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Education as a Moderator of Genetic Mortality Risk*

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Abstract

In this paper we examine whether additional schooling can offset genetic disadvantages in longevity. Using data from the English Longitudinal Study of Ageing (ELSA) and polygenic scores for longevity, we study heterogeneity in the effects of education on panel attrition – a proxy that closely follows mortality for older individuals. Identification comes from the 1947 UK schooling reform, which raised the minimum school-leaving age from 14 to 15 and provides quasi-experimental variation in educational attainment. The results indicate that the reform effect varies by genetic mortality risk. There are no significant effects of an additional year of compulsory schooling on panel attrition among individuals in the group with lowest genetic mortality risk. In contrast, for the group with highest genetic mortality risk an additional year of compulsory schooling increases the probability of survival in the sample up to age 84. This suggests that the reform mitigated the genetic inequalities in longevity.

Keywords: Education, Mortality, Attrition, Gene-Environment Interactions
JEL Classification: *C31, J14, J24*

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1 Introduction

The large and persistent socioeconomic gradient in health is among the most robust empirical regularities in the social sciences, see e.g. Lleras-Muney (2005). Individuals with more education live substantially longer and experience markedly lower morbidity throughout the life cycle. Those with fewer years of schooling die earlier, suffer from earlier onset of chronic disease, and consistently occupy the lower end of the survival distribution. A longstanding literature debates whether these gradients reflect causal effects of education or arise from confounding factors correlated with both schooling and health. Regarding mortality, Clark and Royer (2013) or Meghir et al. (2018), find little evidence for a causal impact of increased compulsory schooling on mortality, whereas others document protective effects (Grytten et al., 2020; Davies et al., 2018).

People also differ in how long they live due to genetic factors: some individuals are "lucky in the genetic lottery," while others are less so. While, in earlier studies, the relationship was assumed to be even stronger, Kaplanis et al. (2018) find that heritability through genes accounts for 12-16% of variations in life expectancy. Using results from gene-wide association studies (GWAS), Ajnakina et al. (2023) find that a one–standard deviation increase in the polygenic index for longevity is associated with a 7% reduction in all-cause mortality over the subsequent ten years.

In this paper, we bring together two areas of research in examining influences on longevity: genetic disposition and education. The central question is: Can additional education compensate for genetic disadvantages? Do individuals with an unfavorable genetic endowment benefit more from longer schooling? Or is the opposite true, that those with favorable genetic predispositions benefit the most? This interaction is commonly referred to as a gene–environment interaction. While gene-environment interactions have become very popular recently in the economics literature, we are, to the best of our knowledge, the first to apply this with respect to longevity. This is the contribution of our paper to the literature. Note, that we use a proxy for mortality: survey panel attrition, because exact mortality information is only available for around 50% of our sample. We show, however, that, for older individuals aged 80 and above, panel attrition is closely related to mortality. For instance, in our subsample with mortality information, almost 60 per cent of those who dropped out of the sample in the age group 80-84 did so due to mortality. We discuss below why the 60 per cent are probably a lower bound.

Despite a rapidly growing interest in gene–environment interactions, existing work has almost entirely focused on outcomes other than panel survival/longevity. A rich literature has studied how the effects of education vary by genetic endowment for educational attainment, cognition, and health behaviors (e.g., Schmitz and Conley, 2017; Barcellos et al., 2018, 2021; Pereira et al., 2022; Barcellos et al., 2025; Hollenbach et al., 2026).

We study gene–environment interactions in panel survival up to age 84 using a well-established natural experiment: the 1947 reform in England and Wales, which raised the minimum school-leaving age from 14 to 15. Individuals born before April 1933 could leave school at the age of 14, while those born just after the cutoff were required to stay for an additional year. This institutional setting has frequently been used to identify causal effects of education (see, e.g., Clark and Royer, 2013; Harmon and Walker, 1995; Oreopoulos, 2006), and its quasi-experimental features allow us to isolate exogenous increases in schooling duration. We combine this natural experiment with genetic information from the English Longitudinal Study of Ageing (ELSA). More than 7,000 respondents of the panel have been genotyped, allowing us to use a polygenic index (PGI) that predicts genetic propensity for longevity.

The results indicate that the reform effect varies substantially by genetic predisposition for longevity. For individuals in the lowest tercile of the longevity PGI, corresponding to the least favorable genetic endowment for longevity and thus the highest genetic mortality risk, an additional year of compulsory schooling increases the probability of remaining in the sample at ages 80 and 84 by more than 30 percentage points. By contrast, the estimated effects are much smaller for individuals with more favorable genetic endowments. For individuals in the highest tercile of the longevity PGI, corresponding to the most favorable genetic endowment for longevity and thus the lowest genetic mortality risk, the effects are statistically indistinguishable from zero. Robustness checks using alternative estimation samples and control variables yield very similar patterns.

These findings suggest that education mitigates, rather than amplifies, genetic inequalities in longevity. This is in line with evidence from other gene–environment studies of education, although these studies have focused on outcomes other than mortality. Most directly, Barcellos et al. (2018) show that additional compulsory schooling reduces health differences related to genetic risk of obesity. At the same time, our findings differ from studies that document complementarities between education and favorable genetic endowments, like Hollenbach et al. (2026) for cognition as the outcome variable. This suggests that the direction of gene–environment interaction is outcome-specific. Taken together, the paper contributes to the literature by showing that gene–environment interactions are not only relevant for educational attainment, cognition, socioeconomic status, or intermediate health outcomes, but also for panel attrition as a proxy for survival at very old ages.

The remainder of the paper proceeds as follows. Section 2 describes the institutional background and the 1947 schooling reform. Section 3 presents the data sources and variable construction. Section 4 outlines the empirical strategy and discusses the empirical setup as well as the main results. Section 5 concludes.

2 Institutional Setting

We draw on exogenous variation created by a change in compulsory schooling legislation in the UK.[1] The 1944 Education Act laid the foundation for two subsequent increases in the minimum school-leaving age in England, Scotland, and Wales. Our empirical strategy relies on the first of these reforms, implemented on April 1, 1947.[2] This reform raised the legal school-leaving age from 14 to 15. Since pupils in the UK usually began full-time schooling at age 5, the reform effectively extended compulsory schooling from nine to ten years. The first birth cohorts legally obliged to attend school for an extra year, often referred to as the "pivotal cohort", were those born in April 1933.

This 1947 reform has served widely as a source of plausibly exogenous variation in education in studies examining its effects on wages (Clark and Royer, 2013; Devereux and Hart, 2010; Harmon and Walker, 1995; Oreopoulos, 2006), labour market outcomes (Clark, 2023), health (Clark and Royer, 2013; Jürges et al., 2013; Powdthavee, 2010; Silles, 2009), health literacy (Johnston et al., 2015), mortality (Clark and Royer, 2013; Gathmann et al., 2015), and cognitive performance (Banks and Mazzonna, 2012; Hollenbach et al., 2026).

