A What 2SLS is estimating

Assume that E_i and G_i are binary. There are four potential outcomes Y_i^{jg}, $j \in \{0,1\}$, $g \in \{0,1\}$ of individual i . Only one is observed. The observation rule is

$$\begin{aligned} Y_i &= E_i \cdot G_i \cdot Y_i^{11} + E_i \cdot (1-G_i) \cdot Y_i^{10} + (1-E_i) \cdot G_i \cdot Y_i^{01} + (1-E_i) \cdot (1-G_i) \cdot Y_i^{00} \\ &= Y_i^{00} + (Y_i^{10} - Y_i^{00})E_i + (Y_i^{01} - Y_i^{00})G_i + (Y_i^{11} - Y_i^{01} - (Y_i^{10} - Y_i^{00}))E_i \cdot G_i \end{aligned}$$

The second equation is the individual potential-outcome representation of the workhorse interaction model

$$Y_i = \beta_0 + \beta_1 E_i + \beta_2 G_i + \beta_3 G_i \times E_i + \varepsilon_i$$

Expressing this interaction equation as separate regressions for $G_i = 0$ and $G_i = 1$ yields

$$\begin{aligned} Y_i &= \beta_0 + \beta_1 E_i + \varepsilon && \text{for } G_i = 0 \\ Y_i &= (\beta_0 + \beta_2) + (\beta_1 + \beta_3)E_i + \varepsilon && \text{for } G_i = 1 \end{aligned}$$

Environment E_i is often a choice variable, therefore endogenous and instrumented by Z_i, a binary instrument. In Wald notation, separately estimating 2SLS regressions for $G_i = 0$ and $G_i = 1$ yields:

$$\begin{aligned} \widehat{\beta}_1 &= \frac{\mathbb{E}[Y_i|Z_i = 1, G_i = 0] - \mathbb{E}[Y_i|Z_i = 0, G_i = 0]}{\mathbb{E}[E_i|Z_i = 1, G_i = 0] - \mathbb{E}[E_i|Z_i = 0, G_i = 0]} && \text{for } G_i = 0 \\ \widehat{\beta}_1 + \widehat{\beta}_3 &= \frac{\mathbb{E}[Y_i|Z_i = 1, G_i = 1] - \mathbb{E}[Y_i|Z =_i 0, G_i = 1]}{\mathbb{E}[E_i|Z_i = 1, G_i = 1] - \mathbb{E}[E_i|Z_i = 0, G_i = 1]} && \text{for } G_i = 1 \end{aligned}$$

Using the LATE theorem (Imbens and Angrist, 1994 – 2SLS estimates are average treatment effects for the compliers), we can rewrite these expressions as:

$$\begin{aligned} \widehat{\beta}_1 &= \mathbb{E}[Y_i^{10} - Y_i^{00}|C(G_i = 0)] \\ \widehat{\beta}_1 + \widehat{\beta}_3 &= \mathbb{E}[Y_i^{11} - Y_i^{01}|C(G_i = 1)] \end{aligned}$$

The mechanics of the LATE require that the group-specific effects ($\widehat{\beta}_1$ and $\widehat{\beta}_1 + \widehat{\beta}_3$) are average treatment effects for the G_i-specific compliers. Without further covariates, the joint interaction regression specification is as flexible as the separate ones. The mechanics of interaction models attribute any difference in the causal effects of E_i on Y_i between $G_i = 0$ and $G_i = 1$ to the interaction coefficient. In essence, the interaction model is numerically identical to separate estimations. In the interaction model, any difference between the

G_i-specific LATEs is mechanically attributed to $\widehat{\beta}_3$. Hence, using the expressions above, this difference amounts to:

$$\widehat{\beta}_3 = (\widehat{\beta}_1 + \widehat{\beta}_3) - \widehat{\beta}_1 = \mathbb{E}[Y_i^{11} - Y_i^{01} | C(G_i = 1)] - \mathbb{E}[Y_i^{10} - Y_i^{00} | C(G_i = 0)]$$

This demonstrates that the interaction coefficient reflects differences in G_i-specific LATEs.

