The Effect of Education on the Relationship between Genetics, Early-Life Disadvantages, and Later-Life SES

Silvia H. Barcellos (USC and NBER), Leandro S. Carvalho (USC) and Patrick Turley (USC)[1]

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We investigate whether education weakens the relationship between early-life disadvantages and later-life SES. Besides early, favorable family and neighborhood conditions, we argue that the genes children inherit also represent a source of advantages. Using a regression discontinuity design, we study a UK compulsory schooling reform that generated exogenous variation in schooling. The reform reduced educational disparities but did not weaken the relationship between early-life disadvantages and wages because advantaged children had higher returns to schooling. Exploiting family-based random genetic variation, we find the policy reduced educational differences driven by both environmental (e.g. credit constraints) and genetic advantages (e.g. innate ability).

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[1] Barcellos: University of Southern California, Center for Economic and Social Research, 635 Downey Way, Los Angeles, CA 90089-3332 (silvia.barcellos@usc.edu); Carvalho: University of Southern California, Center for Economic and Social Research, 635 Downey Way, Los Angeles, CA 90089-3332 (leandro.carvalho@usc.edu); Turley: University of Southern California, Center for Economic and Social Research, 635 Downey Way, Los Angeles, CA 90089-3332(pturley@usc.edu). Michael Bennett, Tami Gjorgjieva, Jiewen Luo and Andrew Yu provided excellent research assistance. This paper benefited from discussions with Dan Benjamin, Pietro Biroli, Dalton Conley, David Cesarini, Ben Domingue, Jason Fletcher, Rachel Griffith, Mireille Jacobson, Phil Koellinger, Adriana Lleras-Muney, Nick Papageorge, Tuomas Pekkarinen, Lauren Schmitz, and from the feedback of seminar participants at Boston University, Lehigh University, Stanford University, University of Rome Tor Vergata, UCLA, USC, University of Houston, and at the 2019 All-CA Labor Economics Conference at UCSC. We thank Hyeokmoon Kweon and Phil Koellinger for help with the ASHE data and Alex Young for providing the imputed polygenic indices of parents. Research reported in this publication was supported through the Russell Sage Foundation grant 98-16-16, NIA grants K99-AG062787-01, K01AG050811, 1K01AG066999-01A1, R21AG060447, RF1AG055654, 3RF1AG055654-01A1S1, and R56AG058726, Open Philanthropy grant 010623-00001, and by the USC Population Research Center. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. This research has been conducted using the UK Biobank Resource under Application Number 15666. The authors declare that they have no relevant or material financial interests that relate to the research described in this paper.

"Education then, beyond all other devices of human origin, is a great equalizer of the conditions of men – the balance wheel of the social machinery."

Horace Mann, pioneering American educator, 1848

"In America, education is still the great equalizer."
Arne Duncan, U.S. Secretary of Education, 2011

1. Introduction

Children born to richer parents are more likely to achieve economic success in adulthood (Jencks 1979; Solon 1999; Almond and Currie 2011). Education is considered by many to be the "great equalizer" and education policy a tool to "level the playing field," ensuring that kids from different backgrounds have similar opportunities for success. Others contend that, while education may reduce poverty, it might be less effective in promoting mobility because children born to richer parents or with higher ability have higher returns to schooling (Cameron and Heckman 2001) and consequently will choose to invest more in education.

This paper investigates whether education weakens the relationship between early-life advantages and later-life socioeconomic status (SES). Using UK Biobank data for over 212,000 individuals, we examine how these relationships were affected by a compulsory schooling change that generated exogenous variation in schooling.

We consider early-life environmental advantages at the *family* and at the *neighborhood* level: being born in a smaller family[2] and in a neighborhood with higher average education[3]. We also consider the genes that one inherits as *individual-level* advantages. Using genetic data, we construct a polygenic index (PGI) for educational attainment (EA), which has been shown to predict a host of SES outcomes (Lee et al. 2018; Belsky et al. 2018). Because of Mendelian inheritance, one's PGI is randomly assigned conditional on the PGI of the parents. We exploit this variation to study a gene-by-environment interaction: how a compulsory schooling change can modify how much of an advantage is conferred by one's luck in the "genetic lottery" (Kweon et al. 2020; Carvalho 2022).

[2] Following the large empirical literature on the trade-off between quantity and quality of children (e.g. Steelman et al. 2002, Chan et al. 2019).

[3] More specifically, the fraction of the adult population in the respondent's birth town around her birth date who stayed in school until at least age 15. Recent work shows that the quality of the neighborhood in which a child grows up affects her later-life SES (e.g. Chetty et al. 2016; Chetty and Hendren 2018a, 2018b).

Consistent with the literature on the importance of initial endowments (e.g., Lee et al. 2018; Black et al. 2005; Mogstad and Wiswall 2016; Chetty and Hendren 2018a, 2018b), our three proxies for early-life advantages are good predictors of education and economic success in adulthood. While each proxy is measured at a distinct level, it can capture in principal variation from the other levels (e.g., since genes are inherited from one's parents, the PGI captures both individual-level and family-level advantages). However, the predictive power of each proxy is remarkably similar if we estimate a joint model where the three proxies are entered together, indicating that each proxy captures an independent dimension of disadvantage.

While these associations do not represent causal relationships (e.g., Kong et al. 2018; Angrist et al. 2010), each proxy is fixed early in life, suggesting that they potentially capture some early-life, causal factors (Rietveld et al. 2013; Okbay et al. 2016; Harden et al. 2020). Indeed, we exploit the random genetic variation mentioned above to show that the EA PGI itself has causal effects on SES outcomes.[4]

The compulsory schooling change we study, the Raising of the School Leaving Age (ROSLA) Order of 1972, increased the minimum age at which students in England, Scotland, and Wales could drop out of school from 15 to 16 years (Oreopoulos 2006; Clark and Royer 2013; Barcellos, Carvalho and Turley 2018, forthcoming). The reform only affected students born after September 1, 1957, generating a discontinuity in the relationship between education and date of birth that can be exploited using a regression discontinuity design. We use data from the UK Biobank to study the effects on schooling, income, and occupational wages 34-38 years after the reform. We estimate that an additional year of schooling increased occupational wages by 5% on average—comparable to previous estimates using the 1972 ROSLA (Grenet 2013).

The ROSLA was successful in reducing educational disparities. For example, those in the most disadvantaged tercile (hereafter, the bottom tercile) of the distribution of the EA PGI stayed on average 0.3 years more in school while it virtually had no effect on those in the most advantaged tercile (hereafter, the top tercile) – there is a similar pattern for the other two proxies for advantages. We find similar effects on education qualifications that are valued in the labor market (namely the CSE and O-Level examinations taken at age 16). These results suggest that the ROSLA effectively narrowed the gap in skills between children from different backgrounds.

[4] This has been shown in previous studies (e.g., Belsky et al. 2018; Lee et al. 2018; Kweon et al. 2020).

However, these educational gains did not translate into reductions in economic disparities. The ROSLA did not narrow the wage gap between disadvantaged and advantaged children. We lay out a simple model that demonstrates that the reduced form effect of the ROSLA on the wage gap can be decomposed into two separate effects: the effect of the ROSLA on educational disparities (the "education effect") and heterogeneity in the returns to schooling (the "returns effect"). The model clarifies that our first-stage results and reduced-form results on wages are consistent as long as children from advantageous backgrounds have higher returns to schooling. As expected, we estimate that an improvement in one standard deviation unit in any of our proxies increases the returns to schooling by 1.8 to 2.8 percentage points.

The lack of exogenous variation in our proxies for early-life advantages complicates the interpretation of the reduction in educational disparities caused by the ROSLA. In the case of the EA PGI, for example, individual differences in the index confound genetic differences with differences in socioeconomic background. As a consequence, the reduction in educational disparities conflates two effects of the compulsory schooling reform: (i) its effect on the causal relationship between genetics and education and (ii) its effect on the association between socioeconomic background and education. As an example of (i), the policy may have reduced educational disparities related to genetically-influenced differences in ability. As an example of (ii), the policy could have also forced students to stay in school who would have otherwise dropped out because of credit constraints. Compulsory schooling laws like the ROSLA aim to reduce (ii) but not necessarily (i).

Family-based genetic data give us a powerful tool to address this question. Exploiting family-based random genetic variation, we separately estimate (i) and (ii), using measures of education quantity (school-leaving age) and qualifications (CSE or O-Level) as educational outcomes. Our results show that the ROSLA weakened the relationship between education and one's socioeconomic background, making the environmental conditions into which a child was born less important for her education. The policy also weakened the relationship between education and genetics, reducing the causal genetic effect. This illustrates how the effect of genetics on education is itself a function of the environment; even causal genetic effects are not fixed or unmodifiable.

Our paper makes a number of contributions. First, we bring together two distinct literatures: a literature in economics on childhood circumstances and adulthood SES (e.g. Almond et al. 2018) and a literature in social genomics on genotypes and later-life outcomes (Barth et al. 2020; Lee et

al. 2018; Belsky et al. 2018). We document that inheriting certain genes, being born into a smaller family, and in a higher-SES neighborhood capture independent dimensions of advantage. For economists, the EA PGI has a number of appealing characteristics as a proxy for advantage. Not only it is objectively measured, it is also determined at conception and invariant to environmental conditions. While the EA PGI may be correlated with one's environment, we illustrate how it is possible to decompose it into individual-level genetic variation and variation in environmental conditions using the EA PGIs of parents (Domingue and Fletcher 2020; Young et al. 2020). More importantly, this individual-level genetic variation is random, providing a source of exogenous variation in early-life advantage.

Second, we study how differences in the *quantity of* schooling and in the *returns to* schooling separately contribute to intergenerational mobility. Previous work on the effects of education on intergenerational mobility was unable to separate the two because the educational reforms studied changed not only the quantity of education but also key elements of the educational system (Meghir and Palme 2005; Pekkarinen et al. 2009; Aakvik et al. 2010; Andreoli et al. 2020; Bertrand, Mogstad and Mountjoy 2020). Our results underlie the importance of making this distinction: in our context advantaged children have substantially higher returns to schooling, which may present a challenge to increasing mobility (Solon 2004).

Finally, to our knowledge, this is the first paper to combine quasi-random variation in environment with random genetic variation to study how genetics interacts with a key environmental factor – education – to shape economic success. Our findings speak to two distinct literatures in economics: (a) on the importance of genetic inheritance for the intergenerational transmission of SES (Bowles and Gintis 2002; Black et al. 2020; Sacerdote 2011; Fagereng et al. 2021) and (b) on gene-by-environment interactions (Papageorge and Thom 2020; Biroli and Zünd 2020; Biroli et al. 2022)[5].

We advance the first literature (a) by showing that educational policy has the potential to affect the contribution of genetic inheritance for the intergenerational transmission of SES. Our work builds on studies in the second literature (b) which have furthered our understanding of how genes and environment interact by exploiting natural experiments (Schmitz and Conley 2016, 2017; Barcellos et al. 2018; Biroli and Zwyssig 2021) or by using within-family designs (Domingue and

[5] There is a large literature in social genetics on gene-by-environment interactions (e.g., Caspi et al. 2002; Caspi et al. 2003; Shanahan et al. 2008). Most studies in this literature are correlational.

Fletcher 2020; Morris et al. 2020; Brumpton et al. 2020; D'onofrio et al. 2013). We take a step forward by combining a natural experiment *with* a family design (Fletcher and Conley 2013).

The rest of the paper is structured as follows. In Section 2, we give more details about the data and introduce our proxies for early disadvantages. Section 3 discusses the interpretation of the genetic proxy (i.e., the EA PGI) and the method used to isolate the random genetic inheritance. Section 4 examines how the compulsory schooling law affected the relationship between early-life disadvantages and later-life SES. Robustness exercises are presented in Section 5. Section 6 concludes.

2. Data and Proxies for Early-Life Advantages

We use data from the UK Biobank (UKB), a large, population-based prospective study initiated by the UK National Health Service (NHS) (Sudlow et al. 2015). More than half a million individuals ages 40 to 69 were assessed between 2006 and 2010 in 22 assessment centers distributed throughout the UK (Allen et al. 2012) – see Appendix A.[6] The assessment included a self-completed touchscreen questionnaire, an in-person interview, physical measurements, and the collection of biological samples. The self-completed questionnaire collected data on, among other things, country of birth and year of immigration; qualifications and school-leaving age; household income; and family history. Information that was not collected via the self-completed touchscreen questionnaire, such as occupation and residence at birth, was collected in a subsequent computer-assisted personal interview (CAPI). Study participants were also genotyped using blood samples collected at the end of the assessment visit.

Our main outcomes of interest will be the participant's age at the time she left school; whether she passed qualification exams that are taken at age 16 – namely the Certificate of Secondary Education (CSE) and the General Certificate of Education (GCE) Ordinary Level (also known as the O-level); annual household income (reported in five brackets); and wages imputed based on one's occupation. During the interview, respondents answered a series of questions about their job (or last job if respondent had retired recently), which the interviewer used to classify the respondent's occupation among more than 400 detailed categories. We use the 2009 Annual

[6] Although the UK Biobank is not nationally representative, our estimates have internal validity because there is no differential selection on the two sides of the September 1, 1957 cutoff – see Appendix B.

Survey of Hours and Earnings (ASHE) to match such categories to median wages for each occupation (Kweon et al. 2020).[7] We view this measure of occupational wages as complementary to our measure of household income: on one hand, income was reported in brackets and occupational wages is a better measure of permanent individual income; on the other, household income is available for a larger fraction of the sample.[8] We find that both measures yield qualitatively similar results. Appendix C shows that the two measures are closely related.

A. Proxies for Early-Life Advantages

We use measures to proxy for early-life advantages at three levels: neighborhood, family, and individual. A recent literature shows that children who grow up in higher-SES neighborhoods have better socioeconomic outcomes, including earnings and college attendance (Chetty and Hendren 2018a, 2018b). As a proxy for neighborhood SES, we use a measure of average education in the individual's birthplace. UKB participants reported the town or district where they first lived when they were born. The coordinates of this locality were used to identify the local district (as of 1961) where the respondent resided at that time. The measure of neighborhood SES corresponds to the fraction of adults in the local district who stayed in school until at least age 15 according to the 1961 Census.[9,10,11] There are 1,436 different neighborhoods in our sample with an average population of about 35,000 and a median population of about 18,000.[12]

We use family size as a proxy for family-level advantages, following the large empirical literature on the trade-off between quantity and quality of children (e.g. Steelman et al. 2002; Chan et al. 2019). Our measure of family size corresponds to the numbers of full brothers and full sisters (i.e., excluding half-siblings, step-siblings, or adopted siblings) reported by the study participant, including those who had already died. Because individuals with fewer siblings tend to have higher

[7] We used gender-specific wages that were calculated among full-time employees and included all labor earnings (such as bonus, tips, overtime, etc.).
[8] Occupational wage is missing for 24.3% of UKB respondents while household income is missing for 11.1%. We show in the Robustness section that results are qualitatively similar if we use income instead of wages. See also Appendix D.
[9] We chose the threshold of age 15 because it maximized the variation in neighborhood characteristics. Importantly, this variable was chosen before assessing its association with our measures of SES and education. It captures a large fraction of the potential neighborhood variation at this level of aggregation.
[10] These data were generously made available by Vision of Britain (www.visionofbritain.org.uk).
[11] For Scotland, these figures were calculated at the county level using data from the 1951 Census.
[12] Chetty and Hendren (2018a) use commuting zones (CZ) as a measure of neighborhood. There are 741 different CZs in the United State with an average population of about 380,000 per CZ.

SES later in life, we reverse code family size (smaller families have a higher proxy value) so it is positively correlated with SES – as it is the case with the other proxies.

As a measure of individual-level advantages, we use a polygenic index[13] (PGI) for educational attainment (EA). A PGI is an individual-level predictor constructed from up to millions of genetic markers. Below, we describe how we constructed the EA PGI and discuss its interpretation.

Human DNA is made up of twenty-three pairs of long molecules, called *chromosomes*. While any pair of individuals are identical for 99.8% of their DNA, there are tens of millions of locations in the genome where individuals differ by a single genetic marker. These locations are called *Single Nucleotide Polymorphisms* (SNPs). At the vast majority of SNPs, people can have one of two possible genetic variants. The variant that a person has is called their *allele*. In genetic data, one of the two possible alleles is arbitrarily chosen as the *reference allele*. Because individuals have two copies of each chromosome, they will either have 0, 1, or 2 copies of the reference allele. The number of reference alleles that an individual has at a SNP is called their *genotype* for that SNP.

