Effects of socio-economic status on genetics: evidence from UK Biobank

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1 Introduction

Charles Murray (1995) warned of "a merging of the cognitive elite with the affluent". On the opposite side of the political spectrum, Karl Marx wrote (1974) "History is the true natural history of man" and (1844) "I am ugly, but I can buy the most beautiful woman.... the effect of ugliness, its repelling power, is destroyed by money." These quotations suggest that social advantages, such as wealth, income, caste or class, may have genetic consequences. If there is assortative mating between socially and genetically advantaged people, then socio-economic status may become encoded in the DNA of subsequent generations.

Figures 1 and 2 illustrate this idea using data for spouse pairs from UK Biobank. Figures 1 plots means of one partner's polygenic score for educational attainment (PSEA), a purely genetic measure, against a measure of the other partner's actual educational attainment: possession of a university degree. University graduates had spouses with higher PSEA.[1] Figure 2 plots partner's PSEA against another measure of own socio-economic status: income. Again, people with higher income had spouses with higher PSEA.

These figures do not prove that genetic encoding of socio-economic status is taking place. Since an individual's own PSEA correlates with both their educational attainment, and their income, both figures could be a result of partner selection on a purely genetic basis. In this paper, we test the theory more rigorously, using environmental shocks to socio-economic status that are unlikely to be correlated with own genetics. First, we develop a simple theory of genetic lock-in, to illustrate how its effects vary with social structure.

2 Theory

There is a large population, whose members have a single genetic trait g_i and a single social trait s_i.

G and S are continuously distributed. Without loss of generality, $EG = ES = 0$ and $Var(G) = Var(S) = 1$.[2] People pair according to an attractiveness function

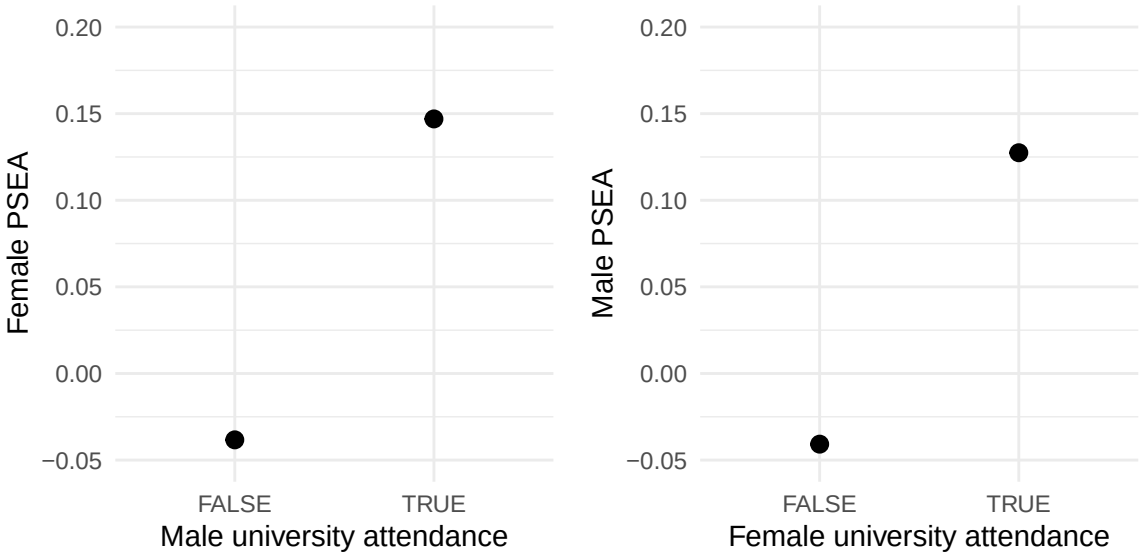
$$A(g_i, s_i) = f((1-k)g_i, ks_i)$$

which is smooth and strictly increasing in both its arguments.

The key parameter is k. This describes the working of the society's marriage market. If $k = 0$, only genetics G are relevant in marriage markets, and social status S has no effect. That is, the marriage market is highly egalitarian. Conversely, if $k = 1$, only social status matters, to the complete exclusion of genetics. This is

[1] To minimize concerns about genetic stratification, i.e. correlations between genetics and non-genetic inherited characteristics such as culture, PSEA is residualized on the first 100 principal components of UK Biobank array data.