B Polygenic scores

The human genome has about 3 billion base pairs, the pairs of nucleic acids that make up the DNA. However, any two people differ by only about 0.1 percent of the base pairs. Most of these genetic differences are substitutions of a single base (adenine, thymine, cytosine, or guanine) for another at a specific location in the genome, called "single nucleotide polymorphisms" (SNPs) that are common across the whole genome. These substitutions result in different genetic variants (alleles) that vary among parts of the population.[10] For example, at a specific SNP location, the DNA sequence might have an adenine base in some individuals, while others may have a thymine base at the same position. One is (arbitrarily) chosen as the reference variant. Then, each SNP can be represented as a count variable of occurrences of the reference variant at this location that can either be 0, 1 or 2, since there are two copies of each chromosome. Large research projects called genome-wide association studies (GWAS), correlate each $j = 1, \ldots, J$ SNPs with a disease or trait, e.g., diabetes, years of education, or smoking. This entails running J regressions of type

$$Y_i = \beta_j S_{ij} + X_i'\delta + \zeta_i \tag{7}$$

where Y_i is the outcome of interest (in our case educational attainment) of individual i, β_j is the individual effect of each SNP j, S_{ij} is the count variable of the reference variant of the SNP with $S_{ij} \in \{0, 1, 2\}$, X_i is a vector of controls that typically include age, gender and principal components of the genetic data, which control for population stratification, i.e., common ancestry[11]. The PGS is then calculated as a weighted sum of all S_{ij}'s, where the weights correspond to the (correlation-adjusted) β_j's obtained in the GWAS:

$$PGS_i = \sum_{j=1}^{J} \tilde{\beta}_j S_{ij} \tag{8}$$

Polygenic scores for various traits or behaviors (personality, mental and physical health, health behaviors, and more) have been calculated for the ELSA sample based on various GWAS and are readily available.

[10] The generally agreed-upon threshold for a substitution to be regarded a SNP is common occurrence in at least one percent of the population.

[11] Principal components are linear combinations of genetic markers that summarize the major patterns of genetic variation *across a population* into fewer dimensions. They reflect population stratification, i.e., different frequencies of genetic variants among subpopulations that could be responsible for spurious correlations with outcomes of interest. Price et al. (2006) show that including principal components as controls can mitigate the confounding effects of population stratification, ensuring that observed associations between genetic variants and traits are not driven by differences in ancestry or population structure.

C Additional sample information

Table C.1: Descriptive statistics (extended)

	Main sample	By E_i		
	Mean (SD)	E_i=1	E_i=0	Difference (SE)
Outcome Y_i				
Recall score	9.67 (3.37)	10.11	8.08	2.03 (0.07)***
Treatment E_i				
Left school ≥ 15	0.78 (0.41)	1.00	0.00	1.00 (0.00)
Polygenic score G_i				
1st PGS quintile	0.20 (0.40)	0.18	0.26	−0.08 (0.01)***
2nd PGS quintile	0.20 (0.40)	0.20	0.21	0.02 (0.01)*
3rd PGS quintile	0.20 (0.40)	0.20	0.19	0.01 (0.01)
4th PGS quintile	0.20 (0.40)	0.20	0.19	0.01 (0.01)
5th PGS quintile	0.20 (0.40)	0.21	0.15	0.07 (0.01)***
Instrument Z_i				
Born 1933 or later	0.66 (0.47)	0.82	0.13	0.69 (0.01)***
Controls				
Female	0.52 (0.50)	0.52	0.50	0.02 (0.01)**
Principal components (standardized):				
– 1 –	0.00 (1.00)	0.00	−0.01	0.02 (0.02)
– 2 –	0.00 (1.00)	0.01	−0.02	0.03 (0.02)
– 3 –	0.00 (1.00)	0.01	−0.04	0.05 (0.02)**
– 4 –	0.00 (1.00)	−0.01	0.02	−0.03 (0.02)
– 5 –	0.00 (1.00)	0.00	0.00	0.00 (0.02)
– 6 –	0.00 (1.00)	0.02	−0.07	0.09 (0.02)***
– 7 –	0.00 (1.00)	0.01	−0.03	−0.04 (0.02)*
– 8 –	0.00 (1.00)	0.00	0.02	−0.02 (0.02)
– 9 –	0.00 (1.00)	0.01	−0.02	0.02 (0.02)
– 10 –	0.00 (1.00)	0.01	−0.02	0.02 (0.02)
Age pattern				
Birth year	1934.89 (5.00)	1936.29	1929.92	6.37 (0.10)***
Age	71.82 (4.29)	70.89	75.10	−4.21 (0.09)***
Observations	11,027	8,590	2,437	

Notes: This table presents extended descriptive statistics including the first 10 principal components of the genetic data. We include mean and standard deviation of the main sample as well as means by E_i, the difference of means and standard errors of a t-test for equality of means. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Table C.2: Descriptive statistics by availability of genetic information

