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Trading Social Status for Genetics in Marriage Markets: Evidence from UK Biobank

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Abstract

If socio-economic status (SES) and genetic variants are both assets in marriage markets, then the two will become associated in spouse pairs, and will be passed on together to future generations. This process provides a new explanation for the surprising persistence of inequality across generations, and for the genes-SES gradient: the genetic differences we observe between high- and low-income people. The gradient includes differences related to human capital and to physical and mental health, so understanding its origins is important for understanding inequality in general, and health inequalities in particular. We model social-genetic assortative mating (SGAM) and test for its existence in a large genetically-informed survey. We compare spouses of individuals with different birth order, which is known to affect socio-economic status and which is exogenous to own genetic endowments among siblings. Spouses of earlier-born individuals have genetic variants that predict higher educational attainment. We provide evidence that this effect is mediated by individuals' own educational attainment and income. Thus, environmental shocks to socio-economic status are reflected in the DNA of subsequent generations. Our work uncovers a new channel by which economic institutions can affect long-run inequality; suggests that genes-SES gradients may be historically widespread; and shows that genetic variation is endogenous to social institutions.

Introduction

Over the long run, inequality is surprisingly persistent across generations (Clark and Cummins 2015; Solon 2018). Intergenerational mobility is correlated with cross-sectional inequality (Becker et al. 2018; Krueger 2012), which has risen dramatically in high-income countries, at the same time as intergenerational absolute mobility has declined

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(Western, Bloome, and Percheski 2008; Chetty et al. 2017).[1] Assortative mating in marriage markets can increase the inequality of human capital and income across families (Breen and Salazar 2011; Greenwood et al. 2014). It follows that how families are formed, and transmit traits and assets to their offspring, are critical for understanding inequality. These processes have been studied from both socio-economic and genetic angles. While educational homogamy is well established, genetic assortative mating has been demonstrated only recently (Hugh-Jones et al. 2016; Robinson et al. 2017). Similarly, wealthy families pass on advantages to their children through both genetic inheritance and environmental influence (Rimfeld et al. 2018; Björklund, Lindahl, and Plug 2006).[2]

This paper examines a plausible, not previously analysed aspect of the spouse matching process: that both social status and genetics contribute to a person's attractiveness in marriage markets, and as a result, genetics and inherited social status may become associated in subsequent generations.[3] For example, suppose that wealth, intelligence and health are positive assets in a potential spouse. Then wealthy people are more likely to marry intelligent or healthy people, and their children will inherit both wealth, and genetic variants associated with intelligence or health. We call this mechanism social-genetic assortative mating (SGAM). SGAM may be an important channel for the transmission of inequality. It leads to a hidden dimension of advantage for privileged families – hidden because most social science datasets do not include genetic information. This dimension may help to explain the surprising long-run persistence of inequality (Clark and Cummins 2015; Solon 2018). At the same time, this advantage is not an exogenous fact of biology, but endogenous to the social structure. Indeed, under SGAM, environmental shocks to an individual's social status may be reflected in the genetics of his or her children.

Below, we first outline a theoretical framework where attractiveness in the marriage market is a function of both socio-economic status (SES) and genetic variants. We show that social-genetic assortative mating in one generation increases the correlation between SES and genetic variants in the offspring generation. This result provides a new explanation of the *genes-SES gradient*, that is, the systematic genetic differences between high- and low-SES people (Belsky et al. 2018; Rimfeld et al. 2018; Björklund, Lindahl, and Plug 2006). The dominant existing explanation for the gradient is meritocratic social mobility: if a genetic variant predicts success in the labour market, then it will become associated with high SES and will be inherited in high-SES families. Under meritocracy, genes causes SES. On the other hand, under SGAM, causality goes both ways, from genes to SES and vice versa. What is more, the strength of the genes-SES gradient depends on economic institutions. Under institutions which increase intergenerational mobility, such as high inheritance tax rates, the genes-SES gradient becomes weaker.

[1] Though relative mobility has been stable (Chetty et al. 2014). In the United Kingdom the Gini coefficient has increased from 26% to 34.6% between 1977 and 2020. The United States has seen a 10 percentage point rise to 43.3% during 1962-2013.

[2] See Sacerdote (2011) for a review of the behavioural genetics and economics literatures on the nature vs nurture debate; for a broader review of intergenerational transmission of income see Black and Devereux (2010).