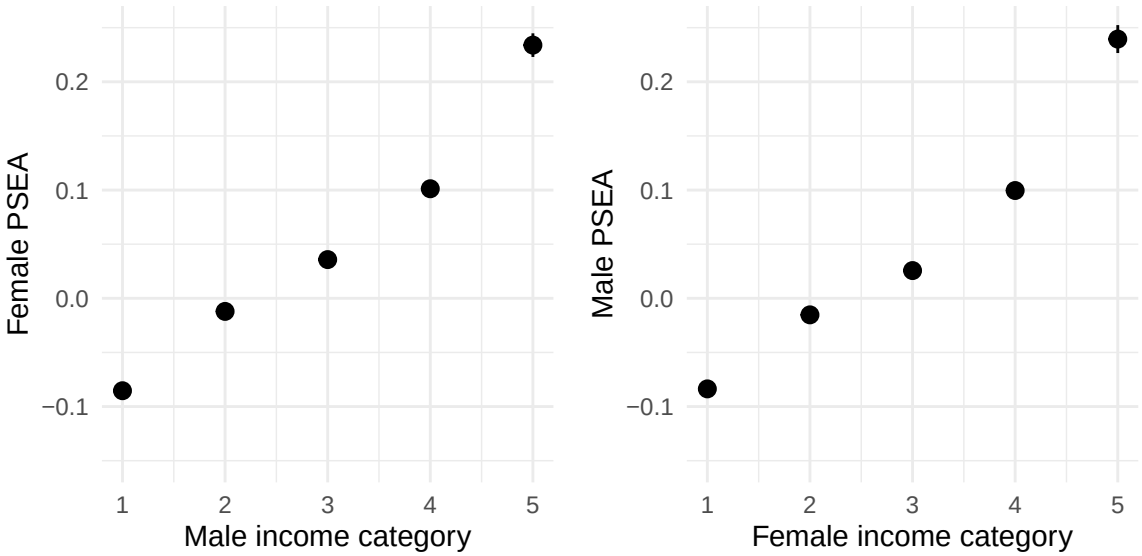
[2] Continuous distribution is not strictly required. All that is needed is for a set of pairs of positive measure to have different values of G and S, along a set of attractiveness curves of positive measure.



(a) Female PSEA by male educational attainment

(b) Male PSEA by female educational attainment

Figure 1: Social and genetic advantage among spouse pairs in UK Biobank



(a) Female EA3 by male income

(b) Male EA3 by female income

Figure 2: Social and genetic advantage among spouse pairs in UK Biobank

the equivalent of a marriage market driven only by "caste" or "class". Realistic societies are between these extremes.

Figure 3 shows the intuition behind our theory. The top row shows a caste marriage market with $k = 1$. A typical pair is shown: children have intermediate values of G and S between their two parents (hollow circle). In this society pairs match only by social status; genetics plays no role. As a result, while the variance of G shrinks within each status group, genetics remain uncorrelated with social status in the children's population distribution, shown on the right. The next row shows a purely egalitarian marriage market with $k = 0$. Parents match only by genetics and ignore social status. Again, as a result there is no correlation between genetics and social status in the children's generation. The bottom row shows a (more realistic) intermediate society, with an intermediate value of k. Because both genetics and social status contribute to attractiveness, matched spouses typically trade them off against each other. As a result, the distribution is squeezed along the gradient of k, and G and S are correlated in the children's generation. We next prove this formally.

If $k = 0$, "indifference curves" of attractiveness are vertical lines in (G, S) space. If $k = 1$, they are horizontal lines. If $k \in (0, 1)$ they are arbitrary downward sloping curves.

Write $p(i)$ for i's partner. Pairs always have the same attractiveness.

$$A(g_i, s_i) = A(g_{p(i)}, s_{p(i)}).$$

Each pair has two children. We assume that both children $c(d)$ of parents d, m have

$$\begin{aligned} g_{c(d)} &= \frac{g_d + g_m}{2}; \\ s_{c(d)} &= \frac{s_d + s_m}{2}. \end{aligned} \tag{1}$$

This is a strong assumption; we relax it later. For real world examples approximated by it, S could be wealth which is equally divided between the children; G could be a highly polygenic trait with many small effects. Write G_p, S_p to denote the population variables in the parents' generation; G_c, S_c for the children's generation.

Proposition 1. *(i) $Cov(G_c, S_c) \geq Cov(G_p, S_p)$, with strict inequality if and only if $0 < k < 1$.*

(ii) If $corr(G_p, S_p) \geq 0$, then $corr(G_c, S_c) \geq corr(G_p, S_p)$, with strict inequality if and only if $0 < k < 1$ or $corr(G_p, S_p) > 0$.

Proof. Within each pair $i, p(i)$ write d for the person with $s_d > s_{p(d)}$ and m for $p(d)$. (Think of these as "dukes" and "milkmaids", or if you prefer "duchesses" and "tennis instructors".) If $k < 1$, then $g_d < g_m$. (If $k = 0$, then define d as the person with $g_d < g_{p(d)}$.)

We integrate over the "dukes" to calculate the covariance in the parents' generation:

$$cov(G_p, S_p) = \int \frac{1}{2}(g_d s_d + g_{p(d)} s_{p(d)}) \, \mathrm{d}d.$$

For the children, the equivalent expression is

$$cov(G_c, S_c) = \int g_{c(d)} s_{c(d)} \mathrm{d}d,$$

observing that $EG_c = ES_c = 0$ from (1).

