

Trading social status for genetics in marriage markets: Evidence from Great Britain and Norway



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Background

Genetics explains c. 50% of variation in many human characteristics.

What's a social scientist to do?

One answer: **make genetics the dependent variable.**

Assortative mating

- Hugh-Jones, Abdellaoui et al. (2016). Assortative Mating on Education Leads to Genetic Spousal Resemblance for Causal Alleles. *Intelligence* 59.
- This paper...

Migration

- Abdellaoui, Hugh-Jones, ..., Visscher (2019). Genetic Correlates of Social Stratification in Great Britain. *Nature Human Behaviour* 3.

Natural selection

- Hugh-Jones and Abdellaoui (2022). Human Capital Mediates Natural Selection in Contemporary Humans. *Behavior Genetics* 52:4.

Goals of this paper

In increasing order of ambition:

- Explain a puzzle about the **intergenerational persistence of inequality**.
- Provide a new explanation of the **genes-SES (socio-economic status) gradient**.
- Rethink the **nature of inequality** in historical human societies.
- Change how we think about **genetic variation**.

Many genetic measures, including polygenic scores for education and health outcomes, differ between people of low and high socio-economic status (SES).

The leading explanation for this **genes-SES gradient** is meritocracy: genetic variants that cause success in *labour markets* lead to upward mobility.

An alternative explanation: both some genetic variants, and high SES, are desirable qualities in **marriage markets**.

If you are rich or privileged, you may marry someone intelligent or good-looking.
Both SES and genetics are then inherited by the next generation.

Under **Social-Genetic Assortative Mating**:

- Shocks to SES are reflected in the DNA of subsequent generations.
- The size of the genes-SES gradient depends on social structure, e.g. on persistence of inherited wealth.
- The genes-SES gradient is likely historically widespread, beyond modern meritocracies.

Literature

Large, mostly separate, literatures on assortative mating in economics and genetics.

Genetics: genetic assortative mating (GAM) (Rao, Morton, and Yee 1976; Heath and Eaves 1985; Otto, Christiansen, and Feldman 1995), including cross-trait assortative mating (Beauchamp et al. 2010; Sundet et al. 2005; Border et al. 2022).

Economics: models and empirics on assortative mating and inequality, including cross-trait assortative mating (Fernández and Rogerson 2001; Fernandez, Guner, and Knowles 2005; Eika, Mogstad, and Zafar 2019; Chiappori, Dias, and Meghir 2018).

Genetic theory predicted in the 1970s that genetically and culturally transmitted traits could become associated in the population (Rao, Morton, and Yee 1976).

Despite this, previous work has not drawn conclusions for the genes-SES gradient (e.g. Rimfeld et al. 2018) or made the link with social structure.

- Ours is the first post-genomic revolution empirics to directly demonstrate SGAM.
- Our model provides a microeconomic foundation for SGAM, which might help bridge the two literatures.

Model

Traits (genetic) and (SES) are normally distributed in the population.

Attractiveness is where α is the relative importance of genes.

People mate assortatively.

