*, *Education Father*, *Age Mother at Birth*, and *Parental Income* from IPUMS CPS 1994 (Flood et al., 2020).

TABLE S.5 – Testing for Selection into Schools (Decomposition)

Outcome: Predicted Years of Education	Teacher w/ MA		Exp. Teacher		New Teacher		Class Size	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	0.328***	0.078	0.212**	0.016	0.175**	0.031	-0.017	0.044
	(0.105)	(0.050)	(0.097)	(0.042)	(0.082)	(0.034)	(0.110)	(0.041)
Difference in coefficients	0.251*** (0.095) [0.064, 0.437]		0.196** (0.087) [0.025, 0.366]		0.144* (0.077) [-0.008, 0.295]		-0.061 (0.102) [-0.261, 0.139]	
Child Controls	×	×	×	×	×	×	×	×
Family Controls	×	×	×	×	×	×	×	×
Control Function	×	✓	×	✓	×	✓	×	✓
N	4,034	4,034	4,034	4,034	4,034	4,034	4,034	4,034
R^2	0.094	0.185	0.080	0.184	0.077	0.184	0.070	0.185
Outcome Mean	14.681	14.681	14.681	14.681	14.681	14.681	14.681	14.681
Outcome SD	1.163	1.163	1.163	1.163	1.163	1.163	1.163	1.163

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the associations of the components of Q^S with predicted years of education. Predicted education is calculated from a regression of completed years of education on all *Child Controls* and *Family Controls*. We test for the equality of coefficients in columns (1) and (2), (3) and (4), (5) and (6), as well as (7) and (8), respectively. Next to point estimates, we present standard errors (in parentheses), and 95% confidence intervals (in brackets). *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.6 – Testing for Influence of Control Variables

Outcome: Years of Education	(1)	(2)	(3)	(4)
PGI^{EA}	0.592***	0.587***	0.373***	0.361***
	(0.040)	(0.039)	(0.029)	(0.028)
Q^S	0.439***	0.430***	0.227***	0.124**
	(0.167)	(0.151)	(0.076)	(0.057)
$PGI^{EA} \times Q^S$	0.002	-0.009	-0.052**	-0.068***
	(0.039)	(0.036)	(0.026)	(0.026)
Child Controls	×	✓	✓	✓
Family Controls	×	×	✓	✓
Control Function	×	×	×	✓
N	4,034	4,034	4,034	4,034
R^2	0.132	0.166	0.325	0.333

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education while sequentially strengthening the set of control variables. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.7 – Robustness to Using Expanded School Quality Indicators

	Baseline	Expanded School Quality Indicators		
Outcome: Years of Education	(1)	+School Policies (2)	+Private School (3)	+Teacher Composition (4)
PGI^{EA}	0.361***	0.364***	0.361***	0.362***
	(0.028)	(0.029)	(0.028)	(0.029)
Q^S	0.124**	0.091**	0.121**	0.086**
	(0.057)	(0.044)	(0.058)	(0.041)
$PGI^{EA} \times Q^S$	-0.068***	-0.072***	-0.067**	-0.057*
	(0.026)	(0.026)	(0.026)	(0.030)
Child Controls	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓
Control Function	✓	✓	✓	✓
N	4,034	3,969	4,034	4,034
R^2	0.333	0.333	0.333	0.333
Outcome Mean	14.681	14.668	14.681	14.681
Outcome SD	2.268	2.269	2.268	2.268

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education when using expanded versions of the school quality indicators. *School policies*: Retention, ability grouping, and school sanctions. *Private School*: Private school indicator. *Teacher Composition*: White teacher, female teacher. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.8 – Robustness to Including Alternative Polygenic Indexes

	Baseline	+ Controls for Other Polygenic Indexes					
Outcome: Years of Education	(1)	Body Mass Index (2)	ADHD (3)	Depressive Symptoms (4)	Intelligence (5)	Ever Smoker (6)	Sleep Duration (7)
PGI^{EA}	0.361***	0.341***	0.330***	0.358***	0.349***	0.341***	0.360***
	(0.028)	(0.031)	(0.028)	(0.028)	(0.031)	(0.031)	(0.028)
Q^S	0.124**	0.121**	0.120**	0.120**	0.124**	0.122**	0.124**
	(0.057)	(0.056)	(0.056)	(0.057)	(0.057)	(0.056)	(0.057)
$PGI^{EA} \times Q^S$	-0.068***	-0.076***	-0.071***	-0.065**	-0.059**	-0.067**	-0.068***
	(0.026)	(0.029)	(0.026)	(0.027)	(0.028)	(0.027)	(0.026)
Other PGI	–	-0.080***	-0.132***	-0.039	0.023	-0.097***	0.026
		(0.026)	(0.028)	(0.030)	(0.030)	(0.036)	(0.028)
Other PGI $\times Q^S$	–	-0.029	0.003	0.035	-0.018	0.017	-0.003
		(0.028)	(0.028)	(0.029)	(0.028)	(0.033)	(0.029)
Child Controls	✓	✓	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓	✓	✓
N	4,034	4,034	4,034	4,034	4,034	4,034	4,034
R^2	0.333	0.334	0.336	0.334	0.333	0.335	0.333

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education when controlling for additional polygenic indexes. We control for other PGIs and their interaction with Q^S. The relevant PGIs are indicated in the column header. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

TABLE S.9 – Robustness to Using Measurement-error-corrected PGI (Becker et al., 2021)

	Coefficient	Standard Error	p-value	Substitutability
Baseline				
PGI^{EA}	0.361	0.032	0.000	
Q^S	0.124	0.062	0.045	
$PGI^{EA} \times Q^S$	-0.068	0.031	0.027	19%
Add Health ($\rho = 1.968$)				
PGI^{EA}	0.747	0.072	0.000	
Q^S	0.097	0.068	0.151	
$PGI^{EA} \times Q^S$	-0.108	0.064	0.089	15%
Health and Retirement Study ($\rho = 1.413$)				
PGI^{EA}	0.566	0.053	0.000	
Q^S	0.111	0.063	0.079	
$PGI^{EA} \times Q^S$	-0.093	0.046	0.044	16%
Wisconsin Longitudinal Study ($\rho = 1.649$)				
PGI^{EA}	0.718	0.069	0.000	
Q^S	0.099	0.067	0.136	
$PGI^{EA} \times Q^S$	-0.106	0.061	0.079	15%
UK Biobank ($\rho = 1.452$)				
PGI^{EA}	0.589	0.055	0.000	
Q^S	0.109	0.063	0.086	
$PGI^{EA} \times Q^S$	-0.096	0.048	0.047	16%

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with completed years of education. We apply the correction method of Becker et al. (2021) to account for measurement error in PGI^{EA}. The correction method is based on $\rho = h^2_{SNP}/R^2$, where h^2_{SNP} indicates SNP heritability and R^2 the share of variation in educational attainment explained by PGI^{EA}. For Add Health, we take ρ from Sanz-de-Galdeano and Terskaya (forthcoming), for all other data sets we take ρ from Becker et al. (2021). Standard errors are bootstrapped with 1,000 draws. These standard errors are likely conservative (Becker et al., 2021; Sanz-de-Galdeano and Terskaya, forthcoming).

TABLE S.10 – Association of Skill Measures with PGI^{EA} and Q^S by Waves

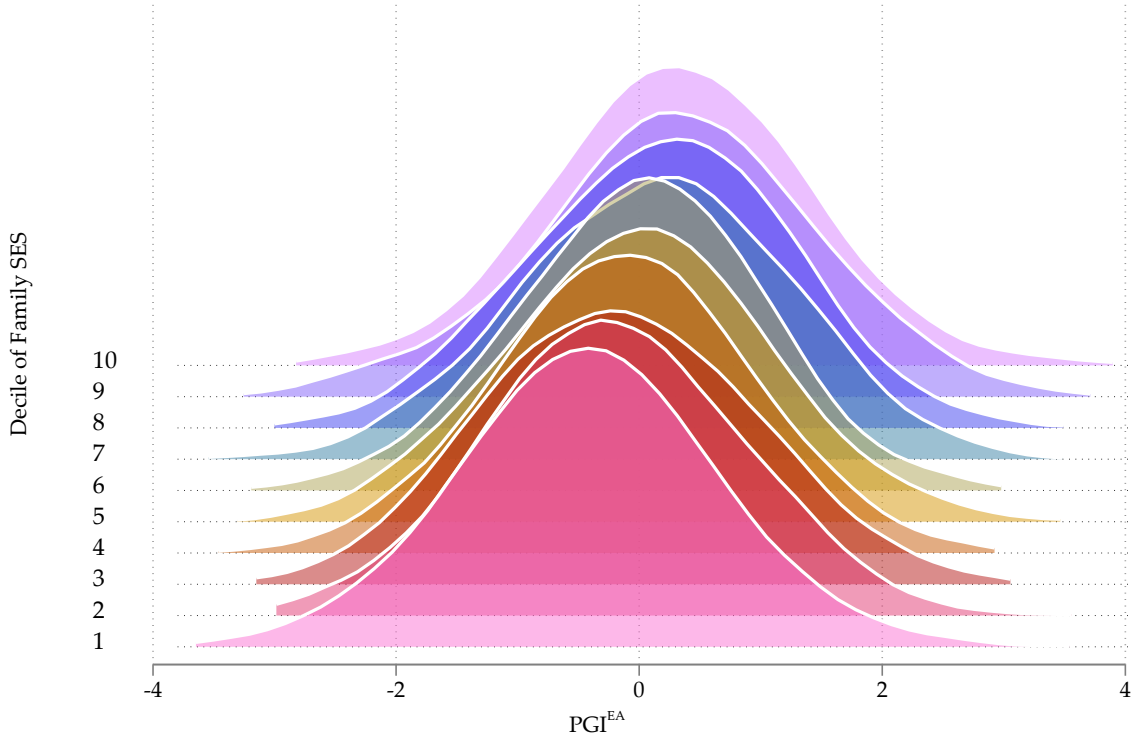
	Wave 1/2				Wave 3/4			
	PVT (1)	Risk (2)	Patience (3)	Subj. health (4)	PVT (5)	Risk (6)	Patience (7)	Subj. health (8)
PGI^{EA}	0.153***	0.038	0.077***	0.040*	0.161***	0.053***	0.103***	0.083***
	(0.020)	(0.026)	(0.026)	(0.022)	(0.016)	(0.020)	(0.022)	(0.019)
Q^S	0.120***	0.052	0.121***	0.100**	0.144***	0.107**	0.081	0.017
	(0.042)	(0.043)	(0.037)	(0.041)	(0.053)	(0.044)	(0.053)	(0.047)
$PGI^{EA} \times Q^S$	-0.025	-0.031	-0.020	-0.049**	-0.062***	-0.032*	-0.046**	-0.066***
	(0.019)	(0.028)	(0.021)	(0.024)	(0.015)	(0.019)	(0.020)	(0.019)
Child Controls	✓	✓	✓	✓	✓	✓	✓	✓
Family Controls	✓	✓	✓	✓	✓	✓	✓	✓
Control Function	✓	✓	✓	✓	✓	✓	✓	✓
N	1,784	1,784	1,784	1,784	1,784	1,784	1,784	1,784
R^2	0.235	0.080	0.085	0.093	0.206	0.118	0.118	0.088