	Full sample	By availability of genetic information		
	Mean (SD)	Yes	No	Difference (SE)
Outcome Y_i				
Recall score	9.32 (3.50)	9.67	8.76	0.91 (0.05)***
Treatment E_i				
Left school ≥ 15	0.76 (0.43)	0.78	0.72	0.06 (0.01)***
Instrument Z_i				
Born 1933 or later	0.65 (0.48)	0.66	0.62	0.04 (0.01)***
Controls				
Female	0.52 (0.50)	0.52	0.52	0.00 (0.01)
Age pattern				
Birth year	1934.67 (5.10)	1934.89	1934.32	0.57 (0.08)***
Age	71.90 (4.26)	71.82	72.02	−0.20 (0.07)***
Observations	17,884	11,027	6,857	

Notes: This table presents descriptive statistics. We include mean and standard deviation of the main sample as well as means by E_i, the difference of means and standard errors of a t-test for equality of means. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Table C.3: Descriptive statistics by PGS quintiles

	1st quintile	2nd quintile	3rd quintile	4th quintile	5th quintile
Outcome Y_i					
Recall score	8.99	9.48	9.73	9.80	10.33
Treatment E_i					
Left school ≥ 15	0.71	0.77	0.79	0.79	0.84
Instrument Z_i					
Born 1933 or later	0.67	0.65	0.65	0.68	0.67
Controls					
Female	0.53	0.51	0.54	0.49	0.50
Age pattern					
Birth year	1934.87	1934.87	1934.96	1934.77	1934.96
Age	71.72	71.84	71.87	71.86	71.81
Observations	2,206	2,205	2,206	2,205	2,205

Notes: This table presents sample means by quintiles of the education polygenic score.

D Additional regression results

Table D.4: The 1947 UK compulsory schooling reform and providing genetic information to ELSA

	Provided genetic information (1)	Left school at 15 or later (E_i) (2)
Z_i	−0.018 (0.030)	0.453 (0.037)***
Provided genetic information $\times Z_i$		0.035 (0.046)
Controls	Yes	Yes
Observations	17,884	17,884

Notes: In this table we show that our instrument, the 1947 UK compulsory schooling reform did not affect the probability of providing genetic information to ELSA and that providing genetic information does not interact with our first stage, the effect of the reform on staying in school until at least 15. Column 1 shows estimates of a linear regression of the instrument Z_i on the probability to provide genetic information to ELSA. Controls include gender, the running variable (distance to 1933 birth cohort) and its interaction with the instrument. Column 2 shows estimates of the first stage interacted with a dummy for providing genetic information to ELSA. Controls include gender, running variable and interactions with the running variable. Both regressions are estimated in a larger sample that fulfils all criteria outlined in section 3.2 but still includes individuals without genetic data available. Standard errors in both regressions are clustered at the individual level. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

Table D.5: Estimates of the first stage by PGS quintile

	Left school at 15 or later (E_i)	
	Coefficient (1)	Standard error (2)
$Z_i \times (G_i = 1)$	0.642	(0.018)***
$Z_i \times (G_i = 2)$	0.537	(0.018)***
$Z_i \times (G_i = 3)$	0.477	(0.018)***
$Z_i \times (G_i = 4)$	0.419	(0.018)***
$Z_i \times (G_i = 5)$	0.356	(0.018)***
Controls	Yes	
Observations	11,027	

Notes: This table presents estimates of the effect of the 1947 UK compulsory schooling reform on the probability of attending school until at least age 15 by quintiles of the education polygenic score. These effects are obtained from the coefficients $\pi^f_{1,\Delta}$ to $\pi^f_{5,\Delta}$ of eq. 4, which correspond to the complier shares in the respective quintile. Standard errors clustered at the individual level shown are in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, and $^{***}p < 0.01$.