A PGI is constructed using estimates from a *Genome Wide Association Study* (GWAS). A GWAS scans the entire genome and estimates associations between individual genotypes and an outcome of interest. Specifically, a GWAS is a series of regressions of some outcome onto the genotype of each SNP, one at a time, and a set of covariates which normally include sex, age, and the first several principal components of the genetic data.[14]

A *polygenic index* (PGI) is a weighted sum of SNP genotypes:

$$PGI_i = \sum_j g_{ij} w_j \tag{1}$$

where PGI_i is the polygenic index for individual i; $g_{ij} \epsilon \{0,1,2\}$ is individual i's genotype at SNP j; and w_j is the weight for SNP j.[15] The weights in a PGI are derived from coefficients estimated

[13] Polygenic indexes are also often called "polygenic scores," "polygenic risk scores," or "genetic risk scores" in the literature. All of these terms correspond to the same thing.
[14] These principal components are included to account for ancestry-related omitted variable bias (see Section 3.A).
[15] GWASs for educational attainment have shown that education is associated to a large number of genetic markers, each with a small influence. PGIs are a way to aggregate these many influences and construct a genetic marker that is sufficiently predictive to use in empirical applications.

in a GWAS.[16] In this paper, we use GWAS coefficients based on Okbay et al. (2022), the largest currently available GWAS for educational attainment (EA).[17] The PGI based on this GWAS can explain 12-16% of the variation in EA.[18]

We note that each of our three measures of advantage may proxy for circumstances at other levels. They may also be imperfect measures of the circumstances they are thought to represent. For example, because the EA PGI is based on estimated associations between educational attainment and each SNP, and these associations are estimated with error, the EA PGI can be thought of as a noisy proxy for the EA PGI that could be constructed if the associations were estimated without error.[19] Finally, with the exception of family size, the proxies are fixed at birth, which means they could not have been affected by the compulsory schooling law that we will study. We show in Appendix B that the reform did not affect family size.

We keep in our sample individuals for whom at least one proxy is available.[20] About 88 percent of them have data on all three proxies. In the regression results below, we include an indicator (and interaction terms when relevant) for whether the individual is missing each proxy. Our results are very similar if we restrict to only those with complete data. The proxy that is more commonly missing is the EA PGI (7.2%). This is for two reasons. First, because genetic data is not available for part of the sample (2.4%). Second, we do not calculate PGIs for those with non-European ancestries (4.8%).[21] As a result, any analyses in this paper based on the PGI only correspond to the

[16] There are several methods for producing PGI weights from GWAS coefficients, but each of them transforms the GWAS coefficients in a way that is meant to account for the correlation structure that exists in the genome. We use a Bayesian method called LDpred (Vilhjálmsson et al. 2015).

[17] We use a version of the GWAS coefficients from Okbay et al. (2022) that omit the subset of individuals from the UK Biobank that we use in our analyses. Specifically, we conduct a GWAS in the UK Biobank that exactly matches the specification in Lee et al., except that we exclude individuals who were born within 10 years of September 1, 1957. We also exclude individuals who had one sibling or parent who also participated in the study. Finally, we excluded first cousins of anyone in these two previously mentioned groups. This is done to avoid overfitting.

[18] Because Okbay et al. (2022) is a GWAS of level of EA, our EA PGI is calibrated to predict levels of EA. Johnson et al. (2020) argue that for interaction studies such as ours, it may be more appropriate to use a GWAS of the variability of the outcome rather than the level. Nevertheless, we use PGI based on levels because Okbay et al. has a much larger sample than any available GWAS on the variability of EA. Using a smaller sample would greatly reduce the power of our study.

[19] Methods have been developed to control for this sort of error in a PGI (Tucker-Drob 2017; DiPrete et al. 2018; Becker et al. 2021). These methods either require additional information that is not known in our context (e.g., the heritability of educational attainment for compliers in our sample) or reduce statistical power (e.g., by requiring to split the GWAS sample in half). Because the PGI and the error of the PGI are independent of the instrument used in our design, we anticipate that correcting for the error in the PGI would not qualitatively affect our results.

[20] 6,775 eligible UKB participants lacked all three proxies.

[21] Samples in genetic research are nearly always restricted to individuals with similar genetic ancestries—defined as a group with tightly clustered first and second principal components of their genetic data. Due to Euro-centric bias in data collection, most currently published GWASs, including Okbay et al. (2022), are based on samples with

European-ancestry sample, though other analyses correspond to the full sample, regardless of ancestry. Family size and neighborhood SES are missing for 1.5% and 5.4% of our sample, respectively. Appendix E shows that the fractions missing each proxy are orthogonal to the schooling reform we study.

B. Proxies for Early-Life Advantages Predict Later-Life SES

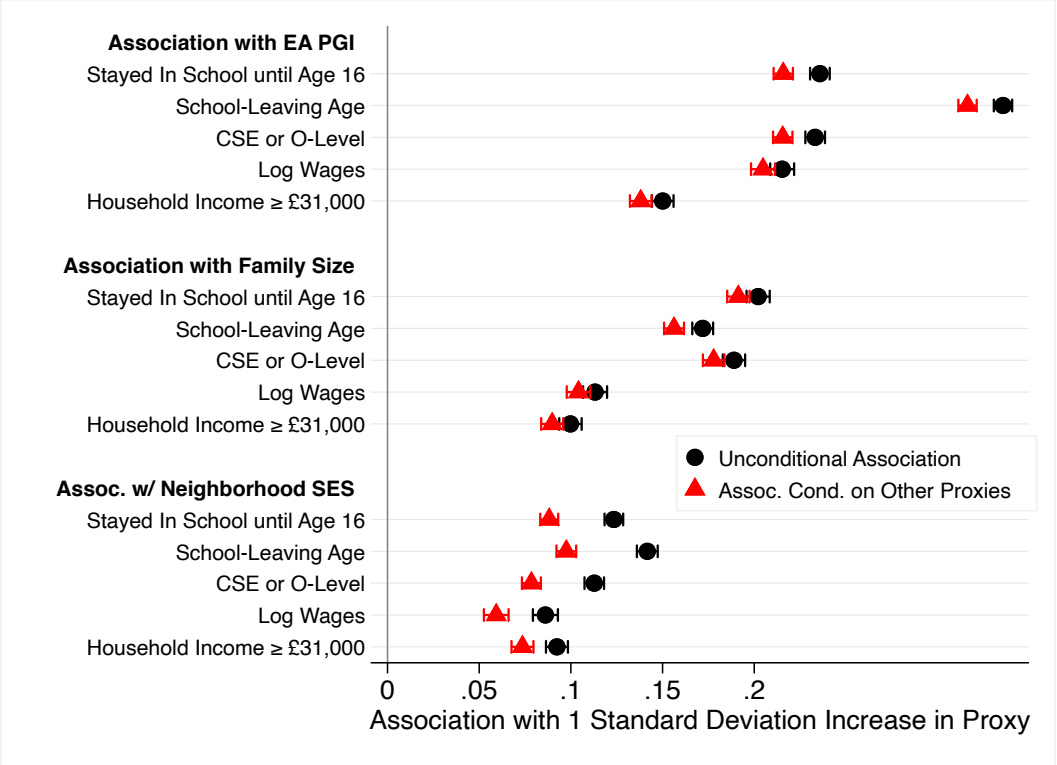
A large body of work shows that genetics, family size, and neighborhood characteristics are associated with SES in adulthood (e.g., Lee et al. 2018; Black et al. 2005; Mogstad and Wiswall 2016; Chetty and Hendren 2018a, 2018b). We begin by documenting these associations in our data. Figure 1 shows the associations of our proxies of disadvantage with different SES measures for cohorts unaffected by the reform. Both the SES measures and the proxies were standardized. The black dots show the unconditional associations. The red triangles show the association of a given proxy conditional on the other two proxies. The correlation between the EA PGI and family size is 0.05; the correlation between the EA PGI and neighborhood SES is 0.12; and the correlation between family size and neighborhood SES is 0.06.

There are two main takeaways. The three proxies – each capturing a different spectrum of disadvantages at the individual, family, and neighborhood levels – are strong predictors of the different measures of long-term SES. They are also independently associated with SES: the association of a given proxy with a SES measure changes little when we condition on the other two proxies. The low correlations between the proxies indicate that they represent distinct sources of advantages.[22] Notice how the EA PGI is as predictive as the other two proxies.

"European" ancestries. These are the primary ancestries found in individuals who self-identify as "White." As a result, PGIs based on currently available GWAS coefficients are substantially more predictive in European-ancestry samples (Martin et al. 2019) and may not generalize to groups with African, Asian, Hispanic, or other non-European ancestries. This is not a major issue in our context since over 95% of our UKB sample born between 1947 and 1967 is of European Ancestry.

[22] To illustrate, imagine to the contrary that the EA PGI and the neighborhood SES were noisy proxies for the same latent measure of advantage and that the low correlation between the two proxies was entirely due to uncorrelated noise. In Figure 3, we estimate that the standardized association between the PGI and school leaving age is 0.32. Using a classical measurement error correction formula, this would imply that the standardized association between the (noiseless) latent measure and school-leaving age would have to be 0.32/0.05 = 6.4. However, standardized associations cannot be greater than one unless they are strongly negatively correlated with other covariates, which is not the case in our setting.

Figure 1: (Pre-Reform) Differences in SES, by Background



Notes: The figure shows coefficients of regressions of the dependent variables listed in the rows on the proxies for disadvantage (the regressions also include pre-reform birth cohort trends). The black circles display coefficients from regressions including just one of the three proxies. The red triangles display coefficients from regressions including all three proxies. Both the dependent variables and the proxies were normalized. The brackets show 95% confidence intervals. The sample is restricted to those born between September 1, 1947 and August 31, 1957 (i.e. the cohorts unaffected by the ROSLA). Wages are imputed based on one's occupation. Sample sizes vary between 117,793 and 166,394 depending on the measure of SES.

Again, these associations do not represent causal relationships between the proxies and later-life SES (e.g., Kong et al. 2018; Angrist et al. 2010). A separate discussion about the interpretation of the association between the EA PGI and SES is warranted.

3. Interpreting the EA PGI and Its Relationship with Later-Life SES

The interpretation of a PGI is subtle and complicated. For example, it is many people's instinct to interpret the EA PGI as a measure of innate ability. Although a PGI can capture these types of individual attributes, the PGI will also be correlated with other individual, familial, and community characteristics. It is, therefore, incorrect to think of it solely as a measure of innate ability.

A. The Association between the EA PGI and SES

Consider a simple regression of an SES outcome on the PGI:

$$Y_i = \kappa_0 + \kappa_1 PGI_i + \chi_i. \quad (2)$$

There are at least two reasons why κ_1 will be a biased estimate of the causal effect of the PGI on SES: population stratification and indirect genetic effects. Population stratification is a form of omitted variable bias where specific genetic variants are more common in a particular group and the average of the outcome of interest Y is higher (or lower) among this group than in the rest of the population. These genetic variants will predict the outcome of interest, but the relationship is not causal.[23] Empirically, it has been shown that controlling for genetic principal components removes much but not all of the bias due to population stratification (Price et al. 2006). We show in the Robustness Section that our results are nearly identical when we control for the first 20 principal components of the participants' genetic matrix.

Another omitted variable in regression (2) is parental genetics: parental genetics drive both a child's own genetics (since the child inherits her genetic variants from her parents) and the child's SES (through parental behaviors and characteristics, such as parental education). Since the parents' and child's PGIs are correlated, when the analysis does not control for parental genotypes, as in Figure 1, the effects of the parental genotypes are partly captured by the offspring's PGI.

B. Controlling for the PGIs of Parents

By using information on the PGIs of an individual's parents, we can however isolate variation in the individual's PGI that is random and therefore orthogonal to her environment. This is possible due to Mendelian inheritance: a biological phenomenon that causes PGIs to be randomly assigned to children conditional on the sum of the PGIs of their parents (Kong et al. 2020). We estimate the following model:

[23] The canonical example of population stratification is chopstick use (Price et al. 2006). Consider a GWAS of whether an individual regularly uses chopsticks and a sample that includes individuals of Asian and non-Asian backgrounds. This GWAS would find many SNPs that are associated with chopstick use, but each of these associations would correspond to SNPs that have alleles that are more common in Asian populations rather than to SNPs that represent any sort of genetic pathway between genes and chopstick use.

$$Y_i = \phi_0 + \phi_1 PGI_i + \phi_2 F_i + e_i. \quad (3)$$

where F_i is the sum of the PGIs of individual i's parents.

The coefficient on the PGI, ϕ_1, estimates "direct genetic effects."[24] Direct genetic effects are defined as the causal effect of inheriting specific genetic variants holding constant one's environment.[25] If we had exogenous variation in the parental PGI, ϕ_2 would pick up "indirect genetic effects" from one's parents. These are the effects of genetic variants of the parents that were not inherited by the child but still affect her because they influence her parents' characteristics and behaviors (Kong et al. 2018).[26] Since we do not have exogenous variation in the parental PGI, it may also represent other environmental advantages, such as indirect genetic effects from other relatives and population stratification.

It is important to stress that the direct genetic effects do not necessarily reflect "ability" or "innate" individual characteristics that may influence productivity. Although the approach identifies the causal effects of genetic variation on SES, we cannot pin down the channel(s) of these effects. For example, suppose that height has a causal effect on wages because *ceteris paribus* employers favor taller people (Persico et al. 2004; Case and Paxson 2008). Then any genetic variant that causes an increase in height will also cause an increase in wages. EA and height have been shown to be genetically correlated (Okbay et al. 2016), such that individuals with higher EA PGIs will tend to be taller and to earn higher wages, but, in this example, this relationship does not necessarily imply that individuals with higher EA PGIs are more productive. However, if some

[24] It is also possible to estimate direct genetic effects using a family fixed-effect model, however the fixed-effect model has several limitations in our context. First, using a fixed-effect model would require us to drop siblings with any missing data or who do not satisfy our sample restrictions. In contrast, our specification only requires genetic data for parents, which can be imputed using only genetic data from at least one other sibling or in some cases was observed because one parent was genotyped. This increases our sample size. Second, if the genotypes of one sibling influences the other sibling, this effect would bias the "direct effect" estimates in a fixed-effect model but not one that controls for the parental PGI. Third, our specification allows us to directly measure the association between the parental PGI and SES and to measure the change in this association as a result of the 1972 ROSLA, which would be impossible in a fixed-effect model. We present fixed-effect results in Appendix F and find qualitatively similar results.

[25] Direct genetic effects are still a function of the environment, however; environmental factors can change the impact of specific genetic variants. Indeed, an important result of this paper is that education can change the direct effect of the EA PGI (see results in section 4.E).

[26] Recent evidence using siblings of parents to estimate the causal effect of the parental PGI on the education of each parent's offspring has found little evidence that the association between the parental PGI and the child's education is due to indirect genetic effects from the parents. Rather, the association between parental PGI and offspring's education seems to be primarily driven by population stratification and assortative mating. (Nivard et al. 2022).

portion of the EA PGI represents "innate" characteristics, this portion would be captured by the direct genetic effect and not the indirect/environmental effect.

C. Imputing the PGIs of Parents

About 5,000 study participants had parents who also participated in the study. For the others, it is possible to impute the sum of the parental genotypes if at least one of the study participant's siblings were also genotyped (Young et al. 2020) - which is true for about 36,000 respondents in our sample . An illustration of this imputation procedure is found in Figure 2. Imagine we would like to impute the sum of the genotypes of a participant's parents at some SNP. Because people normally have two copies of each chromosome, we represent the genotype of Parent 1 and Parent 2 at this SNP as $(A + B)$ and $(C + D)$, respectively, where A, B, C, and D are indicators of whether a parent has a copy of a specified reference allele at the SNP on a certain copy of their chromosome. The sum of the parental genotypes at this locus is therefore $(A + B + C + D)$.

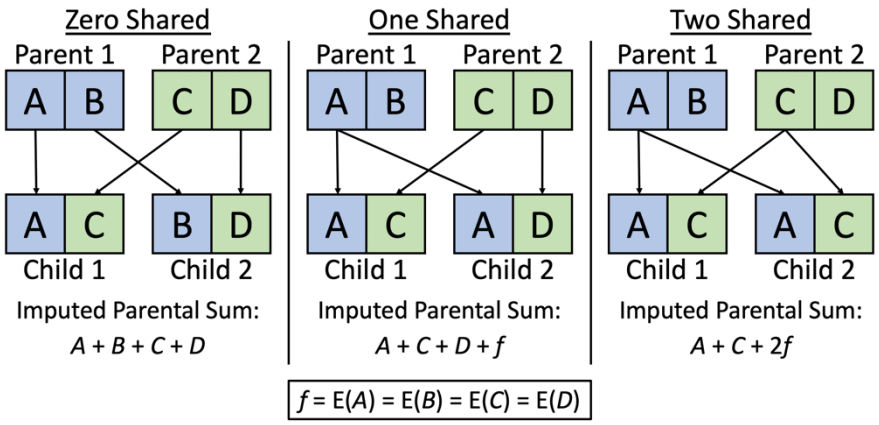
Say we observe two (full) siblings in our data. Each child randomly inherits one of the alleles from Parent 1 (either A or B) and one allele from Parent 2 (either C or D). As Figure 2 illustrates, there are three possible cases. In the first case ("Zero Shared"), the siblings inherited different alleles from each parent, such that all four parental alleles are observed in the two siblings. In the second ("One Shared"), the siblings inherited the same allele from one parent and a different allele from the other, in which case only three parental alleles are observed. Finally, in the third case ("Two Shared"), only two parental alleles are observed because the siblings inherited the same allele from each parent.

It is possible to determine which case we are in at a particular SNP. In the second and third cases, we take the sum of the observed genotypes and impute the unobserved one using the mean genotype for a single allele at that SNP. This mean genotype is sometimes called the *allele frequency* in the population since it corresponds to the frequency at which the allele is found at that SNP on a single copy of a chromosome drawn from the population.[27,28]

[27] We follow a similar procedure when one parent is genotyped but the other parent is unobserved. In these cases, we always observe three of the four parental alleles, so the imputed parental sum is the sum of the three observed genotypes plus a constant equal to the frequency of the missing genotype.