Data: National Longitudinal Study of Adolescent to Adult Health.

Note: Own calculations. This table shows the joint association of PGI^{EA} and Q^S with cognitive skills, preferences, and health. Columns (1)-(4) show skill measures in waves 1 and 2 of Add Health. Columns (5)-(8) show skill measures in waves 3 and 4 of Add Health. We restrict the sample to all individuals in grade 9 and higher during wave 1 of Add Health with non-missing information on the relevant skill measures. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Significance levels: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Standard errors (in parentheses) are clustered at the school level.

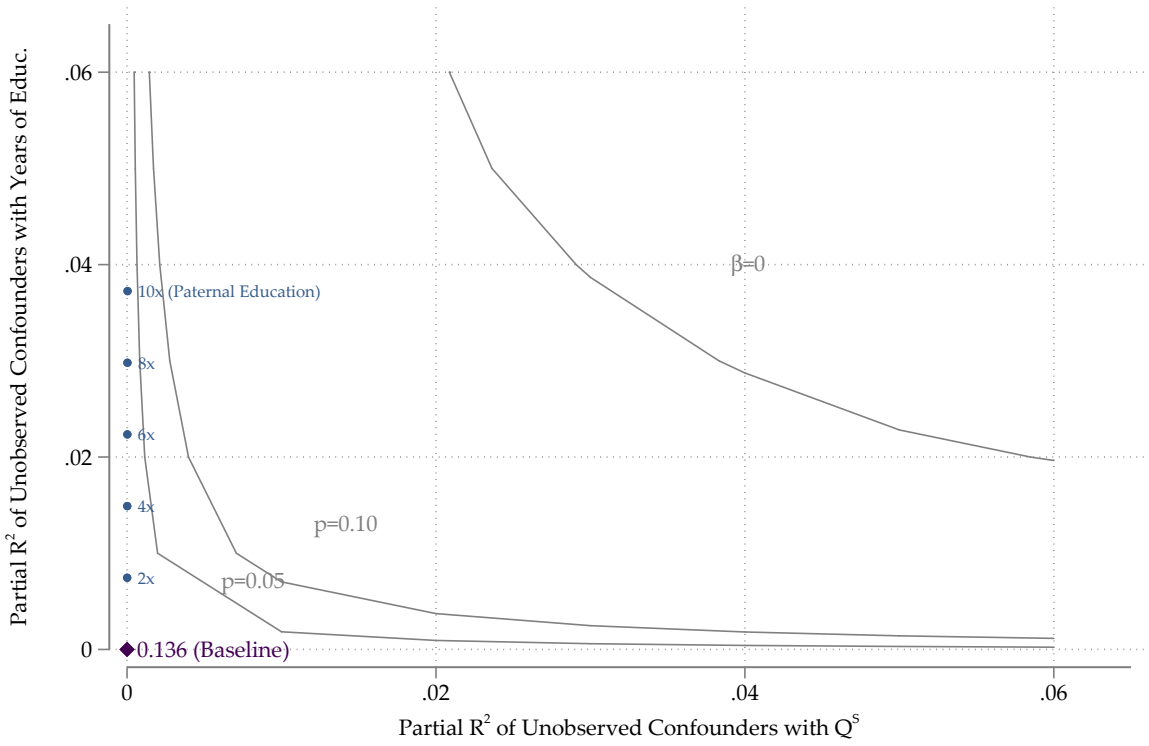
B SUPPLEMENTARY FIGURES

FIGURE S.1 – Distribution of PGI^{EA} by Family SES



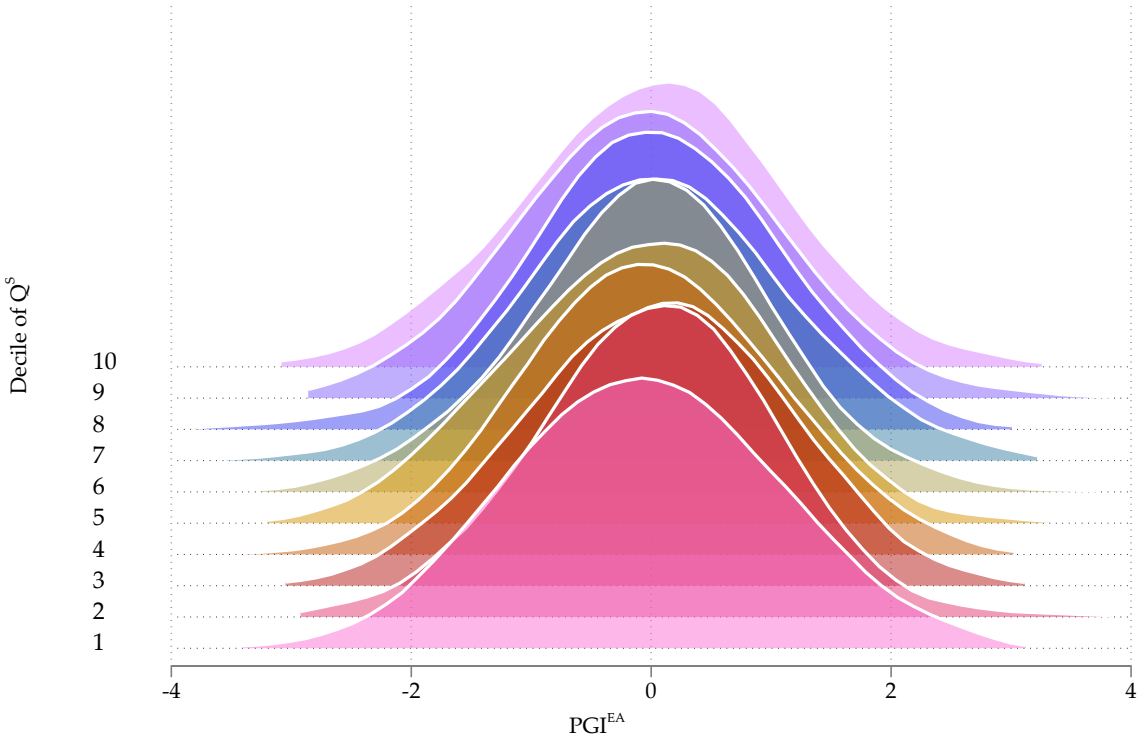
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows unconditional PGI^{EA} distribution by deciles of family SES. Density distributions are smoothed using the Epanechnikov kernel function with a bandwidth of 0.5.