Take an arbitrary pair d, m. Write

$$g_d s_d = (g_c - \Delta g)(s_c + \Delta s);$$
$$g_m s_m = (g_c + \Delta g)(s_c - \Delta s)$$

where

$$\Delta g = \frac{g_m - g_d}{2} \geq 0, \text{strictly so if and only if } k > 0;$$
$$\Delta s = \frac{s_d - s_m}{2} \geq 0 \text{strictly so if and only if } k < 1.$$

Taking the average of the parents gives

$$\frac{1}{2}(g_d s_d + g_m s_m) = g_c s_c - \Delta g \Delta s.$$

This is less than $g_c s_c$ if $0 < k < 1$, and equal to it if $k = 0$ or $k = 1$. Plugging this into the integral shows that

$$cov(G_p, S_p) \leq cov(G_c, S_c)$$

again with strict inequality if and only if $0 < k < 1$. This proves the first part. A similar argument, showing $var(G_c) \leq var(G_p)$ and $var(S_c) \leq var(S_p)$, proves the second part (see the appendix). Figure 6 in the appendix shows that the condition in the second part cannot be relaxed further.

□

2.1 Robustness

We now relax the condition that children are exactly at the mean of their parents' values for G and S. Let

$$g_{c(i)} = \bar{g}_i + \varepsilon_i^G$$
$$s_{c(i)} = \bar{s}_i + \varepsilon_i^S$$

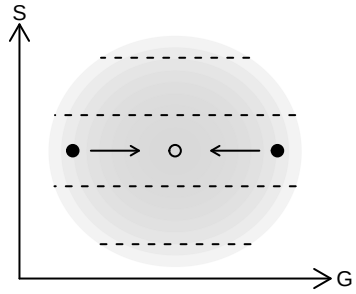
where

$$\bar{g}_i = \frac{g_i + g_{p(i)}}{2}; \bar{s}_i = \frac{s_i + s_{p(i)}}{2}$$

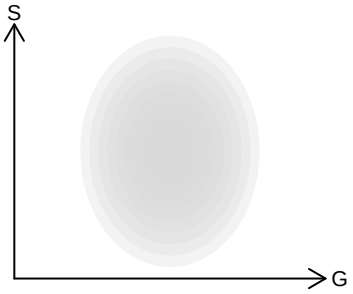
, ε^G has mean 0 and variance σ_G^2 and ε^S has mean 0 and variance σ_S^2.

Proposition 2. *1. if σ_G^2 and σ_S^2 are small enough and $corr(G_p, S_p) \geq 0$, then $corr(G_c, S_c) > corr(G_p, S_p)$ for $k \in (0, 1)$.*

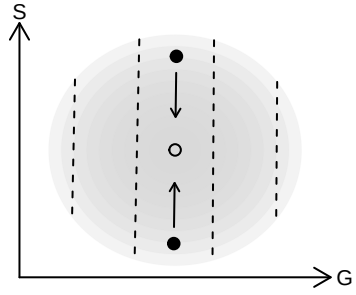
2. if ε^G and ε^S are uncorrelated with each other and with $\bar{G}$ and $\bar{S}$; and if G_p and S_p are uncorrelated, then $corr(G_c, S_c) \geq 0$, with strict inequality if and only if $0 < k < 1$.



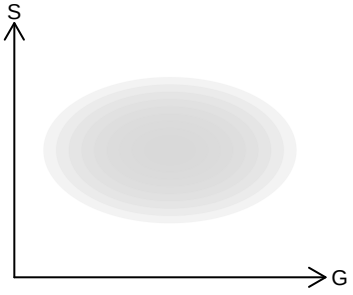
(a) Caste society ($k = 1$): parents



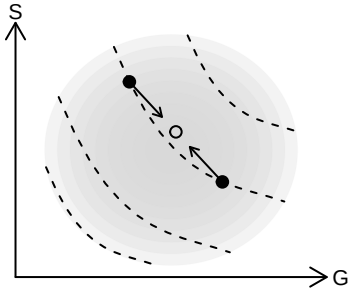
(b) Caste society: children



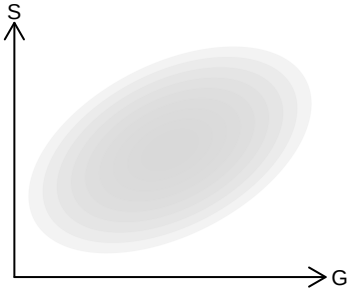
(c) Egalitarian society ($k = 0$): parents



(d) Egalitarian society: children



(e) Intermediate society ($0 < k < 1$): parents



(f) Intermediate society: children

Figure 3: Theory: shaded area is the population distribution. Dotted lines are attractiveness isoquants. Solid dots are example parents, transparent dots are example children. The right hand side shows the children's generation.