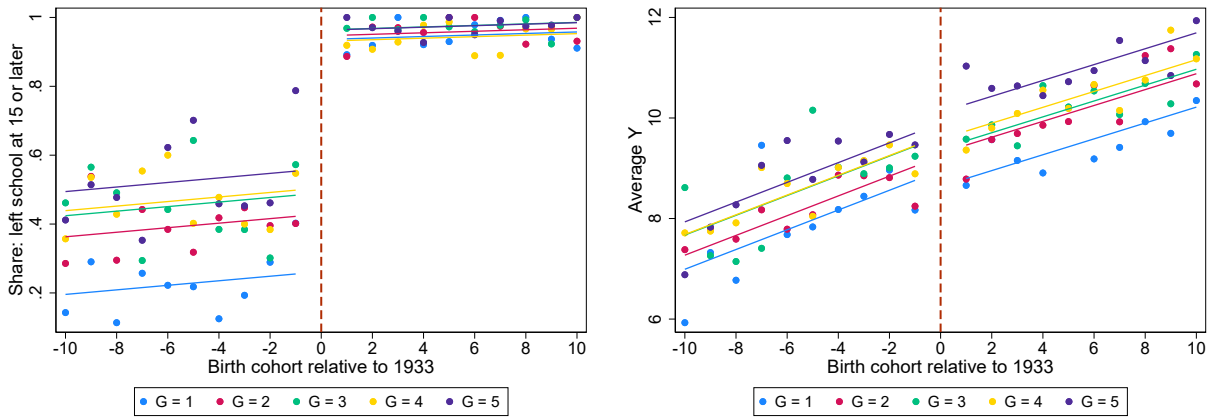

Figure D.1: First Stage and reduced form by G_i

E Testing "no selection into losses" (non-positive MTE slopes)

An important constraint we apply in our linear programming approach is "no selection-into-losses", i.e. no MTEs that increase in U_E. To test this in our setting, we follow Imbens and Rubin (1997) and use the instrument to compute mean outcomes for always-taker, treated and untreated complier, and never-taker. For simplicity, we test this condition globally and do not distinguish between cells of G_i (we show the complete G_i-specific means in Figure 5). We present the results in Table E.6. In Panel A, we focus on differences between always-takers and treated compliers (Column 3) and untreated compliers and never-takers (Column 6). The differences are informative about whether the treated outcome $\mathbb{E}[Y_i^1|U_E = u]$ and the untreated outcome $\mathbb{E}[Y_i^0|U_E = u]$ – the difference of which is the MTE – are heterogeneous in U_E.

Column (3) presents the mean recall difference between always-takers and treated compliers. It shows a substantial and statistically significant heterogeneity: Always-taker recall about 1.25 words more. Intuitively, this is unsurprising, as always-taker to a compulsory schooling reform will, on average, have more years of education, will be more likely to hold advanced degrees, or may be positively selected in terms of unobserved characteristics (if we have selection into gains, which we want to argue). Furthermore, this result shows that $\mathbb{E}[Y_i^1|U_E = u]$ has a negative slope. Likewise, we do the same with untreated compliers and never-taker. Here, the heterogeneity is less pronounced and not statistically significant. If we conclude that both groups do not have different outcomes, we can stop as in this case, the difference in the first two groups proves that we have essential heterogeneity. If the insignificant difference is meaningful, things may change. The difference is also negative, contrasting the existing empirical evidence for the slope of the untreated outcome (see, e.g., Carneiro and Lee, 2009; Westphal et al., 2022). However, it is essential to mention that never-taker should not exist with a compulsory schooling reform, where everyone should be forced to stay in school until age 15. If this group has never existed, this might be a measurement error. If these individuals had special exemptions from the rule change (and therefore existed), the difference between never-taker and untreated compliers may not inform about the global course of the curve. Assessing the multiple complier groups that we gain by stratifying by G_i (see Figure 5) indeed suggests that never-taker are different and $\mathbb{E}[Y_i^0|U_E = u]$ indeed increases when $U_E < 0.95$.

Nonetheless, with only a binary instrument and without exploiting covariate heterogeneity together with the additive separability assumption (which we will do below), an additional linearity assumption is necessary (due to the never-taker) to point-identify a marginal treatment effect via the method introduced by Brinch et al. (2017). We document a formal

Table E.6: Mean outcomes by instrument response types and test for essential heterogeneity

	Unobserved heterogeneity					
	in the treated outcome			in the untreated outcome		
	(1) Always-taker	(2) Treated Complier	(3) Difference (2) − (1)	(4) Untreated Complier	(5) Never-taker	(6) Difference (5) − (4)
Panel A:						
Mean recall:	9.500	8.306	−1.245***	8.109	7.679	−0.353
	(0.215)	(0.332)	(0.454)	(0.215)	(0.340)	(0.396)
Share:	0.456	0.489		0.489	0.055	
	(0.035)	(0.036)		(0.036)	(0.011)	
Panel B:						
Test for essential heterogeneity:						
(sufficient condition, may be uninformative if heterogeneity is nonlinear)						
Slope of $\mathbb{E}[Y_i^1 \mid U_E = u]$			−2.631***			
			(0.961)			
Slope of MTE $\mathbb{E}[Y_i^1 - Y_i^0 \mid U_E = u]$			−1.326			
			(1.423)			