FIGURE S.2 – Sensitivity of Q^S to Unobserved Confounders



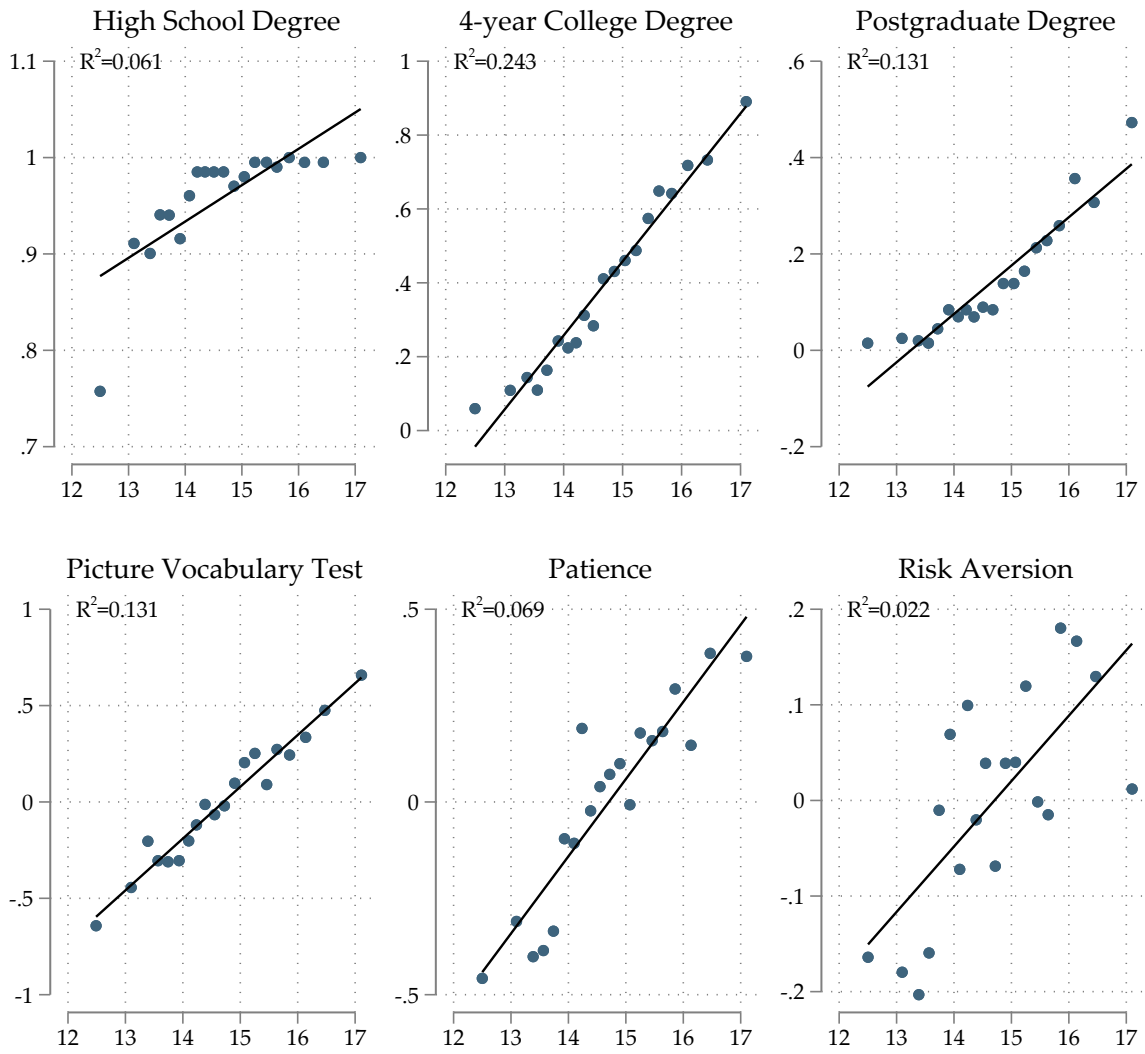
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows the sensitivity of the point estimate for the impact of Q^S on years of education to unobserved confounding variables. The diamond shows baseline estimates from Figure 1. Cinelli and Hazlett (2020) show that omitted variable bias is a function of two partial R^2: (i) the partial R^2 of unobserved confounders with the outcome variable conditional on controls (y-axis), (ii) the partial R^2 of unobserved confounders with the treatment variable conditional on controls (x-axis). We plot three contour lines showing for which combinations of (i) and (ii), the coefficient of Q^S would drop below a significance level of 5%, 10%, and when it would flip signs. For comparison, we also plot adjusted treatment effects of Q^S assuming that we would control for an unobserved confounder with values of (i) and (ii) that are a (2x-, 4x-, 6x-, 8x-, 10x-) multiple of paternal education (measured in years). This comparison suggests that an observed confounder even 10 times as strong as paternal education could not drive the coefficient of Q^S below a significance level of 5%. This result is mainly driven by the low partial R^2 of paternal education with Q^S after partialling out our controls. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Standard errors are clustered at the school level.

FIGURE S.3 – Distribution of PGI^{EA} by Q^S



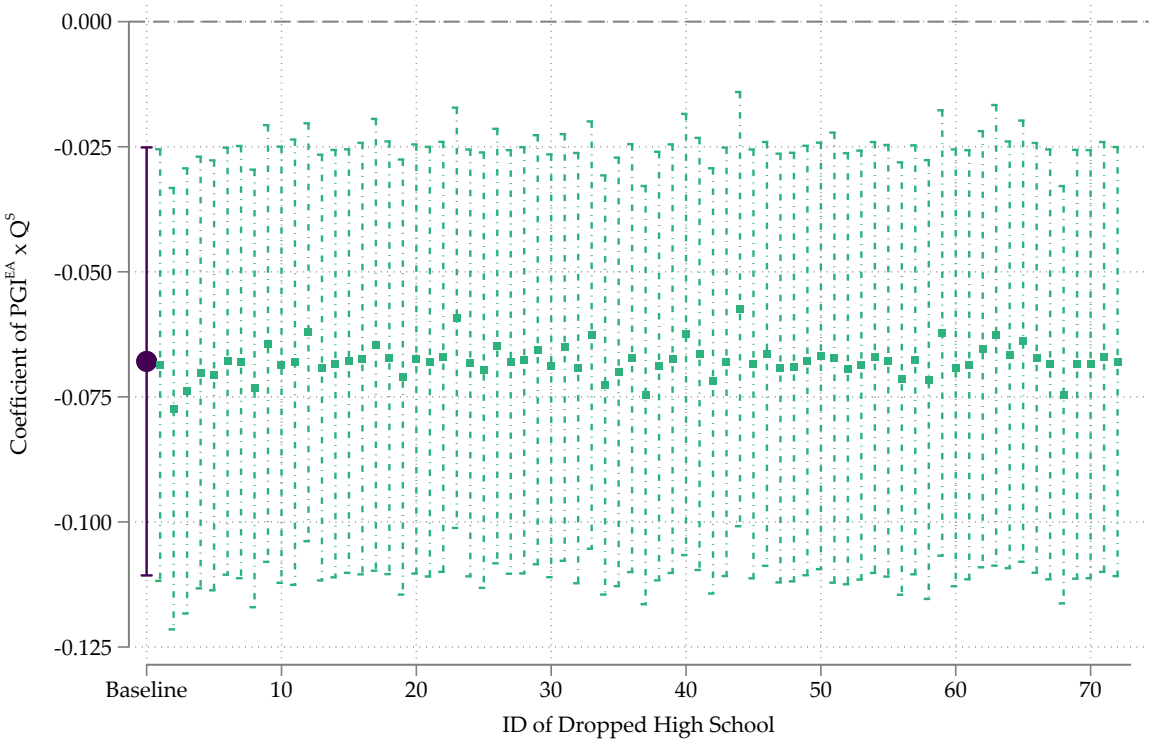
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows PGI^{EA} distribution by deciles of Q^Safter residualizing PGI^{EA} and Q^S by the full set of control variables. Density distributions are smoothed using the Epanechnikov kernel function with a bandwidth of 0.5.

FIGURE S.4 – Association of Educational Attainment and Skills with Predicted Education



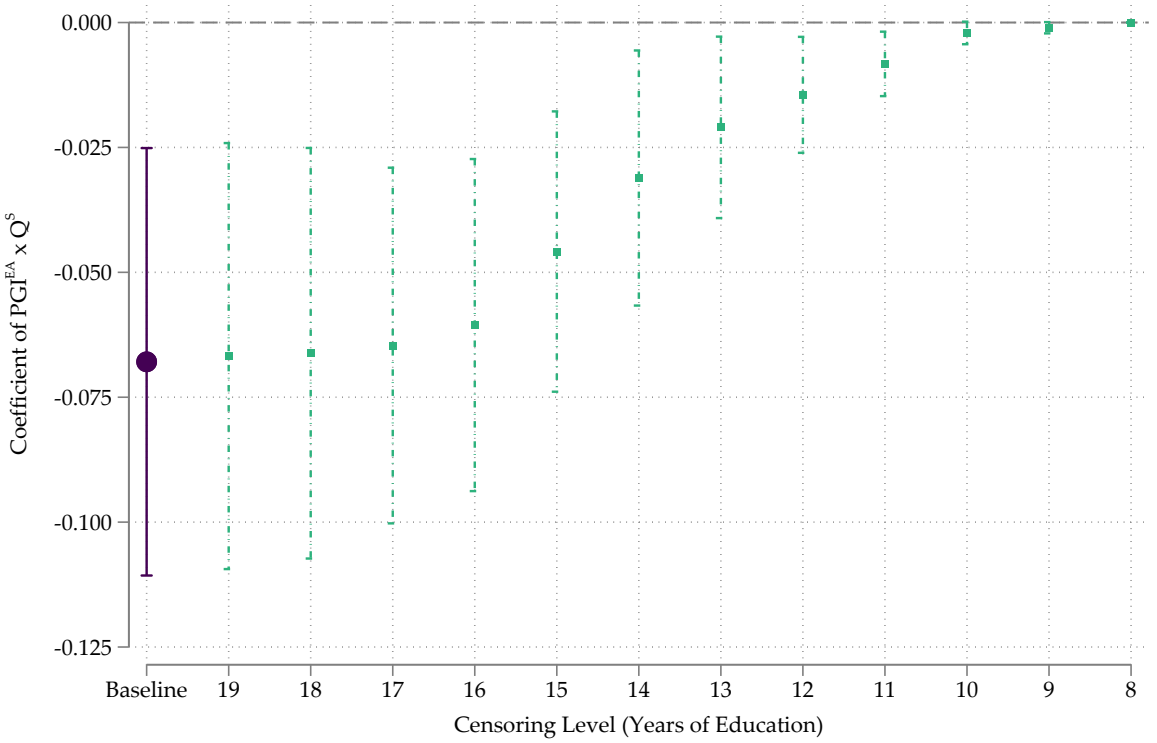
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows the association of various meausures for educational attainment and (non-)cognitive skills with predicted years of education. We bin scatterplots using 20 quantiles of the variable of interest. Black lines are fitted from linear regressions of the variable of interest on predicted education. Predicted education is calculated from a regression of completed years of education on all *Child Controls* and *Family Controls*. Outcomes are measured in Waves 3, 4 and 5. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects.