The conditions in Proposition are quite plausible. For G, they require that either variance in siblings' scores on some summary statistic is not too large, or that it is uncorrelated with the parents' scores. Both of these hold for most polygenic scores, which are additive sums of many small effects of alleles derived randomly from one or other parent. For S, the conditions would hold, for example, if S measures wealth, which is inherited not too unequally between siblings; or if wealth is inherited unequally but not in a way that correlates with S or G.

It is worth considering what kind of social arrangements would violate these conditions. For example, suppose that parents' combined wealth is inherited by the child with the lowest value of $g_{c(i)}$. This creates a negative correlation between $s_{c(i)}$ and $g_{c(i)}$.

2.2 Comparative statics

In the model, intermediate values of k drive increased covariance between genetics and social status. That naturally raises the question whether the change in covariance and/or correlation is increasing in k towards some maximum value in $(0, 1)$, then decreasing, i.e., whether it is quasiconcave in k. In general the answer is no, even on the assumption that

$$A(g_i, s_i) = (1 - k)g_i + ks_i$$

i.e. that indifference curves are straight lines in (G, S) space. However, quasiconcavity in k does hold when indifference curves are straight and (G, S) are jointly normally distributed. We conjecture that it also holds whenever the distribution (G, S) is quasiconcave.

- TODO: I can prove covariance is quasiconcave in k for normal G, S. Prove the same for correlation?

2.3 Discussion

The "marriage market" here is a reduced form mechanism, encompassing that makes a difference to partner choice. For example, if earned income affects attractiveness in the marriage market, then society's level of meritocracy in the labour market will correlate with the value of k: a more meritocratic labour market will allow people with low social status but high human capital (partly genetically determined) to earn more, and therefore to enter the high group.

Also, the contents of G – what counts as "good genes" in the marriage market – are themselves likely to vary across societies. For instance, standards of physical attractiveness vary historically. Similarly, it is plausible that what counted as a "good match", in terms of personality, physical and intellectual characteristics, differed between medieval European nobility and contemporary society.

The model predicts variation in the strength of genetic lock-in. In particular, in "caste societies" where there is complete endogamy within social status groups, there is no scope for genetic lock-in, because marriage partners do not trade off genetics for social status. The model also assumes that social status is inherited randomly from one parent, in the same way a genetic allele is inherited. This assumption can be weakened. For example, if social status is inherited deterministically from the father, then the results remain unchanged (for each pair of parents, just assume that one randomly chosen parent is the father).

Behaviour geneticists often make the point that in meritocratic societies, successful people may transmit relevant genes to their offspring. (TODO: cite relevant papers.) Like genetic genetic lock-in, meritocracy may therefore lead to a correlation between social status and genetics. However, genetic lock-in is a distinct, though overlapping, mechanism. Under meritocracy, certain genetic variants cause higher social status and are then transmitted along with it. This logic does not apply in non-meritocratic societies where social status is ascribed rather than earned. Conversely, genetic variations which cause social status will become associated with it, even in the absence of assortative mating.

By contrast, genetic lock-in applies to genetic variants that are associated with higher social status in the spouse matching process. They do not need to exert any influence whatsoever on an individual's own social status. This process requires assortative mating, but does not require meritocracy. The logic of genetic lock-in therefore applies to a historically much wider range of societies, including societies where social status is wholly ascribed or inherited, such as aristocracies.

In modern societies, both assortative mating and meritocracy are likely to be at play. Genetic variants that cause (e.g.) higher income and wealth will be inherited along with components of social status such as inherited wealth, networks and cultural capital. At the same time, higher social status and "good genes" will assort in the marriage market, even if that higher social status is caused by purely environmental variation. Our empirical analysis shows this latter process at work.

3 Data

As mentioned above, simple correlations between one partner's social status and the other partner's genetics do not prove that genetic lock-in is taking place, because one's social status correlates with one's own genetics. To demonstrate genetic lock-in, we therefore need a source of social advantage which is exogenous to genetics. One possibility is birth order. It is well known that earlier-born children receive more parental care and have better life outcomes. (XXX is it? Go check.) On the other hand, early- and late-born full siblings have the same ex ante expected genetic endowment. [3] We can therefore use birth order as an exogenous shock to social status.

We use data from UK Biobank, a study of about 500,000 individuals.

- TODO: describe N for birth order, describe PSEA calculation.
- TODO: look at mechanisms by which birth order might affect university
- TODO: get IQ data, control for it
- TODO: subset to spouses with children
- TODO: overall index of social status?

4 Results

Ideally we would instrument social status with birth order. However, our measures of social status are noisy and incomplete. For example, we know whether subjects went to university, but not which university they went to, and we only have rough categorical data on household income. Birth order likely affects both these and other measures of social advantage. So, an instrumental variables approach would probably fall foul of the exclusion restriction.

