Genetically Informed Estimation of Marginal Returns to Education

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MARIE CURIE ACTIONS

Is education the great equaliser?

"Education … is the great equalizer of the conditions of men"
— Horace Mann (1848)

- Whether education equalises depends on **two margins**:
  - **Selection:** who enrols
  - **Returns:** how much different individuals gain from attending
- **Challenge:** unobserved characteristics, such as skills, may influence **both** enrolment and returns
- **Genetic data** offer a measure of **initial heterogeneity**:
  - Predetermined with respect to schooling choices
  - Genetic predisposition may shape **both margins**

Research question

Does university **amplify** or **mitigate** inequalities associated with genetic predisposition to education?

- Three interconnected dimensions:
  - (i) Do individuals with different genetic predispositions **select differently** into university?
  - (ii) Do their **returns** to university differ **at comparable margins**?
  - (iii) How much of the difference in average returns reflects **gains** rather than **selection**?

What do we contribute?

(1) Theoretical

- Extend the MTE framework with a mapping function (the warp) to compare **group-specific returns** at a common level of unobserved resistance to treatment
  - Allows the distribution of unobserved resistance to differ across groups
- Decompose treatment-effect gaps into differences in **returns** and **self-selection**

(2) Empirical

- Bring **genetic data** into the study of wage returns to university
- Show whether university education **amplifies or mitigates earnings inequality by genetic predisposition to education**

Related literature

	What we know	What we add
Marginal treatment effects	Selection on gains and heterogeneous treatment effects Björklund and Moffitt (1987); Heckman and Vytlacil (2005); Hollenbach, Schmitz, and Westphal (2026)	**Compare groups at common margins**, without imposing common MTE slopes. Decompose returns vs sorting
Returns to education and university expansions	Heterogeneous gains from schooling; expansions as variation in access Carneiro, Heckman, and Vytlacil (2011); Nybom (2017); Westphal, Kamhöfer, and Schmitz (2022); Braccioli et al. (2024); Blundell, Green, and Jin (2022); Ichino, Rustichini, and Zanella (2026)	**Genetic heterogeneity** in returns to university
Genetics and education	Genetic predisposition and gene–education interactions Papageorge and Thom (2020); Barcellos, Carvalho, and Turley (2021); Hollenbach, Schmitz, and Westphal (2026); Biroli et al. (2026)	From compulsory schooling to the **university margin**, separating **returns** ($G \times E$) from **sorting** (rGE)

Preview of findings

(i) **Different selection:** the warp reveals distinct sorting into university by genetic predisposition to education

(ii) **Different returns:** average returns of **0.69 log points** for those with low genetic predisposition vs **0.31** for those with high

(iii) **Gains rather than selection:** estimates suggest the gap is driven by **differences in returns**, with little contribution from selection

Consistent with university mitigating earnings inequalities associated with genetic predisposition to education

Genetics and Framework

Measuring genetic predisposition to education

▸ More on PGIs

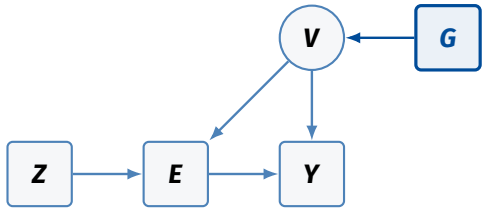
- The **Educational Attainment Polygenic Index (EA PGI)** summarises the association of **millions of genetic variants** with education into a continuous numerical index
- Strongest available predictor using genetic variation alone: it explains **12–16%** of the variation in years of education (Okbay et al., 2022)

Interpretation

- **Fixed at conception**: a dimension of initial heterogeneity
- **Linked to many later socioeconomic outcomes** beyond education (Belsky et al., 2016)
- Predictive on average, but **not deterministic**
- **Doesn't measure innate ability**: may reflect multiple pathways



Overview of the model

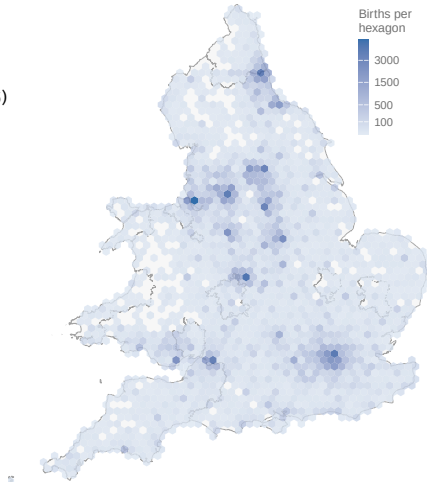


- ***E***: University attainment dummy
- ***Z***: Reduction in distance to university (IV)
- ***Y***: Imputed log hourly wage
- ***V***: Unobserved factors affecting both *E* and *Y* (e.g. skills, motivation)
- ***G***: EA PGI (tertiles)

Data and Context

Data (I): UK Biobank

- Population-based cohort of about **500,000 adults**, recruited in 2006–2010 at ages 40–69 (Sudlow et al., 2015)
- **Genotyped**: we build the EA PGI from genome-wide data
- Education, occupation, **place of birth** and current residence (1 km grid)
- **Analysis sample:** 157,109 individuals of European ancestry, born 1945–1969 in England and Wales



Birthplaces of the analysis sample (10 km hexagons)

Data (II): Universities and local context

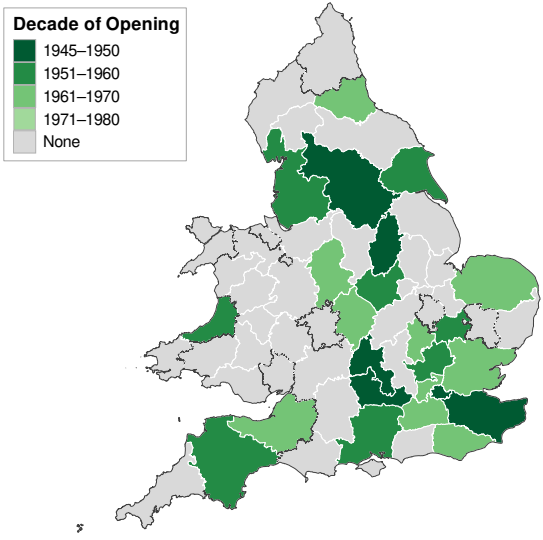
- **Higher education institutions data**
  - We construct a **new geocoded dataset** of UK higher-education institutions (1,072 campus records)
  - Location, opening date, date of university status, closure date; linked to each person's birthplace and cohort

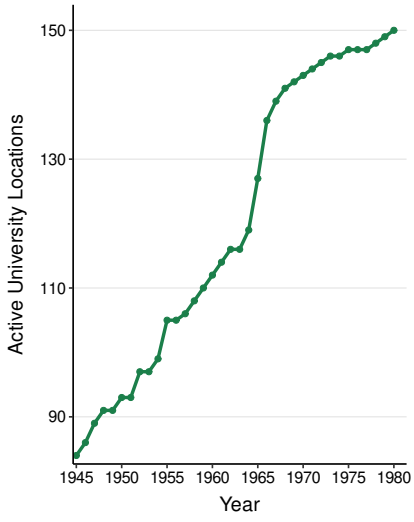
college_data.csv

Institution	Campus	Latitude	Longitude	Teaching on site since	University status since	Closure
University of Oxford	Balliol College	51.755	−1.258	1263	1263	—
Keele University	Rural Campus	53.003	−2.272	1949	1962	—
University of Sussex	Falmer	50.869	−0.088	1961	1961	—
University of York	Campus West	53.946	−1.055	1964	1964	—
Lancaster University	Bailrigg	54.010	−2.788	1964	1964	—
University of Warwick	Main Campus	52.379	−1.561	1965	1965	—

- **Local controls**
  - Pre-expansion socioeconomic characteristics of the district of birth
  - *Sources:* 1951 Census, Great Britain Historical Database, Labour Gazette

Context: University expansion in England and Wales (1945–1980)





How do we identify the returns to university?