FIGURE S.5 – Sensitivity to Outlier Schools



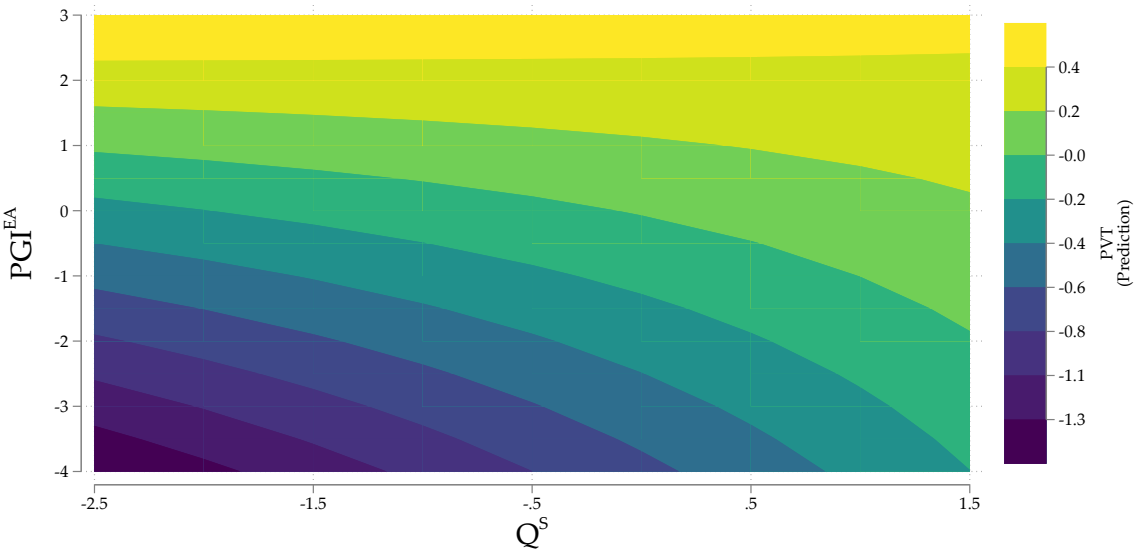
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows point estimates and 90% confidence bands of the interaction of PGI^{EA} with Q^S, and its association with years of education. Each estimate is derived from a subsample of the data in which we drop one High School, respectively. Estimates follow the specification of equation (6). *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Standard errors are clustered at the school level.

FIGURE S.6 – Sensitivity to Ceiling Effects



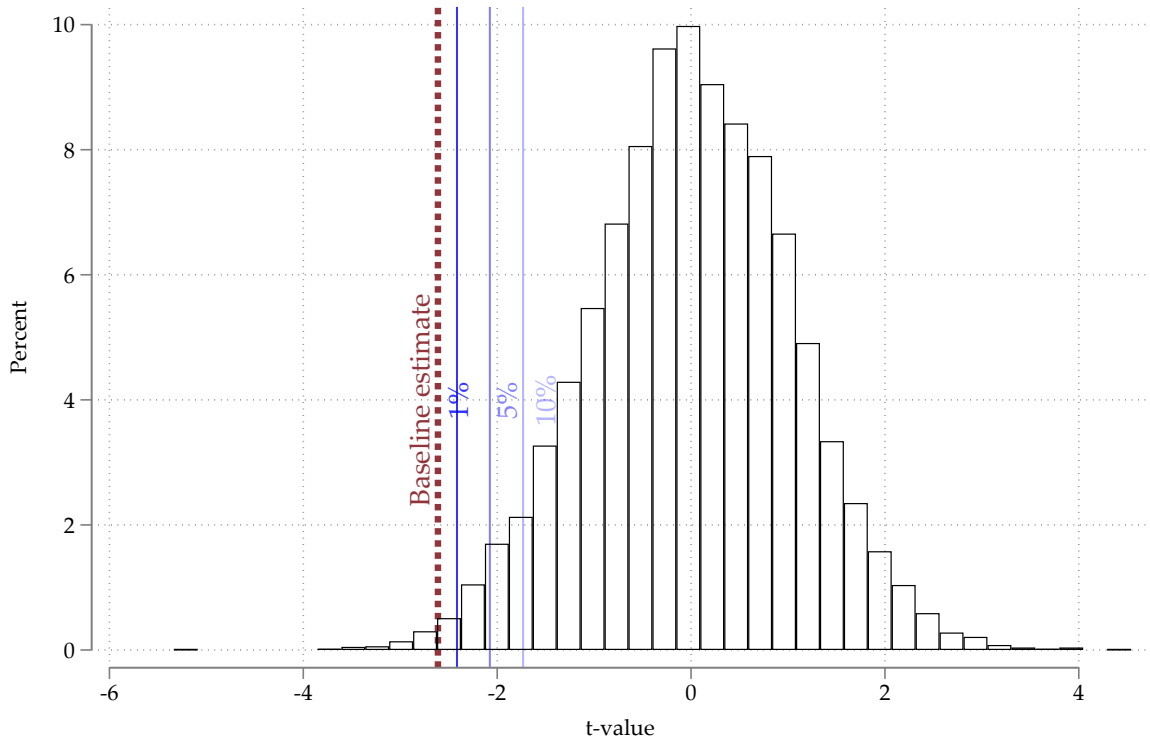
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows point estimates and 90% confidence bands of the interaction of PGI^{EA} and Q^S, and its association with years of education. Each estimate is derived from the full sample while censoring the outcome variable at different levels from above. Estimates follow the specification of equation (6). *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0, \sigma = 1$). Standard errors are clustered at the school level.

FIGURE S.7 – Association of Picture Vocabulary Test with PGI^{EA} by Q^S



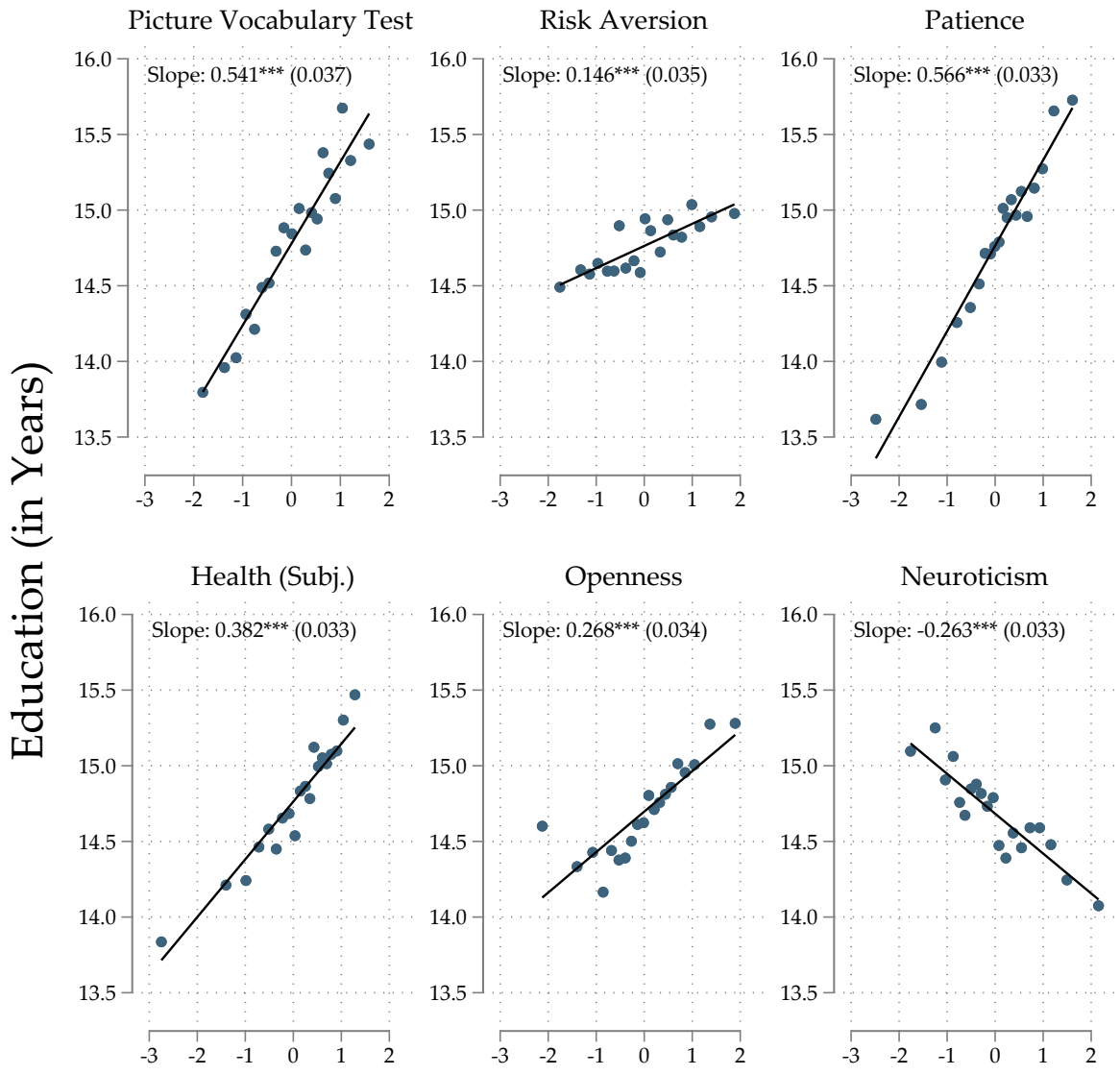
Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows predictions of the PVT by PGI^{EA} and Q^S cell. Predictions are calculated using the model estimated in column (1) in Panel (a) of Table 7 while allowing for non-linear effects over the range of -4.0(0.5)3.0 of PGI^{EA} and -2.5(0.5)1.5 of Q^S.