Instead, we conduct a mediation analysis, following the strategy of Heckman and Pinto (2013). We first regress our measures of own social status (i.e. income and education) on birth order. Then, we regress spouse's PSEA on birth order, with and without controlling for social status. Under the assumption that birth order is exogenous to own genetics, these regressions identify the effect of birth order, plus other environmental variables that correlate with it, on own social status and spouse's genetics. Also, if the estimated effect of birth order on spouse's PSEA changes when social status is included, that is evidence that social status mediates the effect of birth order.

- TODO: clarify the empirical model, you may need help....
- TODO: estimate individual income from job SIC codes? ASHE gives data

[3] This might not be the case, if parents' choice of whether to have more children is endogenous to the genetic endowment of their earlier children. We will check for this below.

Figures 4 and 5 show the relationship between birth order, university education and income, separately for respondents with 1-3 siblings. We test this formally in a linear regression controlling for family size, which may be correlated with parents' characteristics including genetics. Birth order is negatively correlated with both measures (among respondents with 1-7 siblings: university p = 8.3e-64, income p = 4.27e-09).

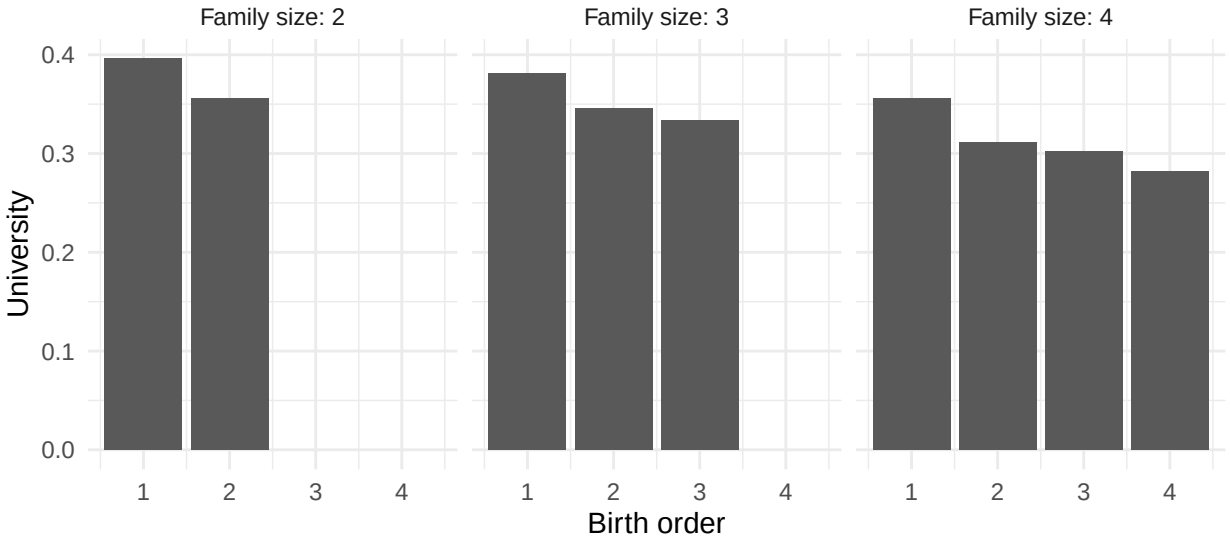


Figure 4: University attendance by birth order and family size

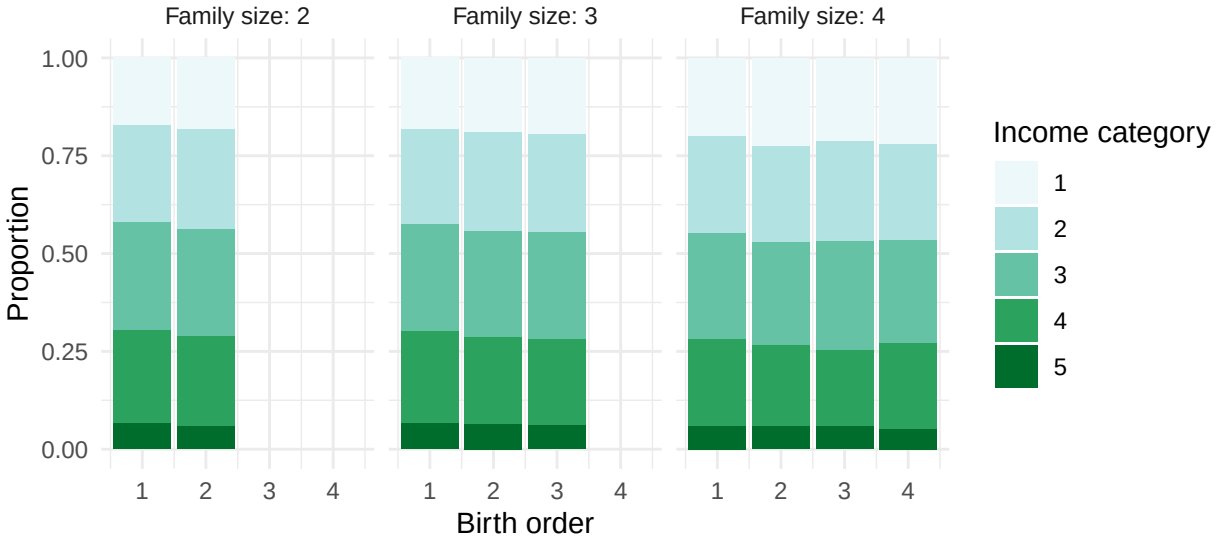


Figure 5: Income by birth order and family size

Next we run regressions of spouse PSEA on birth order, university attendance and income.