[28] This imputation of the unobserved allele is justified under an assumption that the observed alleles are uncorrelated with the unobserved alleles. This assumption would hold if there were random mating in the population, which has been shown to not be the case. However, it has been shown that the expected correlation between parental genotypes

Figure 2: Illustration Imputation of Parental PGIs



Note: The figure illustrates the method used to impute the PGI of parents. In the "Zero Shared" case, the siblings inherited different alleles from each parent, such that all four parental alleles are observed in the two siblings. In the second ("One Shared") case, the siblings inherited the same allele from one parent and a different allele from the other, in which case only three parental alleles are observed. Finally, in the "Two Shared" case, only two parental alleles are observed because the siblings inherited the same allele from each parent. In the last two cases, we impute the unobserved genotypes using the mean genotype for a single allele at that SNP. This mean genotype corresponds to the frequency at which the allele is found at that SNP on a single copy of a chromosome drawn from the population.

The imputation will lead to some error in the parental genotype measure, but it is not classical measurement error. Specifically, under a classical measurement error model, it is assumed that the variable is equal to the true value plus some amount of uninformative noise. In the case of our imputed parental genotypes, our variable is equal to the true value minus some information—the unobserved parental genotypes. Importantly, if the parental PGI is omitted, the portion of the parental PGI that biases the coefficients of (2) are the shared genetic variants between the parents and children. This is the portion of the PGI that we are able to impute without error. This means that, by controlling for the imputed parental PGIs, we can estimate the direct effect, ϕ_1, without bias. More details and proofs of these properties can be found in Young et al. (2020).

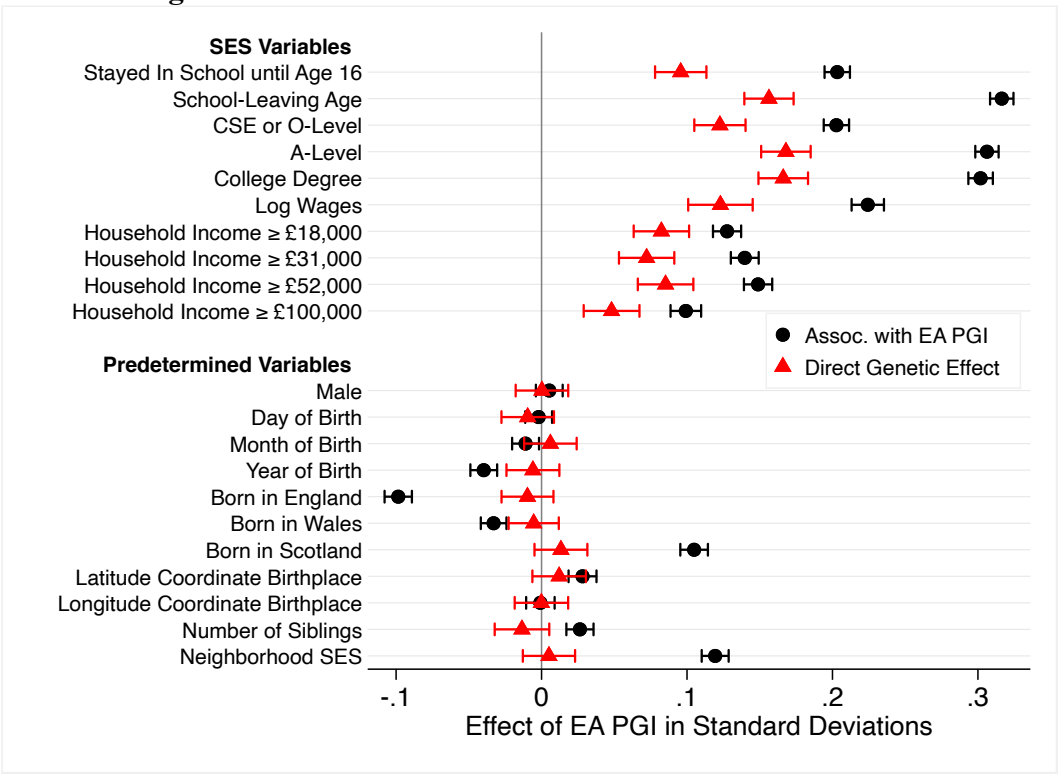
D. Direct Genetic Effects

The red triangles in Figure 3 show estimates of direct genetic effects. The black circles display associations between the EA PGI and the outcomes of interest. The direct genetic effects are

would be on the order of 10^{-6} if parents were sorting at rates implied by the most extreme estimates. We therefore believe that the zero-correlation assumption is a reasonable approximation in this case.

obtained by estimating equation (3) and plotting $\hat{\phi}_1$. The associations correspond to estimating equation (2), which does not control for the parental PGI. For comparison, all outcome variables were standardized. The PGI was also standardized. To maximize statistical power, this particular analysis uses all 43,393 UKB participants of European genetic ancestries with a parent or sibling who had also participated in the UKB.[29]

Figure 3: Direct Genetic Effects vs. EA PGI Associations



Notes: The red triangles shows estimates of the causal effect of the EA PGI. The black circles show associations with the EA PGI. The brackets show 95% confidence intervals. The EA PGI, the SES variables, and the predetermined variables were all normalized. Wages are imputed based on one's occupation. The sample includes all UKB participants of European genetic ancestries for whom the parental EA PGI was available. The number of observations ranges from 27,295 to 43,393 depending on the outcome.

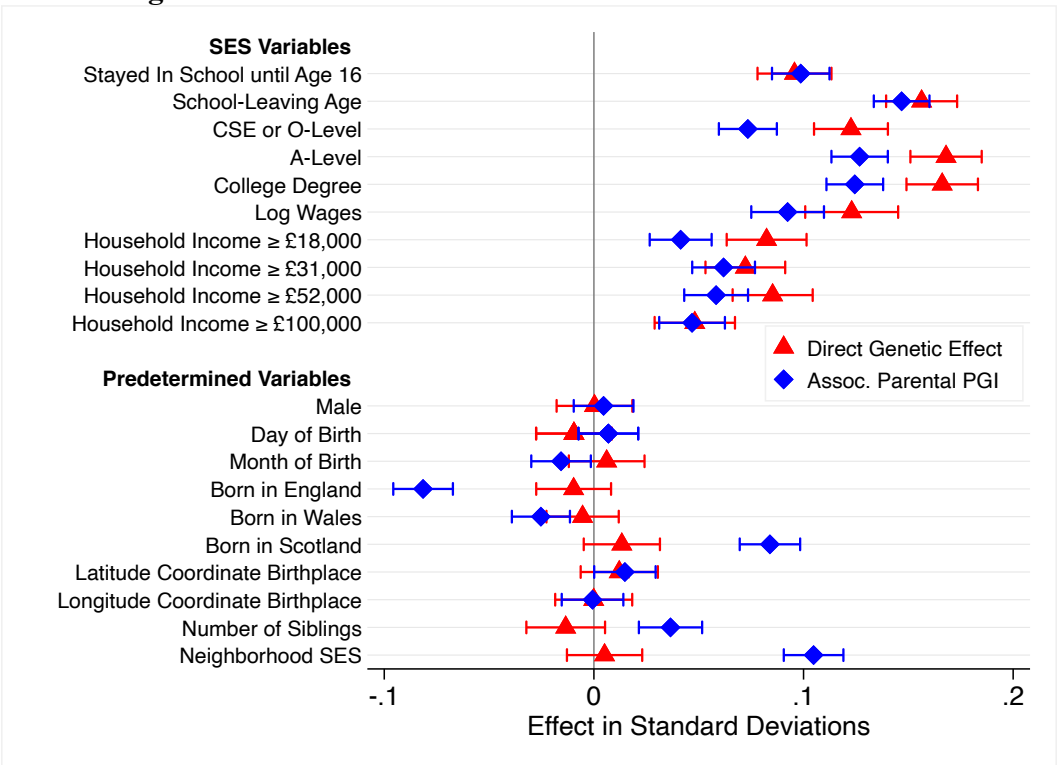
The top panel of the figure shows that, not only the EA PGI predicts several SES outcomes, but that it also has a causal effect on all of them. An increase of one SD in the EA PGI *causes* an increase of 0.16 SD in school-leaving age and an increase of 0.12 SD in log wages. In comparison, an increase of one SD in the EA PGI is *associated* with a 0.32 SD increase in school-leaving age

[29] Figures 1 and 3 were constructed using different samples (e.g., Figure 1's sample includes only those born in England, Scotland, or Wales between September 1, 1947 and August 31, 1957 while Figure 3 includes only study UK Biobank participants for whom parents' genotypes were available or could be imputed). That is why the associations between the EA PGI and the SES outcomes (black circles on both figures) are slightly different in the two figures.

and with a 0.22 SD increase in log wages. The bottom panel of the figure reports results for variables that would have been fixed before or very soon after the genotype, and consequently the PGI, was fixed. Therefore, one would expect no causal relationship between the EA PGI and these variables. Even though the EA PGI is (unconditionally) associated with several of these variables, we cannot reject that the direct genetic effect on any of them is different from zero. This can be seen as a "balance test" that the parental PGI imputation described in Section 3.C jointly with Mendelian inheritance worked as intended and that the conditional EA PGI is indeed random.

The associations with the SES outcomes are larger than the direct genetic effects partly because of indirect genetic effects. The blue diamonds in Figure 4 display the coefficients on the parental PGI when estimating equation (3), i.e., $\hat{\phi}_2$. Having parents with higher EA PGIs is associated with achieving higher success in adulthood (top panel). It is also associated with having fewer siblings and with living in a higher-SES neighborhood (bottom panel). Because we do not have exogenous variation in the parental PGI, these associations may also represent other environmental advantages, such as indirect genetic effects from other relatives and population stratification.

Figure 4: Direct Genetic Effects vs. Environmental Factors



Notes: The red triangles shows estimates of the causal effect of the EA PGI. The blue diamonds show associations with the parental EA PGI. The brackets show 95% confidence intervals. The EA PGI, the parental EA PGI, the SES variables, and the predetermined variables were all normalized. Wages are imputed based on one's occupation. The sample includes all UKB participants of European ancestries for whom the parental EA PGI was available. The number of observations ranges from 27,295 to 43,393 depending on the outcome.

The direct genetic effects from Figure 3 are reproduced in Figure 4 for purposes of comparison. The results *suggest* the association with the parental PGI may be as large as the direct genetic effects, which is broadly consistent with previous findings (Kong et al. 2018; Howe et al. 2021; Lee et al. 2018; Cheesman et al. 2020). In section 4.E, we apply the empirical design explained here to investigate how the compulsory schooling reform affected the causal relationship between genetics and education and how it affected the association between socioeconomic background and education.

4. Education and the Relationship between Early-Life Advantages and Later-Life SES

A. The 1972 Raising of the School-Leaving Age

If, as believed, education levels the playing field, then we would expect it to reduce the gaps between children from disadvantaged and advantaged backgrounds. To investigate this hypothesis, we study a well-known compulsory schooling reform and its effects on these disparities. The 1972 Raising of School Leaving Age (ROSLA) legislation increased the minimum school-leaving age in England, Scotland, and Wales from 15 to 16 years of age.[30] These laws and their implementation have been extensively documented before (see Clark and Royer 2010, 2013; Grenet 2013; Davies et al. 2018) so we only include a brief summary of its main features here. The change took effect in September 1, 1972, implying that those who were 15 or younger before that date (i.e., born on September 1, 1957 or later) had to stay in school until at least age 16 (hereafter, we use the term "stayed in school until age 16" to refer to those who stayed in school until *at least* age 16). Infrastructure investments, such as school building to absorb the additional students, preceded the 1972 ROSLA but key elements of the school system did not change with the reform.

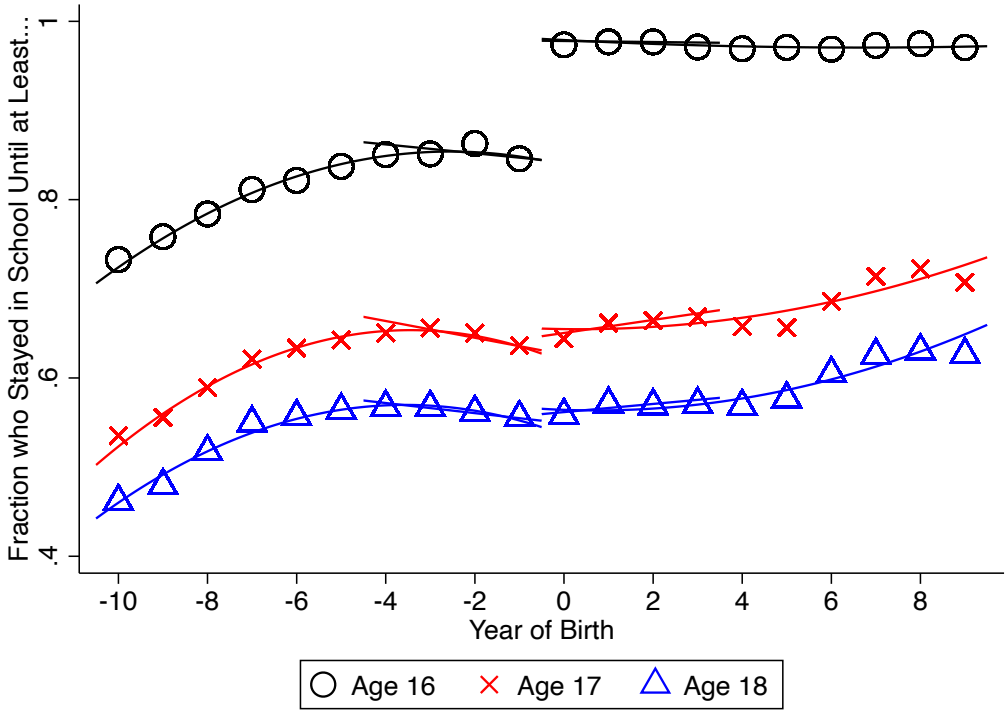
Politically, there were a number of different justifications for the 1972 ROSLA including addressing the British economy's failing competitiveness, raising England's level of education to be on par with other countries, and reducing the number of young people seeking employment at high unemployment times. Interestingly, a 1959 report by the ministry of education advising the Government to increase compulsory schooling to age 16 highlighted the need to tap "all the

[30] http://www.legislation.gov.uk/uksi/1972/444/pdfs/uksi_19720444_en.pdf
http://www.legislation.gov.uk/uksi/1972/59/pdfs/uksi_19720059_en.pdf

available supply of talent" and the social barriers to doing so. The Crowther Report documented that "half of the National Service recruits to the Army who were rated in the two highest ability groups had left school at age 15" and that among recruits "coming from families of manual workers two-thirds of those in the two highest ability groups had left school at 15."[31]

In our analysis, we restrict the sample to participants born between September 1, 1947 and August 31, 1967 who were either born in England, Scotland, or Wales or immigrated to the UK before age 15. Figure 5 illustrates some of the impacts of the ROSLA. It shows the fraction of study participants who stayed in school until age 16 (black circles); age 17 (red Xs); and age 18 (blue triangles) by year of birth. Year of birth runs from September 1 of a given a year to August 31 of the following year. For example, those born in year 0 correspond to those born between September 1, 1957 and August 31, 1958, the first cohort affected by the ROSLA. The figure shows linear trends for a 4-year bandwidth around September 1, 1957 and quadratic dates for a 10-year bandwidth.

Figure 5: Educational Attainment by Year of Birth



Notes: The figure shows the fraction of study participants who stayed in school until age 16 (black circles), age 17 (red crosses), and age 18 (blue triangles) by year of birth. Year of birth runs from September 1 of a given a year to August 31 of the following year. Those born in Year 0 were

[31] See http://www.educationengland.org.uk/documents/crowther/crowther1959-1.html pages 131-132

born between September 1, 1957 and August 31, 1958. Cohorts born after Year 0 had to stay in school until age 16 while cohorts born before could leave at age 15. The figure shows linear trends for a 4-year bandwidth and quadratic trends for a 10-year bandwidth. We use triangular kernel weights that give greater weight to study participants born closer to September 1, 1957. *N* = 86,417 (4-year bandwidth) and 212,290 (10-year bandwidth).

The figure shows that the ROSLA generated a discontinuity in the relationship between staying in school until age 16 and date of birth. There is a sharp increase for those born after September 1, 1957. The discontinuities at ages 17 and age 18 are much smaller but still significant.

We estimate the ROLSA increased the fraction of those staying in school until age 16 by 14 percentage points—see Appendix Table G1. The fraction staying until ages 17 and 18 increased by 3 and 2 percentage points. The ROSLA did not affect the fraction staying in school past 18 or the fraction graduating from college (see Appendix H). On average, the cohort affected by the ROSLA stayed in school approximately 0.18 years more than those who could drop out at age 15.

The increase in schooling generated by the ROSLA resulted in an increase in income, as shown in Appendix Figure G2. We estimate that an additional year of secondary education increased middle-age wages by 5% – see Appendix Table G2. These are consistent with Grenet (2013) who estimated a return of 6-7%.[32,33]

Appendix B shows that predetermined characteristics and our proxies for early-life advantages are smooth around the September 1, 1957 cutoff. These results suggest that the identifying assumption of the regression discontinuity design is satisfied and that we can stratify the results by our proxies for advantage.