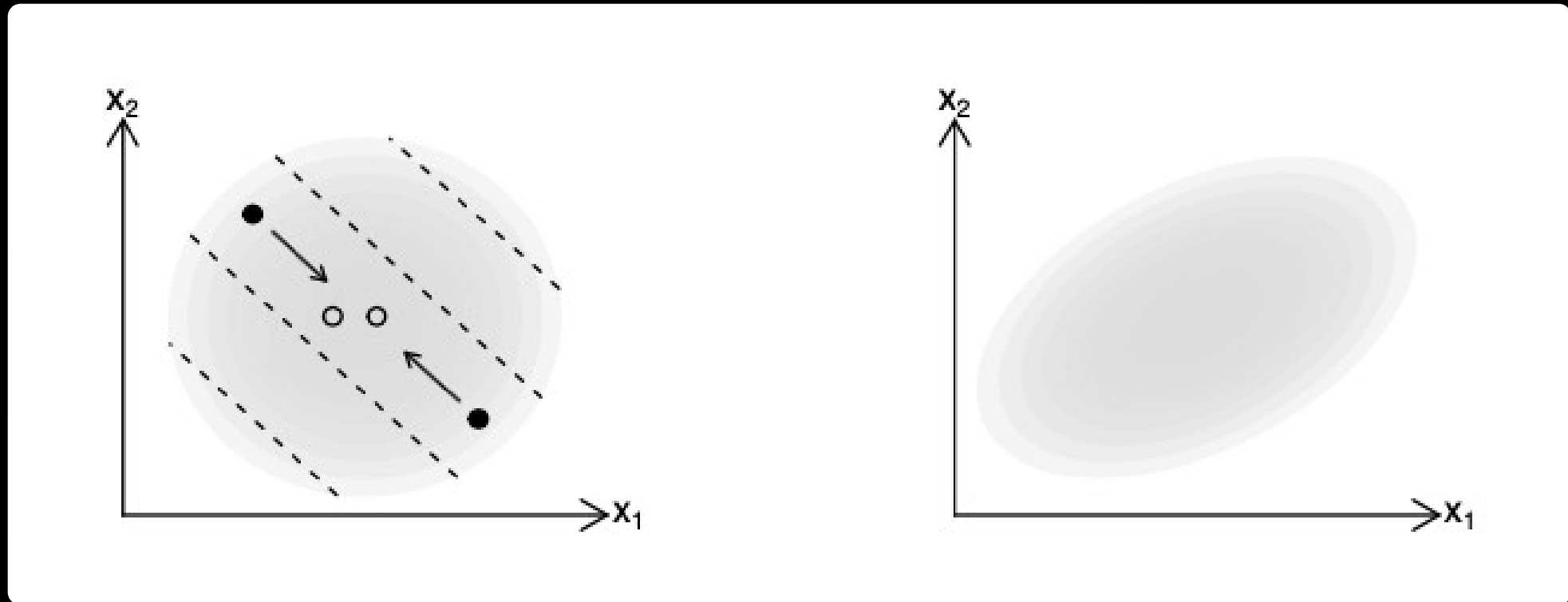
Children inherit

Where:

α is close to 1 (genetic inheritance).

β reflects persistence of SES (e.g. inheritance tax rate τ).

Intuition



Parents (●) mate along iso-attractiveness curves.

Their children (○) are between them.

As a result, the children's distribution is squashed along the attractiveness gradient.