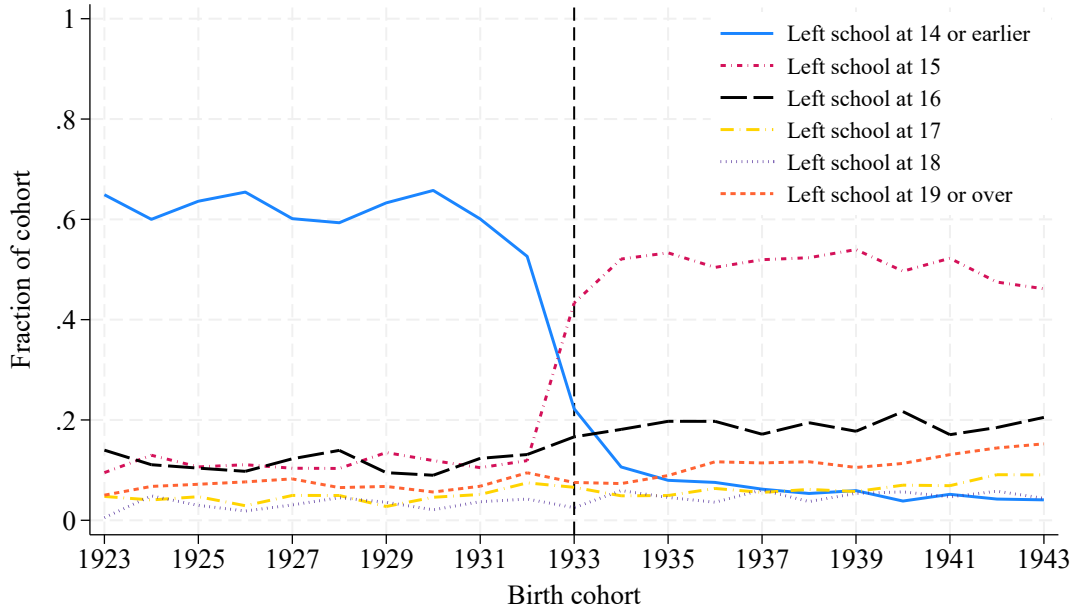
To illustrate the substantial behavioral response to the 1947 reform, Figure 1 plots aggregated cohort-level schooling information from ELSA. It shows the share of people leaving school at different ages (from 14 or younger, at 15 years old, at 16 years old, at 17 years old, at 18 years old, and at 19 or older) varied by each year of birth. The vertical dashed lines indicate the first affected birth cohort of the year 1933. A large impact on schooling leaving behavior could be witnessed. Particularly, the proportion leaving school at age 14 or earlier decreased by over 50 percentage points moving from the 1932 cohort to the 1934 cohort, whereas the fractions leaving school at 16, 17, 18, and 19 or older witness little or no changes at all. This is aligned with descriptions in prior literature (Clark and Royer, 2013; Devereux and Hart, 2010). After the reform, the proportions leaving school at 14 years old or younger remained very low, in return, those who left school at 15 took up a large proportion of each cohort. This trend remains stable for all post-reform cohorts. Furthermore, the spill-over effect of the 1947 reform to longer duration of education, i.e. leaving school at 16 or later, is suggested to be low, as a very little increase in the proportion of people who left school by categories of 16 and 17; for 18, 19 or later, the trend remained fundamentally intact.

Although compliance with the new minimum school-leaving age was extremely high, Figure 1 also reveals some noncompliance. A small number of individuals still report

[1] This section builds in large part on the institutional background described in Hollenbach et al. (2026).

[2] The second increase, enacted in 1972, raised the minimum school-leaving age to 16. Because our analysis focuses on mortality-related outcomes at older ages, only the 1947 reform is usable. Individuals affected by the 1972 reform are—in the period covered by the English Longitudinal Study of Ageing, our data source—still too young for meaningful analysis.

Figure 1: Education by birth cohort



Notes: This figure depicts the school-leaving ages by birth cohorts. The percentage of each cohort that finished their full-time education at age 14 or earlier, at 15, 16, 17, 18 or at 19 or older are reported. The vertical dashed lines indicate the first affected birth cohort of the year 1933. Sample includes all individuals born between 1923 and 1943 in ELSA: wave 0 – 11, without restriction for the availability of individual's genetic data.

leaving school at age 14 even after the reform. As discussed in Clark and Royer (2013), who study these reforms in detail, this is largely attributable to those born in the summer months who turned 15 before the start of the following school year and therefore could legally leave earlier despite the new regulation.

3 Data

3.1 Basic sample selection

For the analysis, we use data from the English Longitudinal Study of Ageing (ELSA). ELSA is an extensive longitudinal panel dataset in England, collecting detailed information on wellbeing and various socioeconomic circumstances among individuals aged 50 and older (Banks et al., 2025). Biennial interviews have been being conducted since 2002, with efforts made to follow up the same set of respondents over time. Currently, eleven waves of the study have been published. The core sample of ELSA was initially taken from the respondents of the Health Survey for England (HSE) in 1998, 1999 or 2001, which corresponds to "Wave 0" in ELSA. We use data from all available waves of ELSA, that is, waves 0 to 11.

We restrict the sample to individuals born within 7 years before and after the pivotal cohort of 1933, covering cohorts born from 1926 to 1940. The 1933 cohort is excluded because

birth-month information is unavailable to ensure the privacy confidentiality, making it impossible to accurately determine whether individuals born in that year fall into the pre- or post-reform group.

The sample is further limited to individuals who were genotyped and for whom genetic data are available in ELSA. Hollenbach et al. (2026) show that being genotyped is not correlated with reform exposure. The final sample consists of 2,360 individuals. Since the last ELSA wave is from 2024, the youngest birth cohort in our sample, born in 1940, can, in principle, be observed until the age of 84. Hence, in order to have all birth cohorts fully covered in the analysis, we study panel survival up to the age of 84.

3.2 Outcome variable: Attrition as a proxy for mortality

Unfortunately, exact mortality information is available in ELSA only for waves 0 to 6. As wave 6 was sampled in year 2012 and 2013, this means that birth cohort 1940, the youngest in our analysis is only 72 or 73 years old by then. That is, we could only estimate gene-environment effects of education on mortality up to age 73. Given that Clark and Royer (2013) find no effects of the reform on mortality up to age 69 this does not seem to be a promising avenue. If any, mortality effects should be detectable at older ages. Thus, we use two different main outcome variables, a binary indicator of *panel survival up to 80* and a binary indicator of *panel survival up to 84*. After explaining how the variables are generated, we show that, in ELSA waves 0 – 6, for individuals older than 80, panel attrition is mostly due to mortality, making it a useful proxy.

The definition of the outcome variables can best be described using the example shown in Table 1. The table shows three example individuals. Person 1 was last observed in wave 8 at age 84. Therefore, we know that this person was still in the sample at ages 80 and 84. For this individual, both outcome variables, therefore, take on the value 1. Person 2 was also last observed in wave 8, at age 82. Therefore, the outcome variable at age 80 takes on the value 1, and at age 84 it takes on the value 0. Person 3 was last observed in wave 11, the final available ELSA wave. At that time, the person was 77 years old, and we have no information about whether they remained in the sample at age 80 and 84. The outcome variable is right-censored and, therefore, takes on the value missing, which means such individual is not included in our sample.

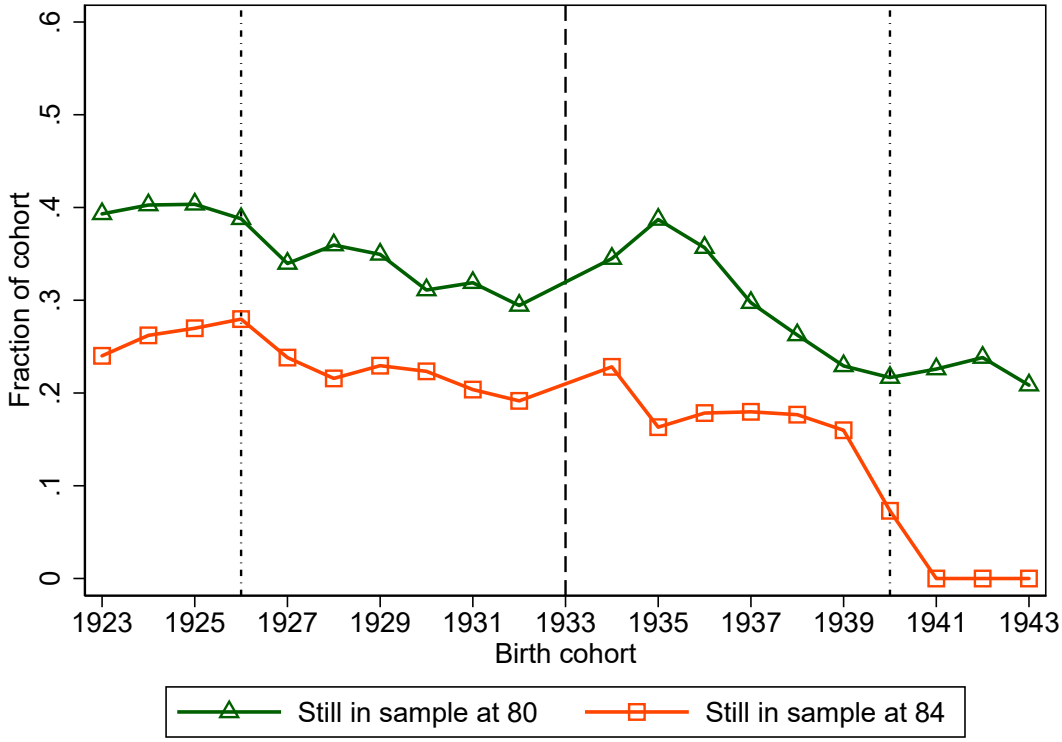
Figure 2 shows sample means of the outcome variable by year of birth. We observe a downward pattern in which the probability of being in the panel at age 80 is highest for the 1923 birth cohort and lowest for the 1943 cohort. This pattern is driven by the construction of the sample: younger cohorts were younger at the time they were sampled. The probability of being in the panel at age 84 is zero for individuals born after 1940, as those who were born in 1940 had only reached age 83 or 84 by the latest wave in 2024.