FIGURE S.8 – Permutation test for placebo assignments of Q^S



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows the frequency distribution of *t*-statistics for the interaction of PGI^{EA} and Q^S under 10,000 permutations of Q^S. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages. All right-hand side variables are standardized on the estimation sample ($\mu = 0$, $\sigma = 1$). Standard errors are clustered at the school level.

FIGURE S.9 – Association of Educational Attainment with (Non-)Cognitive Skills



Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This figure shows the association of educational attainment and various measures of (non-)cognitive skills. We bin scatterplots using 20 quantiles of the variable of interest. Black lines are fitted from linear regressions of educational attainment on the variable of interest and the full set of controls. *Child Controls*: Firstborn dummy, linear birth cohort trend (in months) by gender, 20 principal components of the full matrix of genetic data. *Family Controls*: Age of mother at birth, years of education of both mother and father, average potential wages of both mother and father, the standard deviation of potential wages of both mother and father, dummies for non-US born mothers and fathers, a dummy for Christian religion, state fixed effects. *Control Function*: Share white, share single mothers, maternal education (average), share females, share migrants. All control function variables are calculated as leave-cohort-out school averages.

C BIAS IN GENE-ENVIRONMENT INTERACTION

Assume the following population model:

$$y_i = \alpha + \beta_1 x_{1i} + \beta_2 x_{2i} + \beta_3 (x_{1i} \cdot x_{2i}) + \delta_1 z_i + \delta_2 (x_{1i} \cdot z_i) + \delta_3 (x_{2i} \cdot z_i) + \epsilon_i,$$

with

$$(x_{1i}, x_{2i}, z_i) \sim N\left(0, \begin{bmatrix} 1 & 0 & \sigma^2_{x_1 z} \\ 0 & 1 & \sigma^2_{x_2 z} \\ \sigma^2_{x_1 z} & \sigma^2_{x_2 z} & 1 \end{bmatrix}\right), \; Cov(\epsilon | z, x_1, x_2) = 0.$$

Note we assume $\sigma^2_{x_1 x_2} = 0$ based on the evidence presented in Figure 2. Furthermore, we assume $\sigma^2_{x_1 z} \geq 0, \sigma^2_{x_2 z} \geq 0$ to reflect concerns about i) genetic nurture and ii) selection into schools based on unobservable family background characteristics z.

Since z is unobserved, we estimate the following model:

$$y_i = \tilde{\alpha} + \tilde{\beta}_1 x_{1i} + \tilde{\beta}_2 x_{2i} + \tilde{\beta}_3 (x_{1i} \cdot x_{2i}) + \tilde{\epsilon}_i.$$

What is the bias in the estimated gene-environment interaction $\tilde{\beta}_3$?

By the weak law of large numbers, we know that

$$\hat{\tilde{\beta}} \xrightarrow{p} \beta^* := [\mathbb{E}(X^T X)]^{-1} \mathbb{E}[X^T y].$$

First, under our assumptions about (x_1, x_2), $[\mathbb{E}(X^T X)]$ simplifies to the identity matrix:

$$\mathbb{E}[X^T X] = \begin{bmatrix} 1 & \mathbb{E}[x_1] & \mathbb{E}[x_2] & \mathbb{E}[x_1 x_2] \\ \mathbb{E}[x_1] & \mathbb{E}[x_1^2] & \mathbb{E}[x_1 x_2] & \mathbb{E}[x_1^2 x_2] \\ \mathbb{E}[x_2] & \mathbb{E}[x_1 x_2] & \mathbb{E}[x_2^2] & \mathbb{E}[x_1 x_2^2] \\ \mathbb{E}[x_1 x_2] & \mathbb{E}[x_1^2 x_2] & \mathbb{E}[x_1 x_2^2] & \mathbb{E}[x_1^2 x_2^2] \end{bmatrix} = \begin{bmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{bmatrix}.$$

Therefore, bias will be driven by $\mathbb{E}[X^T y]$ only.

Second, under our assumptions about (x_1, x_2, z), $\mathbb{E}[X^T y]$ reads as follows:

$$\mathbb{E}[X^T y] = \begin{bmatrix} \alpha + \delta_2 \mathbb{E}[x_1 z] + \delta_3 \mathbb{E}[x_2 z] \\ \beta_1 + \delta_1 \mathbb{E}[x_1 z] \\ \beta_2 + \delta_1 \mathbb{E}[x_2 z] \\ \beta_3 + \delta_2 \mathbb{E}[x_1^2 x_2 z] + \delta_3 \mathbb{E}[x_1 x_2^2 z] \end{bmatrix} = \begin{bmatrix} \alpha + \delta_2 \sigma^2_{x_1 z} + \delta_3 \sigma^2_{x_2 z} \\ \beta_1 + \delta_1 \sigma^2_{x_1 z} \\ \beta_2 + \delta_1 \sigma^2_{x_2 z} \\ \beta_3 + \delta_2 \sigma^2_{x_2 z} + \delta_3 \sigma^2_{x_1 z} \end{bmatrix}.$$

Therefore, bias in the gene-environment interaction is captured by:

$$\underbrace{\hat{\tilde{\beta}}_3 - \beta_3}_{=\text{Bias}} = \delta_2 \sigma^2_{x_2 z} + \delta_3 \sigma^2_{x_1 z}.$$

Based on the best evidence from existing empirical literature, we make the following assumptions on the signs of δ_2 and δ_3:

$\delta_2 \geq 0 \rightarrow$ We assume (weak) complementarity between genetic endowments and (unobserved) family background characteristics. This assumption is consistent with existing literature confirming (weakly) positive interactions between children's genetic endowments and parental SES (among others Figlio et al., 2017; Ronda et al., 2022; Turkheimer et al., 2003).

$\delta_3 \leq 0 \rightarrow$ We assume (weak) substitutability between school quality and (unobserved) family background characteristics. This assumption is consistent with existing literature confirming negative interactions between school quality and family SES (among others Cohodes et al., 2021; Jackson et al., 2024).

Under these maintained assumptions, the sign of bias depends on (i) the relative strengths of relationships of the unobserved interaction terms with outcome y, and (ii) the relative magnitudes of the covariances of genetic endowments and school quality with the unobserved confounder:

$$\underbrace{\frac{\delta_2}{\delta_3}}_{=(i)\leq 0} + \underbrace{\frac{\sigma^2_{x_1 z}}{\sigma^2_{x_2 z}}}_{=(ii)\geq 0} \lessgtr 0.$$

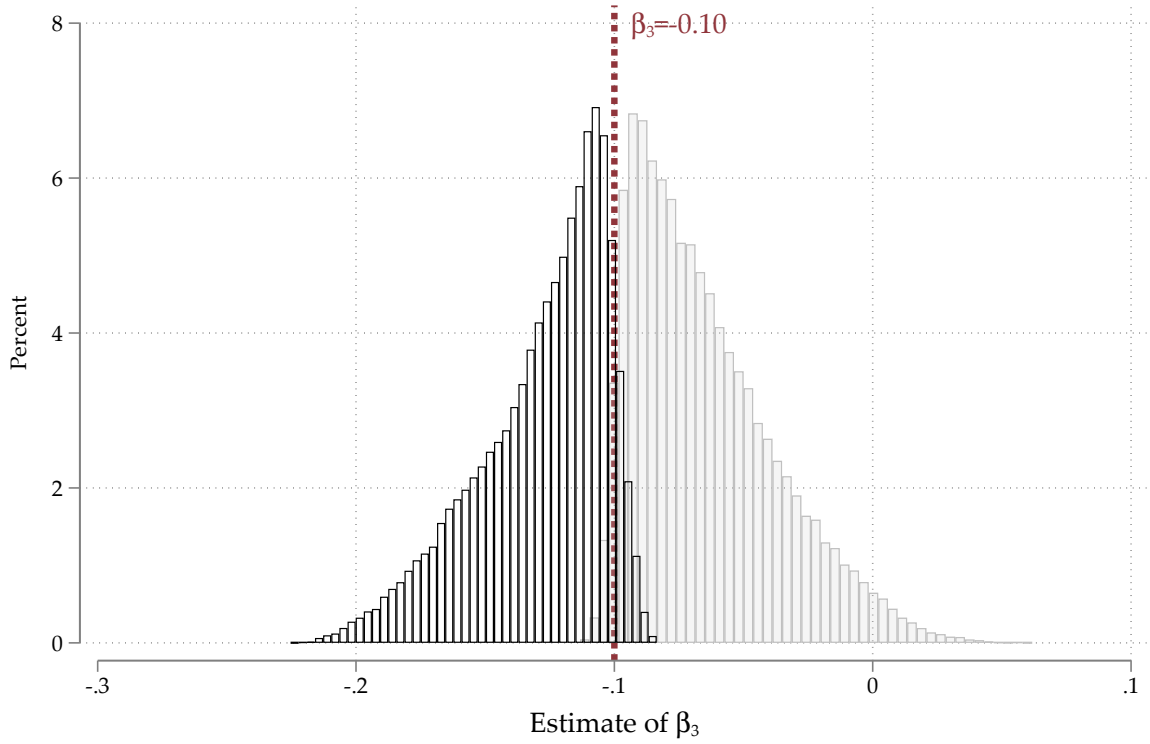
The previous expressions suggest the following conclusions.