Notes: This table presents estimates of mean outcomes for always-taker, treated and untreated complier, and never-taker (panel A) as well as results of a test for essential heterogeneity (panel B). We compute the type-specific shares using the specification of Eq. (2) without G_i. The complier share is the coefficient on Z_i, the always-taker share is the constant (as all variables are demeaned), and the never-taker share is the remainder. For the type-specific outcome means, we compute means by E_i and Z_i (and their interaction) using a reduced-form specification to regress recall on the same controls and full interactions of E_i and Z_i. As compliers never appear alone in these means, we use the formula provided in Imbens and Rubin (1997) and the type-specific shares. Standard errors are computed using 200 bootstrap replications and are shown in parentheses. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$ indicate significance levels for the differences.

test of the slope of $\mathbb{E}[Y_i^1 | U_E = u]$ and $\mathbb{E}[Y_i^1 - Y^0 | U_E = u]$ in Panel B.[12] It shows that the slope of the treated outcome is negative and statistically significant (as shown in Panel A). The slope of the linear MTE is also negative and still large in magnitude. However, likely due to the concerns about never-taker outlined above, it is not statistically significant, albeit with a negative sign. Again, evidence from the G_i-specific complier groups strongly suggests that the $\mathbb{E}[Y_i^0 | U_E = u]$ increases at least for a relevant range when $U_E < 0.95$. We conclude that we likely face essential heterogeneity in our setting. Combined differences in the first stage induced by G_i, the result may be that 2SLS cannot recover the true interaction parameter. We would need to make accurate statements about the interaction effect.

[12]The exact formula reads

$$\frac{\partial \mathbb{E}[Y_i^1 | U_E = u]}{\partial U_E} = \frac{Y_i^{CT} - Y_i^{AT}}{\frac{\pi^C + \pi^{AT}}{2}}, \qquad \frac{\partial \mathbb{E}[Y_i^1 - Y_i^0 | U_E = u]}{\partial U_E} = \frac{Y_i^{CT} - Y_i^{AT}}{\frac{\pi^C + \pi^{AT}}{2}} - \frac{Y_i^{NT} - Y_i^{CU}}{\frac{\pi^C + \pi^{NT}}{2}},$$

where Y_i^{AT}, Y_i^{CT}, Y_i^{CU}, and Y^{NT} are means from Columns (1), (2), (4), and (5), respectively and π^{AT}, π^C, π^{NT} are the corresponding shares (compliers do not need to be differentiated).

F Details on the MTE estimation

We run the following two regressions:

$$E_i = \sum_{g=1}^{5}\sum_{k=0}^{1}\left[\pi^f_{g,k}\mathbb{1}[G_i = g]\times[Z_i = k]\right] + \text{controls} + \omega_i \tag{9}$$

$$Y_i = \sum_{g=1}^{5}\sum_{j=0}^{1}\sum_{k=0}^{1}\left[\delta^f_{g,j,k}\mathbb{1}[G_i = g]\times[E_i = j][Z_i = k]\right] + \text{controls} + \eta_i. \tag{10}$$

The first equation estimates G_i-specific first-stage from which the complier types can be inferred. The second equation estimates conditional means of Y_i, conditional on G_i, Z_i, and Y_i when covariates are fixed. On these estimates, we apply the Imbens and Rubin (1997) formula to compute G_i-specific outcome means for always-taker (AT), never-taker (NT), and (treated on untreated) compliers (C) the are plotted in Figure 5:

$$\begin{aligned}
\mathbb{E}[Y_i^{1g}|C, G_i = g] &= \frac{\delta_{g,1,1}\pi_{g,1} - \delta_{g,1,0}\pi_{g,0}}{\pi_{g,1} - \pi_{g,0}} \\
\mathbb{E}[Y_i^{0g}|C, G_i = g] &= \frac{\delta_{g,0,0}\pi_{g,0} - \delta_{g,0,1}\pi_{g,1}}{\pi_{g,1} - \pi_{g,0}} \\
\mathbb{E}[Y_i^{0g}|NT, G_i = g] &= \delta_{g,0,1} \\
\mathbb{E}[Y_i^{1g}|AT, G_i = g] &= \delta_{g,1,0}
\end{aligned}$$

These linear potential outcome curves could already solve the problems associated with 2SLS estimation of interactions while using richer variations of the polygenic score. Based on them, we can calculate the (interaction) effects according to Table 1 in the interval $0.6 \leq U_D \leq 0.8$. Graphically, this would entail subtracting the blue from the red lines for each quintile. However, this would require extrapolating the lines with $E_i = 0$ to the left or the lines with $E_i = 1$ to the right, demonstrating the general extrapolation problem that we could solve here by a linearity restriction. If we are willing to make this extrapolation, it yields five MTE curves for the effect of E_i on Y_i, one for each quintile, which can then be used to calculate the interaction effects.