B. Did the 1972 Raising of the School-Leaving Age Reduce Educational Gaps?

One of the rationales for the 1972 ROSLA was to reduce educational gaps between children from different socioeconomic backgrounds and to allow high-ability, disadvantaged students to fulfill their potential (Crowther Report 1959). To investigate whether the ROSLA was effective in this regard, Figure 6 shows the average school-leaving age by year of birth for those in the top and bottom terciles of the distributions of the EA PGI (top panel), family size (middle panel), and neighborhood SES (bottom panel). The average education for the top tercile is shown on the right y-axis while the average education for the bottom tercile is shown on the left y-axis. Appendix I shows comparable figures for the fraction of students who stayed in school until ages 16, 17, and

[32] Grenet (2013) studied the 1972 ROSLA using data from the Quarterly Labour Force Survey.

[33] The effects are robust to the alternative bandwidths. Interestingly, these 2SLS estimates are also consistent with the OLS estimates (i.e., regressing log-wages directly onto the endogenous SLA variable), suggesting a limited role for omitted variable bias in this context.

18 – Appendix Figure I1 is of particular interest because age 16 was the margin targeted by the reform.

Figure 6 indicates that the ROSLA was effective in reducing the educational disparities between children from less and more advantaged backgrounds. The average school-leaving age of the bottom tercile (i.e., the children from more disadvantaged backgrounds) jumps discontinuously after September 1, 1957, reducing the difference between the red and black lines. This is true for all three different proxies of disadvantages: individual, family, or neighborhood. The gap in average school-leaving age decreased from 2.3 to 2.03 of a year of schooling between those in the top and the bottom terciles of the EA PGI distribution; from 1 to 0.69 of a year of schooling between those in the top and the bottom terciles of the family size distribution; and from 1 to 0.91 of a year of schooling between those in the top and the bottom terciles of the neighborhood SES distribution. These are large reductions considering that the ROSLA increased school-leaving age on average by approximately 0.18 years—see Appendix Table G1.

The trends shown in Figure 6 are obtained by estimating the following regressions:

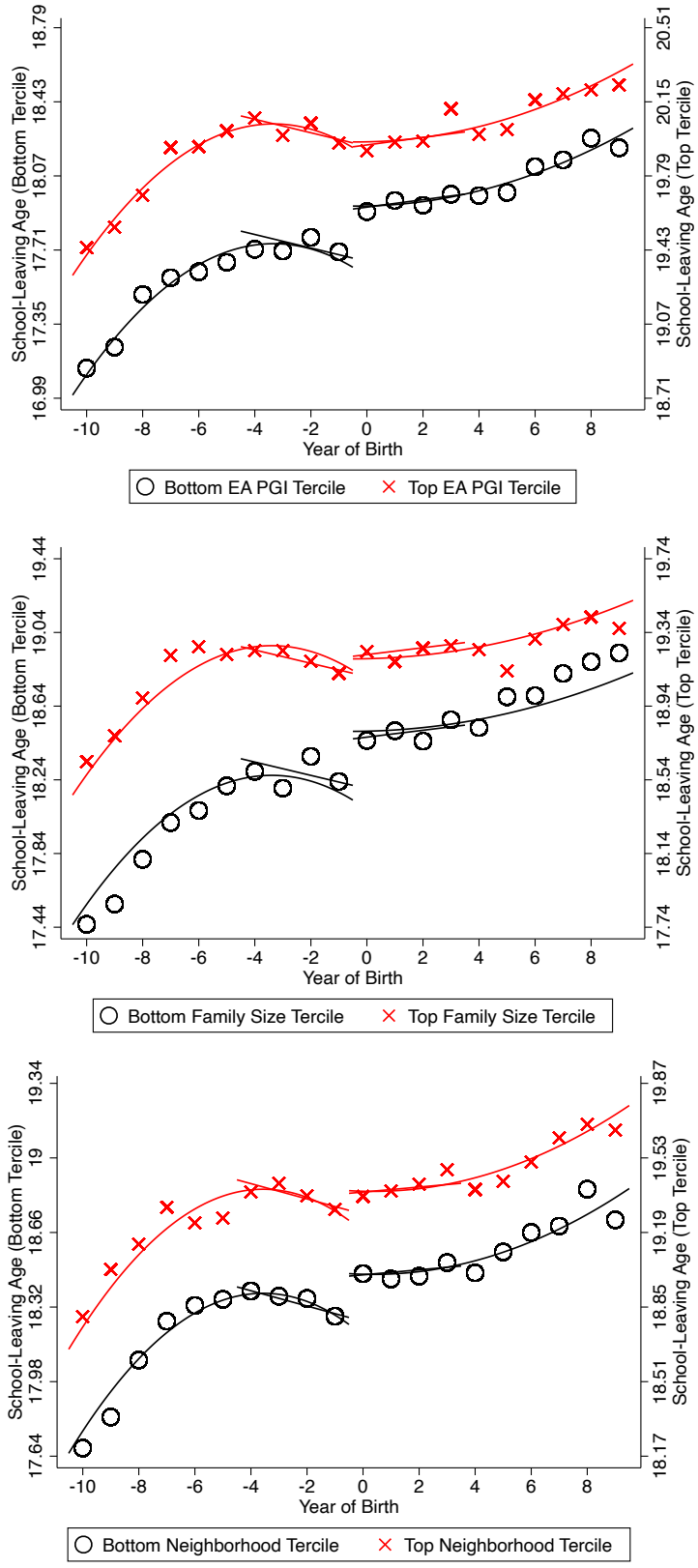
$$SLA_i = \delta_0 + \delta_1 Post_i + \boldsymbol{B}_i'\boldsymbol{\delta}_2 + Post_i \times \boldsymbol{B}_i'\boldsymbol{\delta}_3 + k(DoB_i) + \varepsilon_i. \quad (4)$$

where SLA_i is individual i's school-leaving age; $Post_i$ is 1 if individual i was born on or after September 1, 1957 (and 0 otherwise); $\boldsymbol{B}$ is a vector containing proxies for early-life advantages; and DoB_i is individual i's date of birth. Date of birth is measured in days relative to the cutoff, such that $DoB = 0$ for someone born on September 1, 1957. The function $k(\cdot)$ captures birth cohort trends in educational attainment, which are allowed to differ on either side of the September 1, 1957 cutoff. We run two alternative specifications: (1) a 10-year bandwidth with quadratic trends in date of birth and (2) a 4-year bandwidth with linear trends.[34] We use triangular kernel weights that give greater weight to study participants born closer to the cutoff.[35]

[34] To the best of our knowledge, methods for optimal bandwidth selection for interaction effects have yet to be developed. We worry that the standard CCT optimal bandwidth is too conservative in the case of interactions which is why we consider both a 4-year (the CCT optimal bandwidth) and a 10-year bandwidth. In the Robustness Section we show that our results are robust to the choice of bandwidth.

[35] Even though previous work studying the 1972 ROSLA clustered standard errors by month-year of birth (e.g., Clark and Royer 2013; Davies et al. 2018), we do not need to cluster our standard errors because our data include exact date of birth (Lee and Card 2008).

Figure 6: Effect of the 1972 ROSLA on School-Leaving Age, by EA PGI, Family Size, and Neighborhood SES



Notes: The figures show average school-leaving age by year of birth, separately for those in the bottom tercile (black circles) and in the top tercile (red Xs) of the following distributions: EA PGI (top panel); family size (middle panel); and neighborhood SES (bottom panel). Average education for the top tercile is shown on the right y-axis while average education for the bottom tercile is shown on the left y-axis. Year of birth runs from September 1 of a given a year to August 31 of the following year. Those born in Year 0 were born between September 1, 1957 and August 31, 1958. Cohorts born after Year 0 had to stay in school until age 16 while cohorts born before could leave at age 15. The figure shows linear cohort trends for a 4-year bandwidth and quadratic cohort trends for a 10-year bandwidth. For a given proxy, all three terciles share the same cohort trends. We use triangular kernel weights that give greater weight to study participants born closer to September 1, 1957. N = 79,873 (EA PGI with 4-year bandwidth); 196,727 (EA PGI with 10-year bandwidth); 85,308 (family size with 4-year bandwidth); 209,338 (family size with 10-year bandwidth); 81,454 (neighborhood SES with 4-year bandwidth); and 201,340 (neighborhood SES with 10-year bandwidth).

Table 1: Effect of the 1972 ROSLA on Education, by EA PGI, Family Size, and Neighborhood SES

School-Leaving Age

	4−Year Bandwidth				*10−Year Bandwidth*			
PGI * Post	-0.101			-0.097	-0.115			-0.106
	[0.021]			[0.021]	[0.013]			[0.013]
Family * Post		-0.104		-0.098		-0.144		-0.124
		[0.025]		[0.024]		[0.016]		[0.016]
Neighborhood * Post			-0.032	-0.027			-0.026	-0.020
			[0.023]	[0.022]			[0.015]	[0.014]
Post	0.131	0.121	0.126	0.123	0.179	0.176	0.180	0.166
	[0.042]	[0.043]	[0.044]	[0.042]	[0.039]	[0.040]	[0.040]	[0.038]
Observations	86,417	86,417	86,417	86,417	212,290	212,290	212,290	212,290

1 if Has a CSE or an O-Level

	4−Year Bandwidth				*10−Year Bandwidth*			
PGI * Post	-0.042			-0.039	-0.051			-0.047
	[0.003]			[0.003]	[0.002]			[0.002]
Family * Post		-0.025		-0.024		-0.031		-0.029
		[0.004]		[0.004]		[0.002]		[0.002]
Neighborhood * Post			-0.023	-0.018			-0.026	-0.020
			[0.003]	[0.003]			[0.002]	[0.002]
Post	0.060	0.058	0.060	0.061	0.066	0.063	0.066	0.067
	[0.006]	[0.006]	[0.006]	[0.006]	[0.005]	[0.005]	[0.005]	[0.005]
Observations	85,779	85,779	85,779	85,779	210,633	210,633	210,633	210,633

Notes: In the top panel, the dependent variable is school-leaving age. In the bottom panel, it is an indicator for having a CSE or an O-Level. PGI is the polygenic index for educational attainment. Post is an indicator for being born on or after September 1, 1957. PGI, Family, and Neighborhood are all standardized to have mean zero and standard deviation of one. The first four columns include linear trends in exact date of birth while the last four include quadratic trends. In both cases, trends are allowed to be different before and after September 1, 1957. Robust standard errors between brackets. The coefficients on PGI, Family, and Neighborhood are omitted. The average school-leaving age among those born before September 1, 1957 is 18.9 (4-year bandwidth) and 18.7 (10-year bandwidth). The fraction with a CSE or O-Level among those born before September 1, 1957 is 79% (4-year bandwidth) and 75% (10-year bandwidth).

We use a discrete specification in Figure 6, where $\boldsymbol{B}_i$ is a vector containing indicators for being in the top and in the middle terciles of the distributions of EA PGI (top panel), family size (middle panel), and neighborhood SES (bottom panel). Notice that the birth cohort trends $k(DoB_i)$ are assumed to be the same for individuals in the three different terciles of a given proxy distribution.[36] The Robustness Section discusses that – although relaxing this assumption implies a large loss in precision – the point estimates results are overall very similar. Middle-tercile participants were also used to estimate Figure 6's birth cohort trends (not shown for ease of exposition); this is true not only for Figure 6 but for all figures comparing the bottom and top terciles.

Table 1 reports results from a continuous specification of equation (4), where $\boldsymbol{B}_i$ is a vector containing continuous (standardized) measures of the EA PGI, family size, and/or neighborhood SES (Appendix Table I4 shows results from the discrete specification). Because of space constraints, the estimated coefficients δ_0 and $\boldsymbol{\delta}_2$ are not reported in the table.

The results in the table's top panel confirm that the 1972 ROSLA was successful at leveling the playing field, at least in terms of how long students stayed in school. The interactions of the indicator for being born after September 1, 1957 with the EA PGI, family size, and with neighborhood SES are all negative with most being statistically significant at the 1% (the exception being the interaction with neighborhood SES). Columns (4) and (8) show that the coefficients on the interaction terms are very similar when all the proxy-interactions are included simultaneously.

These reductions in educational differences may not be very meaningful if the students forced to stay in school an extra year put in little effort and gained little with the additional schooling. However, one potential concrete benefit of this additional year of education was the opportunity to sit for a set of qualification exams that are taken at the end of grade ten (when participants are typically 16): the Certificate of Secondary Education (CSE) or the General Certificate of Education (GCE) Ordinary Level (also known as an O-Level).

The bottom panel of Table 1 shows that the 1972 ROSLA also reduced the disparities in terms of educational qualifications. The coefficients on the interaction terms are negative and significant for all proxies, including neighborhood SES. By compelling students to stay in school, the 1972

[36] Notably, this specification excludes any additional control variables, such as principal components of the genetic data. Principal components are normally included as covariates in studies that include PGIs to absorb variation in the PGI and outcome variables related to ancestry differences. Since we do not include principal components in our models, the PGI may also capture at-birth advantages related to ancestry. In the Robustness Section, we show that including principal components has only a negligible effect on our results.

ROSLA led students to get these qualifications, which are valued in the labor market (Dickson and Smith 2011). This is consistent with the estimated reduction in school-leaving age disparities and provides evidence that the students compelled to stay in school for an additional year acquired concrete benefits as a result.

C. Did the 1972 Raising of the School-Leaving Age Reduce Wage Gaps?

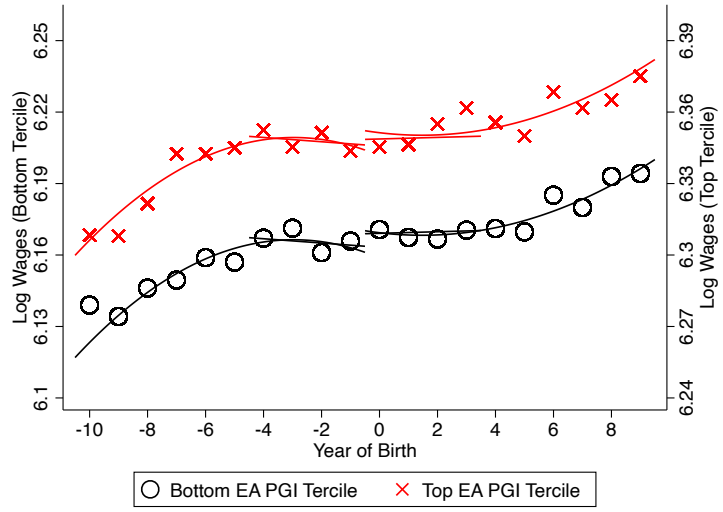
We documented in the previous section that the ROSLA was successful in reducing educational disparities between children from different backgrounds. What would be the expected impact of such reduction on wage disparities *if the returns to schooling were the same for everyone*? At the eve of the program, the wage gap between the top and bottom terciles of each proxy distribution was 0.183 (EA PGI), 0.083 (family size), and 0.078 log points (neighborhood SES). The ROSLA reduced the top-bottom gap in school-leaving age by 0.270 (EA PGI), 0.310 (family size), and 0.096 years of schooling (neighborhood SES) – see Appendix Table I4. If the return to this additional year of schooling were 5% (see Appendix Table G2), we would expect the wage gaps described above to decrease by 7.4% (EA PGI),[37] 18.6% (family size), and 6.1% (neighborhood SES).

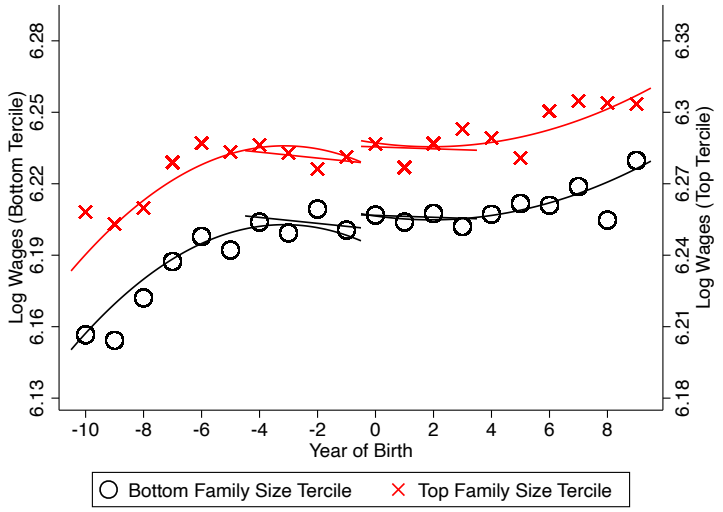
In practice, we find no evidence that there was a reduction in the wage disparities. Figure 7 shows the average log wage by year of birth, separately for those in the bottom and in the top terciles of each proxy distribution. The ROSLA did not narrow the gap between the two groups. Appendix Table J2 confirms these results.