We follow a growing literature that exploits **university expansions** to instrument education (e.g., Westphal, Kamhöfer, and Schmitz, 2022; Braccioli et al., 2024; Bratti, Cottini, and Ghinetti, 2022)

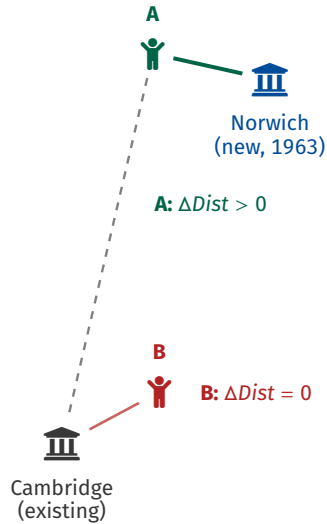
Reduction in distance to university

Instrument

The reduction in distance to the nearest university between birth and age 15

$$\Delta Dist = Dist^{\text{birth}} - Dist^{\text{age 15}}$$

- **29%** of the sample got closer to a university by age 15, by **13 km** on average ▸ Summary statistics
- **Caveat:** residence at 15 is not observed, but UKB families rarely moved: 1.2% a year moved more than 1 km, 0.6% more than 50 km (von Hinke and Vitt, 2024)



What we control for

- **Pre-expansion local socioeconomic conditions** (1946–1951)
  - Baseline access: number of universities within 10 km of the birthplace at birth
  - Birth district: urban status, education level, social-class composition, population, density and growth, unemployment
- **Individual:** sex, number of siblings, 20 genetic principal components
- **Fixed effects:** birth region $\times$ academic cohort
- **Caveat:** parental information is not available, so local conditions may not fully capture family background

First stage: better access raises university attainment

▸ Robustness	(1) Overall	(2) Low EA PGI	(3) Medium EA PGI	(4) High EA PGI
$\Delta Dist$	0.014^{***}	0.012^{***}	0.015^{***}	0.012^{***}
	(0.003)	(0.002)	(0.003)	(0.003)
Observations	157,109	52,370	52,369	52,370
First-stage F	35.47	17.14	29.57	11.77
Controls (X)	Yes	Yes	Yes	Yes
Region × cohort FE	Yes	Yes	Yes	Yes
Test of equal effects across tertiles			$p = 0.374$	

Notes: Effect of $\Delta Dist$ (per 10 km) on completing university, with UKB weights. Columns (2)–(4) interact $\Delta Dist$ with EA-PGI-tertile indicators. Baseline controls and birth-region-by-cohort FE throughout. SEs clustered by district of birth. First-stage F: cluster-robust Wald statistic for the excluded instrument. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

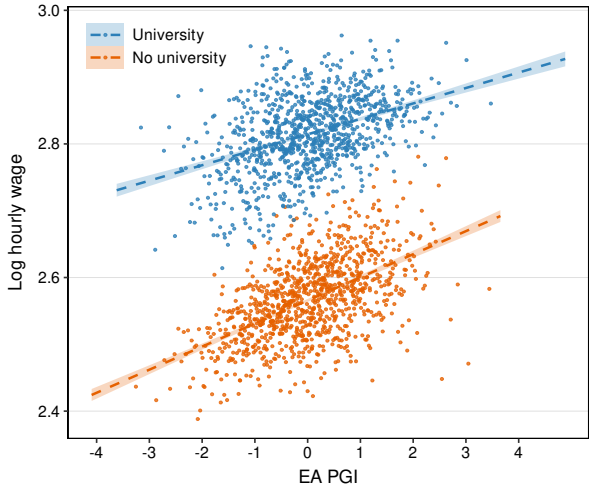
First stage present in every EA-PGI group, with similar effects across groups

Identifying assumptions

- **Relevance:** Z shifts university attainment ✓
- **Conditional exogeneity:** given X, Z is independent of G and of unobservables in E, Y
  - Openings predicted by only a few pre-expansion district characteristics ▸ Predictors
  - Balanced on the EA PGI and other predetermined traits ▸ Balance
  - Robust to more granular geographic FE ▸ Fixed effects
  - Later openings do not predict university ▸ Placebo
- **Exclusion:** Z affects Y only through E
  - Later openings do not predict wages ▸ Placebo
- **Monotonicity:** better access weakly increases university attendance for every individual
  - Attainment rises with $\Delta Dist$, with no reversal ▸ Bins

A first look at the evidence

Raw data: the university wage gap narrows with the EA PGI



Notes: Each point is the mean log hourly wage within one of 1,000 EA-PGI quantile bins, separately by university status. Dashed lines are linear fits on the individual data with 95% confidence bands. UKB weights throughout.

Benchmark: the conventional IV interaction

▸ Specification ▸ Full table

	Log wage
University	0.512***
	(0.076)
EA PGI	0.034*
	(0.017)
University × EA PGI	−0.093**
	(0.041)
Observations	157,109
First-stage *F* (joint)	19.25
Controls (*X*)	Yes
Region × cohort FE	Yes

Notes: 2SLS: University and University × EA PGI instrumented with Δ*Dist* and Δ*Dist*× EA PGI. Joint *F*: both excluded instruments in the University equation. Continuous, standardised EA PGI. 20 genetic PCs, UKB weights. SEs clustered by district of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

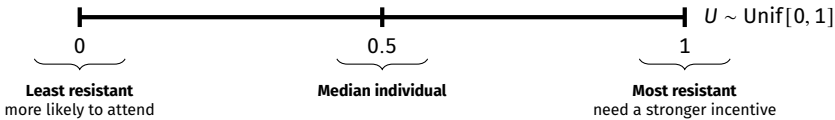
- **Selection on unobserved gains** in the returns to education is widely documented (e.g., Carneiro, Heckman, and Vytlacil, 2011)
- With such selection, the IV interaction **may compare different complier populations** across PGI levels: it need not isolate differences in returns at comparable margins (Hollenbach, Schmitz, and Westphal, 2026)
- We therefore turn to the Marginal Treatment Effect (MTE) framework (Heckman and Vytlacil, 2005)

Marginal treatment effects (MTE)

Accounting for selection on unobserved gains

Resistance to treatment and treatment rule

- Following Heckman and Vytlacil (2005) we can **rank individuals** based on their **unobserved resistance to university** (U)



- An individual **attends university whenever** their **propensity,** $P(Z)$, to attend exceeds their latent resistance:

$$E = \mathbb{1}\{P(Z) \geq U\}$$

▸ Derivation

Marginal treatment effects

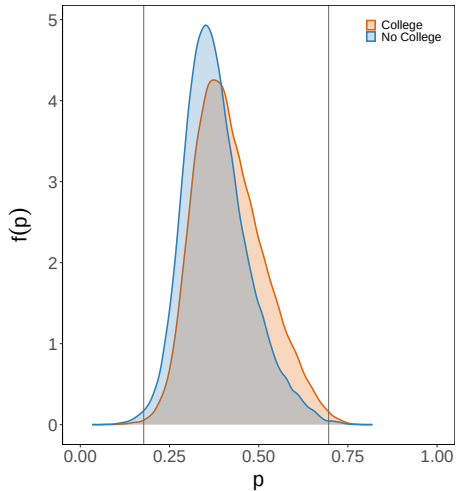
- Individuals with $U = P(Z)$ are at the **margin of indifference** between attending university and not attending