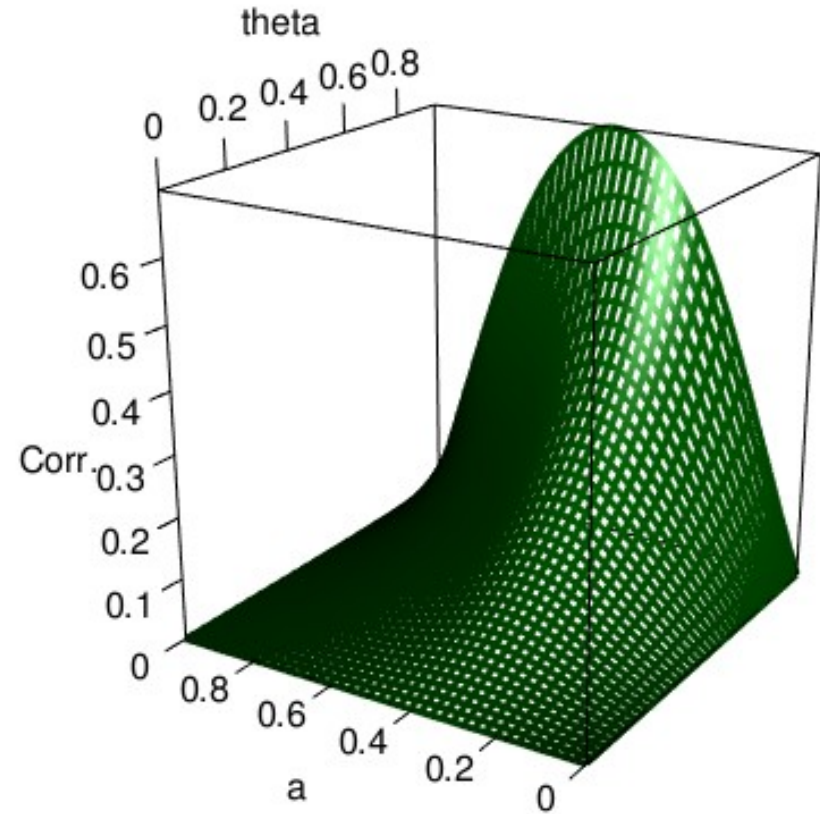
Result

If parents' θ and a are independent, children's θ and a are positively correlated for θ .

The correlation increases in persistence of SES (θ).

The same holds for the long-run distribution.

Extensions



Long-run correlation between θ and a , by θ and a

Data

- UK Biobank, a study of about 500,000 individuals born 1935-1970.
UKBB has no explicit information on spouse pairs. We match pairs manually, and check using genetic children of the pairs. This gives 35,682 spouse pairs.
- MOBA, a study of about 100,000 mothers and babies born in Norway.

Our dependent variable is spouse's **Polygenic Score for Educational Attainment** (PSEA).

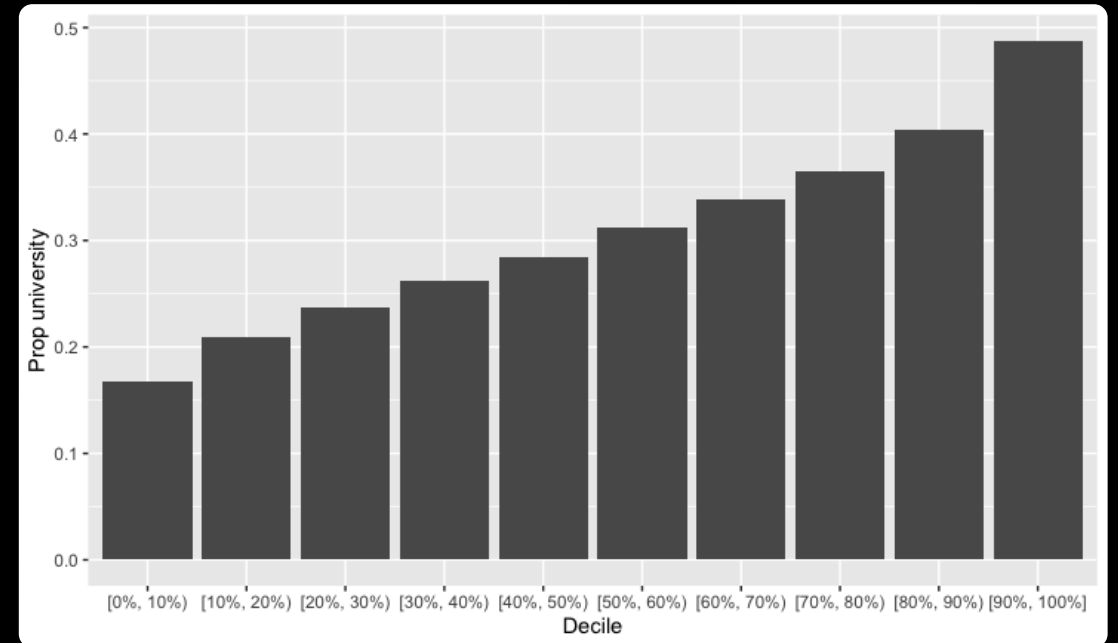
This is a DNA-derived summary statistic which predicts people's level of educational attainment.