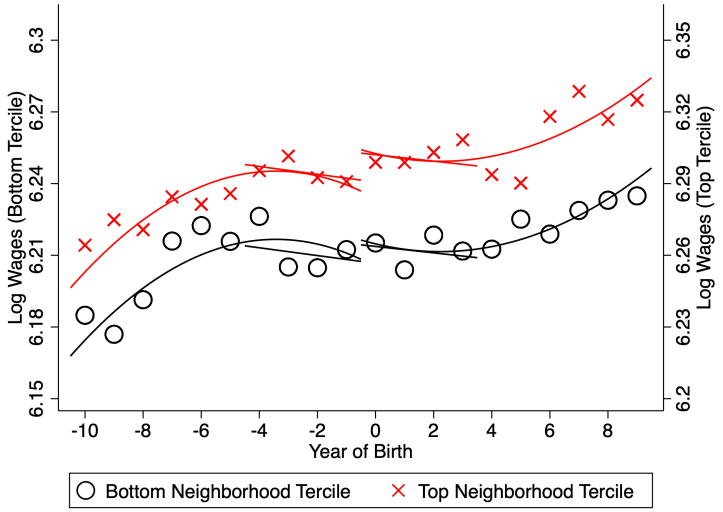
Figure 8 summarizes the first-stage (Appendix Table I4) and reduced-form results (Appendix Table J2). For any given proxy – EA PGI (black), family size (blue), or neighborhood SES (red), it reports the difference between those in the top and bottom terciles of the proxy distribution on the eve of the ROSLA (Xs) and immediately after (circles). The left y-axis and left panel show top-bottom differences in the schooling-leaving age. The right y-axis and right panel show top-bottom differences in log wages. The brackets report 95% confidence intervals testing whether the post-reform difference is equal to the pre-reform difference.[38]

[37] 0.074=(0.05)*(0.27)/0.183, where 0.05 is the return to schooling, 0.27 is the reduction in the educational gap between the top and bottom terciles of the EA PGI distribution, and 0.183 is the original wage gap between them.
[38] Because the figure illustrates a test of the difference between the points indicated by the X's and the solid circles, we do not report confidence intervals for the pre-ROSLA levels.

Figure 7: Effect of the 1972 ROSLA on Log Wages, by EA PGI, Family Size, and Neighborhood SES

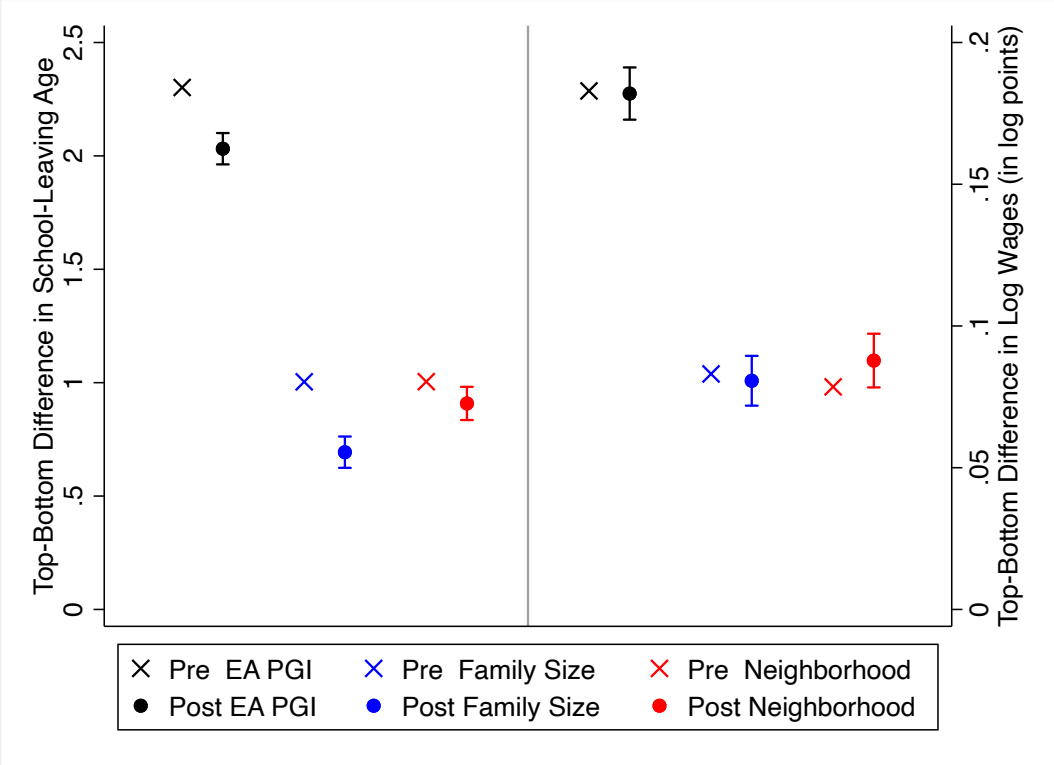






Notes: The figures show average log wages by year of birth, separately for those in the bottom tercile (black circles) and in the top tercile (red crosses) of the following distributions: EA PGI (top panel); family size (middle panel); and neighborhood SES (bottom panel). Average wages for the top tercile is shown on the right y-axis while average wages for the bottom tercile is shown on the left y-axis. Wages are imputed based on one's occupation. Year of birth runs from September 1 of a given a year to August 31 of the following year. Those born in Year 0 were born between September 1, 1957 and August 31, 1958. Cohorts born after Year 0 had to stay in school until age 16 while cohorts born before could leave at age 15. The figure shows linear cohort trends for a 4-year bandwidth and quadratic cohort trends for a 10-year bandwidth. For a given proxy, all three terciles share the same cohort trends. We use triangular kernel weights that give greater weight to study participants born closer to September 1, 1957. N = 79,873 (EA PGI with 4-year bandwidth); 196,727 (EA PGI with 10-year bandwidth); 85,308 (family size with 4-year bandwidth); 209,338 (family size with 10-year bandwidth); 81,454 (neighborhood SES with 4-year bandwidth); and 201,340 (neighborhood SES with 10-year bandwidth).

Figure 8: Summary of First-Stage and Reduced-Form Results



Notes: The figure shows differences in average school-leaving age (left y-axis) and in average log wages (right y-axis) between the top and bottom terciles of the distributions of the EA PGI (black), family size (blue), and neighborhood SES (red) at the eve of the ROSLA (Xs) and immediately after (circles). Wages are imputed based on one's occupation. The brackets report 95% confidence intervals testing whether the post-reform difference is equal to the pre-reform difference. Estimates from specification with 10-year bandwidth and quadratic trends. N = 196,727 (EA PGI); 209,338 (family size); and 201,340 (neighborhood SES).

The left panel shows that the 1972 ROSLA was effective in reducing disparities in education. The pre-ROSLA top-bottom differences in school-leaving age decreased by 10-31% across our three proxies. The right panel shows that, contrary to what was expected, disparities in wages did not reduce. The estimates suggest instead that the wage gap did not change and may have even increased. In the next section, we lay out a model that clarifies that these two results are consistent with each other as long as advantaged children have higher returns to their additional schooling.

D. Early-Life Advantages and The Returns to Schooling

This paper's main question of interest is whether educational policies, such as the ROSLA, can weaken the relationship between early-life advantages and later-life SES. In particular, we are interested in the effect of the ROSLA on the relationship between early-life advantages and wages:

$$\frac{\partial E[Y \mid \boldsymbol{X}, Post = 1]}{\partial B} - \frac{\partial E[Y \mid \boldsymbol{X}, Post = 0]}{\partial B} \tag{5}$$

where Y is log wages, B is a proxy for early-life advantages, $\boldsymbol{X}$ is a vector of observables (including B and birth cohort trends), and $Post$ is an indicator for whether the student was born after September 1, 1957 and had therefore to stay in school until age 16.

If education is a "great equalizer", we would expect (5) to be negative, that is, we would expect the relationship between early-life advantages and wages to be weaker for the cohorts born after September 1, 1957 than for the cohorts born before this date. This is not what we find. Our analyses (see Figures 7 and 8) indicate that (5) is approximately equal to zero.

We show next that the difference in equation (5) can be decomposed into two effects. One effect depends on how the reform affects educational disparities between advantaged and disadvantaged children ("the education effect"). The other depends on how the returns to schooling among compliers vary with early-life advantages ("the returns effect"). The model clarifies that our apparently conflicting results are consistent with each other as long as advantaged children have higher returns to schooling.

For simplicity, we consider the decision of whether to stay in school until age 16 – the margin targeted and most affected by the policy. Let Y_1 be the potential log wage if the individual were to stay in school until age 16, and Y_0 her potential log wage if she were to drop out before age 16. Define the potential outcomes as:

$$Y_1 = \mu_1(\boldsymbol{X}) + E_1 \text{ and } Y_0 = \mu_0(\boldsymbol{X}) + E_0 \tag{6}$$

$$\text{where } \mu_1(\boldsymbol{x}) = E[Y_1 \mid \boldsymbol{X} = \boldsymbol{x}] \text{ and } \mu_0(\boldsymbol{x}) = E[Y_0 \mid \boldsymbol{X} = \boldsymbol{x}].$$

The return to schooling for the individual is:

$$\beta \equiv Y_1 - Y_0 = \mu_1(\boldsymbol{x}) - \mu_0(\boldsymbol{x}) + E_1 - E_0. \tag{7}$$

Let Δ be the net benefit to the individual of staying in school until age 16:

$$\Delta = \Pi(\boldsymbol{X}) - V \tag{8}$$

where $\Pi(\boldsymbol{X})$ is the part of the net benefit that is explained by the observables $\boldsymbol{X}$ and V is the unobserved (net) cost of staying in school until age 16. In (8), V is uncorrelated with $\boldsymbol{X}$ by construction. The individual decides to stay in school until age 16 if $\Delta \geq 0$:

$$S = 1 \iff \Delta \geq 0 \tag{9}$$

The actual wage is $Y = SY_1 + (1 - S)Y_0$.

One can show that (5) is equal to:[39]

$$\overbrace{\frac{\partial E[Y \mid \boldsymbol{X}, Post = 1]}{\partial B} - \frac{\partial E[Y \mid \boldsymbol{X}, Post = 0]}{\partial B}}^{\text{Total Effect}}$$

$$= \overbrace{E(\beta \mid \boldsymbol{X}, Complier) \times \frac{\partial}{\partial B}\left[E(S \mid \boldsymbol{X}, Post = 1) - E(S \mid \boldsymbol{X}, Post = 0)\right]}^{\text{Education Effect}}$$

$$+ \overbrace{\left[E(S \mid \boldsymbol{X}, Post = 1) - E(S \mid \boldsymbol{X}, Post = 0)\right] \times \frac{\partial E(\beta \mid \boldsymbol{X}, Complier)}{\partial B}}^{\text{Returns Effect}} \tag{10}$$

We refer to the left hand side of (10) as the "total effect." The first line on the right hand side is "the education effect." The education effect is a product of two terms: the average returns to schooling among compliers, which is positive, times the effect of the reform on educational

[39] The schooling decisions of non-compliers are the same irrespective of whether they born after September 1, 1957 or not. Thus, any post-pre difference in wages is driven by wage gains of compliers of staying in school until age 16:
$E[Y \mid \boldsymbol{X}, Post = 1] - E[Y \mid \boldsymbol{X}, Post = 0]$
$= Pr(Complier \mid \boldsymbol{X}) \times \{E[Y \mid \boldsymbol{X}, Complier, Post = 1] - E[Y \mid \boldsymbol{X}, Complier, Post = 0]\}$.
The actual wage Y can be rewritten as $Y = SY_1 + (1 - S)Y_0 = Y_0 + S\beta$, giving:
$E[Y \mid \boldsymbol{X}, Post = 1] - E[Y \mid \boldsymbol{X}, Post = 0]$
$= Pr(Complier \mid \boldsymbol{X}) \times \{E[S\beta \mid \boldsymbol{X}, Complier, Post = 1] - E[S\beta \mid \boldsymbol{X}, Complier, Post = 0]\}$
$= Pr(Complier \mid \boldsymbol{X}) \times E[\beta \mid \boldsymbol{X}, Complier, Post = 1]$
where we have used that $E[Y_0 \mid \boldsymbol{X}, Complier, Post] = E[Y_0 \mid \boldsymbol{X}, Complier]$, that $E[S \mid \boldsymbol{X}, Complier, Post = 0] = 0$, and that $E[S \mid \boldsymbol{X}, Complier, Post = 1] = 1$. Finally, we get equation (10) by taking the derivative of the expression above and using the product rule. Notice $Pr(Complier \mid \boldsymbol{X}) = E(S \mid \boldsymbol{X}, Post = 1) - E(S \mid \boldsymbol{X}, Post = 0)$ because only compliers change their decisions of whether to stay in school until age 16 as a consequence of the ROSLA.

disparities. The second line on the right hand side is the "returns effect." The returns effect is also a product of two terms: the effect of the reform on education, which is positive, times a second term that captures how the returns to schooling (among compliers) varies with advantage. In sum, equation (10) shows that the effect of the ROSLA on the wage gap depends on both the education and returns effects. This model extends to other specifications described above. For example, the vector of observables, $\boldsymbol{X}$, could include the three proxies for advantage. In this case, equation (10) would correspond to the effect of the ROSLA on the relationship between log wages and *a particular proxy* for early-life advantage, *holding the other two proxies constant*.

Our results in the previous section imply that the total effect is approximately zero and that the education effect is negative. Therefore, we would expect the returns effect to be positive, that is, that the returns to schooling should increase with advantage. We investigate next the heterogeneity in returns to schooling. The technology of skill formation framework (Heckman 2000; Cunha et al. 2006) provides a rationale for why the returns to schooling may vary with the EA PGI, family size, or neighborhood SES. Conti and Heckman (2010), for example, propose that genetics may affect the stock of skills both as an input and by shaping the production function, which in turn may increase the returns to schooling (Heckman, Stixrud, and Urzua 2006; Urzua 2006).

To estimate how the returns to schooling vary with our proxies, we estimate the following equation:

$$lnW_i = \eta_0 + \eta_1 SLA_i + \boldsymbol{B}_i'\boldsymbol{\eta}_2 + SLA_i \times \boldsymbol{B}_i'\boldsymbol{\eta}_3 + l(DoB_i) + \epsilon_i, \quad (11)$$

We estimate (11) through two-stage least squares (2SLS), using the indicator for being born on or after September 1, 1957 and its interactions with $\boldsymbol{B}_i$ to instrument for school-leaving age and its interactions with $\boldsymbol{B}_i$. The results are shown in Table 2. The top panel shows results for the full sample. The bottom panel restricts the sample to those who left school at age 18 or younger in order to increase statistical power (see Appendix K). As shown in Appendix H, the ROSLA did not affect the fraction of students who stayed in school past age 18.

Table 2: Effect of an Additional Year of Schooling on Log Wages, by EA PGI, Family Size, and Neighborhood SES

	Log Wages							
Top Panel: Full Sample	*4−Year Bandwidth*				*10−Year Bandwidth*			
PGI * SLA	0.026			0.059	0.042			0.044
	[0.049]			[0.143]	[0.025]			[0.031]
Family * SLA		0.023		0.033		0.020		0.023
		[0.018]		[0.100]		[0.010]		[0.014]
Neighborhood * SLA			0.122	0.107			0.056	0.046
			[0.140]	[0.150]			[0.020]	[0.020]
SLA	0.063	0.061	0.089	0.143	0.072	0.061	0.076	0.097
	[0.066]	[0.053]	[0.092]	[0.234]	[0.037]	[0.031]	[0.032]	[0.051]
Observations	86,417	86,417	86,417	86,417	212,290	212,290	212,290	212,290
Bottom Panel: SLA < 19								
PGI * SLA	0.023			0.022	0.025			0.025
	[0.013]			[0.013]	[0.008]			[0.008]
Family * SLA		0.020		0.018		0.019		0.018
		[0.010]		[0.010]		[0.006]		[0.006]
Neighborhood * SLA			0.028	0.025			0.022	0.020
			[0.015]	[0.015]			[0.009]	[0.009]
SLA	0.066	0.060	0.063	0.083	0.062	0.055	0.056	0.072
	[0.025]	[0.026]	[0.024]	[0.029]	[0.021]	[0.021]	[0.021]	[0.023]
Observations	46,308	46,308	46,308	46,308	114,025	114,025	114,025	114,025

Notes: The dependent variable is log wages, which are imputed based on one's occupation. SLA is school-leaving age. PGI is the polygenic index for educational attainment. The bottom panel is restricted to participants who dropped out at age 18 or younger. PGI, Family, and Neighborhood are all standardized to have mean zero and standard deviation of 1. The first four columns include linear trends in exact date of birth while the last four include quadratic trends. In both cases, trends are allowed to be different before and after September 1, 1957. Robust standard errors between brackets. We omit the coefficients on PGI, Family, and Neighborhood.

We find larger returns to schooling for advantaged children, as measured by our proxies. Full sample results, presented in the top panel of Table 2 show that all interactions are positive, even if not always statistically significant. Once we condition to the group that was affected by the reform (those who left school before age 19, bottom panel) coefficients are similar but more precisely estimated.[40] Overall, an improvement in one standard deviation unit in any of our proxies increases the returns to schooling by approximately 1.8 to 2.8 percentage points (bottom panel of Table 2), consistent with the implications of the decomposition of equation (10). Furthermore, the coefficients on the interactions are very similar in the specifications that include all proxy variables

[40] Appendix Table J3 shows the discrete version of Table 2.

at once (bottom panel, columns 4 and 8). This suggests that the gradients in returns to schooling with respect to the three different proxies are independently important. We show in the Robustness Section that we find qualitatively similar results if we use income instead of wages as the dependent variable.