First, our maintained assumptions are still insufficient to sign the bias due to the opposite signs of (i) and (ii).

We confirm this conjecture empirically in a simulation where we estimate β_3 under different assumptions about $\delta_2, \delta_3, \sigma^2_{x_1 z}, \sigma^2_{x_2 z}$. Figure S.10 shows that estimates fall symmetrically to both sides of the true parameter value $\beta_3 = -0.1$, i.e., bias can be either positive or negative. Furthermore, we show that positive bias most likely occurs when we set the simulation parameters such that $|(i)| < |(ii)|$ (gray-shaded bars) and that negative bias most likely occurs when we set the simulation parameters such that $|(i)| > |(ii)|$ (dark-lined bars).

Second, our expression of bias illustrates that the likely direction of bias crucially depends on the extent of confounding through genetic nurture. As $\sigma^2_{x_1 z} \rightarrow 0$, bias depends more strongly on the magnitudes of δ_2 and $\sigma^2_{x_2 z}$ and less strongly on the magnitude of δ_3. Therefore, bias is

FIGURE S.10 – Simulation for $\beta_3 = -0.10$



Data: Simulated data.
Note: Own calculations. This figure presents estimates of β_3 under different assumptions about $\delta_2, \delta_3, \sigma^2_{x_1z}, \sigma^2_{x_2z}$. The simulation is based on the following data generating process: $y_i = 0.4x_{1i} + 0.2x_{2i} - 0.1(x_{1i} \cdot x_{2i}) + 0.2z_i + \delta_2(x_{1i} \cdot z_i) + \delta_3(x_{2i} \cdot z_i) + \epsilon_i$, where $\sigma^2_{x_1x_2} = 0$, $\sigma^2_{x_1z} = \sigma^2_{x_2z} \in [0.0(0.05)0.50]$, $\delta_2 \in [0.0(0.01)0.20]$, $\delta_3 \in [-0.20(0.01)0.00]$. We run 50 iterations for each combination of parameters. Gray-shaded bars represent estimates for parameter combinations such that $|(i)| < |(ii)|$. Dark-lined bars represent estimates for parameter combinations such that $|(i)| > |(ii)|$.

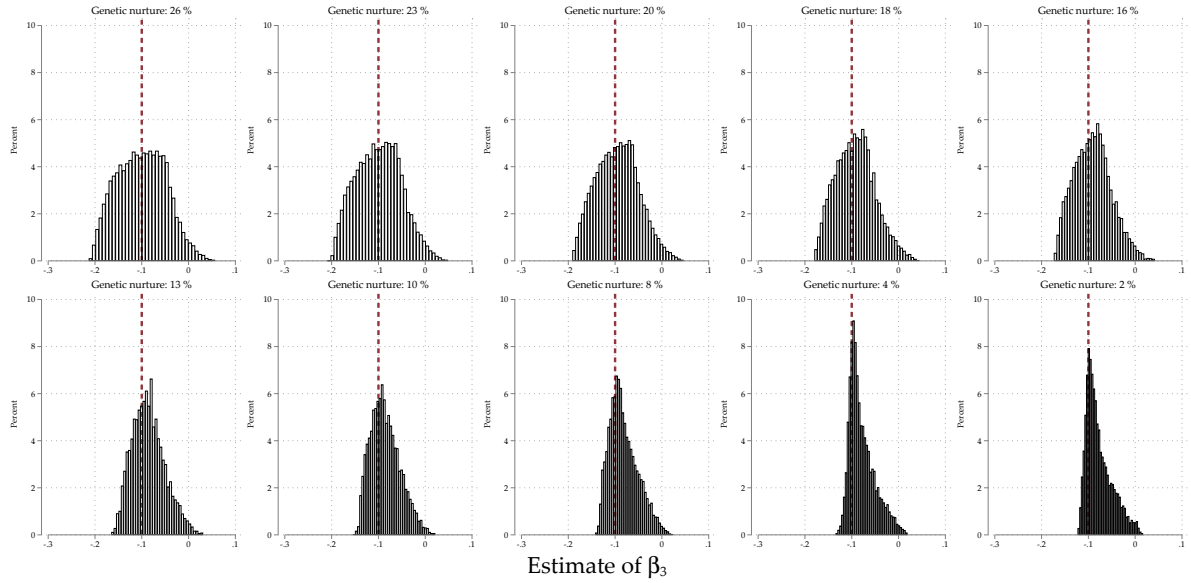
more likely to be positive (less likely to be negative) if the extent of confounding by genetic nurture decreases.

Again, we confirm this conjecture empirically through our simulation. Figure S.11 shows the distribution of estimates under different assumptions about $\sigma^2_{x_1z}$. As $\sigma^2_{x_1z} \to 0$, the implied extent of confounding through genetic nurture decreases, and the distribution of estimates shifts to the right of the true parameter value $\beta_3 = -0.1$.

Third, if we assume the absence of genetic nurture, i.e., $\sigma^2_{x_1z} = 0$, the extent of positive bias increases with $\sigma^2_{x_2z}$ and δ_2.

Again, we confirm this conjecture empirically through our simulation. Figure S.12 shows that estimates of β_3 increase with increasing $\sigma^2_{x_2z}$ and δ_2. That is, the stronger the residual selection into schools that is not accounted for by our control variables ($\sigma^2_{x_2z}$) and the stronger the predictive power of interactions between children's genetic endowments and parental SES for outcome y (δ_2), the more would our estimated gene-environment interaction be attenuated to-

FIGURE S.11 – Simulation for $\beta_3 = -0.10$ under decreasing $\sigma^2_{x_1z}$

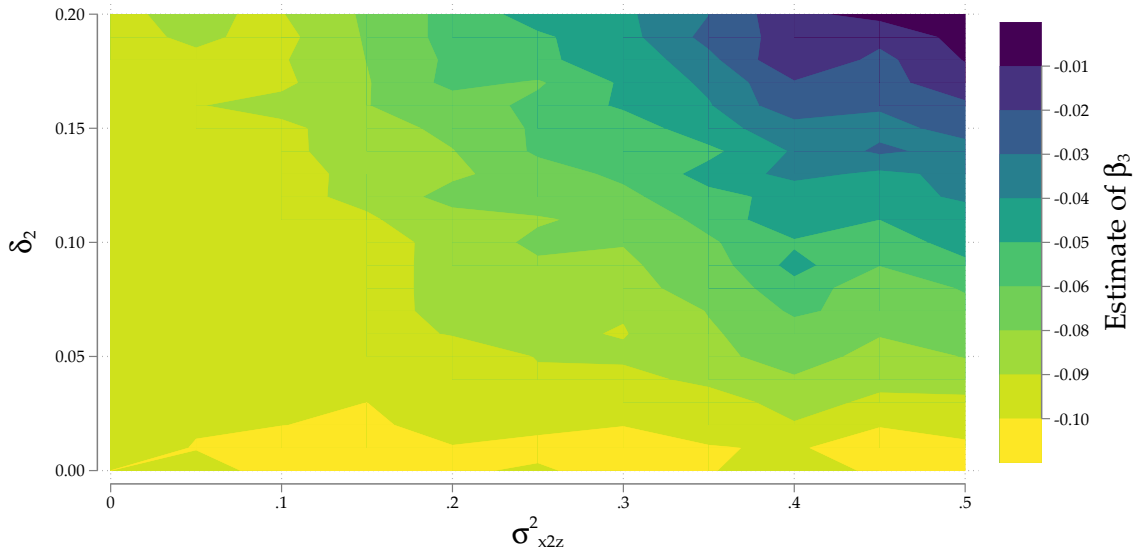


Data: Simulated data.
Note: Own calculations. This figure presents estimates of β_3 under different assumptions about $\delta_2, \delta_3, \sigma^2_{x_1z}, \sigma^2_{x_2z}$. The simulation is based on the following data generating process: $y_i = 0.4x_{1i} + 0.2x_{2i} - 0.1(x_{1i} \cdot x_{2i}) + 0.2z_i + \delta_2(x_{1i} \cdot z_i) + \delta_3(x_{2i} \cdot z_i) + \epsilon_i$, where $\sigma^2_{x_1x_2} = 0$, $\sigma^2_{x_1z} = \sigma^2_{x_2z} \in [0.0(0.05)0.50]$, $\delta_2 \in [0.0(0.01)0.20]$, $\delta_3 \in [-0.20(0.01)0.00]$. We run 50 iterations for each combination of parameters. Each panel represent simulation results for a different specification of $\sigma^2_{x_1z}$ over the interval $\sigma^2_{x_1z} = [0.05(0.05)0.50]$. We denote the implied extent of confounding through genetic nurture in the table header.

wards zero.

The assumption of $\sigma^2_{x_1z} = 0$ is supported by the similarity of results from between- and within-family models presented in Table 2. Therefore, we interpret our estimates as a lower bound of the substitutability between genetic endowments and school quality.