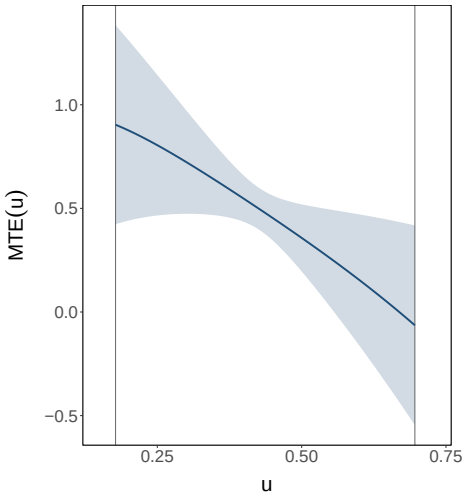
The Marginal Treatment Effect (MTE) is the return to university for individuals at a given point of that margin (Heckman and Vytlacil, 2005):

$$\text{MTE}(u) \equiv \mathbb{E}(Y_1 - Y_0 \mid U = u)$$

- It tells us how the returns to university vary across individuals with different latent resistance to university

Results (I): Pooled propensity $P(Z)$ and MTE





Treatment effects and MTE

- Following Carneiro, Heckman, and Vytlacil (2011) we can compute different treatment effects, τ, as weighted averages of the MTE:

$$\tau = \int_0^1 \text{MTE}(u)\, w^{\tau}(u)\, du$$

- For instance:

$$\text{ATE} = \mathbb{E}(Y_1 - Y_0) = \int_0^1 \text{MTE}(u)\, du$$

$$\text{ATT} = \mathbb{E}(Y_1 - Y_0 \mid E = 1) = \int_0^1 \text{MTE}(u)\, w^{ATT}(u)\, du$$

$$\text{ATU} = \mathbb{E}(Y_1 - Y_0 \mid E = 0) = \int_0^1 \text{MTE}(u)\, w^{ATU}(u)\, du$$

Results (II): Returns to university

	Support		Treatment effects		
Pooled MTE	$u_{\min}$	$u_{\max}$	ATE†	ATT†	ATU†
Unconditional	0.16	0.71	0.457^{***}	0.738^{***}	0.246^{*}
			(0.067)	(0.177)	(0.149)
Conditional on G	0.06	0.90	0.439^{***}	0.927^{***}	0.113
			(0.089)	(0.219)	(0.215)

Notes: Effects on log hourly wages, in log points. †Support-restricted treatment effects. The no-G interval uses trimmed overlap; the continuous-G interval is fixed at $[0.06, 0.90]$. Bootstrap standard errors in parentheses (250 replications per specification). Conditional on G defines the MTE model that includes the EA PGI (G) as an additional observed covariate in X. Stars: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$; two-sided normal tests using bootstrap means and standard deviations.

Large average returns, highest for those who attend: selection on gains

(as in Carneiro, Heckman, and Vytlacil, 2011; Nybom, 2017)

ATE† in line with the **raw graduate wage gap** (Blundell, Green, and Jin, 2016)

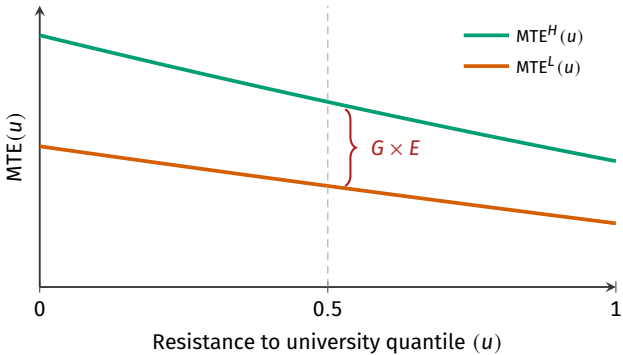
Who gains more from university?

Does selection on gains vary across EA-PGI groups?

Estimating $G \times E$ via MTE

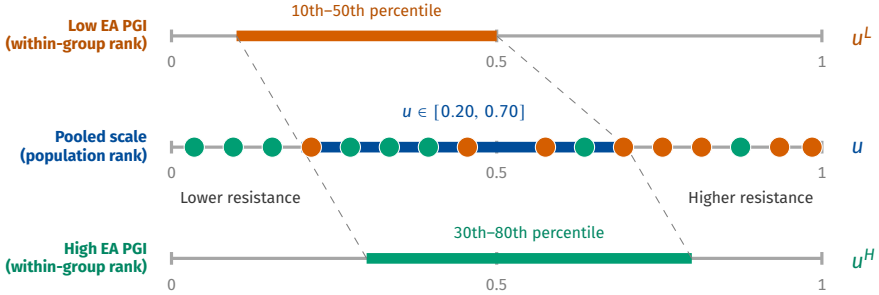
- We want to compare High PGI ($G = H$) individuals to Low PGI ($G = L$)
- **Existing approach:** take the difference between the MTE^g curves at the same rank u

(Hollenbach, Schmitz, and Westphal, 2026)



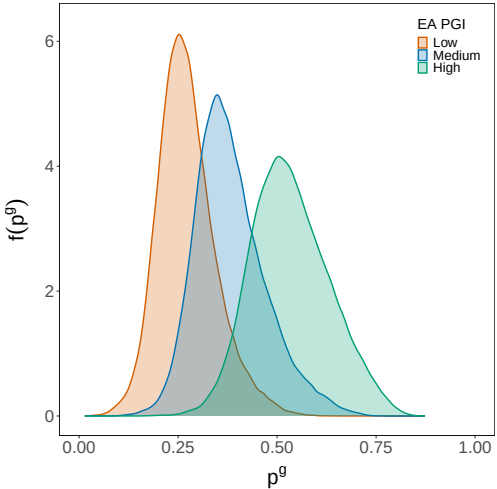
The problem: rank comparability

- **Issue:** the **same quantile** can mean very **different latent resistance** across groups



- Differences in MTE may combine genuine heterogeneity and selection patterns

Results (III): Propensity to university by EA PGI tertile



Our approach: a common resistance scale

- **Novelty:** we allow group-specific resistances (u^g) by introducing a **warp** function:

Definition 1 (warp function)

$$u^g(u) = F^g\left(F^{-1}(u)\right) \; : \; [0,1] \to [0,1]$$

maps the pooled rank u to the group-specific rank u^g

- **Intuition:** $u^g(u)$ tells us the equivalent resistance rank in group g for someone at pooled rank u