E. Genetics, Environment, and Educational Disparities

The results presented so far show that the compulsory schooling reform weakened the relationship between early-life advantages and education. As discussed in Section 2, we do not have however exogenous variation in our proxies for early-life advantages, which complicates the interpretation of our results. Take the case of the EA PGI for example. In terms of the model presented above, the effect of the ROSLA on the relationship between the EA PGI and education is given by:

$$\frac{\partial E[S \mid PGI, Post = 1]}{\partial PGI} - \frac{\partial E[S \mid PGI, Post = 0]}{\partial PGI}. \tag{12}$$

For ease of exposition, we suppress $\boldsymbol{X}$ that contains the birth cohort trends and may include the other proxies for early-life advantages. Empirically, this effect corresponds to the coefficient δ_3 estimated in Section 4.B:

$$S_i = \delta_0 + \delta_1 Post_i + \delta_2 PGI_i + \delta_3 (PGI_i \times Post_i) + k(DoB_i) + \varepsilon_i. \tag{4'}$$

However, as discussed in Section 3, individual differences in the EA PGI confound genetic differences with differences in socioeconomic background: for example, individuals with higher EA PGIs tend to have parents with higher EA PGIs, who are on average more educated and have higher average incomes. As a result, δ_3 conflates two effects of the compulsory schooling reform: (i) its effect on the causal relationship between genetics and education and (ii) its effect on the association between socioeconomic background and education. Compulsory schooling laws such as the ROSLA typically aim to reduce (ii) but not necessarily (i).

This section leverages the biological processes of Mendelian inheritance to separately estimate (i) and (ii). During meiosis, the genes that a person inherits are randomly assigned conditional on the genes of their parents. Using the random inheritance of a person's genes from their parents, we can decompose the EA PGI into two components: its random component and the parental PGI F,

which proxies for one's socioeconomic background. In this context, the effects (i) and (ii) correspond respectively to the following derivatives:

$$\frac{\partial E[S \mid PGI, F, Post = 1]}{\partial F} - \frac{\partial E[S \mid PGI, F, Post = 0]}{\partial F} \tag{13}$$

$$\frac{\partial E[S \mid PGI, F, Post = 1]}{\partial PGI} - \frac{\partial E[S \mid PGI, F, Post = 0]}{\partial PGI} \tag{14}$$

The first is the effect of the reform on the relationship between education and one's socioeconomic background, holding one's genetics constant. The latter is the effect of the reform on the relationship between education and one's genetics, holding socioeconomic background constant – in contrast to equation (12), which did not hold the socioeconomic background constant.

In practice, we estimate:

$$S_i = \theta_0 + \theta_1 Post_i + \theta_2 PGI_i + \theta_3 (PGI_i \times Post_i) + \theta_4 F_i + \theta_5 (F_i \times Post_i) + m(DoB_i) + \xi_i. \tag{15}$$

The coefficient on $PGI_i \times Post_i$, θ_3, estimates (ii), the effect of the ROSLA on the causal relationship between education and genetics (also known as the "direct genetic effect"). The coefficient on $F_i \times Post_i$, θ_5, estimates (i) the effect of the ROSLA on the association between education and one's socioeconomic background (proxied by the parental PGI).

This design is unique and powerful. To our knowledge, this study is the first to estimate a gene-by-environment interaction using credibly exogenous variation in both genotypes and environment. One potential explanation for this gap in the literature is that the design is very demanding in terms of statistical power. It requires having a large enough sample that satisfies two conditions: (a) participants were affected by an exogenous variation in environmental circumstances and (b) there are data on the genotypes of participants and their parents. As discussed in Section 3.C, while few UK Biobank study participants had a parent who was also part of the study, it is possible to impute the parental genotypes if at least one of the study participant's siblings is also genotyped (Young et al. 2020). Measured or imputed data on parental PGIs are available for about 10% of our sample. Furthermore, the child PGI and imputed parental PGI are

highly correlated ($r = 0.86$).[41] This means that the residual variation in the children's PGI after controlling for the parental PGI is small, leading to even larger standard errors than would be obtained in a sample of the same size that did not control for the parental PGIs. For this reason, the analyses in this section are not as well-powered as the other analyses in the paper. Given the statistical power concerns, we focus on the specification with a 10-year bandwidth. Appendix F assesses the sensitivity of results to alternative bandwidths and to alternative specifications, including a family fixed effects specification.

Results are shown in Table 3. The top panel shows first-stage results for both the fraction of participants who stayed in school until age 16 and for school-leaving age. The bottom panel shows reduced-form results for the fraction of participants who got a CSE or an O-Level (the educational qualification exams that were typically taken at age 16) and for log wages. Columns (1), (4), (7), and (10) do not condition on the parental PGI. All the remaining columns include both the parental PGI and its interaction with the indicator for being born after September 1, 1957, which permits separately estimating the effects of the ROLSA on the relationships of education with both one's socioeconomic background as well as with one's genetics. Columns (3), (6), (9), and (12) also add family size and neighborhood SES, our other proxies for early-life advantages, and their interactions with the indicator.

The main take away from Table 3 is that the ROSLA reduced educational differences driven by both environmental (e.g. credit constraints or school quality) and genetic advantages (e.g. innate ability or physical attractiveness). In other words, the ROSLA reduced not only the parental PGI's predictive power but also the direct genetic effect. First, in column (1), we learn that the policy reduced the association between the EA PGI and whether a student stayed in school until age 16 by more than 80%. When we condition on the parental PGI in column (2), we further learn that the policy reduced both the causal relationship between staying in school until age 16 and one's genetics as well as the association between staying until age 16 and the parental PGI. The coefficients on the parental PGI and its interactions are very similar when controls for family size and neighborhood SES are added in column (3), suggesting again that the non-causal component

[41] Normally, the correlation between a person's PGI and the mean PGI of their parents is $\sqrt{0.5}$. The correlation is higher with imputed parental PGIs because only the genotypes that are transmitted to at least one child are observed. The untransmitted genotypes, which are approximately uncorrelated with the transmitted ones, are not included in the imputed parental PGI.

of the PGI captures additional variation in socioeconomic background beyond what is captured by family size and neighborhood SES.

Table 3: Genetics, Environment, and Educational Disparities

	Stayed in School until 16			*School-Leaving Age*		
	(1)	(2)	(3)	(4)	(5)	(6)
Top Panel: First Stage						
PGI * Post	-0.074	-0.026	-0.025	-0.078	-0.035	-0.029
	[0.004]	[0.008]	[0.008]	[0.039]	[0.083]	[0.082]
Parental PGI * Post		-0.044	-0.040		-0.040	-0.033
		[0.006]	[0.006]		[0.065]	[0.064]
Family * Post			-0.049			-0.086
			[0.005]			[0.047]
Neighborhood * Post			-0.023			0.023
			[0.004]			[0.047]
Post	0.144	0.142	0.116	0.234	0.216	0.168
	[0.012]	[0.012]	[0.012]	[0.125]	[0.125]	[0.125]
PGI	0.090	0.037	0.036	0.986	0.466	0.459
	[0.004]	[0.008]	[0.007]	[0.029]	[0.059]	[0.058]
Parental PGI		0.049	0.045		0.476	0.442
		[0.006]	[0.006]		[0.045]	[0.045]
Family			0.059			0.37
			[0.005]			[0.033]
Neighborhood			0.029			0.266
			[0.004]			[0.033]
	CSE or O-Level			*Log Wages*		
	(7)	(8)	(9)	(10)	(11)	(12)
Bottom Panel: Reduced-Form						
PGI * Post	-0.054	-0.024	-0.023	-0.008	-0.011	-0.010
	[0.006]	[0.011]	[0.011]	[0.006]	[0.011]	[0.011]
Parental PGI * Post		-0.027	-0.024		0.002	0.003
		[0.009]	[0.009]		[0.009]	[0.009]
Family * Post			-0.020			-0.006
			[0.007]			[0.005]
Neighborhood * Post			-0.025			0.002
			[0.006]			[0.006]
Post	0.071	0.069	0.060	0.005	0.004	0.000
	[0.017]	[0.017]	[0.017]	[0.016]	[0.016]	[0.017]
PGI	0.096	0.054	0.053	0.089	0.051	0.050
	[0.004]	[0.009]	[0.009]	[0.004]	[0.008]	[0.007]
Parental PGI		0.038	0.033		0.035	0.033
		[0.007]	[0.007]		[0.006]	[0.006]
Family			0.055			0.032
			[0.005]			[0.004]
Neighborhood			0.033			0.021
			[0.005]			[0.004]

Notes: The top panel shows first stage-estimates. The bottom panel shows reduced-form estimates. PGI is the polygenic index for educational attainment. It is standardized to have a mean of zero and a standard deviation of one. Parental PGI, which is the sum of the PGIs of parents, is standardized using the same statistics. See Section 3.B about how the parental PGI is imputed. Family and Neighborhood are also standardized to have mean zero and standard deviation of one. Log wages is imputed based on one's occupation. Ten-year bandwidth including quadratic trends in exact date of birth. Trends are allowed to be different before and after September 1, 1957. Robust standard errors between brackets. N = 21,418 in columns (7)-(9) and 21,491 in all other columns.

Columns (4)-(6) are broadly consistent with columns (1)-(3): the point estimates also suggest that the ROSLA reduced the direct genetic effect on school-leaving age and its association with the parental PGI. However, these effects are not statistically significant; they are less precisely estimated than the effect on staying until age 16 because there is more variation at higher school-leaving ages that the ROSLA reform did not affect.

The results in columns (1) to (3) are consistent with how the ROSLA affected the distribution of schooling. There was near perfect compliance with the compulsory schooling law; therefore, there is very little variation in the fraction of those cohorts born after September 1, 1957 who stayed in school until age 16 to explain. For this reason, these negative interactions are perhaps to be expected. In this sense, the results in columns (7) to (9), the reduced-form effect of the reform on the fraction of participants with a CSE or an O-Level, are more interesting. In contrast to a person's school-leaving age, the compulsory schooling law did not establish any regulations regarding these educational qualifications; students were not required by law to obtain either one of them. In other words, there is no reason *a priori* to expect that the ROSLA would have changed the direct genetic effects and the environmental effects on the fraction of participants with a CSE or O-Level.

The ROSLA increased the fraction of students obtaining one of these qualifications by 7.1 percentage points – see column (7). It also reduced the association between the EA PGI and the likelihood of having a CSE or an O-level. For participants born before September 1, 1957, a one standard deviation increase in the EA PGI was associated with a 9.6 percentage-point *increase* in this likelihood. For participants born after September 1, 1957, this association is reduced to less than half of that.

More interestingly, the ROSLA weakened the relationship between having one of these qualifications and one's socioeconomic background, as column (8) shows. It also weakened the relationship between these qualifications and one's genetics, reducing the direct genetic effect.

This cleanly illustrates how the effect of genetics on education is itself a function of the environment; even causal genetic effects are not fixed or unmodifiable.

The similarity of the coefficients on the interaction terms in column (8) may give the impression that the compulsory schooling reform impacted the genetic and environmental effects similarly. However, when we control for family size and neighborhood SES in column (9), we notice that the impact of the reform on environmental effects was much larger, considering that it also weakened the relationship of qualifications with family size and with neighborhood SES.[42]

For completeness, we also revisit in columns (10) to (12) the question of whether the schooling reform reduced wage disparities. Was the ROSLA successful in making the environmental conditions into which a child was born less important for her wage prospects? We cannot reject the hypothesis that the relationship between environmental advantages and wages remained unchanged, but then again the standard errors are large. Because of these statistical power concerns, we refrain from estimating the two-stage least squares counterparts of (11) and (12).

5. Robustness

In this section, we report on a series of exercises to investigate the robustness of our results. We show how the results (do not) change when we (a) Vary the regression discontinuity bandwidth; (b) Allow different terciles to have different birth cohort trends; (c) Include controls; and (d) Use income or the Townsend Deprivation Index as the SES outcome.

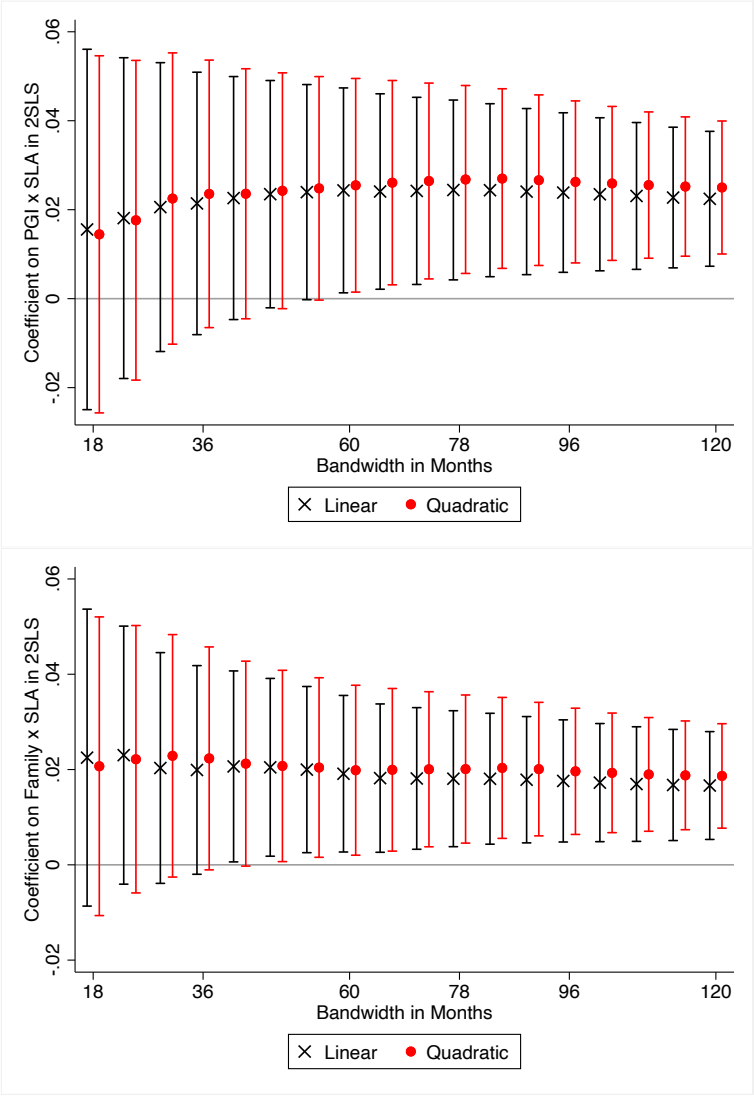
A. Bandwidth Choice

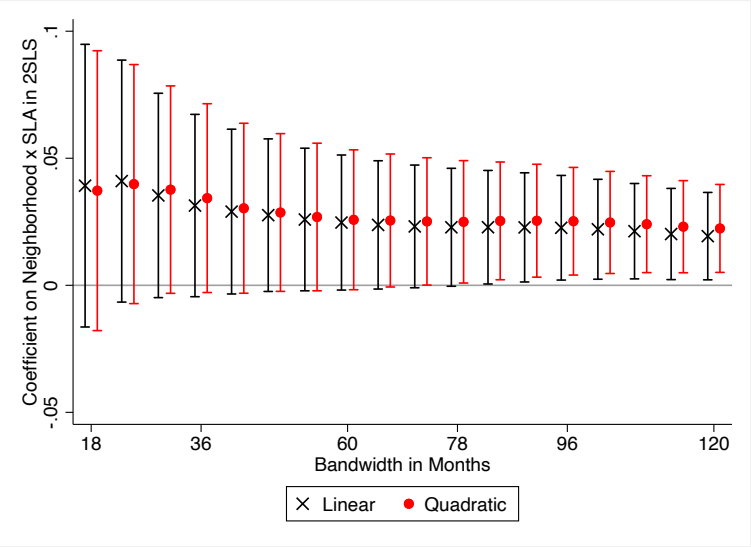
Figure 9 assesses the sensitivity of the results to the choice of bandwidth. It shows two-stage least square estimates for different bandwidths (measured in months). The markers display the coefficient on the interaction of one of the proxies with school-leaving age (i.e., $\boldsymbol{\eta_3}$ in the continuous specification of equation (11)) with 95% confidence intervals around it. The black Xs

[42] On the other hand, the PGI does not represent all genetic factors that influence educational attainment (e.g., rare variants) and it is only a noisy proxy for the true contribution of the effect of the included variants (Becker et al. 2021). As such, compulsory schooling laws could have had a larger impact on genetic effects than environmental effects. That said, we believe this is unlikely since a more "complete" PGI may also better reflect environmental factors such as indirect effects and population stratification, just as they do for the "incomplete" PGI used in this paper. Therefore, we anticipate that the effect of the ROSLA on environmental effects is larger than its effect on genetic effects.

are estimated using linear trends. The red circles are estimated using quadratic trends. The figure shows that the point estimates are remarkably similar across bandwidths and trend specifications. They are not statistically different from zero at the 5% confidence level for narrower bandwidths, but this is expected because the confidence intervals increase as the bandwidth shrinks. The corresponding figures for the first-stage estimates and for the reduced-form estimates are shown in Appendix L.

Figure 9: Sensitivity to Bandwidth Choice





Notes: These figures investigate how the 2SLS estimates (from continuous specification) vary with the bandwidth size (in months). It shows the coefficient on the interaction of school-leaving age with the EA PGI (top panel), family size (middle panel), and neighborhood SES (bottom panel). Black Xs show estimates using linear trends. Red circles show estimates using quadratic trends. Wages are imputed based on one's occupation. The sample is restricted to participants who dropped out at age 18 or younger. *N* varies from 17,783 (18-month bandwidth) to 114,077 (120-month bandwidth).