FIGURE S.12 – Simulation for $\beta_3 = -0.10$ with $\sigma^2_{x_1z} = 0$



Data: Simulated data.
Note: Own calculations. This figure presents estimates of β_3 under different assumptions about $\delta_2, \delta_3, \sigma^2_{x_2z}$, while fixing $\sigma^2_{x_1z} = 0$. The simulation is based on the following data generating process: $y_i = 0.4x_{1i} + 0.2x_{2i} - 0.1(x_{1i} \cdot x_{2i}) + 0.2z_i + \delta_2(x_{1i} \cdot z_i) + \delta_3(x_{2i} \cdot z_i) + \epsilon_i$, where $\sigma^2_{x_1x_2} = 0$, $\sigma^2_{x_1z} = 0$, $\sigma^2_{x_2z} \in [0.0(0.05)0.50]$, $\delta_2 \in [0.0(0.01)0.20]$, $\delta_3 \in [-0.20(0.01)0.00]$. We run 50 iterations for each combination of parameters.

D DATA APPENDIX

D.1 Outcome variables

Educational attainment. We measure educational attainment by total *years of education*. In each wave, respondents were asked about their highest level of education at the time of the interview. We use the most recent information for each respondent and transform education levels into years of education following the mapping suggested by Domingue et al. (2015). Numeric values in parentheses: eighth grade or less (8), some high school (10), high school graduate (12), GED (12), some vocational/technical training (13), some community college (14), some college (14), completed vocational/technical training (14), associate or junior college degree (14), completed college (16), some graduate school (17), completed a Master degree (18), some post-baccalaureate professional education (18), some graduate training beyond a master's degree (19), completed post-baccalaureate professional education (19), completed a doctoral degree (20).

We use the most recent available information to construct the following measures for educational degrees: *High School* (including GED), *2-year College*, *4-year College*, and *Post-Graduate*. Two-year college degrees include associate and junior college degrees and vocational and technical training after high school. Four-year college degrees include bachelor's degrees. Post-graduate degrees include master's, doctoral, and post-baccalaureate professional degrees. If available, information is taken from wave 5; otherwise, we take it from wave 4 or 3, respectively. We only include respondents for whom we observe educational degrees when they are at least 27 years old at the time of observation. We assume an ordinal ranking of degrees (high school < 2-year college < 4-year college < post-graduate) and assign the possession of a lower-ranked degree if a respondent obtained a higher-ranked degree. For example, we assume that a respondent has finished high school if he or she has obtained a college degree, even if we do not have explicit information about high school graduation status.

Health. We proxy *subjective health* by quality-adjusted life years (QALY) that we derive from self-assessed health (SAH) measures. We use information from waves 3 and 4, where participants were asked "in general, how is your health?" We convert their (categorical) responses into a continuous measure using a mapping proposed by Van Doorslaer and Jones (2003). Using information about objective health—the Health Utility Index Mark III—Van Doorslaer and Jones (2003) scale the intervals of the SAH categories. This approach yields "quality weights" for health between 0 and 1. The values for each health status category are as follows (quality weights in parentheses): "excellent" (0.9833), "very good" (0.9311), "good" (0.841), "fair" (0.707), and "poor" (0.401).[1] We average resulting QALY measures across waves 3 and 4.

[1] See Table 4 in Van Doorslaer and Jones (2003).

We construct an index of *objective health* based on information from wave 4. Specifically, we sum the standardized values about whether a respondent (i) is obese, (ii) has stage one hypertension, and (iii) has high cholesterol (as indicated by the respondent). Each item was answered with either "yes" (= 1) or "no" (= 0). We reverse-code our measure of objective health such that higher values indicate better health.

Cognitive skills. The *Picture Vocabulary Test* (PVT) is a test for receptive hearing vocabulary and a widely used proxy for verbal ability and scholastic aptitude. To administer the PVT, an examiner presents a series of pictures to the respondent. There are four pictures per page, and the examiner speaks a word describing one of the pictures. The respondent then has to indicate the picture that the word describes. Our analysis uses age-adjusted PVT percentile ranks from wave 3 (Harris, 2020).

Preferences. We construct two measures of preferences: *risk aversion* and *patience*. In waves 3 and 4, participants were asked (i) whether they like to take risks and (ii) whether they live without much thought for the future. Questions were answered on a five-point Likert scale ranging from "strongly agree" to "strongly disagree." We reverse-code both measures and use averages from waves 3 and 4 in our analysis.

Personality. The Big Five personality traits are openness to experience, conscientiousness, extraversion, agreeableness, and neuroticism (Almlund et al., 2011). We use information from wave 4 to construct personality measures. Participants were asked questions related to one of the five personality traits. Questions were answered on a five-point Likert scale ranging from "strongly agree" to "strongly disagree." We use averages of the following questions in our analysis. *Openness*: (i) "I have a vivid imagination," (ii) "I have difficulty understanding abstract ideas" (reverse-coded), (iii) "I am not interested in abstract ideas" (reverse-coded), (iv) "I do not have a good imagination" (reverse-coded). *Conscientiousness*: (i) "I get chores done right away," (ii) "I like order," (iii) "I often forget to put things back in their proper place" (reverse-coded), (iv) "I make a mess of things" (reverse-coded). *Extraversion*: (i) "I am the life of the party," (ii) "I talk to a lot of different people at parties," (iii) "I don't talk a lot" (reverse-coded), (iv) "I keep in the background" (reverse-coded). Agreeableness: (i) "I sympathize with others' feelings," (ii) "I feel others' emotions," (iii) "I am not interested in other people's problems" (reverse-coded), (iv) "I am not really interested in others" (reverse-coded). *Neuroticism*: (i) "I have frequent mood swings," (ii) "I get upset easily," (iii) "I am relaxed most of the time" (reverse-coded), (iv) "I seldom feel blue" (reverse-coded).

Family investments. To measure *parental time investments*, we use the information on a series of activities children have done with their mother or father in the last four weeks. Specifically,

the child is asked whether he or she has (i) gone shopping, (ii) played a sport, (iii) gone to a religious service or church-related event, (iv) talked about someone he or she is dating, or a party he or she went to, (v) gone to a movie, play, museum, concert, or sports event, (vi) had a talk about a personal problem he or she was having, (vii) talked about his or her school work or grades, (viii) worked on a project for school, (ix) talked about other things he or she is doing in school. Questions were answered with "yes" (= 1) or "no" (= 0). We standardize answers to $\mu = 0$ and $\sigma = 1$ on the full sample of Add Health respondents and then sum across dimensions (Anderson, 2008; Kling et al., 2007).

To measure *parenting style*, we follow Agostinelli et al. (forthcoming) and use child responses on whether they can make the following decisions on their own without interference from their parents: (i) the time they must be home on weekend nights, (ii) the people they hang around with, (iii) what you wear, and (iv) what time they go to bed on weeknights. Questions were answered with "yes" (= 1) or "no" (= 0). We standardize answers to $\mu = 0$ and $\sigma = 1$ on the full sample of Add Health respondents and then sum across dimensions (Anderson, 2008; Kling et al., 2007).

D.2 Variables of interest

Polygenic indexes. Add Health obtained saliva samples from consenting participants in wave 4. After quality control procedures, genotyped data is available for 9,974 individuals and 609,130 SNPs. Add Health uses this data to calculate a set of different PGIs using summary statistics from existing GWAS. Our baseline measure PGI^{EA} is based on statistics from Lee et al. (2018).

School characteristics. In waves 1 and 2, Add Health administered questionnaires to head-teachers of Add Health schools. We use this information to construct an indicator for high school quality using principal components analysis (PCA). We extract the first component from a PCA that includes the following school-level information (component loadings in parentheses): (i) the average class size (-0.31), (ii) the share of teachers with a master's degree (+0.63), (iii) the share of new teachers in the current school year (-0.36), (iv) the share of teachers with school-specific tenure of more than five years (+0.62). We apply a factor rotation for interpretability reasons (oblique oblimin rotation of the Kaiser normalized matrix with $\gamma = 0$; see Gorsuch, 1983). The calculated factor is standardized to $\mu = 0$ and $\sigma = 1$ on the entire sample of Add Health respondents in wave 1.

Alternatively, we use the same information and aggregate across dimensions using the procedure suggested in Anderson (2008) and Kling et al. (2007). The calculated factor is standardized to $\mu = 0$ and $\sigma = 1$ on the entire sample of Add Health respondents in wave 1.

TABLE S.11 – Summary Statistics (Outcomes)

	Obs.	Mean	SD	Min	Max
Educational Attainment					
Years of Education	4,034	14.68	2.27	8.00	20.00
High School Degree	4,032	0.96	0.20	0.00	1.00
2-year College Degree	4,034	0.50	0.50	0.00	1.00
4-year College Degree	4,034	0.39	0.49	0.00	1.00
Post-Graduate Degree	4,034	0.14	0.35	0.00	1.00
Health					
Subjective	3,471	0.91	0.07	0.40	0.98
Objective	4,034	0.05	1.94	-6.46	1.62
Cognitive Skills					
Picture Vocabulary Test	3,380	59.25	26.34	0.00	100.00
Preferences					
Risk Aversion	3,465	2.82	0.86	1.00	5.00
Patience	3,465	3.91	0.73	1.00	5.00
Personality					
Openness	3,996	3.63	0.63	1.00	5.00
Conscientousness	4,028	3.64	0.69	1.25	5.00
Extraversion	4,027	3.33	0.77	1.00	5.00
Agreeableness	4,026	3.86	0.59	1.00	5.00
Neuroticism	4,022	2.57	0.70	1.00	5.00
Family Investments					
Parental Time Investment	4,034	0.64	6.88	-13.68	28.53
Parenting Style	4,034	-0.21	2.25	-2.87	7.36

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows summary statistics for outcome variables in our core analysis sample. The sample is restricted to genotyped individuals who (i) are of European descent, and (ii) attended an Add Health high school or an associated feeder school in wave 1.