Polygenic Score for Educational Attainment

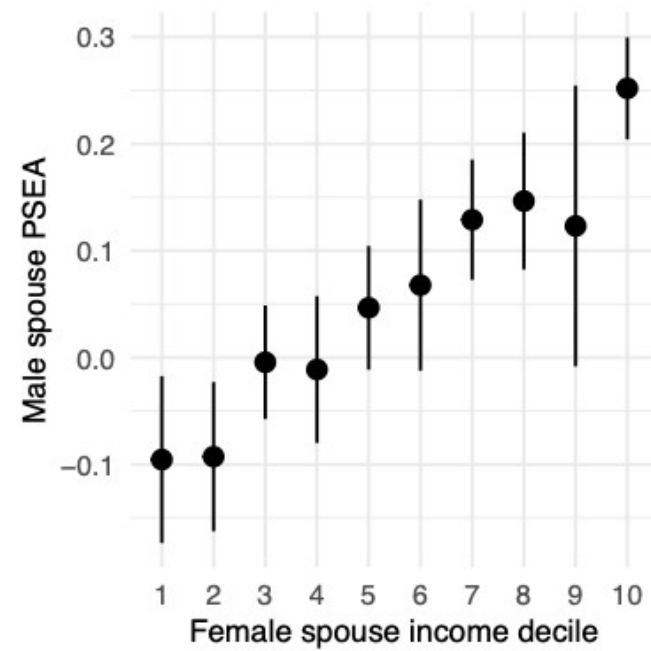
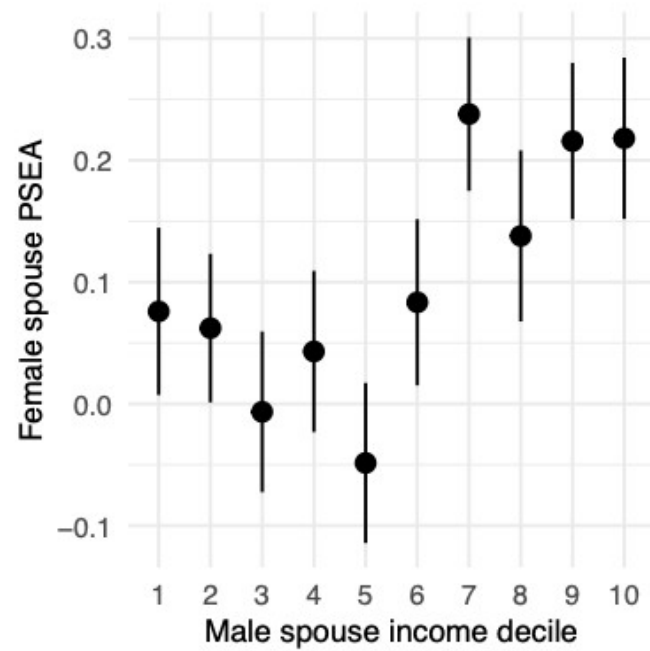
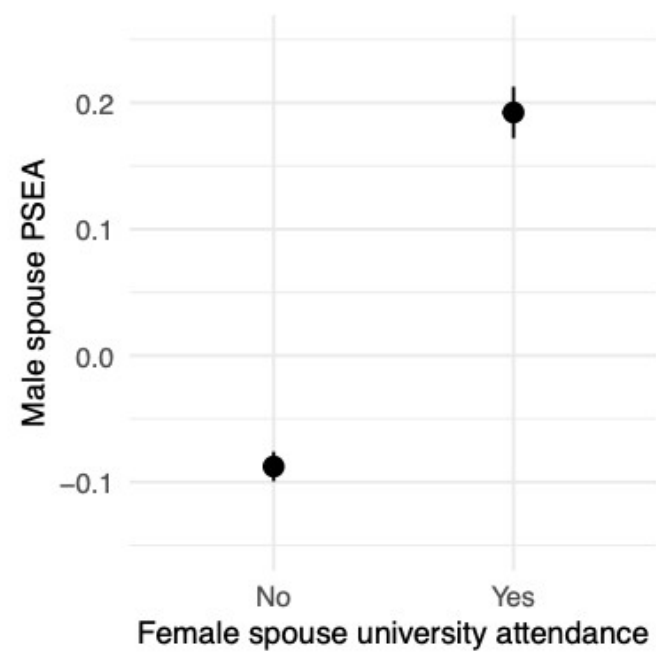
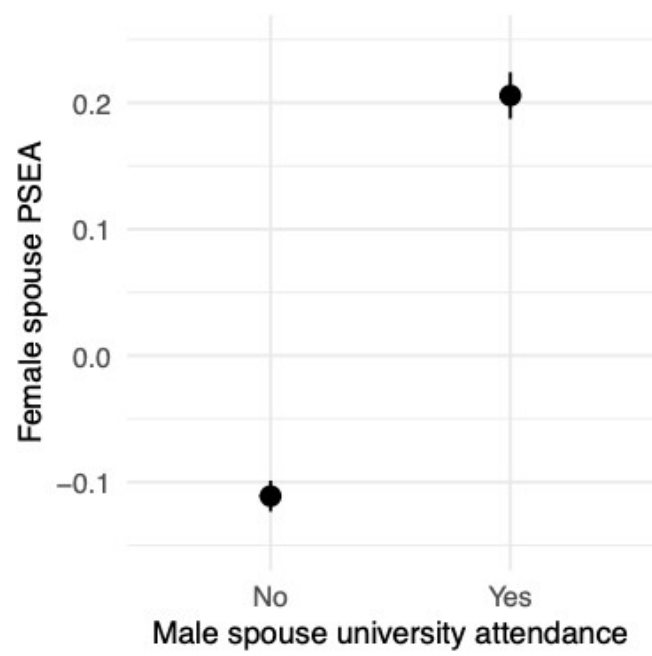
Linear regression of probability of attending university on PSEA:
1 sd PSEA = 9.2 percentage points.