B. Allowing Birth Cohort Trends to Vary with Proxies for Early-Life Advantages

To maximize statistical power, the previous analyses assumed that the birth cohort trends were invariant to early-life advantages. Figure 10 assesses the sensitivity to this assumption. It shows two sets of quadratic trends for the 10-year bandwidth. The solid curves show the birth cohort trends for log wages under this assumption. The dashed curves show comparable trends when the top and bottom terciles of a given proxy are allowed to have distinct birth cohort trends (the middle tercile also has its own birth cohort trend; it is not shown in the figure for ease of exposition). The two sets of trends are in general quite similar. Appendix Figure M1 and Appendix Figure M2 show birth cohort trends for school-leaving age and for qualifications, yielding the same conclusion. Appendix Figure M3, M4, and M5 show that the results also hold for the 4-year bandwidth with linear trends. Tables in Appendix M show corresponding estimates. Importantly, the p-values reported in the tables show that we cannot reject at the 5% level that the different terciles have the same birth cohort trends.

C. Controls

Table 4 investigates whether the results are robust to the inclusion of controls. For conciseness, we only report results for the continuous specification using a 10-year bandwidth with quadratic trends. Column (1) of Table 4 reproduces the first-stage benchmark (which corresponds to the last column, top panel of Table 1) while column (4) reproduces the 2SLS benchmark (which corresponds to the last column, bottom panel of Table 2). Columns (2) and (5) of Table 4 add the following controls: male, age, age squared, male $\times$ age, male $\times$ age squared, and indicator variables for the calendar month of birth. To address concerns about population stratification (see Section 3.A), columns (3) and (6) include, besides the controls previously mentioned, the first twenty principal components (PCs) of the participant's genetic matrix and the interactions of these PCs with either the indicator for being born after September 1, 1957 (in the first-stage) or with school-leaving age (in the 2SLS). The first-stage results in columns (2) and (3) are not sensitive to these inclusions. Nor are the 2SLS results shown in columns (5) and (6). That said, there is evidence that some social stratification in a PGI may remain between historically poorer and richer regions of the UK even after controlling for principal components (Abdellaoui et al. 2019).

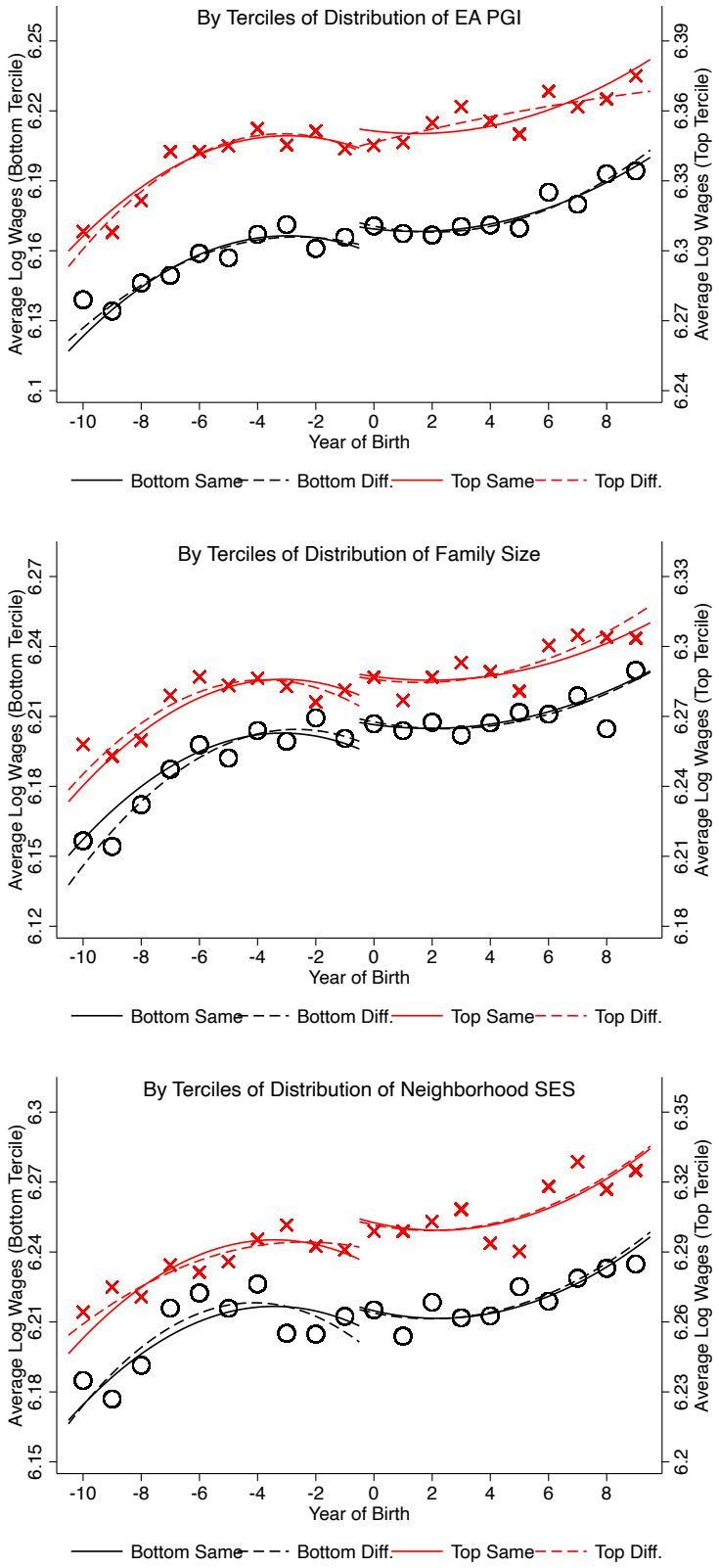
D. Alternative SES Measures

Finally, we investigate the concern that data on wages are available only for those UKB participants who reported an occupation. The last two columns of Table 4 report two-stage least square estimates that use as dependent variables either an indicator for having an annual household income of £31,000 or more or a Townsend Deprivation Index (reverse coded). Among the 155,807 UKB participants who dropped out at age 18 or younger, wages are missing for 26.8% of them, income is missing for 14.5% and the Townsend Index for 0.1%. The Townsend Deprivation Index is a measure of the material deprivation of the neighborhoods in which study participants lived.[43] Overall, we reach similar conclusions irrespective of whether we use occupational wages, household income (see also Appendix D), or the Townsend index. The exception is that, when we use these alternative outcomes, the returns to schooling no longer vary with neighborhood SES.

[43] The Townsend Deprivation Index is constructed from four rates measured at the neighborhood level: (1) unemployment, (2) non-car ownership, (3) non-home ownership, and (4) household overcrowding. Rates were estimated for each output area using the 2001 Census. Participants were assigned the scores of the output areas where their residential postcodes were located.

These results are consistent with the evidence shown in the Appendix E that the fraction missing wages and the fraction missing household income are smooth around the September 1, 1957 birthday cutoff.

Figure 10. Different Birth Cohort Trends for Bottom and Top Terciles



Notes: The figure assesses the sensitivity of the results to the assumption that the birth cohort trends were invariant to early-life advantages. The solid curves show 10-year birth cohort trends for log wages under this assumption. The dashed curves show comparable trends when the top and

bottom terciles are allowed to have distinct birth cohort trends (the middle tercile also has its own birth cohort trend; it is not shown for ease of exposition). N = 196,813 (EA PGI); 209,425 (family size); and 201,425 (neighborhood SES). Wages are imputed based on one's occupation.

Table 4: Robustness

	First Stage			2SLS				
	School-Leaving Age			*Log Wages*			*Income ≥ £31,000*	*Townsend Index*
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PGI * Post	-0.106	-0.108	-0.115					
	[0.013]	[0.013]	[0.014]					
Family * Post	-0.124	-0.122	-0.120					
	[0.016]	[0.016]	[0.016]					
Neighborhood * Post	-0.020	-0.018	-0.013					
	[0.014]	[0.014]	[0.015]					
Post	0.166	0.119	0.114					
	[0.038]	[0.038]	[0.039]					
PGI * SLA				0.025	0.021	0.021	0.027	0.064
				[0.008]	[0.007]	[0.008]	[0.011]	[0.022]
Family * SLA				0.018	0.019	0.018	0.034	0.056
				[0.006]	[0.005]	[0.005]	[0.008]	[0.016]
Neighborhood * SLA				0.020	0.019	0.017	-0.014	0.011
				[0.009]	[0.008]	[0.009]	[0.011]	[0.023]
SLA				0.072	0.075	0.081	0.148	0.119
				[0.023]	[0.024]	[0.026]	[0.037]	[0.073]
Controls?		✓	✓		✓	✓	✓	✓
PCs?			✓			✓	✓	✓
Observations	212,290	212,290	212,290	114,025	114,025	114,025	133,086	155,490

Notes: The first six columns of the table assess the robustness to including controls. All columns except for columns (1) and (4) include the following controls: male, age, age squared, male × age, male × age squared, and indicator variables for the calendar month of birth. To address concerns about population stratification, columns (3) and (6) include in addition the first twenty principal components (PCs) of the participant's genetic matrix and the interactions of these PCs with either the indicator for being born after September 1, 1957 or with school-leaving age. The dependent variable in columns (1) to (6) is log wages, which are imputed based on one's occupation. To investigate concerns about missing wages, columns (7) and (8) use as the dependent variable an indicator for having an annual household income of £31,000 or more and the Townsend Deprivation Index (reverse coded). Columns (4) to (8) are restricted to participants who dropped out at age 18 or younger. Robust standard errors between brackets.

6. Conclusion

Many consider education to be the "great equalizer" that "levels the playing field," giving children from different backgrounds similar opportunities of economic success. We investigated whether education weakens the relationship between early-life advantages and later-life SES. In particular, we examined whether the ROSLA, a 1972 compulsory schooling reform in the UK that increased secondary education, reduced disparities between disadvantaged and advantaged children. Besides early, favorable family and neighborhood circumstances, we argue that the genes

a child inherits also represent a source of individual-level advantages. We find that the ROSLA was successful in reducing the differences in education between children from advantaged and disadvantaged backgrounds.

The lack of exogenous variation in our proxies for early-life advantages complicates however the interpretation of these results. In the case of the EA PGI, for example, individual differences in the EA PGI confound genetic differences with differences in socioeconomic background. We exploited that a person's PGI is randomly assigned conditional on the PGI of their parents to separate between two conflated effects of the reform: (i) its effect on the causal relationship between education and genetics and (ii) its effect on the association between education and one's socioeconomic background. We find that the ROSLA weakened the relationship between education and one's socioeconomic background, making the environmental conditions into which a child was born less important for her education. The policy also weakened the relationship between education and one's genetics, reducing the direct genetic effect. This illustrates how the effect of genetics on education is itself a function of the environment. Even causal genetic effects are not fixed and can be modified by policy.

While the ROSLA reduced educational disparities, the reform was not a "great equalizer." It did not reduce the wage gaps between individuals from different backgrounds. This is consistent with the idea that education might be more effective in reducing poverty than in promoting mobility (Cameron and Heckman 2001). Our finding contrasts with the impacts of educational reforms in Sweden, Finland, and Norway. The Swedish reform increased the wages of children of less-educated fathers and decreased the wages of children of more-educated fathers (Meghir and Palme 2005); the Finish reform reduced the correlation between the earnings of fathers and sons (Pekkarinen et al. 2009); and the Norwegian reform reduced the gap in earnings between disadvantaged and advantaged children (Bertrand, Mogstad and Mountjoy 2020).

In contrast to the reforms in Sweden, Norway and Finland, the key elements of the British school system did not change with the 1972 ROSLA. This distinction enables us to estimate how returns to schooling vary with children's background and to identify the role that such differences may play in the intergenerational transmission of SES. We find that advantaged children had higher returns to schooling, which explains why, despite reducing educational disparities, the UK reform did not weaken the relationship between early advantages and wages. In contrast, the Swedish, Finish, and Norwegian reforms shifted a large fraction of disadvantaged children from a

vocational stream with lower returns to schooling to a more academic stream with higher returns.[44] The contrast between these two types of reforms suggests that differences in the returns to schooling may be an important obstacle in increasing intergenerational mobility (Solon 2004).

There are several possible explanations for why advantaged children had higher returns to schooling. One is school quality. We found higher returns to schooling for children born in more-educated neighborhoods; these children might have gained more skills from the additional schooling if these neighborhoods had higher-quality schools.[45] An alternative hypothesis is that, due to frictions in migration, children born in less-educated neighborhoods may not have benefited as much from additional education because there were fewer high-paying jobs where they lived (Bergman et al. 2019). Additionally, environmental advantages could have played out at the family-level. Heckman and co-authors have argued that parents play a crucial role in fostering non-cognitive and cognitive skills of their children (Heckman 2000; Cunha et al. 2006), and that these skills increase the returns to schooling of their offspring (Heckman, Stixrud, and Urzua 2006; Urzua 2006). Finally, differences in genetically-influenced characteristics, such as innate ability or physical attractiveness, might also help explain the EA PGI gradient in returns to schooling. Understanding the reasons behind the SES gradient on returns to schooling is crucial for the design of policies to increase mobility and would be an important topic for further research.

One potential way to further our understanding of such underlying factors would be to exploit random family genetic variation coupled with quasi-experimental designs, following the approach implemented in this study. To our knowledge, this study is the first to estimate a gene-by-environment interaction using credibly exogenous variation in both genotypes and environment. Going forward, this design will be essential to deepen our understanding of how nature and nurture jointly shape socioeconomic status.

[44] Starting in seventh grade, students in Sweden who attended the old school system were tracked into an academic or a vocational stream based on their grades. The reform abolished this selection, giving students the option to choose between three different streams. In Finland, students could also choose between an academic and a vocational track. The reform postponed this choice from age 11 to age 16. In Norway, the reform aimed to improve the quality of the vocational track by integrating more general education into the curriculum and offering vocational students a pathway to college.

[45] In the US, returns to schooling are larger at regions with higher-quality schools (Card and Krueger 1992; DeCicca and Krashinsky 2020). That said, the limited existing evidence for the UK suggests that the returns to schooling in England and Wales do not vary with school quality (Campbell 2001).

References

Aakvik, Arild, Kjell G. Salvanes, and Kjell Vaage. "Measuring heterogeneity in the returns to education using an education reform." *European Economic Review* 54, no. 4 (2010): 483-500.

Abdellaoui, Abdel, David Hugh-Jones, Loic Yengo, Kathryn E. Kemper, Michel G. Nivard, Laura Veul, Yan Holtz et al. "Genetic correlates of social stratification in Great Britain." *Nature human behaviour* 3, no. 12 (2019): 1332-1342.

Allen, Naomi, Cathie Sudlow, Paul Downey, Tim Peakman, John Danesh, Paul Elliott, John Gallacher et al. "UK Biobank: Current status and what it means for epidemiology." *Health Policy and Technology* 1, no. 3 (2012): 123-126.

Almond, Douglas, Janet Currie, and Valentina Duque. "Childhood circumstances and adult outcomes: Act II." *Journal of Economic Literature* 56, no. 4 (2018): 1360-1446.

Altonji, Joseph G., and Thomas A. Dunn. "Using siblings to estimate the effect of school quality on wages." *The Review of Economics and Statistics* (1996): 665-671.

Andreoli, Francesco, Markus Jantti, and Arnaud Lefranc. "Does Comprehensive Compulsory Schooling Equalize Opportunities? Evidence from the Swedish Compulsory Schooling Reform," 2020.

Angrist, Joshua, Victor Lavy, and Analia Schlosser. "Multiple experiments for the causal link between the quantity and quality of children." *Journal of Labor Economics* 28, no. 4 (2010): 773-824.

Ashenfelter, Orley, and Cecilia Rouse. "Income, schooling, and ability: Evidence from a new sample of identical twins." *The Quarterly Journal of Economics* 113, no. 1 (1998): 253-284.

Banks, James, and Fabrizio Mazzonna. "The effect of education on old age cognitive abilities: evidence from a regression discontinuity design." *The Economic Journal* 122, no. 560 (2012): 418-448.

Banks, James, Leandro S. Carvalho, and Francisco Perez-Arce. "Education, decision making, and economic rationality." *Review of Economics and Statistics* 101, no. 3 (2019): 428-441.

Barcellos, Silvia H., Leandro S. Carvalho, and Patrick Turley. "Education can reduce health differences related to genetic risk of obesity." *Proceedings of the National Academy of Sciences* 115, no. 42 (2018): E9765-E9772.

Barcellos, Silvia H., Leandro S. Carvalho, and Patrick Turley. "Distributional effects of education on health." Forthcoming at the *Journal of Human Resources*.

Barth, Daniel, Nicholas W. Papageorge, and Kevin Thom. "Genetic endowments and wealth inequality." *Journal of Political Economy* 128, no. 4 (2020): 1474-1522.

Becker, Joel, Casper AP Burik, Grant Goldman, Nancy Wang, Hariharan Jayashankar, Michael Bennett, Daniel W. Belsky et al. "Resource profile and user guide of the Polygenic Index Repository." *Nature human behaviour* (2021): 1-15.

Belsky, Daniel W., Benjamin W. Domingue, Robbee Wedow, Louise Arseneault, Jason D. Boardman, Avshalom Caspi, Dalton Conley et al. "Genetic analysis of social-class mobility in five longitudinal studies." *Proceedings of the National Academy of Sciences* 115, no. 31 (2018): E7275-E7284.

Benabou, Roland. "Unequal societies: Income distribution and the social contract." *American Economic Review* 90, no. 1 (2000): 96-129.