Table 1: Definition of the outcome variables

Individual	Last observed in wave	Age last observed	In sample at 80	In sample at 84
1	8 (2017)	84	1	1
2	8 (2017)	82	1	0
3	11 (2024)	77	.	.

Notes: Own artificial example for illustration purposes.

Figure 2: Sample development



Notes: This graph shows the sample development for the outcome variables of interest. It depicts fraction of individuals who remained in the original sample at the age of 80 and 84 by birth cohort. The vertical dashed line marks the first affected birth cohort (1933). The vertical dash-dotted lines mark the bandwidth for our analysis, where the sample is more balanced. ELSA: wave 0 – 11, without restriction for the availability of individual's genetic data.

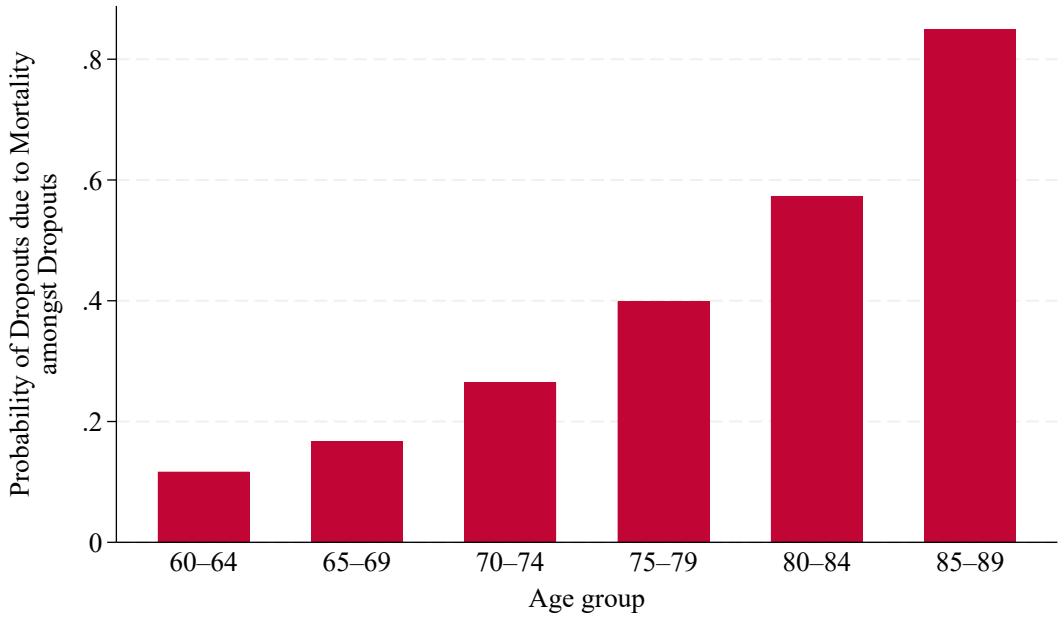
Since later, in the regression analysis, we use a regression discontinuity design and control for cohort trends on both sides of the reform cutoff – thus focusing on the discontinuity in 1933 – this pattern does not pose a problem for the econometric design. After generating the outcome variables, each individual is only used once in the analysis, that is, the 2,360 observations represent different unique individuals.

The relationship between panel attrition and mortality

Next, we assess whether panel survival can serve as a meaningful proxy for general survival. To do so, we draw on the exact mortality information available in ELSA for waves 0 to 6 and examine how often panel exit occurs because a respondent is verified

as deceased. Using only cases when the ELSA group was able to verify if a dropout was alive or dead, we get the results shown in Figure 3. Among individuals who dropped out of the sample at ages 70 - 74, 25 percent did so due to verified mortality. Note that, the overall dropout rate at that age group is about 10 percent, so that absolute numbers of mortality are low, as expected. The relationship between panel attrition and mortality gets closer, the older individuals get. It reaches around 60 percent in the age group 80 - 84 and exceeds 80 percent among individuals aged 85 - 89, indicating that mortality becomes the predominant reason for dropout at very old ages. The link between attrition and mortality is therefore particularly pronounced among the oldest individuals.

Figure 3: Share of attrition due to mortality by age group



Notes: This figure shows the proportion of mortality-related dropouts relative to the total number of dropouts within each age group. Dropout is coded as one if an individual does not appear in the subsequent wave. Dropout due to mortality is coded as one when a year of death is recorded in the corresponding wave. Sample includes all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA (Harmonized ELSA): wave 0 – 6, where the mortality status is available, without restriction for the availability of individual's genetic data.

Although panel attrition occurs for several reasons, the majority of attrition in ELSA among older individuals seems to be mortality-related, which is why we believe that analyses based on panel attrition can reasonably be used to infer general patterns of mortality at the very old ages. Given that many dropouts among alive persons in the age group 80-84 are also health related, we assume – but this is necessarily speculative – that many of the remaining 40% alive dropouts are also at high risk of mortality. Thus, the 60% may be considered a lower bound.