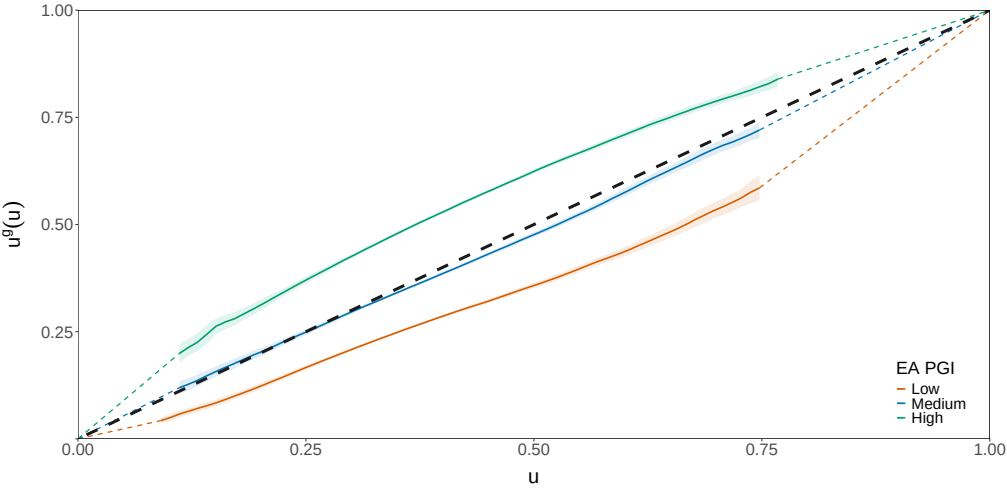
Estimation in three steps

1. **Propensity scores:** probit of university on $\Delta Dist$ and the controls, pooled ($P(Z)$) and within each EA-PGI tertile ($P^g(Z)$)

2. **Warp:** the calibration identity $u^g(P(Z)) = P^g(Z)$ identifies it from the two propensity scores; we estimate it by **isotonic regression** of $\widehat{P}^g(Z)$ on $\widehat{P}(Z)$ ▸ Details
   - Steps 1–2 determine the **supported range** common to all groups

3. **MTE curves:** **local IV** (Heckman and Vytlacil, 1999): the MTE is the slope of $\mathbb{E}[Y \mid P(Z) = p]$ in p, estimated by local polynomial regression, pooled and within each tertile; group curves are then placed on the common scale with the warp ▸ Details

Results (IV): Estimated relationship between resistances



Comparing group-specific MTEs

- Groups exhibit **clearly different latent resistances** to university
- We therefore express all MTEs on a **common resistance scale** using the **warp** $u^g(u)$:

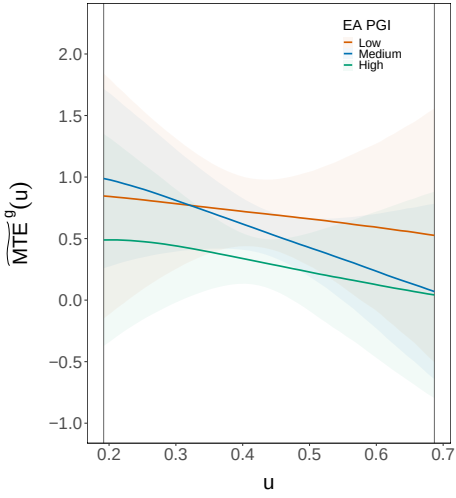
$$\widetilde{\text{MTE}}^{\,g}(u) = \text{MTE}^g\big(u^g(u)\big)$$

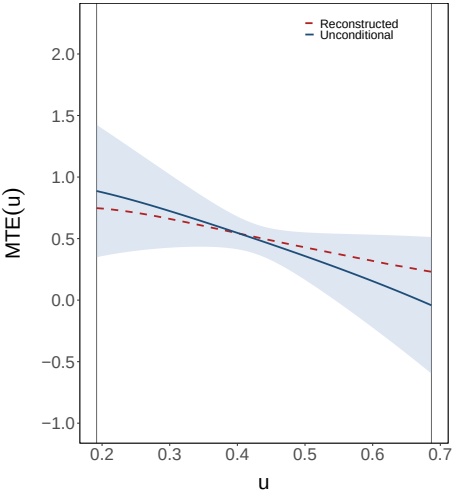
Corollary 1 (reconstruction of the pooled MTE)

The pooled MTE is a **weighted, warped average** of the group-specific MTEs:

$$\text{MTE}(u) = \sum_{g \in \mathcal{G}} \pi_g \, \text{MTE}^g\big(u^g(u)\big) \, \omega_g(u)$$

Results (V): Group-specific MTE





Results (VI): Returns to university by EA PGI

	Support				Treatment effects		
Group-specific MTE	$u^g_{\min}$	$u^g_{\max}$	$u_{\min}$	$u_{\max}$	ATE†	ATT†	ATU†
Low EA PGI	0.10	0.53	0.17	0.70	0.689***	0.774**	0.629*
					(0.177)	(0.390)	(0.336)
Med EA PGI	0.17	0.68	0.17	0.70	0.531***	0.823***	0.330
					(0.120)	(0.285)	(0.218)
High EA PGI	0.28	0.79	0.17	0.70	0.310**	0.427	0.192
					(0.128)	(0.317)	(0.265)

Notes: Effects on log hourly wages, in log points. †Treatment effects are evaluated over the observed common support of the resistance distribution. In the absence of full support, the reported estimates correspond to integrals over the identified region and therefore approximate the population treatment parameters defined over the full support. Bootstrap standard errors in parentheses (250 replications). Support bounds refer to the original sample; bootstrap bounds vary by draw. Stars: $^*p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$; two-sided normal tests using bootstrap means and standard deviations.

Largest returns for the low EA-PGI group, smallest for the high

What drives the gap in returns?

Differences in **gains**, or differences in **selection**?

Disentangling gene-environment interplay

Two pathways of gene-environment interplay



EA PGI variation is associated with **different selection** into university

(*rGE*)

Returns to university vary with one's EA PGI

($G \times E$)

Can they be disentangled?

Decomposition of $G \times E$: Example for the ATE (I)

- The **difference in average returns to university** between the high and low EA PGI groups:

$$\Delta^{\text{ATE}} = \text{ATE}^H - \text{ATE}^L = \underbrace{\int_0^1 \widetilde{\text{MTE}}^H(u)\,\omega_H(u)\,du}_{\text{ATE}^H} - \underbrace{\int_0^1 \widetilde{\text{MTE}}^L(u)\,\omega_L(u)\,du}_{\text{ATE}^L}$$

- The weights $\omega_g(u)$ depend only on **selection** into university:
  - $\omega_H(u) - \omega_L(u)$ reflects different **selection patterns** (***rGE***)
- $\widetilde{\text{MTE}}^g(u)$ is the **return at a given resistance**:
  - $\widetilde{\text{MTE}}^H(u) - \widetilde{\text{MTE}}^L(u)$ reflects different **returns** (***G* × *E***)

Decomposition of $G \times E$: Example for the ATE (II)

Proposition 3 (decomposition; ATE case)

Derivation

$$\Delta^{\text{ATE}} = \underbrace{\int_0^1 \left(\widetilde{\text{MTE}}^{H}(u) - \widetilde{\text{MTE}}^{L}(u)\right) \overline{\omega}(u)\, du}_{G \times E} + \underbrace{\int_0^1 \overline{\text{MTE}}(u) \left(\omega_H(u) - \omega_L(u)\right) du}_{rGE}$$

$\widetilde{\text{MTE}}^{g}$: group MTE on the common scale; ω_g: group weights; $\overline{\omega}$, $\overline{\text{MTE}}$: their averages across groups.

- This **approach is also applicable to other treatment effects** See proposition

Results (VII): Decomposition of ATE

Comparison	Return heterogeneity $G \times E$ (MTE)	Selection *rGE* (Composition)	ATE difference Total
Medium vs Low	−0.161	0.010	−0.151
	(0.215)	(0.013)	(0.218)
High vs Low	−0.403*	0.026	−0.377*
	(0.215)	(0.047)	(0.219)
High vs Medium	−0.262	0.035	−0.226
	(0.177)	(0.031)	(0.179)

Notes: This table decomposes differences in average treatment effects (ATE) across EA PGI tertiles into a sorting component (*rGE*) and a return heterogeneity component ($G \times E$) following Proposition 3. Bootstrap standard errors in parentheses (250 replications). Stars: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$; two-sided normal tests using bootstrap means and standard deviations.