Bergman, Peter, Raj Chetty, Stefanie DeLuca, Nathaniel Hendren, Lawrence F. Katz, and Christopher Palmer. *Creating moves to opportunity: Experimental evidence on barriers to neighborhood choice*. No. w26164. National Bureau of Economic Research, 2019.

Bertrand, Marianne, Magne Mogstad, and Jack Mountjoy. "Improving Educational Pathways to Social Mobility: Evidence from Norway's "Reform 94"." Forthcoming at *Journal of Labor Economics*.

Biroli, Pietro, Titus J. Galama, Stephanie von Hinke, Hans van Kippersluis, Cornelius A. Rietveld, and Kevin Thom. "The Economics and Econometrics of Gene-Environment Interplay." *arXiv preprint arXiv:2203.00729* (2022).

Biroli, Pietro, and Christian Lukas Zünd. "Genes, Pubs, and Drinks: Gene-environment interplay and alcohol licensing policy in the UK." (2020).

Biroli, Pietro, and Laura Zwyssig. "Moral Hazard Heterogeneity: Genes and Health Insurance Influence Smoking after a Health Shock." (2021).

Black, Sandra E., Paul J. Devereux, and Kjell G. Salvanes. "The more the merrier? The effect of family size and birth order on children's education." *The Quarterly Journal of Economics* 120, no. 2 (2005): 669-700.

Black, Sandra E., Paul J. Devereux, Petter Lundborg, and Kaveh Majlesi. "Poor little rich kids? The role of nature versus nurture in wealth and other economic outcomes and behaviours." *The Review of Economic Studies* 87, no. 4 (2020): 1683-1725.

Bowles, Samuel, and Herbert Gintis. "The inheritance of inequality." *Journal of economic Perspectives* 16, no. 3 (2002): 3-30.

Brinch, Christian N., Magne Mogstad, and Matthew Wiswall. "Beyond LATE with a discrete instrument." *Journal of Political Economy* 125, no. 4 (2017): 985-1039.

Brumpton, Ben, Eleanor Sanderson, Karl Heilbron, Fernando Pires Hartwig, Sean Harrison, Gunnhild Åberge Vie, Yoonsu Cho et al. "Avoiding dynastic, assortative mating, and population stratification biases in Mendelian randomization through within-family analyses." *Nature communications* 11, no. 1 (2020): 1-13.

Cameron, Stephen V., and James J. Heckman. "The dynamics of educational attainment for black, hispanic, and white males." *Journal of political Economy* 109, no. 3 (2001): 455-499.

Campbell, David. *Rates of return to schooling and the quality of education in England and Wales*. No. 01, 15. Department of Economics Discussion Paper, 2001.

Card, David. "Estimating the return to schooling: Progress on some persistent econometric problems." *Econometrica* 69, no. 5 (2001): 1127-1160.

Card, David, and Alan B. Krueger. "Does school quality matter? Returns to education and the characteristics of public schools in the United States." *Journal of political Economy* 100, no. 1 (1992): 1-40.

Carneiro, Pedro, James J. Heckman, and Edward J. Vytlacil. "Estimating marginal returns to education." *American Economic Review* 101, no. 6 (2011): 2754-81.

Carvalho, Leandro. "Genetics, Economic Choices, and Income." Unpublished manuscript (2022).

Case, Anne, and Christina Paxson. "Stature and status: Height, ability, and labor market outcomes." *Journal of political Economy* 116, no. 3 (2008): 499-532.

Caspi, Avshalom, Joseph McClay, Terrie E. Moffitt, Jonathan Mill, Judy Martin, Ian W. Craig, Alan Taylor, and Richie Poulton. "Role of genotype in the cycle of violence in maltreated children." *Science* 297, no. 5582 (2002): 851-854.

Caspi, Avshalom, Karen Sugden, Terrie E. Moffitt, Alan Taylor, Ian W. Craig, HonaLee Harrington, Joseph McClay et al. "Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene." *Science* 301, no. 5631 (2003): 386-389.

Chan, Tak Wing, Morag Henderson, and Rachel Stuchbury. "Family size and educational attainment in England and Wales." *Population studies* 73, no. 2 (2019): 165-178.

Cheesman, Rosa, Avina Hunjan, Jonathan RI Coleman, Yasmin Ahmadzadeh, Robert Plomin, Tom A. McAdams, Thalia C. Eley, and Gerome Breen. "Comparison of adopted and nonadopted individuals reveals gene–environment interplay for education in the UK Biobank." *Psychological science* 31, no. 5 (2020): 582-591.

Chetty, Raj, Nathaniel Hendren, and Lawrence F. Katz. "The effects of exposure to better neighborhoods on children: New evidence from the Moving to Opportunity experiment." *American Economic Review* 106, no. 4 (2016): 855-902.

Chetty, Raj, and Nathaniel Hendren. "The impacts of neighborhoods on intergenerational mobility I: Childhood exposure effects." *The Quarterly Journal of Economics* 133, no. 3 (2018a): 1107-1162.

Chetty, Raj, and Nathaniel Hendren. "The impacts of neighborhoods on intergenerational mobility II: County-level estimates." *The Quarterly Journal of Economics* 133, no. 3 (2018b): 1163-1228.

Clark, Damon, and Heather Royer. *The effect of education on adult health and mortality: Evidence from Britain*. No. w16013. National Bureau of Economic Research, 2010.

Clark, Damon, and Heather Royer. "The effect of education on adult mortality and health: Evidence from Britain." *American Economic Review* 103, no. 6 (2013): 2087-2120.

Conti, Gabriella, and James J. Heckman. "Understanding the early origins of the education–health gradient: A framework that can also be applied to analyze gene–environment interactions." *Perspectives on Psychological Science* 5, no. 5 (2010): 585-605.

Crowther, G. "The Crowther report, 15 to 18: A report of the Central Advisory Council for Education (England)." (1959).

Cunha, Flavio, James J. Heckman, Lance Lochner, and Dimitriy V. Masterov. "Interpreting the evidence on life cycle skill formation." *Handbook of the Economics of Education* 1 (2006): 697-812.

Currie, Janet, and Douglas Almond. "Human capital development before age five." In *Handbook of labor economics*, vol. 4, pp. 1315-1486. Elsevier, 2011.

D'onofrio, Brian M., Benjamin B. Lahey, Eric Turkheimer, and Paul Lichtenstein. "Critical need for family-based, quasi-experimental designs in integrating genetic and social science research." *American journal of public health* 103, no. S1 (2013): S46-S55.

Davies, Neil M., Matt Dickson, George Davey Smith, Gerard J. Van Den Berg, and Frank Windmeijer. "The causal effects of education on health outcomes in the UK Biobank." *Nature human behaviour* 2, no. 2 (2018): 117-125.

DeCicca, Philip, and Harry Krashinsky. *Do Differences in School Quality Generate Heterogeneity in the Causal Returns to Education?*. No. w27089. National Bureau of Economic Research, 2020.

Deschênes, Olivier. "Estimating the effects of family background on the return to schooling." *Journal of Business & Economic Statistics* 25, no. 3 (2007): 265-277.

Dickson, Matt, and Sarah Smith. "What determines the return to education: an extra year or a hurdle cleared?." *Economics of education review* 30, no. 6 (2011): 1167-1176.

DiPrete, Thomas A., Casper AP Burik, and Philipp D. Koellinger. "Genetic instrumental variable regression: Explaining socioeconomic and health outcomes in nonexperimental data." *Proceedings of the National Academy of Sciences* 115, no. 22 (2018): E4970-E4979.

Domingue, Benjamin W., and Jason Fletcher. "Separating measured genetic and environmental effects: Evidence linking parental genotype and adopted child outcomes." *Behavior genetics* 50, no. 5 (2020): 301-309.

Fagereng, Andreas, Magne Mogstad, and Marte Rønning. "Why do wealthy parents have wealthy children?." *Journal of Political Economy* 129, no. 3 (2021): 703-756.

Fletcher, Jason M., and Dalton Conley. "The challenge of causal inference in gene–environment interaction research: Leveraging research designs from the social sciences." *American journal of public health* 103, no. S1 (2013): S42-S45.

Fletcher, Jason, Yuchang Wu, Zijie Zhao, and Qiongshi Lu. "The production of within-family inequality: Insights and implications of integrating genetic data." *BioRxiv* (2020).

Grenet, Julien. "Is extending compulsory schooling alone enough to raise earnings? Evidence from French and British compulsory schooling laws." *The Scandinavian Journal of Economics* 115, no. 1 (2013): 176-210.

Harden, K. Paige, Benjamin W. Domingue, Daniel W. Belsky, Jason D. Boardman, Robert Crosnoe, Margherita Malanchini, Michel Nivard, Elliot M. Tucker-Drob, and Kathleen Mullan Harris. "Genetic associations with mathematics tracking and persistence in secondary school." *NPJ science of learning* 5, no. 1 (2020): 1-8.

Hause, John C. "Earnings profile: Ability and schooling." *Journal of political economy* 80, no. 3, Part 2 (1972): S108-S138.

Heckman, James J. "Policies to foster human capital." *Research in economics* 54, no. 1 (2000): 3-56.

Heckman, James J., Jora Stixrud, and Sergio Urzua. "The effects of cognitive and noncognitive abilities on labor market outcomes and social behavior." *Journal of Labor economics* 24, no. 3 (2006): 411-482.

Heckman, James J., John Eric Humphries, and Gregory Veramendi. "Returns to education: The causal effects of education on earnings, health, and smoking." *Journal of Political Economy* 126, no. S1 (2018): S197-S246.

Howe, Laurence, David Evans, Gibran Hemani, George Davey Smith, and Neil Davies. "Evaluating indirect genetic effects of siblings using singletons." (2021).

Imbens, GW, and JD Angrist. "Identification and estimation of local average treatment effects." *Econometrica* 62, no. 2 (1994): 467-475.

Jencks, Christopher. "Who gets ahead? The determinants of economic success in America." (1979).

Johnson, Rebecca A., Ramina Sotoudeh, and Dalton Conley. "Polygenic scores for plasticity: A new tool for studying gene-environment interplay." *bioRxiv* (2020).

Kong, Augustine, Gudmar Thorleifsson, Michael L. Frigge, Bjarni J. Vilhjalmsson, Alexander I. Young, Thorgeir E. Thorgeirsson, Stefania Benonisdottir et al. "The nature of nurture: Effects of parental genotypes." *Science* 359, no. 6374 (2018): 424-428.

Kong, Augustine, Stefania Benonisdottir, and Alexander I. Young. "Family analysis with Mendelian imputations." *BioRxiv*(2020).

Kowalski, Amanda E. “Reconciling seemingly contradictory results from the Oregon health insurance experiment and the Massachusetts health reform.” Forthcoming in the *Review of Economics and Statistics*.

Kweon, Hyeokmoon, Casper Burik, Richard Karlsson Linnér, Ronald De Vlaming, Aysu Okbay, Daphne Martschenko, Kathryn Harden Harden, Thomas A. DiPrete, and Philipp Koellinger. "Genetic Fortune: Winning or Losing Education, Income, and Health." (2020).

Lee, David S., and David Card. "Regression discontinuity inference with specification error." *Journal of Econometrics* 142, no. 2 (2008): 655-674.

Lee, James J., Robbee Wedow, Aysu Okbay, Edward Kong, Omeed Maghzian, Meghan Zacher, Tuan Anh Nguyen-Viet et al. "Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals." *Nature genetics* 50, no. 8 (2018): 1112-1121.

Lee, David L., Justin McCrary, Marcelo J. Moreira, and Jack Porter. "Valid t-ratio Inference for IV." *arXiv preprint arXiv:2010.05058* (2020).

Loury, Glenn C. "Intergenerational transfers and the distribution of earnings." *Econometrica: Journal of the Econometric Society* (1981): 843-867.

Martin, Alicia R., Masahiro Kanai, Yoichiro Kamatani, Yukinori Okada, Benjamin M. Neale, and Mark J. Daly. "Clinical use of current polygenic risk scores may exacerbate health disparities." *Nature genetics* 51, no. 4 (2019): 584-591.

Meghir, Costas, and Mårten Palme. "Educational reform, ability, and family background." *American Economic Review* 95, no. 1 (2005): 414-424.

Mogstad, Magne, and Matthew Wiswall. "Testing the quantity–quality model of fertility: Estimation using unrestricted family size models." *Quantitative Economics* 7, no. 1 (2016): 157-192.

Morris, Tim T., Neil M. Davies, Gibran Hemani, and George Davey Smith. "Population phenomena inflate genetic associations of complex social traits." *Science Advances* 6, no. 16 (2020): eaay0328.

Okbay, Aysu, Jonathan P. Beauchamp, Mark Alan Fontana, James J. Lee, Tune H. Pers, Cornelius A. Rietveld, Patrick Turley et al. "Genome-wide association study identifies 74 loci associated with educational attainment." *Nature* 533, no. 7604 (2016): 539-542.

Okbay, Aysu, Yeda Wu, Nancy Wang, Hariharan Jayashankar, Michael Bennett, Seyed Moeen Nehzati, Julia Sidorenko, et al. "Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals." *Nature Genetics*, *forthcoming*.

Oreopoulos, Philip. "Estimating average and local average treatment effects of education when compulsory schooling laws really matter." *American Economic Review* 96, no. 1 (2006): 152-175.

Papageorge, Nicholas W., and Kevin Thom. "Genes, education, and labor market outcomes: evidence from the health and retirement study." *Journal of the European Economic Association* 18, no. 3 (2020): 1351-1399.

Pekkarinen, Tuomas, Roope Uusitalo, and Sari Kerr. "School tracking and intergenerational income mobility: Evidence from the Finnish comprehensive school reform." *Journal of Public Economics* 93, no. 7-8 (2009): 965-973.

Persico, Nicola, Andrew Postlewaite, and Dan Silverman. "The effect of adolescent experience on labor market outcomes: The case of height." *Journal of Political Economy* 112, no. 5 (2004): 1019-1053.

Price, Alkes L., Nick J. Patterson, Robert M. Plenge, Michael E. Weinblatt, Nancy A. Shadick, and David Reich. "Principal components analysis corrects for stratification in genome-wide association studies." *Nature genetics* 38, no. 8 (2006): 904-909.

Rietveld, Cornelius A., Sarah E. Medland, Jaime Derringer, Jian Yang, Tõnu Esko, Nicolas W. Martin, Harm-Jan Westra et al. "GWAS of 126,559 individuals identifies genetic variants associated with educational attainment." *science* 340, no. 6139 (2013): 1467-1471.

Sacerdote, Bruce. "Nature and Nurture Effects on Children's Outcomes: What Have We Learned from Studies of Twins and Adoptees?." In *Handbook of social economics*, vol. 1, pp. 1-30. North-Holland, 2011.

Schmitz, Lauren, and Dalton Conley. "The long-term consequences of Vietnam-era conscription and genotype on smoking behavior and health." *Behavior genetics* 46, no. 1 (2016): 43-58.

Schmitz, Lauren, and Dalton Conley. "Modeling gene-environment interactions with quasi-natural experiments." *Journal of personality* 85, no. 1 (2017): 10-21.

Shanahan, Michael J., Stephen Vaisey, Lance D. Erickson, and Andrew Smolen. "Environmental contingencies and genetic propensities: Social capital, educational continuation, and dopamine receptor gene DRD2." *American Journal of Sociology* 114, no. S1 (2008): S260-S286.

Solon, Gary. "Intergenerational mobility in the labor market." In *Handbook of labor economics*, vol. 3, pp. 1761-1800. Elsevier, 1999.

Solon, Gary. "A model of intergenerational mobility variation over time and place." *Generational income mobility in North America and Europe* 2 (2004): 38-47.

Steelman, Lala Carr, Brian Powell, Regina Werum, and Scott Carter. "Reconsidering the effects of sibling configuration: Recent advances and challenges." *Annual review of sociology* 28, no. 1 (2002): 243-269.

Sudlow, Cathie, John Gallacher, Naomi Allen, Valerie Beral, Paul Burton, John Danesh, Paul Downey et al. "UK biobank: an open access resource for identifying the causes of a wide range of complex diseases of middle and old age." *Plos med* 12, no. 3 (2015): e1001779.

Tucker-Drob, Elliot M. "Measurement error correction of genome-wide polygenic scores in prediction samples." *bioRxiv* (2017): 165472.

Urzua, Sergio. "The effects of cognitive and noncognitive skills on racial and ethnic wage gaps." *Unpublished manuscript, Department of Economics, University of Chicago* (2006).

Vilhjálmsson, Bjarni J., Jian Yang, Hilary K. Finucane, Alexander Gusev, Sara Lindström, Stephan Ripke, Giulio Genovese et al. "Modeling linkage disequilibrium increases accuracy of polygenic risk scores." *The american journal of human genetics* 97, no. 4 (2015): 576-592.

Vytlacil, Edward. "Independence, monotonicity, and latent index models: An equivalence result." *Econometrica* 70, no. 1 (2002): 331-341.

Welland, Jon D. "Cognitive abilities, schooling and earnings: the question of functional form." *The Review of Economics and Statistics* (1978): 622-627.

Young, Alexander I., Seyed Moeen Nehzati, Chanwook Lee, Stefania Benonisdottir, David Cesarini, Daniel J. Benjamin, Patrick Turley, and Augustine Kong. "Mendelian imputation of parental genotypes for genome-wide estimation of direct and indirect genetic effects." *BioRxiv* (2020).