- TODO: include age FTE as well as university - to check university has an "extra effect" - this slightly suggests that uni is a "marriage market" and not just granting extra skills
- TODO: get profession data from Abdel for ASHE
- TODO: enquire about job history data - does it predict current profession well?
  - otherwise we risk the critique that current profession is endogenous to spouse PSEA

- TODO: consider alternative exogenous shocks to income. For example, some professions are more "cyclical" than others wrt recessions. If we could do predicted income at age 21-25 from business cycle X profession, that might count as exogenous. (Could use an independent source to estimate evolution of incomes, e.g. GHS or BHPS)
- TODO: get IQ data from Abdel - again this controls for skills
- TODO: get 2178 "overall health" and 2188 "longstanding illness/disability/infirmity"
  - control for effect on health
- Overall idea here is to unpack the different effects of birth order, separate them from university/income.
- TODO: control for age (polynomial) and birth month (seasonality)
- TODO: check whether sunshine is well established in the medical/health literature as an environmental effect. If not, probably skip it.
- TODO: split up by gender.

Table 1: Regressions of spouse PSEA

	(1)	(2)	(3)	(4)
Birth order	-0.0043 *	0.0005	-0.0002	0.0026
	(0.0020)	(0.0020)	(0.0020)	(0.0020)
University		0.1644 ***		0.1258 ***
		(0.0039)		(0.0040)
Income			0.0762 ***	0.0607 ***
			(0.0017)	(0.0018)
Own EA3	0.0466 ***	0.0303 ***	0.0365 ***	0.0260 ***
	(0.0023)	(0.0023)	(0.0023)	(0.0023)
Family size dummies	Yes	Yes	Yes	Yes
N	301319	301319	301319	301319
R2	0.003	0.009	0.010	0.013
logLik	-427246.003	-426337.107	-426237.365	-425744.187
AIC	854514.006	852698.214	852498.730	851514.373

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. Standard errors clustered by spouse pair.

Table 1 shows the results. Column 1 shows the effect of birth order controlling only for own PSEA and family size. It establishes that earlier-born children have spouses with higher PSEA. The effect size is small. This is to be expected, because (a) the effects of birth order on university, income and (presumably) other variables are small, and (b) PSEA is measured with a lot of error. We aim to test theory rather than estimating an effect size, so we focus more on statistical significance.

Column 2 includes university attendance. Column 3 includes income. Column 4 includes both. We estimate the percentage decrease in the effect of birth order across the columns, along with 95% confidence intervals for this figure, by running bootstraps (N = 199).[4] Including university attendance alone reduces the effect

[4]The sample percentage decrease calculated from the figures in Table 1 is not the correct estimate, since $E(X/Y) \neq EX/EY$.

of birth order by 36.4% (CI 13.0% – 135.2%). Including income alone reduces the effect of birth order by 53.6% (CI 19.4% – 205.6%). Including both decreases the effect by 78.5% (CI 28.3% –297.0%).

Our next regressions split up the data into subsets. We regress male birth order on female spouses' PSEA; female birth order on male spouses' PSEA; and the subset of individuals who had children. Table 2 shows the results.

- TODO: describe results
- TODO: very few couple pairs (< 50%) report the same number of children. Why?
  - equally likely to be males or females reporting more.

Table 2: Regressions of spouse PSEA: subsets

	(1)	(2)	(3)	(4)	(5)	(6)
Birth order	-0.003	0.002	-0.006 *	-0.001	-0.005	0.001
	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)
University		0.173 ***		0.156 ***		0.179 ***
		(0.009)		(0.012)		(0.005)
Own EA3	0.045 ***	0.028 ***	0.048 ***	0.033 ***	0.047 ***	0.030 ***
	(0.002)	(0.001)	(0.002)	(0.002)	(0.003)	(0.003)
N	156448	156448	144871	144871	206849	206849
R2	0.003	0.010	0.004	0.009	0.004	0.011
logLik	-221781.953	-221253.087	-205451.599	-205067.133	-293338.059	-292612.558
AIC	443585.906	442530.174	410925.198	410158.265	586698.119	585249.116