In the paper, we are unwilling to make such an assumption and apply the partial identification method by Mogstad et al. (2018). As one input, the method uses the conditional means that the coefficients ($\delta^f_{g,j,k}$ and the corresponding $\pi^f_{g,k}$) reflect. These are the "moments" for the linear programming method by Mogstad et al. (2018). Figure F.2 plots the results of this approach, where the slightly transparent, horizontal lines are the "moments" (G_i-specific outcome means and their placement on the unit-interval, which we derive from the complier shares). The blue (for the treated outcome) and red (for the untreated) lines

are the output of this linear programming approach. They reflect the minimal (the dashed lines) and maximal (the solid lines) possible interaction effect (defined in the main text) that the MTR lines (Bernstein polynomials, see Figure F.3) produce while being consistent with the shape restrictions and matching the moments.

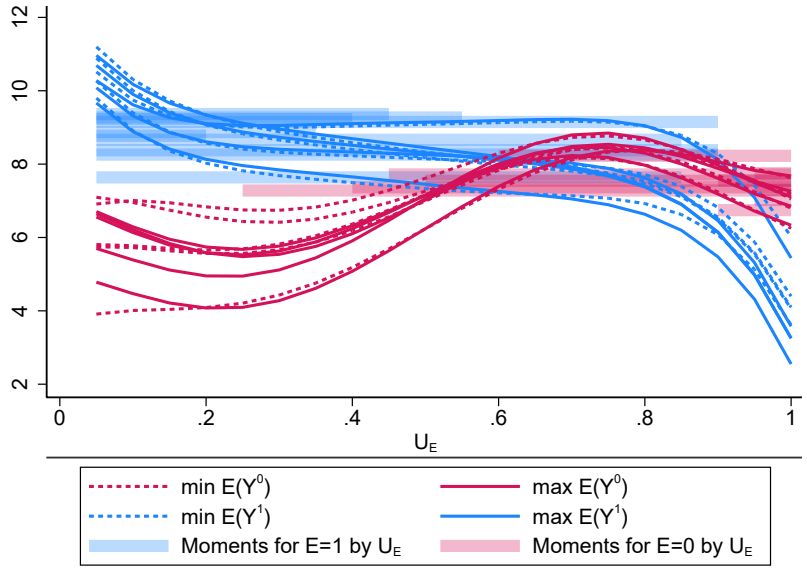

Figure F.2: Potential outcome curves estimated with Bernstein polynomials

Notes: This figure shows the minima and maxima of the ten potential outcome curves estimated via linear program with Bernstein polynomials. Blue indicates curves and moments for $E_i = 1$, and red indicates $E_i = 0$. Solid lines are maxima; dashed lines are minima of the potential outcome curves. There are five pairs of curves for $E_i = 1$ and five for $E_i = 0$, one pair for every PGS quintile. Every pair consists of a minimum and a maximum that bound the potential outcome curve for its respective quintile. The vertical bars indicate the moments the curves must pass and the U_E ranges of individuals contributing to these means.

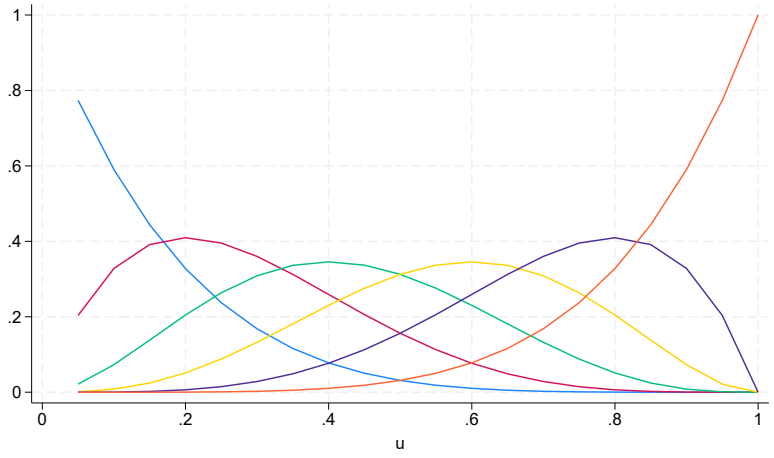

Figure F.3: Graphical representation of the Bernstein base functions

Notes: This figure depicts the six Bernstein base functions that compose a Bernstein polynomial of degree five. The formula for each base function reads $b_{v,n}(u) = \binom{n}{v} u^v (1-u)^{n-v}$, where $n = 5$ is the degree, v denotes the specific base function and u is a specific grid point on the unit interval. The formula that obtains the MTE by the sum of all base functions weighted by the corresponding parameter θ_v^{jg} reads $m^j(u, g) = \sum_{v=0}^{n} \theta_v^{jg} b_{v,n}(u)$, where G_i is the genetic bin, j the treatment state (as defined above) in addition to the variables and parameters defined above.