The estimates point mainly to return heterogeneity

Limitation (I): earnings are not observed directly

- Following Kweon et al. (2020) and Barcellos, Carvalho, and Turley (2021), we impute **log hourly wages from 400+ detailed (4-digit) occupations**, using a wage model estimated on the LFS and ASHE

- **Validated out of sample** in an independent survey (BHPS, $N = 32{,}947$)
  - Imputed and observed weekly wages: $R^2 \approx 0.75$ (Kweon et al., 2020)

Limitation (II): sample representativeness

- **Volunteer participation**
  - Participants are healthier and more educated than the population (Fry et al., 2017)
  - Census-based **inverse-probability weights** address participation bias; the MTE estimates shown here are unweighted (van Alten et al., 2024)
- **European ancestry**
  - PGIs are ancestry-specific, so we restrict to unrelated individuals of European ancestry
  - 20 genetic principal components control for population structure (Price et al., 2006)
  - **Looking ahead:** more diverse discovery samples and improved PGI portability offer a route to extending this analysis

Conclusions

Recap

(i) **Selection differs.** The warp reveals distinct sorting into university across EA-PGI groups and places their returns on a common resistance scale

- High EA-PGI individuals are **less resistant to university overall**

(ii) **Estimated returns differ.** Average gains are **0.69 vs 0.31 log points** for low vs high EA-PGI individuals over the common support

(iii) **The decomposition points to differences in gains.** The point estimates attribute most of the gap to differences in returns; sorting plays a smaller role

- High minus low: -0.403 returns $+\,0.026$ sorting $=\,-0.377$ log points

Takeaways

"The natural distribution is neither just nor unjust … What is just and unjust is the way that institutions deal with these facts"

— John Rawls (1971)

- We can now **compare groups at common margins** and **decompose** between-group gaps into differences in **returns** and in **sorting**, for any grouping
- In our context, individuals with a **lower genetic propensity for educational attainment** seem to have gained more from university
- These larger gains suggest that university may **partly offset earnings differences** associated with genetic predisposition to education

University may indeed act as an equaliser

Thank you for listening!

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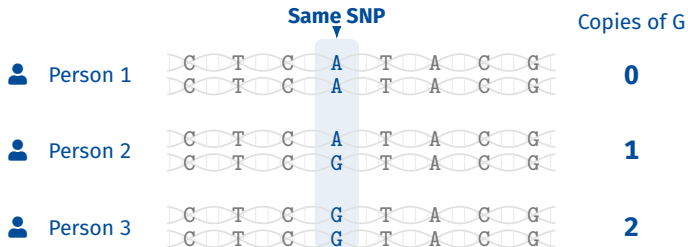
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Genetic variants: differences at the same DNA position

Back

- Human genome: about **3 billion base pairs**, written in four letters (A, C, G, T)
- A **single nucleotide polymorphism (SNP)** is a difference in one letter at the same position across individuals
- **84.7 million SNPs** catalogued by the 1000 Genomes Project (The 1000 Genomes Project Consortium, 2015)



Each person carries two copies of each autosome: the genotype counts 0, 1 or 2 copies of a specified allele.

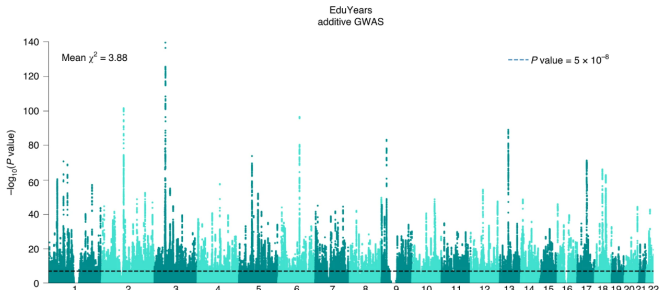
Constructing a PGI (1): discovery

Back

Estimate SNP–education associations in a large discovery sample

$$\text{Education}_i = \alpha_j + \beta_j G_{ij} + C_i' \gamma_j + \varepsilon_{ij}$$

- One regression per SNP j
- G_{ij}: allele count
- C_i: age, sex, ancestry PCs and other cohort-specific controls
- $\widehat{\beta}_j$: **adjusted association**, not necessarily causal



Each dot is a SNP; height indicates statistical significance. (Okbay et al., 2022)

Schematic regression: specifications vary by cohort and can use mixed models. Association estimates are combined across discovery cohorts.

Constructing a PGI (2): weighted sum

Back

- Turn GWAS associations into **prediction weights**
  - Account for correlation between SNPs (linkage disequilibrium) and shrink noisy estimates, e.g. using LDpred (Okbay et al., 2022)
- Apply these weights to each individual's genotypes in the target sample

$$PGI_i = \sum_{j=1}^{J} \widehat{w}_j \, G_{ij}$$

$\widehat{w}_j$: GWAS-derived prediction weight G_{ij}: allele count (or imputed dosage)

- Aggregates many small associations, including SNPs below genome-wide significance
- Standardised to mean zero and standard deviation one in the target sample

Reduction in distance: summary statistics

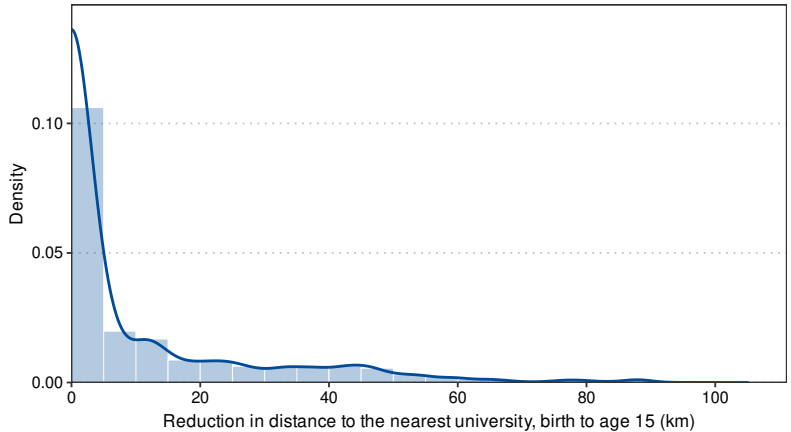
Distribution Back

	(1) Overall	(2) Positive reduction	(3) Negative reduction	(4) No reduction
Mean reduction	3.848	13.275	−0.537	0.000
Standard deviation	11.316	17.758	0.723	0.000
Min	−3.948	0.001	−3.948	0.000
Q1	0.000	1.281	−0.452	0.000
Q2 (median)	0.000	4.288	−0.317	0.000
Q3	1.010	19.391	−0.159	0.000
Max	96.897	96.897	−0.001	0.000
Observations	157,109	48,087	1,834	107,188
Frequency	100.00%	29.06%	1.70%	69.24%

Notes: The reduction is the difference, in kilometres, between the distance to the nearest university active at birth and the distance to the nearest university active by age 15.

Distribution of the instrument

Back



Notes: Histogram (5 km bins) and reflected kernel density of the reduction in distance to the nearest university between birth and age 15, for individuals with a positive reduction (29% of the analysis sample; mean 13 km, median 4 km). The remaining 69% experienced no change and 2% a small increase. Analysis sample (N = 157,109), UKB weights.