Within-siblings regression (causal):
4.5 percentage points.

Substantial confounding with family environment, but large causal effects remain.



University attendance by PSEA decile



These results could be confounded by the individual's own genetics.

- We already know that there is assortative mating on PSEA (Hugh-Jones et al. 2016).

We need an independent variable which is

- independent of genetics;
- available for a large enough N.
 - Polygenic scores and causes of variation in SES are noisy
 - The spouse matching process is unpredictable (Shakespeare 1595)

We use **birth order**.

- Siblings have the same expected polygenic scores, by the “**lottery of meiosis**”.
- Early-born siblings receive more parental care and have better life outcomes, including **socio-economic status (SES)**.

Estimation strategy

Hard to justify instrumental variables:

- Birth order affects other things than SES.
- We only have imperfect measures of SES (estimated income, educational attainment).

Instead we run a **mediation analysis**:

- Does birth order affect spouse's PSEA?
- Is the effect mediated by measures of SES?

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Controls and mediators

SES mediators

University attendance

Median earnings of first job (guesstimated from SOC code)

Non-SES mediators

Fluid IQ

Height

BMI

Self-reported health

Controls

Family size

Month of birth, year of birth

Parent's age at birth (only available for some respondent)



Table 1: Regressions of mediators on birth order

	University	Income	Fluid IQ	Height	BMI	Health
Birth order	−0.0790 *** (0.0067)	−1.0899 * (0.4264)	−0.2733 *** (0.0304)	−0.7012 *** (0.1355)	0.1907 ** (0.0662)	−0.0430 *** (0.0103)
PSEA	0.0889 *** (0.0046)	1.5144 *** (0.3307)	0.3180 *** (0.0200)	0.1970 * (0.0921)	−0.4281 *** (0.0456)	0.0533 *** (0.0068)
Parents' age at birth	0.0163 *** (0.0012)	0.2623 *** (0.0722)	0.0588 *** (0.0053)	0.1514 *** (0.0241)	−0.0989 *** (0.0117)	0.0110 *** (0.0018)
Family size dummies	Yes	Yes	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes	Yes	Yes
N	10220	3412	10220	10220	10220	10220
R2	0.074	0.026	0.058	0.017	0.023	0.018