*** p < 0.001; ** p < 0.01; * p < 0.05. Standard errors for columns 5 and 6 clustered by spouse pair.

4.1 Robustness

Although all children of the same parents have the same polygenic scores in expectation, it could still be possible that genetics correlates with birth order within the sample. This could happen if parents select family size on the basis of genetics. For example, if the first child had a phenotype reflecting a high (or low) polygenic score, then that might affect the parents' decision to have a second child. Alternatively, respondents might select into the sample on the basis of a combination of birth order and genetics. We check this by regressing 33 different polygenic scores on birth order, controlling for family size.[5] No scores were significant at $p < 0.10/33$. 3 scores were significant at $p < 0.10$ (body mass index, conscientousness, and neuroticism). Coefficients were never greater than 0.01 of a standard deviation. Table 3 in the appendix reruns regressions controlling for these scores. Results are almost unchanged.

One early explanation for birth order effects was that these could be due to genetic mutations in older parents. More recent research has mostly rejected this explanation in favour of "social" explanations. (TODO: cite that paper.) Nevertheless, we rerun regressions controlling for father's age at birth, using the subset

[5]Polygenic scores were residualized on the first principal components of the genetic data.

of respondents who reported this data. (Cochran and Harpending (2013) report that mutational load is approximately linear in father's age, while it is constant in mothers' age. This is because female eggs are created once, before the female's own birth, while male sperma cells continue dividing throughout the lifespan.) Table 4 in the appendix shows that the effect of birth order remains significant and surprisingly increases in size.

- TODO: given that father's age correlates with birth order and has an opposite signed effect, should we use it as a control in the main regressions?
  - Problem is selection: people who answer father's age question are those whose father is still alive.

5 Conclusion

Behaviour geneticists have pointed out that in meritocratic societies, genetics and social status will be correlated, because some genetic variants will be causally linked to success in the labour market. We argue that causality can also go the other way. If social factors and genetics both matter in marriage markets, then "good genes" will become associated with markers of social status, such as class, education or income.

Our empirical analysis shows that in a modern Western democracy, earlier-born children had spouses with higher PSEA. Thus, an environmental shock has effects on the genetics of people's spouses and becomes encoded in the genetics of their children. We also provided evidence that these effects are mediated by social status: income and education.

Over history, our marriage market mechanism has probably operated in a much greater variety of societies than the labour market mechanism, because societies in which status can be earned are relatively rare compared to those in which status is inherited. (TODO: cite WEIRD? Or "status vs contract"?) As a result, we would expect long-standing correlations between status and genetic variation. We have not tested this here, but it could be tested using ancient DNA data.

Our analysis also has implications for the practice of controlling polygenic scores by partialling out principal components of genetic data. This is done so as to avoid confounding the effects of genetic variation with social stratification. However, insofar as the geneticists' concept of stratification (mating which is non-random with respect to genetics) overlaps with the sociological concept of stratification (a hierarchical ranking of individuals), stratification will predictably associate with causally relevant genetic variants. For this reason, we would expect principal components to contain real information about causally relevant variants. Of course, they may also contain information about variants of no effect, which only correlate with outcomes because of social stratification. So, it is an empirical question whether controlling for principal components improves or weakens the predictive power of polygenic scores. Within-family analyses could help to resolve this question.

In popular media, and sometimes by social scientists, genetics are often thought of as an external, "natural" constraint on social structure. This is one reason why behaviour genetics remains such a controversial topic. The broadest message of our research is that individual genetics are a social outcome like any other. They can even be viewed as another form of capital, alongside human, social and cultural capital: that is, a resource to be sought, accumulated and competed over. The analysis of this kind of capital is an exciting area for further research to which both social scientists and geneticists can contribute.

6 Appendix

6.1 Second part of Proposition 1

Proof. Write

$$corr(G_j, S_j) = \frac{cov(G_j, S_j)}{\sqrt{var(G_j)var(S_j)}} \text{ for both generations } j \in \{p, c\}. \tag{2}$$

where

$$var(G_p) = \frac{1}{2}\int g_d^2 + g_{p(d)}^2 \mathrm{d}d;$$
$$var(G_c) = \int g_{c(d)}^2 \mathrm{d}d.$$

Much as before,

$$\begin{aligned} g_d^2 + g_m^2 &= (g_c - \Delta g)^2 + (g_c + \Delta g)^2 \\ &= 2g_c^2 + 2(\Delta g)^2 \\ &\geq 2g_c^2. \end{aligned}$$

This shows that $var(G_c) \leq var(G_p)$ and a similar argument shows $var(S_c) \leq var(S_p)$. Thus the covariance is higher (and positive) in the children's generation, while the variances are lower. Combining these ensures that

$$corr(G_c, S_c) \geq corr(G_p, S_p).$$

Since for any k, either $var(G_c) < var(G_p)$ or $var(S_c) < var(S_p)$, the only way to get strict equality for the above is if $k \in \{0, 1\}$ and $cov(G_c, S_c) = cov(G_p, S_p) = 0$.