[3] *Social status* refers to characteristics that an individual possesses in virtue of their social position. For example, my wealth is a fact about me that holds in virtue of my relationship to certain social institutions (bank deposits, title deeds et cetera). Other examples include caste, class, income, and educational qualifications. *Socio-economic status* (SES) is a specific type of social status which exists in economically stratified societies, covering variables such as educational attainment, occupational class, income and wealth (e.g. White 1982).

Next, using data on matched spouses born between 1935 and 1970 from UK Biobank (a large genetically-informed survey), we test the hypothesis that an individual's higher social status attracts spouses with higher genetic potential for educational attainment. Our genetic measure, the polygenic score for educational attainment (PSEA), derives from large-scale genome-wide association studies (Lee et al. 2018). PSEA reflects a bundle of polygenic effects on underlying traits, including intelligence, personality, and physical and mental health (Demange et al. 2021). PSEA predicts, and causes, educational attainment itself, as well as intelligence and labour market outcomes. It is already known that humans mate assortatively on PSEA (Hugh-Jones et al. 2016; Robinson et al. 2017), which makes it a likely candidate for detecting SGAM.

The endogeneity of socio-economic status is the main challenge in identifying the causal effect of SES on the spouse's genetic endowment. For instance, individuals with high education qualifications tend to also have high PSEA, and as mentioned above, they may take partners based on genomic similarity. Indeed, recent studies show strong assortative mating on PSEA, much more than we would expect if spouses matched only on (our observed measures of) actual educational attainment (Okbay et al. 2022). To isolate the causal link from own SES to partner genes, we use the "accident of birth" as a shock to SES which is independent of own genetics. Specifically, we use an individual's birth order as a "treatment" which affects their partner choice through a range of mechanisms, including by affecting their own SES. It is well documented that earlier-born children enjoy higher parental investment and have better life outcomes, including measures of SES such as educational attainment and occupational status (Black, Devereux, and Salvanes 2011; Booth and Kee 2009; Lindahl 2008). At the same time, the facts of biology, in particular the so-called "lottery of meiosis", guarantee that siblings' birth order is independent of their genetic endowments.[4]

Birth order could affect partner choice both through SES, and through non-SES mechanisms. So, we run a mediation analysis similar to Heckman, Pinto, and Savelyev (2013), decomposing the treatment effect into effects of measured and unmeasured mediating variables. Specifically, we estimate a reduced-form model with spouse polygenic scores for educational attainment (PSEA) as the dependent variable, and own birth order as the main independent variable. We then estimate a model which also includes measures of own socio-economic status, including university attendance and estimated income in one's first job. Under certain assumptions, these variables can be interpreted as mediating the effect of birth order on spouse genetics.

We find that later-born children have spouses with significantly lower PSEA in the reduced-form regressions. When we add mediators, including university attendance and/or income, birth order is no longer independently significant, while the SES mediators increase the spouse's PSEA at 0.1% significance. University attendance explains an estimated 38-55% of the effect of birth order, and income explains about 10-13%. Thus, SES appears to mediate the effect of birth order on spouse genetics. The results are robust to the inclusion of several controls, including non-SES mediators,

[4]Though Muslimova et al. (2020) find that PSEA and birth order do interact to produce human capital.

and a rich set of own genetic traits.

Our paper contributes to several literatures. Firstly, we study a new kind of assortative mating. The economics literature on matching in marriage markets has typically focused on educational similarities (e.g. Pencavel 1998; Chiappori, Salanié, and Weiss 2017) or social class or caste (e.g. Abramitzky, Delavande, and Vasconcelos 2011; Banerjee et al. 2013), but also sorting based on age, physical traits and ethnicity (Hitsch, Hortaçsu, and Ariely 2010). Matching decisions on the marriage market follow multiple criteria, with some degree of substitutability between them.[5] For instance, Chiappori, Oreffice, and Quintana-Domeque (2012) showed that individuals trade off BMI for partners' income or education and that the marginal rate of substitution between these characteristics is different for males and females. The genetics literature has focused on genetic assortative mating (GAM), the phenomenon that people with similar genes marry each other. Recent research has confirmed the long-standing conjecture that GAM takes place in contemporary human populations (Howe et al. 2019; Hugh-Jones et al. 2016; Robinson et al. 2017). Geneticists have also developed the concept of cross-trait assortative mating (Beauchamp et al. 2010; Sundet et al. 2005; Border et al. 2022). This happens when e.g. people with genes for height marry people with genes for intelligence, or people with genes for two different types of disease trait marry each other. As a result, the two types of genetic variation become associated. In this paper we bring the two literatures together, extending cross-trait assortative mating to encompass social status as well as genetic variants. Our results confirm that individuals with higher social status attract spouses with genetics for higher educational attainment.