University placement was not random

Back

- **A sparse starting point:** in 1962 only about 4% of each generation entered university, and large areas had none nearby
- **Where new universities went:**
  - new campuses in cities without a university (e.g., Sussex, York, Warwick) (Willetts, 2017)
  - Robbins: favour large centres of population (Robbins, 1963)
  - technical colleges upgraded where capacity already existed (Calhoun, 2014)
- **Distance remained a real cost:** grants (1962) and admissions (UCCA) became national, but attending usually meant relocating and finding lodgings (Robbins, 1963; Hillman, 2013)

Placement followed population and existing provision: we control for both

Which districts got a university?

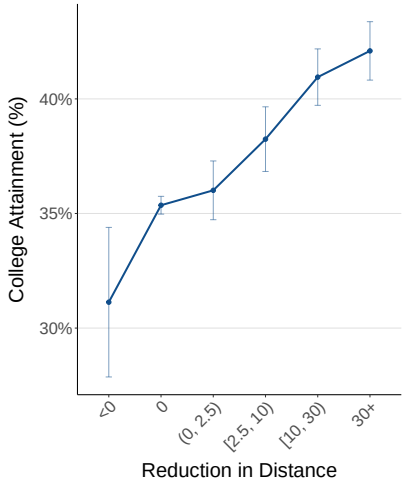
Back

	(1) Any opening	(2) Opening count
Log population, 1951	0.034*** (0.010)	0.072*** (0.022)
Baseline universities within 10 km	0.028* (0.014)	0.079* (0.041)
Share educated over age 15	0.013** (0.007)	0.019 (0.012)
Unemployment rate, 1951	−0.010 (0.008)	−0.025* (0.015)
Urban district	−0.010 (0.021)	0.029 (0.035)
Log population density, 1951	0.006 (0.009)	−0.007 (0.018)
Population growth, 1946–1951	−0.006 (0.004)	−0.006 (0.007)
Low-SES share	0.007 (0.006)	0.010 (0.009)
High-SES share	0.006 (0.006)	0.013 (0.010)
Region fixed effects	Yes	Yes
Districts	1,468	1,468
R^2	0.087	0.114

Notes: Unit: 1951 England–Wales local-government district (full district universe with complete characteristics). Outcome: any campus opening or university-status acquisition in the district in 1945–1980, or their count. Baseline universities: number active in 1944 within 10 km. Continuous predictors standardised; urban is a dummy. HC1 SEs in parentheses. Without region FE, unemployment also predicts openings (across regions; absorbed by our region × cohort FE). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

Reduction in distance and university attainment

▸ Functional forms ◂ Back



Alternative functional forms

Back

	(1) Baseline	(2) Quadratic	(3) Log
Panel A: First stage			
Dependent variable: University			
$\Delta Dist$	0.014^{***}	0.013^{**}	
	(0.002)	(0.007)	
$\Delta Dist^2$		0.000	
		(0.001)	
$\Delta \log(Dist)$			0.014^{**}
			(0.006)
First-stage F	35.47	21.49	5.45
Panel B: Reduced form			
Dependent variable: Log wage			
$\Delta Dist$	0.006^{***}	0.008^{**}	
	(0.001)	(0.004)	
$\Delta Dist^2$		−0.000	
		(0.001)	
$\Delta \log(Dist)$			0.009^{***}
			(0.003)
Observations	157,109	157,109	157,109

Notes: $\Delta Dist$ per 10 km; column (3) uses the change in log distance. In column (2) the F-statistic tests $\Delta Dist$ and $\Delta Dist^2$ jointly. Baseline controls, birth-region-by-cohort FE, 20 genetic PCs and UKB weights in all columns. SEs clustered by district of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

First-stage robustness to geographic fixed effects

▸ 2SLS ◂ Back

	(1) Region × cohort FE	(2) Region + cohort FE	(3) Region + cohort FE + region trends	(4) County × cohort FE
$\Delta Dist$	0.014^{***}	0.011^{***}	0.012^{***}	0.010^{***}
	(0.002)	(0.002)	(0.002)	(0.003)
Observations	157,109	157,109	157,109	157,109
R^2	0.090	0.087	0.088	0.108
First-stage F	35.47	28.89	31.68	10.78
Controls (X)	Yes	Yes	Yes	Yes

Notes: Dependent variable: completed university. $\Delta Dist$ per 10 km. Column (1) is the baseline; column (4) absorbs birth-county-by-cohort fixed effects. All columns use the same sample, baseline controls, genetic PCs and UKB weights. SEs clustered by district of birth; F is the cluster-robust Wald statistic for the excluded instrument. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

Balance of predetermined characteristics

Back

	(1) EA PGI	(2) Male	(3) Number of siblings
$\Delta Dist$	0.007 (0.005)	−0.001 (0.002)	−0.008 (0.005)
Mean dep. var.	0.022	0.476	1.860
Observations	157,109	157,109	157,109

Notes: Each column is a separate weighted OLS regression of the predetermined outcome on $\Delta Dist$ (per 10 km), with baseline controls and birth-region-by-cohort FE. An outcome that is itself a baseline control is dropped from the control set. Mean dep. var. is unweighted. SEs clustered by district of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

Placebo tests: openings after age 25

Back

	(1) 25 → 30	(2) 25 → 35	(3) 25 → 40
Panel A: First stage			
Dependent variable: University			
Δ*Dist*	0.003	0.004	0.004
	(0.004)	(0.004)	(0.005)
Placebo *F*-statistic	0.51	0.74	0.69
Joint Wald test (*p*-value)		0.843	
Panel B: Reduced form			
Dependent variable: Log wage			
Δ*Dist*	0.001	0.002	0.001
	(0.003)	(0.002)	(0.002)
Joint Wald test (*p*-value)		0.831	
Observations		157,109	

Notes: Placebo Δ*Dist* (per 10 km) counts only universities gaining university status after age 25 and by the upper age indicated, relative to the nearest university at birth. Baseline controls, EA PGI and its interaction with sex, birth-region-by-cohort FE, 20 genetic PCs and UKB weights in all columns. SEs clustered by district of birth. The joint Wald test is for all three windows jointly, accounting for their overlap. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

Alternative exposure windows and university definitions

Back

	Exposure window					University definition		
	(1) Birth→11	(2) Birth→12	(3) Birth→13	(4) Birth→14	(5) Birth→15	(6) All	(7) Pure new	(8) Other
Panel A: First stage								
Dependent variable: University								
Δ*Dist*	0.014***	0.014***	0.014***	0.014***	0.014***	0.014***	0.020***	0.010***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.004)	(0.003)
First-stage *F*	38.37	37.06	35.58	35.93	35.47	35.47	22.11	14.50
Panel B: Reduced form								
Dependent variable: Log wage								
Δ*Dist*	0.006***	0.006***	0.006***	0.006***	0.006***	0.006***	0.009***	0.005***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)
Observations	157,109							

Notes: Δ*Dist* per 10 km. Columns (1)–(5) vary the age by which openings enter the instrument; (5) and (6) are the baseline. Columns (7)–(8) split the baseline instrument by type of entry: purpose-built post-war universities (e.g., Sussex, York, Warwick) vs. status conversions and other entries. Baseline controls, birth-region-by-cohort FE, 20 genetic PCs and UKB weights in all columns. SEs clustered by district of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$.