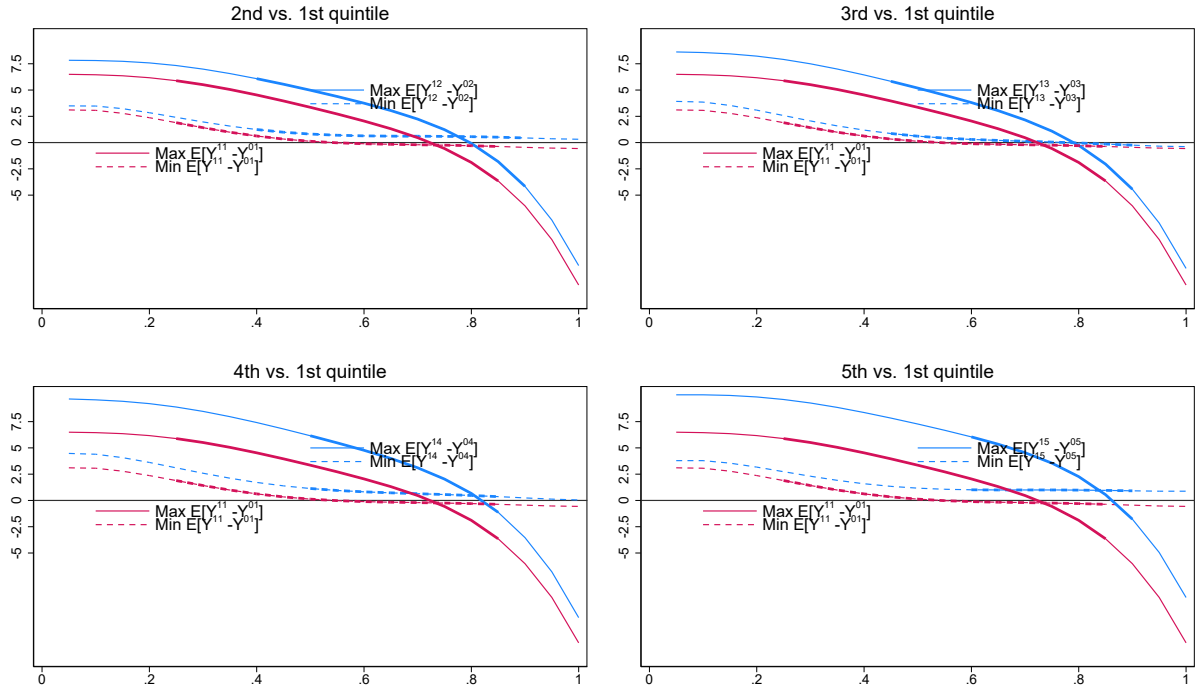

Figure F.4: Quintile comparisons of the interaction effect without never-taker

Notes: This figure shows the quintile comparisons of the interaction effect from Figure 6 when never-taker (their sample moments) are not used to construct the MTE bounds. For every PGS quintile, we estimate bounds: maxima (solid lines) and minima (dashed lines) at which the interaction effect is maximized/minimized. The bounds for quintiles 2-4 (in blue) are compared to those of the bottom quintile (in red), our reference category, yielding four comparisons. The gene-environment interaction is the difference between the blue and red curves at $U_E \in [0.6, 0, 8]$. The thick part of the curves indicates the size of the complier share and its location on the U_E scale, both of which differ by PGS quintile.