3.3 Genetic data

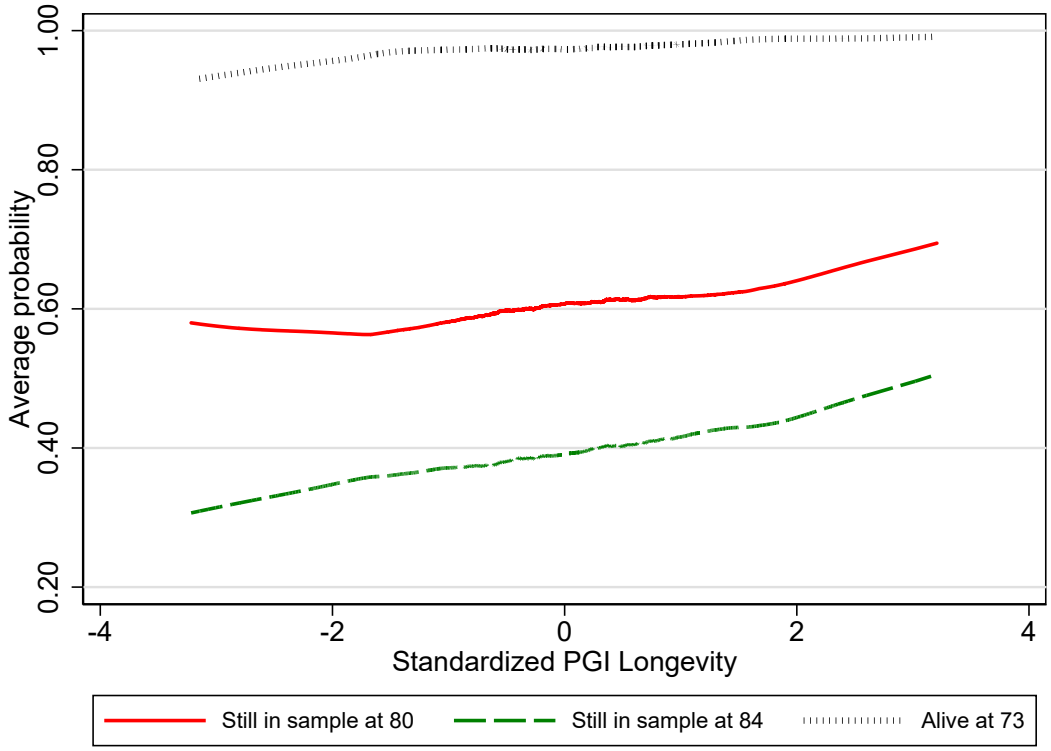
To capture genetic information, we draw on a Polygenic Index (PGI) for longevity provided in ELSA. The score is constructed based on the GWAS meta-analysis by Deelen et al. (2019), which at the time represented the most recent and comprehensive genetic study of the longevity phenotype. Their analysis defines longevity in an "extreme" way: individuals are classified as long-lived if they survive up to the age corresponding to the 90th or 99th percentile of the survival distribution. In contrast, controls are those whose age at death or age at last follow-up falls at or below the 60th survival percentile. These survival thresholds are not fixed chronological ages but are derived from cohort-, sex-, and country-specific life tables. This approach avoids bias that may arise from heterogeneity in mortality patterns across demographic groups (Deelen et al., 2019).

A recent study by Ajnakina et al. (2023), which uses the same ELSA data and applies a similar method to construct the longevity PGI, shows that a one–standard deviation increase in the longevity PGI is associated with a 7% reduction in all-cause mortality over the subsequent ten years. In cause-specific analyses, the estimated reduction is even larger, around 19%. The indicator can be interpreted as a genome-wide measure of genetic mortality risk. A single individual's PGI aggregates their propensity across thousands of variants, rather than reflecting any single marker. A more detailed discussion of the construction and interpretation of PGIs is provided in Appendix A. The distribution of the longevity PGI is approximately normal (NatCen Social Research, 2022). Individuals positioned on the lower tail exhibit, on average, a lower genetic endowment for longevity, whereas those on the upper tail display a higher genetic endowment.

Figure 4 shows the bivariate relationship between the longevity PGI and our outcome variables using local polynomial regression to smooth. We use the standardized longevity PGI, that is, PGI minus the sample mean divided by the sample standard deviation. Generally, we observe a positive relationship between the two: individuals with a genetic position indicating higher life expectancy also have a higher probability of remaining in the sample.

This holds for age 80, where – roughly speaking – the difference between the worst and the best genetic disposition is about 10 percentage points. The red-dotted line depicts the mean of the binary outcome variable *still in sample at age 80*, which varies between roughly 60 and 70 percent. Thus, the relationship is clearly present, but not extremely large. The difference between the worst and the best genetic disposition for age 84, showed in the green long-dashed line, is larger, at around 20 percentage points. For comparison, the figure also shows, in the yellow dash-dotted line, the average probability of being alive at age 73. Here, too, we observe a positive relationship, which is quantitatively somewhat smaller at around 5 percentage points. This is likely because individuals are younger at that age and genetically driven longevity differences tend to manifest themselves more

Figure 4: Bivariate relationship between outcome variables and PGI longevity



Notes: This figure depicts how the average probability of the outcome variables varies with the standardized PGI longevity. The red solid line, and the green dashed line represent the probability of still being observed in the sample at age 80 and at 84 respectively, while the black dotted line represents the probability of being alive at age 73. The sample is our main sample selection, including all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA: wave 0 – 11. The outcome variable of being alive at age 73 is generated based on real mortality data provided by ELSA available up to wave 6.

strongly at later ages, exceeding the average life expectancy (Ajnakina et al. (2023); van den Berg et al. (2019)).

Overall, we find a positive association between genetic predisposition and actual survival probability, as well as the panel survival probabilities. Once again, this can be interpreted as evidence that our panel-attrition measure is a usable proxy for mortality.

In this paper, we focus on a nonlinear specification of the PGI for longevity by using terciles, as done in Barcellos et al. (2018). Specifically, we divide the distribution of the longevity PGI in our main sample into three equal groups: the lowest, middle, and highest terciles. The lowest tercile represents individuals with the least favorable genetic endowment for longevity, while the highest tercile represents those with the most favorable endowment.

3.4 Further variables and descriptive statistics

Regarding education, ELSA does not report respondents' total years of schooling. Instead, it records the age at which individuals finished continuous full-time education. These values are top- and bottom-coded, with ages 14 or below grouped together and ages 19

or above combined. We therefore construct the treatment variable E_i as an indicator that takes the value one for respondents who remained in school until at least age 15, and zero otherwise. As shown in Figure 1, the share of individuals who left school at age 15 or later – corresponding to at least ten years of schooling – changes sharply with the 1947 reform that increased the compulsory school-leaving age from 14 to 15.

Because this measure is based on retrospective reporting, concerns about recall error naturally arise, particularly in samples of older adults. Nonetheless, recalling the age of school completion – especially in the historical context of the immediate post-World War II period – may be easier than recalling total years of education. Furthermore, Figure 1, along with our regression results, aligns closely with the findings of Clark and Royer (2013), who rely on data collected between 1991 and 2004, well before our observation window. This consistency suggests that recall bias in the education measure is likely limited.

In the regression analysis, in addition to the PGI, we include the first ten principal components (PCs) of the genetic data as controls. These PCs condense genome-wide variation into a small number of orthogonal dimensions and adjust for population stratification – that is, systematic differences in allele frequencies across ancestral subgroups that might otherwise induce spurious associations. Such confounding can arise when both a certain genotype and a given outcome are disproportionately represented within the same population subgroup (Barth et al., 2022). Following Price et al. (2006), who show that PCs effectively mitigate this issue, the inclusion of PCs has become standard practice in gene–environment studies (see, e.g., Barcellos et al., 2018; Barth et al., 2020, 2022; Biroli et al., 2025; Pereira et al., 2022). Although both PGIs and PCs summarize genetic variation, they serve different purposes: PCs capture broad population structure, whereas PGIs are designed to predict specific outcomes such as longevity.

Although determined at conception, an individual's genetic endowment is not entirely exogenous with respect to later-life outcomes. Genetic traits are inherited from parents, whose own genotypes shape the home and social environment in which children grow up. We also include parental longevity as an additional control. ELSA contains data on the ages at death of respondents' parents. Based on this information, we construct a three-category measure of parental longevity: one category capturing cases in which both parents died before age 80, having short longevity; one category indicating that at least one parent survived at or beyond age 80, and a third category for observations with missing information on one or both parents.

Table 2 shows descriptive statistics of our sample, together with descriptive statistics for the "treatment" ($E_i = 1$) and "control" ($E_i = 0$) groups separately. Generally, almost 75 percent of the observations in the sample are in the treatment group, or in other words, leaving school at the age of 15 or older; 60 percent were born in 1933 or later, and 47 percent are male. Taking a look into the treatment and control group, a large percentage of

those who left school at the age of 15 or older were born in 1933 or later, at around almost 80 percent. This shows that there might be a strong correlation between the instrument and the treatment variable.

As could be inferred from Figure 4, the probability of being in the sample at age 80 is 60 percent, and it drops to 39 percent for being in the sample after age 84. The probability of being in the sample is, interestingly, higher in the control group with less education ($E = 0$) than in the group with more education ($E = 1$). However, this reflects the sample-composition artifact described above and also shown in Figure 2. This issue will later be addressed in the regression by controlling for birth cohorts.