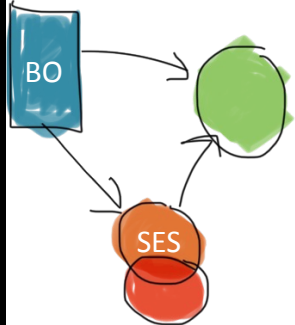


Table 2: Regressions of spouse PSEA on birth order

	(1)	(2)	(3)
Birth order	−0.0091 (0.0074)	−0.0075 (0.0074)	−0.0314 * (0.0146)
Own PSEA		0.0650 *** (0.0065)	0.0573 *** (0.0100)
Parents' age at birth			0.0116 *** (0.0026)
Family size dummies	Yes	Yes	Yes
Birth month dummies	No	Yes	Yes
Birth year dummies	No	Yes	Yes
N	23840	23797	10206
R ²	0.003	0.010	0.013

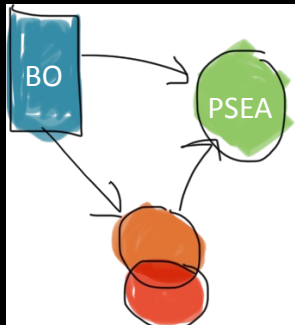


Table 3: Regressions of spouse PSEA on birth order and mediators

	(1)	(2)	(3)	(4)
Birth order	−0.0314 *	−0.0045	−0.0106	−0.0042
	(0.0146)	(0.0146)	(0.0270)	(0.0270)
University		0.2179 ***		0.1538 ***
		(0.0225)		(0.0377)
Income			0.0037 ***	0.0031 **
			(0.0011)	(0.0011)
Fluid IQ		0.0172 **	0.0201 *	0.0112
		(0.0053)	(0.0094)	(0.0097)
Height		0.0029 **	0.0046 *	0.0043 *
		(0.0011)	(0.0020)	(0.0019)
BMI		−0.0109 ***	−0.0114 **	−0.0109 **
		(0.0022)	(0.0040)	(0.0040)
Self-reported health		0.0181	0.0145	0.0077
		(0.0151)	(0.0272)	(0.0271)
Own PSEA	0.0573 ***	0.0263 **	0.0218	0.0118
	(0.0100)	(0.0101)	(0.0184)	(0.0185)
Parents' age at birth	0.0116 ***	0.0053 *	0.0091 +	0.0078 +
	(0.0026)	(0.0026)	(0.0047)	(0.0047)
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
N	10206	10206	3407	3407
R ²	0.013	0.032	0.030	0.034
logLik	−14297.465	−14197.703	−4810.934	−4802.396
AIC	28694.930	28505.406	9731.869	9716.791

SES mediators

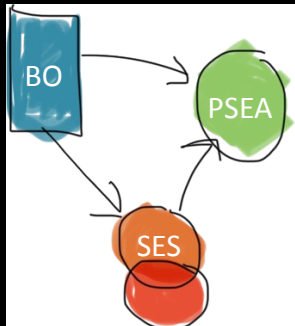
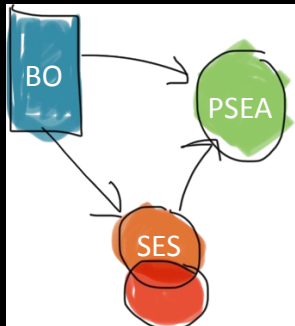
Non-SES
mediators

Table 4: Percent of birth order effects accounted for by mediators, models 2-4

	Model 2 (%)	Model 3 (%)	Model 4 (%)
University	54.9		38.7
Income		13.0	10.6
Fluid IQ	15.0	17.6	9.7
Height	6.6	10.4	9.5
BMI	6.6	7.0	6.6
Self-reported health	2.5	2.0	1.1