G Robustness checks for the linear programming approach

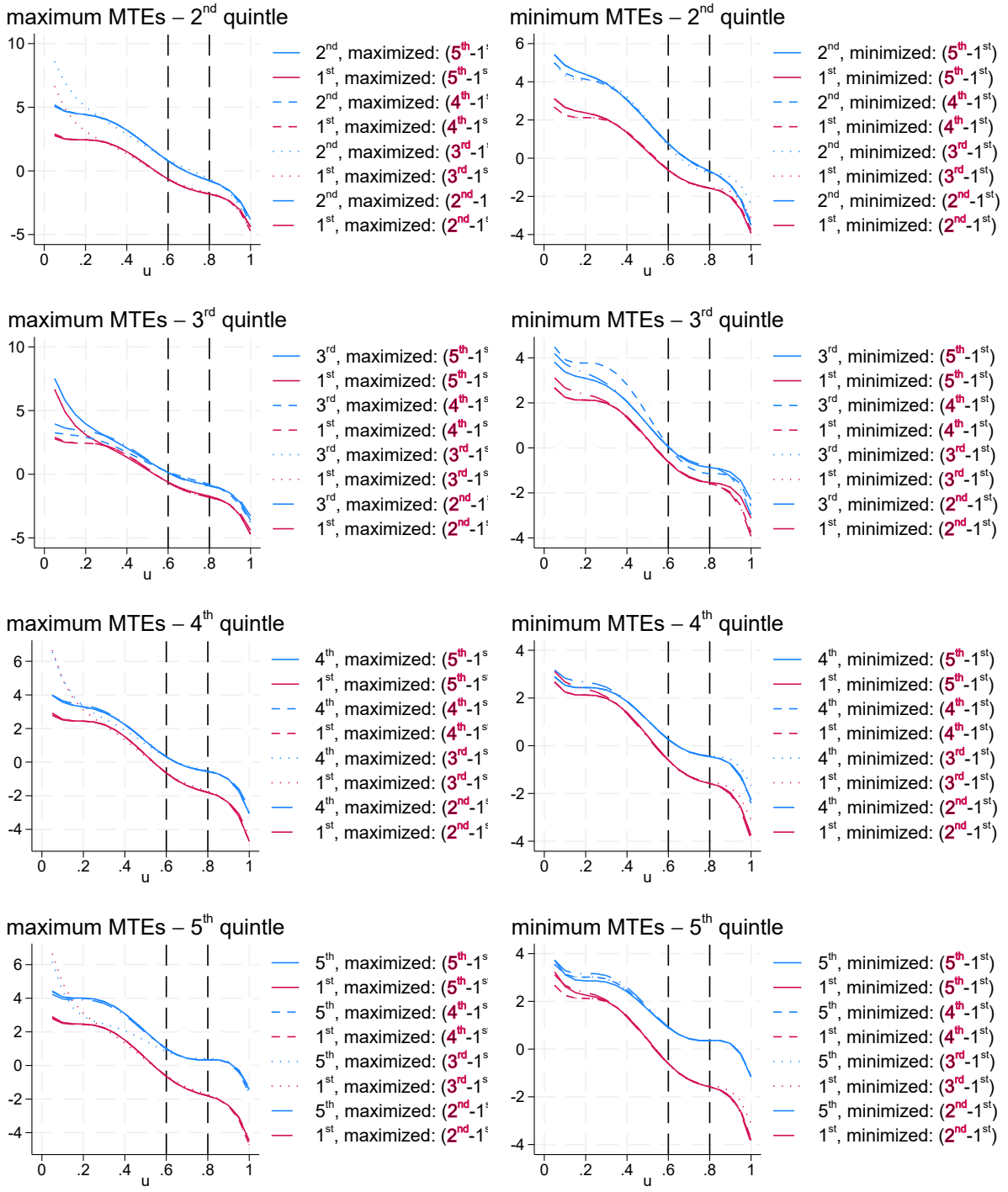

Figure G.5: MTEs when the target $G \times E$ parameter is adjusted to specific quintiles

Notes: This figure shows robustness checks for our main result in Figure 6. Here, we optimize the interaction effect for different comparisons. Whereas our preferred specification optimizes the difference between the first and the fifth quintile (see Eq. 6), we generalize this approach and optimize differences between the first any other quintile such that $\beta_{G\times E}(0.6, 0.8, g) = \frac{1}{g-1}\int_{0.6}^{0.8}[m^1(u,g) - m^0(u,g)] - [m^1(u,1) - m^0(u,1)]du \quad \forall g \in \{2,3,4,5\}$. The solid lines correspond to optimizing $g = 5$, our main result. The dashed lines show the optimization for $g = 4$, the dotted for $g = 3$, and the dashed-dotted line for $g = 2$. The respective quintile G_i used for the target parameter $\beta_{G\times E}(0.6, 0.8, g)$ is highlighted in bold. Maximized and minimized MTEs are shown separately, maximized MTEs in the left and minimized MTEs in the right column. The rows present pairwise comparisons between the first and another PGS quintile (the second quintile in the first row, the third in the second row, ...).