4 Empirical Analysis

4.1 Empirical Strategy

We run the following regressions:

$$Y_i = \sum_{g=1}^{3} \alpha_{g,0} \mathbb{1}[G_i = g] + \alpha_{1,1} E_i + \sum_{g=2}^{3} \alpha_{g,1} \mathbb{1}[G_i = g] \times E_i + \gamma_1 \widetilde{bc}_i + \gamma_2 \widetilde{bc}_i \times Z_i \tag{1}$$
$$+ \sum_{g=1}^{3} \left[\gamma_{g,3} \widetilde{bc}_i \times \mathbb{1}[G_i = g] + \gamma_{g,4} \widetilde{bc}_i \times Z_i \times \mathbb{1}[G_i = g] \right] + \alpha X' + \varepsilon_i$$

where Y_i are the outcome variables of individual i, which are the status of being present in the sample at the age of 80/84. E_i is a dummy variable for educational attainment, taking on the value 1 if an individual has left school at 15 or later. The genetic predisposition is presented by tercile indicators for the PGI ($G_i = g$ with $g \in \{1, 2, 3\}$ where 1 corresponds to the lowest tercile, 2 to the middle tercile, and 3 to the highest tercile). The gene-environment interplay $G \times E$ is captured by interacting E_i with the full set of indicators for the three terciles of the PGI, so that $\alpha_{1,1}$ shows the effect for the lowest tercile of the PGI, while $\alpha_{g,1}$ with $g \in \{2, 3\}$ show the average additional effects for these groups relative to that of the lowest tercile. We include the linear birth cohort, centered around the year 1933, that is $\widetilde{bc}_i$ = birth cohort - 1933. Z_i is the indicator of being born after 1933. Additionally, we allow the effect of the cohort trends and its interaction with Z_i to vary accross different groups of the PGI to increase the flexibility, by interacting them further with the tercile indicators. Vector X' includes a set of control variables, namely gender, parental longevity, as well as the standardized values of the first ten principal components of the genetic data. Finally, ε_i denotes the error term.

Table 2: Descriptive statistics

	Main sample	By E_i		
	Mean (SD)	E_i=1	E_i=0	Difference (SE)
Outcome Y_i				
Still in sample at 80	0.60 (0.49)	0.59	0.65	0.07 (0.02)**
Still in sample at 84	0.39 (0.49)	0.38	0.44	0.06 (0.02)**
Treatment E_i				
Left school ≥ 15	0.74 (0.44)	1.00	0.00	−1.00 (0.00)
Instrument Z_i				
Born from 1933 onwards	0.60 (0.49)	0.78	0.12	−0.66 (0.02)***
Polygenic index G_i				
1st PGI tertile	0.33 (0.47)	0.33	0.33	0.00 (0.02)
2nd PGI tertile	0.33 (0.47)	0.33	0.35	0.03 (0.02)
3rd PGI tertile	0.33 (0.47)	0.34	0.31	−0.03 (0.02)
Selected Controls				
Male	0.47 (0.50)	0.46	0.49	0.02 (0.02)
Birth year	1933.94 (4.27)	1935.29	1930.17	−5.12 (0.17)***
Parental longevity:				
Both had age at death < 80	0.35 (0.48)	0.34	0.38	0.05 (0.02)*
At least one had age at death ≥ 80	0.60 (0.49)	0.62	0.56	−0.07 (0.02)**
Missings	0.05 (0.21)	0.04	0.06	0.02 (0.01)*
Principal components (standardized):				
-1-	0.00 (1.00)	0.01	−0.02	−0.03 (0.05)
-2-	0.00 (1.00)	0.00	−0.01	−0.02 (0.05)
-3-	0.00 (1.00)	0.00	0.00	0.00 (0.05)
-4-	0.00 (1.00)	0.01	−0.02	−0.02 (0.05)
-5-	0.00 (1.00)	0.01	−0.03	−0.04 (0.05)
-6-	0.00 (1.00)	0.01	−0.03	−0.03 (0.05)
-7-	0.00 (1.00)	0.02	−0.04	−0.06 (0.05)
-8-	0.00 (1.00)	−0.02	0.04	0.06 (0.05)
-9-	0.00 (1.00)	0.01	−0.03	−0.04 (0.05)
-10-	0.00 (1.00)	0.01	−0.03	−0.03 (0.05)
Observations	2,360	1,737	623	

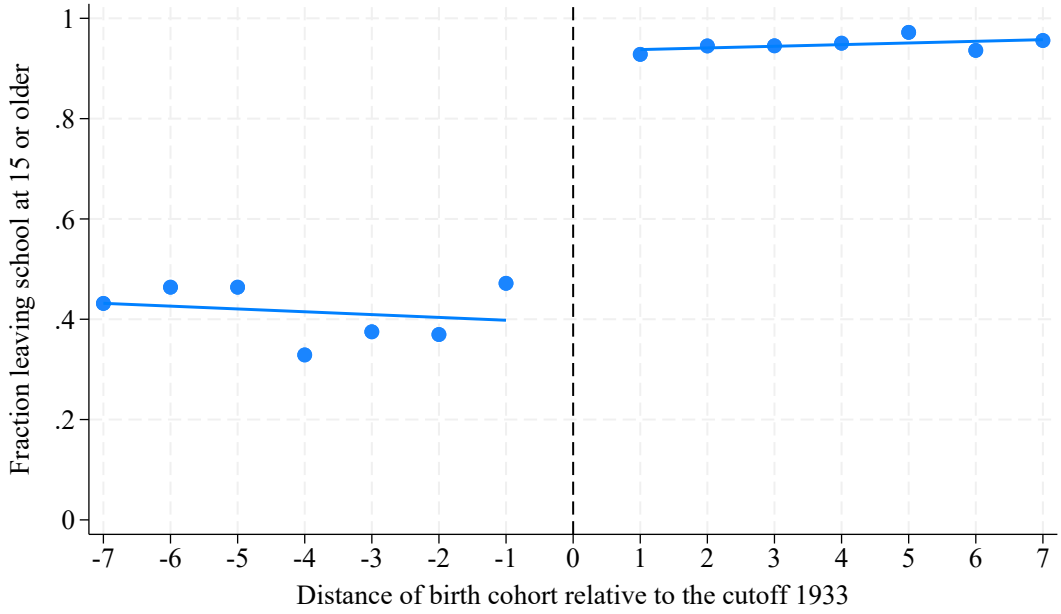
Notes: This table shows descriptive statistics of our main sample selection as well as the control and treatment group by E_i separately. All variables are included. The mean and standard deviation are included, together with the mean difference between control and treatment group and their standard errors through a t-test. The sample is our main sample selection, including all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA: wave 0 – 11. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Instrumenting E_i and the gene-environment interactions ($\mathbb{1}[G_i = g] \times E_i$) in Equation (1) with Z_i and Z_i interacted with terciles indicators of PGI ($\mathbb{1}[G_i = g] \times Z_i$) in a two stage least squares regression essentially gives us a fuzzy regression discontinuity design with birth cohorts as the running variable and the pivotal cohort of 1933 as the cutoff.

4.2 Graphical evidence of first stage and reduced form

Figure 5 illustrates the impact of the 1947 compulsory schooling reform in the UK on individual educational attainment where each dot represents the fraction of leaving school at age 15 or later (that is $E = 1$) for each year-of-birth cohort. A remarkable discontinuity at the cutoff is visible here, repeating the result shown in Figure 1. The reform increased the share of people completing their formal education at the age of 15 or older from only over 40 percent of 1932 cohort to more than 90 percent in 1934 cohort. The fraction of people leaving school at 15 or later remained stable at over 90 percent for every post-reform cohort. Overall, there was an increase of around 50 percentage points in the proportion of people finishing their full-time education at 15 or later in post-reform cohort compared to in pre-reform cohort. This suggests that the compliance with this regulation was high, meaning, a strong first stage in the regression.