Robustness

Norway

	(1)	(2)	(3)	(4)
Birth order	−0.0684 *** (0.0051)	−0.0426 *** (0.0054)	−0.0652 *** (0.0054)	−0.0428 *** (0.0054)
University		0.3049 *** (0.0075)		0.3091 *** (0.0076)
Income			0.0053 *** (0.0013)	−0.0043 ** (0.0014)
Height		0.0033 *** (0.0004)	0.0061 *** (0.0004)	0.0032 *** (0.0004)
BMI		−0.0206 *** (0.0009)	−0.0240 *** (0.0009)	−0.0208 *** (0.0009)
Own PSEA	0.1193 *** (0.0034)	0.0699 *** (0.0036)	0.1068 *** (0.0035)	0.0704 *** (0.0036)
Parents' age at birth	0.0132 *** (0.0008)	0.0085 *** (0.0008)	0.0125 *** (0.0008)	0.0085 *** (0.0008)
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
N	89308	80465	80503	80465
R^2	0.032	0.060	0.041	0.060

Norway

Table 7: Percent of birth order effects accounted for by mediators
(Norway)

	Model 2 (%)	Model 3 (%)	Model 4 (%)
University	35.5		36.0
Income		0.9	−0.7
Height	2.3	4.3	2.3
BMI	0.7	0.8	0.7

Socio-Genetic Assortative Mating

Explain a puzzle about the **intergenerational persistence of inequality**.

- Inequality can persist because of unmeasured genetic variation (Clark 2021). Genetics can be a mediator, not just a confound, for transmission of SES over generations.

Provide a new explanation of the **genes-SES gradient**.

- In modern meritocracies, genes affect SES.
- Under SGAM, in all societies, SES can affect genes.
- Shocks to SES are reflected in the DNA of subsequent generations.

Rethink the **nature of inequality** in historical human societies.

- Prediction: a genes-status gradient should be visible in ancient DNA.

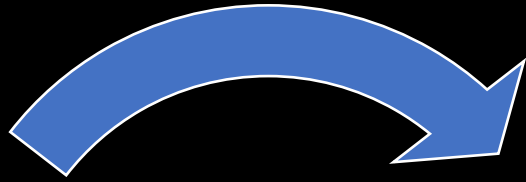
Change how we think about **genetic variation**.

- Yes, genes are “biological” ...
- But across generations, **genetic variation is a social outcome**.
- The size of the genes-SES gradient is affected by socio-economic institutions.

Results from human genetics have been controversial, and will likely continue to be so.

Rather than banning research we think is harmful (Nature Human Behavior 2022), perhaps it would help to take the perspective above.

Thank you!



Society

Genetics



“Part of the beauty of me is that I am very rich.”

Extensions

The basic result extends to non-normal/discontinuous distributions, and non-linear attractiveness.

We can allow “meritocracy”, where adult SES depends directly on own genes.

- Genes-SES correlation may either increase or decrease in θ .

The α parameter can differ for men and women.

- Genes-SES correlation is maximized when parameters are most different. (Intuition: then e.g. high-status males almost always match high-genetics females.)

Spouse pairs

Some respondents in the Biobank sample have a genetic child who is also in the sample.

Among our spouse pairs, 511 have a genetic child of at least one partner in the sample.

For 86% (441) of these, the child is the genetic child of both partners.

Comparison: 11% of families with dependent children included a stepchild in England and Wales in 2011 (National Statistics 2014).

Robustness

Extra mediators: BMI, self-reported health.

Birth order is independent of 33 different polygenic scores.

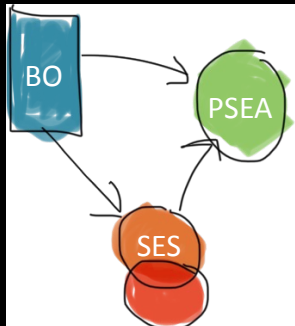
Results are qualitatively robust...

- ... if we use birth order dummies: strongest effect for first child versus subsequent children.
- ... using age left full-time education as the key mediator
- ... for males and females only (initial birth order coefficient is not significant)
- ... for couples with children

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SES mediators

Non-SES
mediators

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