Benchmark specification

Back

- **First stages:**

$$E_i = \pi_0 + \pi_1 G_i + \pi_2 Z_i + \pi_3 (Z_i \times G_i) + \delta X_i + \mu_{r(i)} \times \alpha_{c(i)} + v_{1,i}$$
$$(G_i \times E_i) = \theta_0 + \theta_1 G_i + \theta_2 Z_i + \theta_3 (Z_i \times G_i) + \eta X_i + \mu_{r(i)} \times \alpha_{c(i)} + v_{2,i}$$

- **Second stage:**

$$Y_i = \beta_0 + \beta_1 G_i + \beta_2 \widehat{E_i} + \boldsymbol{\beta_3} \widehat{(G_i \times E_i)} + \gamma X_i + \mu_{r(i)} \times \alpha_{c(i)} + \varepsilon_i$$

Y	Log-hourly imputed wage	Z	Reduction in distance to university
E	University dummy	X	Vector of controls
G	Educational Attainment PGI	$\mu_{r(i)} \times \alpha_{c(i)}$	Region-of-birth $\times$ cohort fixed effects
		v_1, v_2, ε	Idiosyncratic error terms

Benchmark: full second-stage results

Back

	Continuous G		Discrete G
	(1) Baseline	(2) County × cohort FE	(3) Baseline
University	0.512^{***}	0.588^{***}	0.515^{***}
	(0.076)	(0.115)	(0.128)
EA PGI	0.034^{*}	0.029	
	(0.017)	(0.019)	
University × EA PGI	-0.093^{**}	-0.104^{***}	
	(0.041)	(0.038)	
University × EA PGI Medium			−0.052
			(0.124)
University × EA PGI High			−0.168
			(0.146)
EA PGI Medium			0.020
			(0.036)
EA PGI High			0.073
			(0.062)
Num. Obs.	157,109	157,109	157,109
First-stage F-statistic	19.25	5.53	12.43
Adjusted R^2	0.188	0.195	0.183
Fixed effects	Region × cohort	County × cohort	Region × cohort

Notes: Outcome: log hourly wage. All specifications include controls (X) and birth-geography-by-cohort fixed effects. SEs in parentheses, robust to heteroskedasticity and clustered by district of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.10$. UKB weights applied (van Alten et al., 2024). University and its interactions are instrumented using Reduction in Distance and its interactions with G. Discrete G denotes EA-PGI tertiles, with Low as the reference category.

IV model with G setup

- Modified Roy model (Roy, 1951):
  - **Choice equation:** $E = f_E(Z, V), \quad E \in \{0, 1\}$
  - **Outcome equation:** $Y = f_Y(E, V, \varepsilon_Y)$
  - **Confounding vector:** $\boldsymbol{V} = f_V(G, \varepsilon_V)$
  - **Independence condition:** $G, Z, \varepsilon_Y, \varepsilon_V$ are mutually independent
- Standard IV conditions:
  - Relevance
  - Exclusion
  - Exogeneity: $Z, G, \varepsilon_Y, \varepsilon_V$ are mutually independent
  - Monotonicity

Resistance to treatment ranks

- For any measurable $\psi : \mathbb{R}^d \to \mathbb{R}$ we have that $S \equiv \psi(\boldsymbol{V})$ is a random variable:

$$F_S(s) = Pr(S \leq s)$$

- For individuals in group $g \in \mathcal{G}$:

$$F_S^g(s) = Pr(S \leq s \mid G = g)$$

- Which allows to **rank individuals based on their unobservables**:

$$U \equiv F_S(S) \sim \text{Unif}(0,1)$$
$$U^g \equiv F_S^g(S) \mid G = g \ \sim \ \text{Unif}(0,1)$$

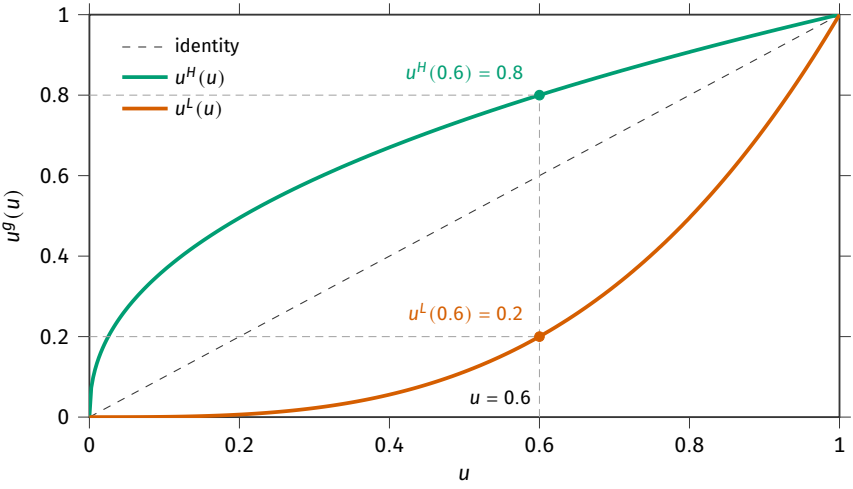
Treatment rules

- **Latent index:** $E = \mathbb{1}\{\zeta(Z) \geq S\}$ for some function ζ and $S \equiv \psi(\boldsymbol{V})$
  - Equivalent to independence and monotonicity (Vytlacil, 2002); writing ranks as $U \sim \text{Unif}(0,1)$ also needs S continuously distributed (A5)
- Since $Z \perp S$, $P(Z) = \Pr(S \leq \zeta(Z)) = F_S(\zeta(Z))$, and applying F_S to both sides of the index:

$$E = \mathbb{1}\{P(Z) \geq U\}, \qquad E = \mathbb{1}\{P^g(Z) \geq U^g\} \text{ on } \{G = g\}$$

- $P(Z) \equiv P(E = 1 \mid Z)$: **pooled propensity**; $P^g(Z) \equiv P(E = 1 \mid Z, G = g)$: **group-specific propensity**
- U and U^g rank individuals by unobserved resistance, in the pooled population and within group g
- Individuals at $P(Z) = U$ (or $P^g(Z) = U^g$ on $\{G = g\}$) are indifferent

The warp: illustration



Properties of the warp

Proposition 1 (properties of the warp)

Let F_S and F_S^g, $g \in \mathcal{G}$, be continuous and strictly increasing on a common support, with densities $f_S > 0$ and $f_S^g > 0$ on the interior. Then:

(i) **Monotonicity and derivative.** u^g is strictly increasing, with

$$\omega_g(u) \equiv (u^g)'(u) = \frac{f_S^g(F_S^{-1}(u))}{f_S(F_S^{-1}(u))} > 0$$

(ii) **Endpoints.** $u^g(0) = 0$ and $u^g(1) = 1$, under common support and no boundary atoms

(iii) **Mixture identity.** $\sum_g \pi_g \, u^g(u) = u$ for all $u \in [0, 1]$, where $\pi_g \equiv \Pr(G = g)$

(iv) **Calibration identity.** $u^g\big(P(Z)\big) = P^g(Z)$ a.s. in Z, under the treatment rules

(v) **Posterior composition.** $\Pr(G = g \mid U = u) = \pi_g \, \omega_g(u)$, and therefore $\sum_g \pi_g \, \omega_g(u) = 1$

- The **tilt weight** $\omega_g(u)$: how much group g contributes to the pooled resistance distribution at rank u

Estimation: warp and tilt weights

Back

- By the calibration identity, $u^g(\cdot)$ is the monotone regression function of $P^g(Z)$ on $P(Z)$:

$$\hat{u}^g(\cdot) \equiv \arg\min_{m \in \mathcal{M}} \sum_{i:\, G_i = g} \left(\hat{P}_i^g - m(\hat{P}_i) \right)^2, \qquad \mathcal{M}: \text{ monotone functions } [0,1] \to [0,1]$$