Figure 5: Education by birth cohort

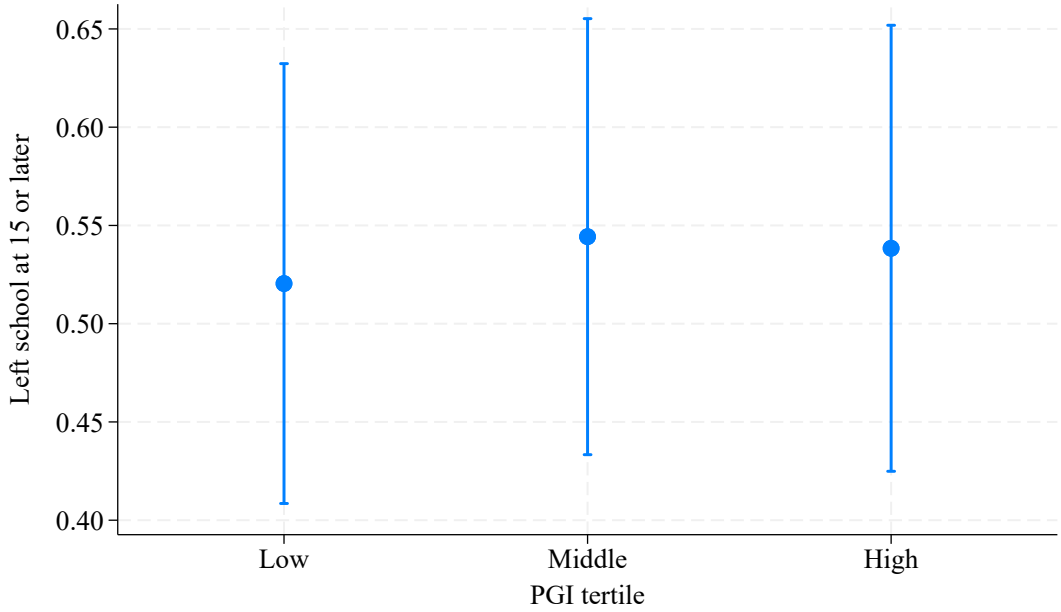


Notes: This graph shows percentage of leaving school at 15 or older by re-centered birth cohort. Each dot represents the mean of each year-of-birth cohort. The horizontal axis represents the running variable, or the re-centered birth years relative to the cutoff of 1933. Distance of Birth Cohort Relative to the Cut-Off = Birth Year - 1933. The vertical dashed line marks the cutoff of 1933. The fitted lines are included to show the trends in pre-reform and post-reform cohorts. The sample is our main sample selection, including all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA: wave 0 – 11, excluded the year 1933.

Figure 6 reports first stage coefficients by terciles of the PGI in order to address a concern regarding IV estimations of gene–environment interactions discussed in Hollenbach et al. (2026). IV estimates of gene–environment interactions may be biased when complier groups differ systematically across the genetic distribution, such that the instrument shifts different subpopulations into treatment at different values of the PGI. When this is coupled with treatment effects that exhibit essential heterogeneity, meaning that the magnitude of the causal effect depends on unobserved factors that also shape the likelihood of taking

the treatment, the 2SLS estimator confounds true interaction effects with changes in the underlying complier population.

Figure 6: First stage effect by terciles of longevity PGI



Notes: This figure illustrates the heterogeneity of first stage results regarding the terciles PGI longevity. Each point represents the mean probability of staying in school longer (until 15 years old or later) for individuals in post-reform cohorts in a given PGI tercile, with vertical lines indicating confidence intervals. The sample is our main sample selection, including all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA: wave 0 – 11, excluded the year 1933.

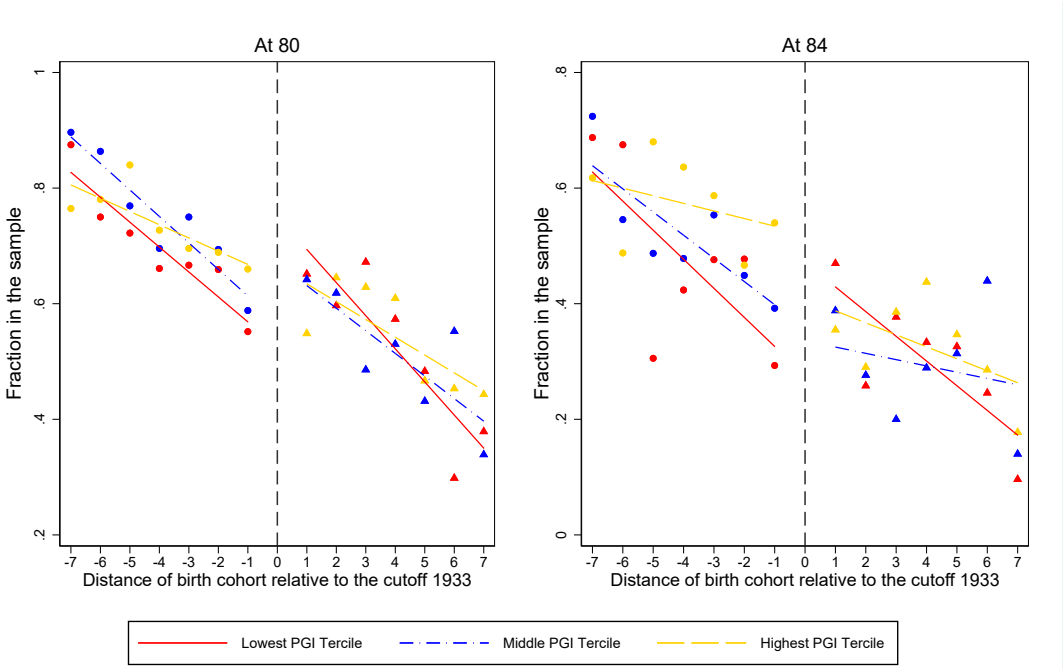
To assess whether this issue is relevant in our setting, we inspect whether the first stage differs across groups defined by the longevity polygenic score. Figure 6 shows the corresponding first-stage estimates for terciles of the longevity polygenic score. The estimated effects of the reform on the probability of staying in school until at least age 15 are very similar across all three genetic groups: approximately 0.52 in the lowest tercile, 0.55 in the middle tercile and 0.54 in the highest tercile. Unlike the substantial gradients documented in Hollenbach et al. (2026) for an education PGI, there is here no meaningful variation in complier shares across genetic strata. Consequently, the identification problem highlighted by Hollenbach et al. (2026) does not seem to present in our context. We therefore proceed with standard 2SLS estimation, as the first stage is sufficiently stable across polygenic-score terciles and does not generate variation in complier composition that could contaminate the estimated $G \times E$ interaction.

Figure 7 provides an impression of the "reduced form" of the reform effect. It shows how the schooling reform measured at the 1933 cutoff affects the probability of being present in the sample at later ages. Separately by terciles of the longevity PGI, sample means of the outcome *still in the sample at age 80* and *age 84* are plotted against the relative birth year (birth year - 1933), together with linear fits on both sides of the cutoff. The overall downward slopes seen in all lines reflect the specific sample construction described above

in 3.2. However, the key question is whether there is a discontinuity at the cutoff since cohort effects are accounted for in the regression design.

Three findings are noteworthy and transparently already foreshadow the IV regression results shown below. First, the red lines show a remarkable upward shift in sample survival among individuals in the lowest PGI tercile implying a strong effect of the compulsory schooling reform among those with the least favorable genetic endowments for longevity. In contrast, there is a smaller jump for the middle tercile and no (age 80) or even a downward jump (age 84) in the highest terciles.