□

To show that the condition in the second part cannot be relaxed further, consider the distribution in Figure 6. There is negative correlation in the parents' generation (the shaded area). If $k = 1$ or is close enough to 1, then assortative mating along the dotted lines will reduce the variance of S along those lines, pushing the distribution towards the darker central area, without affecting the covariance. This will make the correlation more negative. After repeated generations the horizontal variance within values of G will almost disappear and the correlation will approach -1.

6.2 Proposition 2.1

Proof. Note that in proposition 1, we took $g_{c(i)} = \bar{g}_i$ and $s_{c(i)} = \bar{s}_i$. Write

$$\begin{aligned} cov(G_c, S_c) &= cov(\bar{G} + \varepsilon^G, \bar{S} + \varepsilon^S) \\ &= cov(\bar{G}, \bar{S}) + cov(\varepsilon^G, \bar{S}) + cov(\bar{G}, \varepsilon^S) + cov(\varepsilon^G, \varepsilon^S). \end{aligned} \tag{3}$$

For any X and Y, $cov(X, Y)$ is bounded by $\sqrt{var(X)var(Y)}$. Plugging σ_G^2 and σ_S^2 into this formula shows that under condition 1, $cov(G_c, S_c)$ will be arbitrarily close to $cov(\bar{G}, \bar{S})$. Similarly, writing

$$var(G_c) = var(\bar{G}) + var(\varepsilon^G) + 2cov(\bar{G}, \varepsilon^G)$$

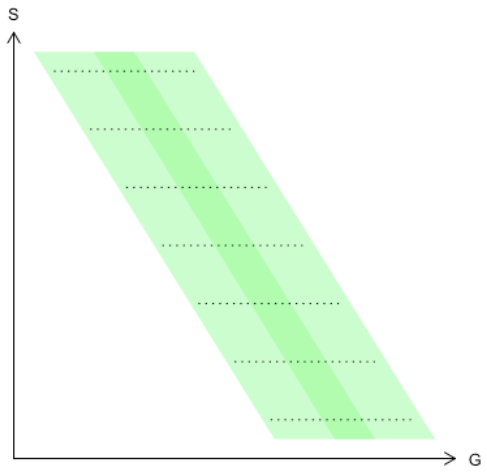


Figure 6: Correlation counterexample

shows that $var(G_c)$ will approach $var(\bar{G})$ as σ_G^2 grows small, and similarly for $var(S_c)$. Plugging these facts into (2) shows that $corr(G_c, S_c)$ approaches $corr(\bar{G}, \bar{S})$ as σ_G^2 and σ_S^2 grow small. Proposition 1 then shows $corr(\bar{G}, \bar{S}) < corr(G_p, S_p)$ for $k \in (0, 1)$.

Under condition 2, $cov(G_c, S_c) = cov(\bar{G}, \bar{S})$ since the last three terms of the sum in (3) are zero. Then since

$$cov(\bar{G}, \bar{S}) \geq cov(G_p, S_p) = 0$$

with strict inequality iff $k \in (0, 1)$, the covariance signs the correlation.

□

6.3 Robustness checks

```
## `summarise()` regrouping output by 'n_sibs' (override with `.groups` argument)
```

Table 3: Regressions of spouse PSEA with controls for polygenic scores

	(1)	(2)	(3)	(4)
Birth order	-0.0044 *	0.0004	-0.0003	0.0026
	(0.0020)	(0.0020)	(0.0020)	(0.0020)
University		0.1641 ***		0.1255 ***
		(0.0039)		(0.0040)
Income			0.0760 ***	0.0606 ***
			(0.0017)	(0.0018)
Own EA3	0.0443 ***	0.0283 ***	0.0344 ***	0.0242 ***
	(0.0023)	(0.0024)	(0.0023)	(0.0024)
Family size dummies	Yes	Yes	Yes	Yes
Polygenic score controls	Yes	Yes	Yes	Yes
N	301319	301319	301319	301319
R2	0.004	0.010	0.010	0.013
logLik	-427224.610	-426320.385	-426220.787	-425730.024
AIC	854477.221	852670.771	852471.573	851492.048

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. Standard errors clustered by spouse pair.

Table 4: Regressions of spouse PSEA controlling for father's age at birth

	(1)	(2)
Birth order	-0.0256 ***	-0.0137 **
	(0.0050)	(0.0050)
University		0.1552 ***
		(0.0073)
Father's age at birth	0.0092 ***	0.0063 ***
	(0.0009)	(0.0009)
Own EA3	0.0421 ***	0.0267 ***
	(0.0038)	(0.0039)
Family size dummies	Yes	Yes
N	81838	81838
R2	0.004	0.010
logLik	-116308.673	-116083.314
AIC	232641.346	232192.629

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$. Standard errors clustered by spouse pair.