Family socioeconomic status. We use the *social origins factor score* constructed by Belsky et al. (2018). Their measure uses information about parental education, occupation, household income, and household receipt of public assistance in wave 1. The score is standardized to $\mu = 0$ and $\sigma = 1$ on the entire sample of Add Health respondents in wave 1.

D.3 Control variables

Child characteristics. The child's *gender* (female or male, as indicated by the interviewer) is taken from the in-home questionnaire in wave 1.

We calculate the child's *age* (in months) at each wave by subtracting the child's birth date from

TABLE S.12 – Summary Statistics (Variables of Interest)

	Obs.	Mean	SD	Min	Max
Polygenic Scores					
PGI^{EA}	4,034	0.02	0.99	-4.13	3.39
School Characteristics					
Q^S (PCA)	4,034	0.26	1.23	-3.17	2.49
Q^S (Anderson, 2008)	4,034	0.25	0.90	-2.59	1.98
Teacher w/ MA (%)	4,034	51.20	24.11	0.00	95.00
Experienced Teacher (%)	4,034	66.65	23.43	0.00	98.00
New Teacher (%)	4,034	7.88	7.28	0.00	47.00
Class Size	4,034	24.40	4.50	12.00	38.00
Family SES					
Social Origins Factor Score	3,958	0.33	1.14	-4.51	3.51

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows summary statistics for variables of interest in our core analysis sample. The sample is restricted to genotyped individuals who (i) are of European descent, and (ii) attended an Add Health high school or an associated feeder school in wave 1.

the interview date. Because birth dates have minor inconsistencies across waves, we take averages across waves 1 to 4.

We use the first 20 *principal components* of the full matrix of the genetic data. The components are obtained from a principal components analysis on the matrix of SNPs in Add Health (see Braudt and Harris, 2020, for a discussion). The principal components are standardized to $\mu = 0$ and $\sigma = 1$ on the entire sample of genotyped Add Health respondents.

Family socioeconomic status. We use information from wave 1 to construct measures of *parents' education*. We transform parents' highest degree into years of education following the mapping suggested by Domingue et al. (2015). Numeric values in parentheses: never went to school (0), eighth grade or less (8), some high school (10), completed vocational/technical training instead of high school (10), went to school but level unknown (12), respondent does not know (12), high school graduate (12), GED (12), completed vocational/technical training after high school (14), some college (14), completed college (16), professional training beyond a master's degree (19). Where available, mothers' and fathers' education refers to the resident parent. If this information is unavailable, we use the biological parents' education instead.

Information about *mother's age at birth* (in years) is obtained from wave 1 if available and wave 2 otherwise. To calculate age at birth, we take information about the mother's age (as indicated by the child) and subtract the child's age at the respective wave.

Information about religion (*Christian* or not) is obtained from wave 1 (as indicated by the child).

We calculate *potential wages* for population group g in time period t according to the following formula (Shenhav, 2021):

$$\hat{w}_{gt} = \sum_{j} \frac{E_{jg,1970}}{E_{g,1970}} \times \sum_{o} \frac{E_{ojg,1970}}{E_{jg,1970}} \left(\pi_{ojt,-r}\right) \times w_{ojt,-r},$$

where $\frac{E_{jg,1970}}{E_{g,1970}}$ describes the group-specific employment share of industry j in 1970, $\frac{E_{ojg,1970}}{E_{jg,1970}}$ describes the group- and industry-specific employment share of occupation o in 1970, $\pi_{ojt,-r}$ describes the leave-region-out industry-specific employment growth in occupation o for the year t relative to 1970 (scaled by the overall employment growth in occupation o for the year t relative to 1970), and $w_{ojt,-s}$ describes the leave-region-out average hourly wage paid in year t for each occupation/industry/region cell. We define groups g by individuals that are homogeneous in gender (male, female), educational attainment ($<$ High School, High School, $>$ High School), and ethnicity (Non-Hispanic White, Hispanic, Non-Hispanic Black). We define regions r by census regions (North-East, Midwest, South, West). Employment shares in 1970 are taken from the 1970 decennial census. Employment shares and wages in periods t are taken from the March Supplements of the Current Population Survey (CPS) from 1975-2000. We match the time series of $\hat{w}_{gt}$ to the parents of respondents in Add Health based on information about g. Then, we calculate (i) the mean potential wages across respondents ages 0–14 and (ii) the standard deviation in potential wages across respondents ages 0–14.

Control function We construct the control function variables from the in-school questionnaires in wave 1. Specifically, for each school, we calculate the (i) *share of white students*, (ii) the *share of students in single-parent households*, (iii) *average years of education of students' mothers*, (iv) the *share of female students*, and (v) the *share of students with a migration background*. We transform mothers' highest degree into years of education following the mapping suggested by Domingue et al. (2015). Numeric values in parentheses: never went to school (0), eighth grade or less (8), some high school (10), went to school but level unknown (12), respondent does not know (12), high school graduate (12), GED (12), completed vocational/technical training after high school (14), some college (14), completed college (16), professional training beyond a four-year college (19). We calculate all control function variables to prevent mechanical correlation while excluding the respondent cohort (leave-cohort-out).

Other school characteristics. We use information from the school administrator questionnaire in wave 1 to construct proxies for other school policies. Specifically, we construct a binary indicator that assumes a value of 1 if the school uses *ability groups* based on English ability, a proxy for the strictness of *retention policy* by calculating the school average of retained students in 1993, and construct a *strictness index* for sanction policies. The strictness index is constructed as follows. School administrators were asked what happens to first-time offenders in the following domains: (i) cheating, (ii) fighting with another student, (iii) injuring another student,

(iv) possessing alcohol, (v) possessing an illegal drug, (vi) possessing a weapon, (vii) drinking alcohol at school, (viii) using an illegal drug at school, (ix) smoking at school, (x) verbally abusing a teacher, (xi) physically injuring a teacher, and (xii) stealing school property. Responses are "minor action", "in-school suspension", "out-of-school suspension", and "expulsion." We standardize answers to $\mu = 0$ and $\sigma = 1$ on the entire sample of Add Health respondents and then sum across dimensions (Anderson, 2008; Kling et al., 2007).

We also use information from the school administrator questionnaire in wave 1 to construct measures for teacher composition. Specifically, we calculate the schools' share of full-time classroom teachers that the school administrator identifies as (i) *White*, (ii) *Hispanic*, and (iii) *Female*.

Other PGI. We use the PGI for body mass index (*BMI*) (Yengo et al., 2018), attention deficit hyperactivity disorder (*ADHD*) (Demontis et al., 2019), *depressive symptoms* (Howard et al., 2019), *intelligence* (Savage et al., 2018), *smoking* (Liu et al., 2019), and *sleep duration* (Jansen et al., 2019). All polygenic indexes are standardized to $\mu = 0$ and $\sigma = 1$ on the full sample of genotyped Add Health respondents.

TABLE S.13 – Summary Statistics (Controls)

	Obs.	Mean	SD	Min	Max
Child Controls					
Female	4,034	0.54	0.50	0.00	1.00
Age in Months (Wave 1)	4,034	192.41	19.62	144.00	256.00
Firstborn	4,034	0.48	0.50	0.00	1.00
Family Controls					
Education Mother (in Years)	4,034	13.54	2.48	0.00	19.00
Education Father (in Years)	4,034	13.56	2.68	0.00	19.00
Maternal Age at Birth	4,034	25.33	4.83	16.00	46.08
Christian	4,034	0.82	0.38	0.00	1.00
Foreign-born Father	4,034	0.03	0.16	0.00	1.00
Foreign-born Mother	4,034	0.03	0.17	0.00	1.00
Potential Wage/Hour Mother (Mean)	4,034	12.57	1.39	9.40	14.27
Potential Wage/Hour Father (Mean)	4,034	15.40	1.32	11.14	17.11
Potential Wage/Hour Mother (SD)	4,034	0.35	0.11	0.12	0.51
Potential Wage/Hour Father (SD)	4,034	0.39	0.08	0.20	0.65
Control Function					
White Student (School Share)	4,034	0.81	0.18	0.05	1.00
Single Parents (School Share)	4,034	0.24	0.08	0.03	0.58
Educ. Mother (School Average)	4,034	13.40	0.68	12.03	16.25
Female (School Share)	4,034	1.48	0.05	1.00	1.72
Migration Background (School Share)	4,034	0.95	0.07	0.38	1.00
Other School Characteristics					
Strictness Index	4,034	0.19	0.46	-3.09	0.78
Ability Groups	4,034	0.66	0.47	0.00	1.00
Retention Policy (%)	3,969	3.56	3.72	0.00	20.75
White Teacher (%)	4,034	93.23	11.49	18.00	100.00
Female Teacher (%)	4,034	57.08	13.85	25.00	96.00
Private School	4,034	0.09	0.28	0.00	1.00
Polygenic Scores					
BMI	4,034	-0.02	1.00	-3.42	3.56
ADHD	4,034	-0.04	1.00	-3.82	3.48
Depressive Symptoms	4,034	-0.02	1.00	-3.79	3.55
Intelligence	4,034	0.02	0.99	-3.57	4.64
Ever Smoker	4,034	-0.03	1.00	-4.25	4.25
Sleep Duration	4,034	0.01	0.99	-3.82	2.97

Data: National Longitudinal Study of Adolescent to Adult Health.
Note: Own calculations. This table shows summary statistics for control variables in our core analysis sample. The sample is restricted to genotyped individuals who (i) are of European descent, and (ii) attended an Add Health high school or an associated feeder school in wave 1.

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