Figure 7: Reduced form by terciles of longevity PGI: In sample at 80/84



Notes: This graph shows effect of the 1947 schooling reform on the probability of still being present in the sample at age 80/84 by longevity PGI terciles. Each point represents the sample mean for a given longevity PGI tercile in each birth cohort. The lowest tercile is shown in red, the middle tercile in blue, and the highest tercile in yellow. The vertical dashed lines mark the cutoff of 1933. The fitted lines are included to show the trends in pre-reform and post-reform cohorts for each group. Distance of Birth Cohort Relative to the Cut-Off = Birth Year - 1933. The sample is our main sample selection, including all individuals born between 1926 and 1940 (bandwidth of 7 years before and after the cutoff of 1933) in ELSA: wave 0 – 11, excluded the year 1933.

4.3 Regression results

In Table 3, we present benchmark results based on OLS estimations from Equation (1). The coefficient of E_i points at a significant positive correlation of education with the probability at age 80 and at age 84 for the reference group, that is the lowest longevity PGI tercile. Looking at the gene–environment interactions, the mark-ups relative to the lowest PGI tercile for individuals in the higher terciles are negative but without a clear pattern accross outcome variables.

Table 3: OLS

	(1) Still in sample at 80		(2) Still in sample at 84	
E_i	0.170	(0.046)***	0.144	(0.045)***
$G_i = 1$	reference category		reference category	
$G_i = 2$	0.066	(0.067)	−0.029	(0.067)
$G_i = 3$	0.044	(0.072)	0.154	(0.073)**
$E_i \times (G_i = 1)$	reference category		reference category	
$E_i \times (G_i = 2)$	−0.115	(0.061)*	−0.007	(0.062)
$E_i \times (G_i = 3)$	−0.065	(0.063)	−0.123	(0.065)*
Controls	Yes		Yes	
Observations	2,360		2,318	

Notes: This table shows OLS estimates of Equation (1) where E_i is remaining in school until at least age 15 with non-linear G_i as terciles built from the PGI for longevity ($G_i = g$ with $g \in \{1, 2, 3\}$ where 1 corresponds to the lowest tercile, 2 to the middle tercile, and 3 to the highest tercile). Robust standard errors shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

In Table 4, we apply the same approach to the IV/RDD specification. The overall pattern is consistent accross both outcome variables.

First, and as expected, we observed a clear gradient in mortality by genetic endowment for longevity. Individuals in the highest tercile have a by up to 45 percentage points higher likelihood to be in the sample at age 84 than individuals in the lowest tercile, see the coefficient of $G_i = 3$.

However, this difference is completely equalized by different effects of education. We observe a positive effect of education for individuals in the lowest PGI longevity tercile, see the coefficient of E_i. The effect is substantial, amounting to roughly 37 and 32 percentage points, on average, respectively.

The effect of education on panel survival for the middle tercile is 0.373 - 0.145 = 0.228 and 0.316 - 0.359 = - 0.043. Even clearer, the effect for the highest tercile is 0 regarding sample survival until 80 and even negative regarding sample survival until 84. Both are statistically insignificant, however, as confirmed by post-estimation test. Taken together, and this is the main finding of this paper, these results suggest that the schooling reform reduced inequalities associated with genetic endowments for longevity.

In Table 5, we report the robustness checks, changing the specification in four dimensions. First and second, we run the same IV version model in Equation 1 but use a wider bandwidth of 8 years and a narrower bandwidth of 6 years before and after the cutoff respectively, instead of 7 years like in the main analysis. Third, we run the main analysis excluding the ten principal components as control variables. And finally, we use the

Table 4: 2SLS Regression results

	(1) Still in sample at 80		(2) Still in sample at 84	
E_i	0.373	(0.145)**	0.316	(0.144)**
$G_i = 1$	reference category		reference category	
$G_i = 2$	0.086	(0.148)	0.213	(0.152)
$G_i = 3$	0.245	(0.155)	0.451	(0.160)***
$E_i \times (G_i = 1)$	reference category		reference category	
$E_i \times (G_i = 2)$	−0.145	(0.200)	−0.359	(0.204)*
$E_i \times (G_i = 3)$	−0.348	(0.202)*	−0.534	(0.205)***
Controls	Yes		Yes	
Observations	2,360		2,318	

Notes: This table shows 2SLS estimates of Equation (1) where E_i is remaining in school until at least age 15 (instrumented with Z_i) with G_i as terciles built from the PGI for longevity ($G_i = g$ with $g \in \{1, 2, 3\}$ where 1 corresponds to the lowest tercile, 2 to the middle tercile, and 3 to the highest tercile). Robust standard errors shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Table 5: Robustness checks

	(1) Baseline	(2) Bandwidth 8	(3) Bandwidth 6	(4) Without principal components	(5) Quadratic cohort trends
E_i	0.373**	0.340**	0.363**	0.368**	0.296
	(0.145)	(0.135)	(0.157)	(0.144)	(0.235)
$G_i = 1$			Reference category		
$G_i = 2$	0.086	0.069	0.111	0.088	0.092
	(0.148)	(0.142)	(0.165)	(0.148)	(0.141)
$G_i = 3$	0.245	0.216	0.187	0.236	0.261*
	(0.155)	(0.141)	(0.166)	(0.154)	(0.146)
$E_i \times (G_i = 1)$			Reference category		
$E_i \times (G_i = 2)$	–0.145	–0.102	–0.234	–0.146	–0.135
	(0.200)	(0.191)	(0.223)	(0.200)	(0.198)
$E_i \times (G_i = 3)$	–0.348*	–0.345*	–0.286	–0.334*	–0.351*
	(0.202)	(0.186)	(0.218)	(0.201)	(0.200)
Observations	2,360	2,687	2,061	2,360	2,360

Notes: This table reports robustness checks using the outcome *Still in sample at 80* for the gene-environment interactions from different specifications, using non-linear $G \times E$ effect with G_i as terciles built from the PGI for longevity ($G_i = g$ with $g \in \{1, 2, 3\}$ where 1 corresponds to the lowest tercile, 2 to the middle tercile, and 3 to the highest tercile). The main results from Table 4 are reported in column (1) for reference. The subsequent columns present four alternative specifications: estimating the main model in Equation 1 using a wider bandwidth of ±8 years around the 1933 cutoff (2), using a narrower bandwidth of ±6 years around the 1933 cutoff (3), excluding all principal components (4), and using quadratic cohort trends instead of linear cohort trends (5). Robust standard errors shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

quadratic cohort trends instead of the linear trends to increase the flexibility. We provide the main baseline results from Table 4 for reference.

For sake of brevity, only the results for the outcome *Still in sample at 80* are reported. The robustness checks for the other outcome variable yield similar patterns and point in the same direction. They are reported in the Appendix. Overall, the results remain stable across different specifications. The estimated reform effects for the lowest tercile are consistently positive and statistically significant, amounting to around 34 to 36 percentage points, which is comparable to the baseline model. Importantly, the interaction effects are also consistent. Individuals in the highest PGI longevity tercile experience a significantly smaller effect of an additional year of schooling on the probability of remaining in the sample at age 80 compared to those in the lowest tercile. The only exception occurs in the specification using a narrower bandwidth of ±6 years around the cutoff, where the interaction term becomes statistically insignificant. Despite a relatively large estimate, the smaller sample size in this specification leads to larger standard errors and reduces statistical significance. All in all, these results support the interpretation that additional compulsory schooling has a compensatory effect for individuals with the least favorable genetic endowments for longevity.

5 Conclusion

This paper examines whether additional compulsory schooling can offset genetic disadvantages in longevity. We combine quasi-experimental variation from the 1947 UK compulsory schooling reform with genetic information from the English Longitudinal Study of Ageing and estimate gene–environment interactions in survival at older ages. Because exact mortality information is only available for part of the observation period, we use panel survival as our main outcome. We show that this measure is closely related to mortality at very old ages: among individuals aged 80–84 who leave the panel and for whom mortality status can be verified, almost 60 percent do so because of death, and this share exceeds 80 percent among those aged 85–89. Panel survival is therefore an imperfect but informative proxy for survival at the ages most relevant for our analysis.