- Solved by **isotonic regression**: a nonparametric fit that preserves the order of the resistance ranks
- The isotonic fit is piecewise constant, so we smooth it with a kernel-weighted local quadratic estimator and take the derivative:

$$\hat{\omega}_g(u) = \frac{\partial\, \hat{u}^g(u)}{\partial u}, \qquad \text{normalised so that } \sum_g \pi_g\, \hat{\omega}_g(u) = 1$$

Estimation: local IV

Back

- Under the model, the MTE is identified as the derivative of the outcome in the propensity score (Heckman and Vytlacil, 1999):

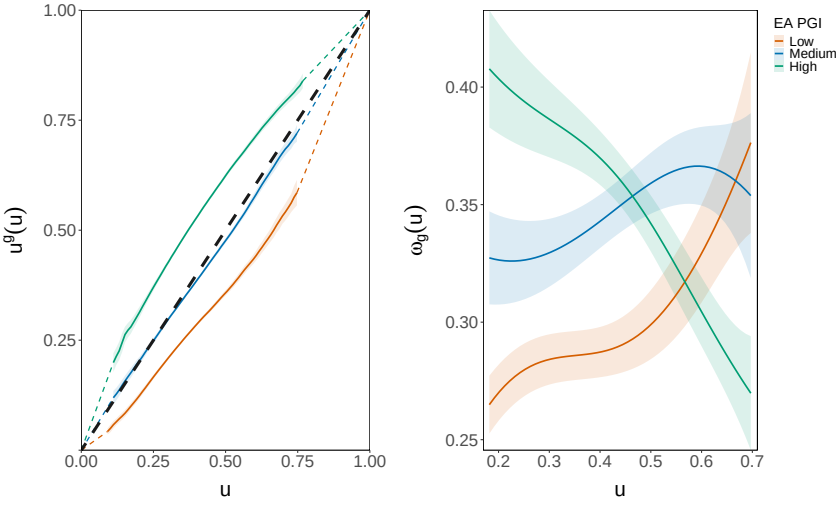
$$\text{MTE}(x, u) = \left. \frac{\partial \, \mathbb{E}\big[Y \mid X = x, \; P(Z) = p\big]}{\partial p} \right|_{p=u}$$

1. **Propensity score:** probit for $\widehat{P}(Z)$; restrict to the trimmed common support of treated and untreated
2. **Unobservable part:** partial out $\widehat{P}(Z)$ from Y, X and $X \cdot \widehat{P}(Z)$ with local linear smoothers; regress residualised Y on the residualised regressors to estimate the observable part, and subtract it from Y to obtain $\tilde{Y}$
3. **Estimate the slope:** fit a **local quadratic** to $\tilde{Y}$ in $\widehat{P}(Z)$ around each p: its **linear coefficient** estimates the slope $\partial \, \mathbb{E}[\tilde{Y} \mid \widehat{P}(Z) = p] / \partial p$; combine with the coefficients on X to obtain $\widehat{\text{MTE}}(x, u)$

- Repeated within each EA-PGI tertile for MTE^g, with **no separability or parallel-slope restrictions** across groups

Implemented in our extension of the `localIV` R package (Zhou, 2020)

Estimated warp and tilt weights



Decomposition of $G \times E$: Example for the ATE (I)

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- We are interested in the **difference in average returns to university** between the high and low EA PGI groups ($G = H$ vs. $G = L$) in the full population, i.e.,

$$\Delta^{\text{ATE}} = \text{ATE}^{H} - \text{ATE}^{L}$$

- The group-specific ATE is:

$$\text{ATE}^{g} = \int_0^1 \text{MTE}^{g}(u)\, \omega_g(u)\, du, \quad g \in \{H, L\}$$

- Hence,

$$\Delta^{\text{ATE}} = \underbrace{\int_0^1 \text{MTE}^{H}(u)\, \omega_H(u)\, du}_{\text{ATE}^{H}} - \underbrace{\int_0^1 \text{MTE}^{L}(u)\, \omega_L(u)\, du}_{\text{ATE}^{L}}$$

Decomposition of $G \times E$: Example for the ATE (II)

- The **tilt weights** $\omega_G(u) = \dfrac{f_S^G(F_S^{-1}(u))}{f_S(F_S^{-1}(u))}$ depend only on selection:
  - Variation in G linked to variation in $E \implies$ ***rGE***
- $\text{MTE}^g(u) = \mathbb{E}[Y_1 - Y_0 \mid U^g = u, G = g]$ measures the **treatment effect at a given resistance** level
  - Different returns at a given resistance (linked to propensity) to university $\implies$ $\boldsymbol{G \times E}$

Decomposition of $G \times E$: Example for the ATE (III)

- Suppose there are no differences in $\text{MTE}^g(u)$ across G:

$$\text{MTE}^H(u) = \text{MTE}^L(u) = \overline{\text{MTE}}(u)$$

- Then,

$$\Delta^{\text{ATE}} = \int_0^1 \overline{\text{MTE}}(u) \left(\omega_H(u) - \omega_L(u)\right) du$$

- The difference in the ATE arise from the weights (***rGE***)

Decomposition of $G \times E$: Example for the ATE (IV)

- Instead, suppose there are no distributional differences across G, then:

$$\omega_H = \omega_L = \overline{\omega}(u)$$

- Then,

$$\Delta^{\text{ATE}} = \int_0^1 \left(\text{MTE}^H(u) - \text{MTE}^L(u) \right) \overline{\omega}(u)\, du$$

- All differences in the ATE arise from the difference in the MTE ($\boldsymbol{G \times E}$)

Decomposition of $G \times E$: Example for the ATE (V)

- Put together:

$$\Delta^{\text{ATE}} = \underbrace{\int_0^1 \left(\text{MTE}^H(u) - \text{MTE}^L(u)\right) \overline{\omega}(u)\, du}_{G \times E} + \underbrace{\int_0^1 \overline{\text{MTE}}(u) \left(\omega_H(u) - \omega_L(u)\right) du}_{rGE}$$

- Where:
  - $\overline{\omega}(u) = \frac{1}{2}\left(\omega_H(u) + \omega_L(u)\right)$
  - $\overline{\text{MTE}}(u) = \frac{1}{2}\left(\text{MTE}^H(u) + \text{MTE}^L(u)\right)$
- This **approach is also applicable to other treatment effects** ▸ See proposition

Generalisation of the decomposition of differences in TE

- Let $A_g(u) \equiv \text{MTE}^g\big(u^g(u)\big)$, $W_g(u) \equiv B_g(u)\, C_g(u)$
  - where $B_g(u) \equiv w_\tau^g\big(u^g(u)\big)$ and $C_g(u) \equiv \omega_g(u)$
- Then, the g-specific TE is: $\tau^g = \int_0^1 A_g(u)\, B_g(u)\, C_g(u)\, du$

Proposition 3 (decomposition)

The difference $\tau^g - \tau^{g'}$ admits the following exact decomposition:

$$\tau^g - \tau^{g'} = \int_0^1 \left(\frac{W_g + W_{g'}}{2}\right)(A_g - A_{g'})\, du \qquad \text{(MTE channel)}$$

$$+ \int_0^1 \left(\frac{A_g + A_{g'}}{2} \cdot \frac{C_g + C_{g'}}{2}\right)(B_g - B_{g'})\, du \qquad \text{(Target-weight channel)}$$

$$+ \int_0^1 \left(\frac{A_g + A_{g'}}{2} \cdot \frac{B_g + B_{g'}}{2}\right)(C_g - C_{g'})\, du \qquad \text{(Composition channel)}$$

Decomposition of differences in treatment effects

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