Our main finding is that education does not raise panel survival uniformly. Rather, the effect of additional compulsory schooling differs strongly by genetic predisposition for longevity. Individuals in the lowest tercile of the longevity PGI, who have the least favorable genetic endowment and therefore the highest genetic mortality risk, benefit substantially from the reform. For this group, an additional year of compulsory schooling increases the probability of remaining in the sample up to ages 80 and 84 by more than 30 percentage points. By contrast, individuals in the highest tercile of the longevity PGI, who have the most favorable genetic endowment and therefore the lowest genetic mortality risk, experience no additional gain. The implied effects for this group are close to zero or

even negative (but statistically insignificant). Thus, education appears to matter most for those who are genetically most disadvantaged with respect to longevity.

Education does not appear to amplify genetic differences in longevity. In contrast, education may reduce the extent to which genetic endowments translate into later-life survival differences. This interpretation is consistent with the idea that social policy can moderate the relationship between biological predispositions and health outcomes, rather than merely shifting average outcomes.

Our findings therefore speak to two literatures. First, they contribute to the literature on the causal effect of education on health and mortality. Previous evidence on the mortality effects of compulsory schooling reforms is mixed, with some studies finding little effect and others documenting protective effects. Our results suggest one possible reason why average effects may be difficult to detect or interpret: the benefits of education may be concentrated among particular groups. In our setting, the average effect masks substantial heterogeneity by genetic mortality risk.

Second, the results contribute to the growing literature on gene–environment interactions in economics and health. Barcellos et al. (2018) show that additional compulsory schooling reduced health differences related to genetic risk of obesity, with larger improvements among individuals with higher genetic risk. Our findings are closely aligned with this compensatory interpretation, but extend it to a different outcome and a later stage of the life cycle. While Barcellos et al. (2018) study middle-age health outcomes such as body size and lung function, we examine survival-related outcomes at very old ages. The similarity in the qualitative pattern suggests that education can reduce the consequences of genetic disadvantage not only for intermediate health outcomes, but also for survival-related outcomes later in life. At the same time, our results differ from evidence of complementarity between education and favorable genetic endowments in other domains, such as cognition. This highlights that the direction of gene–environment interaction is not necessarily universal, but depends on the outcome, the relevant genetic endowment, and the life-cycle stage under study.

The findings also speak to the interpretation of genetic risk. A longevity PGI is predictive of (panel) survival in our data, and individuals with more favorable genetic endowments are more likely to remain in the sample at older ages. However, the fact that the effect of schooling is largest among those with the least favorable genetic endowment shows that genetic risk is not destiny. Genetic predispositions may shape baseline risks, but their consequences can be moderated by social institutions. Compulsory schooling is one such institution: by increasing education among cohorts that would otherwise have left school earlier, the reform appears to have weakened the link between genetic mortality risk and later-life survival.

Our study has several limitations. First, our outcome is panel survival rather than verified mortality for the full sample. Although we provide evidence that panel attrition is strongly related to mortality at older ages, especially beyond age 80, it remains a proxy and may also capture health-related nonresponse or other forms of attrition. Second, the analysis is based on a genotyped subsample of ELSA and therefore faces the usual trade-off between rich genetic information and sample size. This limits statistical power, especially for detecting interaction effects. Third, our estimates are local to the cohorts affected by the 1947 UK compulsory schooling reform and to the margin of increasing the minimum school-leaving age from 14 to 15. The results need not generalize to other educational reforms, other countries, or other historical contexts.

Despite these limitations, the results point to a broader lesson. Educational policies may affect health inequality not only by changing average levels of schooling, but also by changing how strongly pre-existing vulnerabilities translate into later-life outcomes. Education can act as an equalizing force in the production of longevity. Future research should examine whether similar compensatory patterns arise in larger genetic samples, with directly observed mortality, and for other institutional changes in education. More generally, the results indicate that understanding the health returns to education requires attention not only to whether education matters, but also to for whom it matters most.

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A Polygenic indices

The human genome consists of roughly three billion base pairs—the nucleotide pairs that form DNA.[3] Yet any two individuals differ in only about 0.1 percent of these base pairs. Most of this variation arises from single–base substitutions at specific positions in the genome. These substitutions, known as *single nucleotide polymorphisms* (SNPs), represent common genetic variants observed throughout the population.[4] For instance, at a given genomic position, some individuals might carry an adenine, while others carry a thymine. One allele is then arbitrarily designated as the reference variant, and each SNP can be encoded as a count of how many copies (0, 1, or 2) of this reference allele an individual possesses, reflecting the two copies of each chromosome.

Genome-wide association studies (GWAS) relate these SNPs to phenotypes such as diabetes, years of education, or smoking behavior. Conceptually, GWAS involve estimating a regression of the form

$$Y_i = \beta_j S_{ij} + X_i'\delta + \zeta_i, \tag{2}$$

for each SNP j, where Y_i denotes the phenotype of individual i, $S_{ij} \in \{0, 1, 2\}$ is the count of the reference allele, and β_j is the estimated association between SNP j and the outcome. The vector X_i contains control variables such as age, sex, and principal components of the genetic data, which adjust for population structure to avoid spurious associations.[5]

A polygenic index (PGI) aggregates the estimated contributions of many SNPs into a single measure. Formally, it is constructed as a weighted sum of the relevant J SNPs:

$$PGI_i = \sum_{j=1}^{J} \beta_j S_{ij}. \tag{3}$$

Polygenic scores for a range of traits and behaviors—including personality, mental and physical health, and health-related behaviors—have been constructed for the ELSA sample using results from multiple GWAS and are readily available for research use.

[3] This description closely follows Hollenbach et al. (2026).

[4] A genetic substitution is typically classified as a SNP if it occurs in at least one percent of the population.

[5] Principal components are linear combinations of genetic markers that summarize major dimensions of genetic variation *across the population*. They capture population stratification—systematic differences in allele frequencies across subgroups that may induce false associations if both the phenotype and certain variants are more common in one subgroup. Price et al. (2006) show that including principal components reduces such confounding.

B Additional Results

Table B1: Robustness checks for *Still in sample at 84*

	(1) Baseline	(2) Bandwidth 8	(3) Bandwidth 6	(4) Without principal components	(5) Quadratic cohort trends
E_i	0.316**	0.381***	0.220	0.320**	0.066
	(0.144)	(0.135)	(0.152)	(0.144)	(0.242)
$G_i = 1$			Reference category		
$G_i = 2$	0.213	0.234	0.304*	0.225	0.218
	(0.152)	(0.148)	(0.175)	(0.152)	(0.151)
$G_i = 3$	0.451***	0.426***	0.428**	0.451***	0.461***
	(0.160)	(0.148)	(0.170)	(0.160)	(0.157)
$E_i \times (G_i = 1)$			Reference category		
$E_i \times (G_i = 2)$	–0.359*	–0.363*	–0.489**	–0.373*	–0.346*
	(0.204)	(0.193)	(0.234)	(0.204)	(0.210)
$E_i \times (G_i = 3)$	–0.534***	–0.510***	–0.492***	–0.536***	–0.538**
	(0.205)	(0.189)	(0.221)	(0.205)	(0.210)
Observations	2,318	2,573	2,059	2,318	2,318

Notes: This table reports robustness checks using the outcome *Still in sample at 84* for the gene-environment interactions from different specifications, using non-linear $G \times E$ effect with G_i as terciles built from the PGI for longevity ($G_i = g$ with $g \in \{1, 2, 3\}$ where 1 corresponds to the lowest tercile, 2 to the middle tercile, and 3 to the highest tercile). The main results from Table 4 are reported in column (1) for reference. The subsequent columns present four alternative specifications: estimating the main model in Equation 1 using a wider bandwidth of ±8 years around the 1933 cutoff (2), using a narrower bandwidth of ±6 years around the 1933 cutoff (3), excluding all principal components (4), and using quadratic cohort trends instead of linear cohort trends (5). Robust standard errors shown in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.