Secondly, SGAM may affect economic inequality and intergenerational mobility. Clark and Cummins (2015) show using a database of surnames that long-run intergenerational persistence of wealth, over five generations, is much higher than would be predicted by the statistics for parent-child correlations in wealth. Clark (2021) argues that the data can be explained by an underlying process where unobserved genetic variation determines wealth. We show below that SGAM could also generate these patterns. The mechanism again is unobserved genetic variation, but the interpretation is different, since we view genetic endowments not an exogenous source of variation, but as an asset effectively "traded" in marriage markets in exchange for wealth and social status. Put another way, for Clark, in analysing the effect of ancestral wealth on descendant wealth, genetics is a *confound*; under SGAM, it can be a *mediator*.

SGAM also affects cross-sectional inequality, like other forms of assortative mating (Fernández and Rogerson 2001; Fernandez, Guner, and Knowles 2005; Eika, Mogstad, and Zafar 2019; Chiappori, Dias, and Meghir 2018). We know that there is a "genes-SES gradient": many genetic variants are associated with high or low SES. From twin studies, the heritability of occupational class and educational attainment, i.e. the proportion of variance explained by genetic differences between individuals, is around 50% (Tambs et al. 1989). Genome-wide Complex Trait Analysis (GCTA)

[5] Oreffice and Quintana-Domeque (2010) show that height and BMI are associated with spouse earnings. Dupuy and Galichon (2014) find spouse matching on multiple independent dimensions, including education, height, BMI and personality.

shows that the family socio-economic status of 2-year-old children can be predicted from their genes (Trzaskowski et al. 2014). Children born into higher-income families have more genetic variants predicting educational attainment (Belsky et al. 2018). Studies comparing adoptees to non-adoptees show that both post-birth environment and pre-birth conditions (genetics and to a lesser extent prenatal environment) contribute to the transmission of wealth and human capital (e.g. Björklund, Lindahl, and Plug 2006). Thus, the genes-SES gradient is an important source of inequality. Genetic variation in human capital is key, since a likely cause of the recent rise in inequality is the increase in market returns to human capital (e.g. Kaplan and Rauh 2013; Eika, Mogstad, and Zafar 2019). Another important aspect of the genes-SES gradient is in genetic associations with health. DNA-derived scores predictive of several health outcomes are associated with regional economic deprivation (Abdellaoui et al. 2019). There is a correlation between education and health, which may be mediated by shared genetic causes (Amin, Behrman, and Kohler 2015; Boardman, Domingue, and Daw 2015). Family SES correlates with several health-related polygenic scores (Selzam et al. 2019), and genetic effects associated with SES significantly alter the genetic relationships between many mental health outcomes (Marees et al. 2021). Understanding the aetiology of health and disease requires disentangling the effects of genetics and environments. In turn, that requires understanding how the two can become correlated.

SGAM shows how marriage markets can lead high SES to be associated with different genetic variants. Thus, it can explain the genes-SES gradient. The standard explanation for the genes-SES gradient is returns to talent in labour markets, a.k.a. meritocracy. Parents with higher ability reap higher market returns, and they may then pass both higher socio-economic status and their genes to their children, leading to an association between the two (Belsky et al. 2018). This mechanism depends on the level of meritocracy in social institutions (Branigan, McCallum, and Freese 2013; Heath et al. 1985): in a society where social status was ascribed rather than earned, it could not take effect. Indeed, after the fall of communism in Estonia, the heritability of SES increased, presumably because post-communist society allowed higher returns to talent (Rimfeld et al. 2018). By contrast, SGAM does not require meritocracy. Even when social status is entirely ascribed, it may still become associated with certain genetic variants, so long as their associated phenotypes are prized assets in marriage markets. Since meritocracy is historically rare, while assortative mating is universal, this suggests that genes-SES gradients are likely to be historically widespread.

Both meritocracy and SGAM may increase social inequality overall, if there are complementarities between genetic and environmental components of human capital, for example if higher-ability parents make more productive investments in children's human capital (Cunha and Heckman 2007; Cunha, Heckman, and Schennach 2010; Heckman and Mosso 2014; Kong et al. 2018), or if high-income parents are able to invest more in transmitting their human capital (Becker et al. 2018). Thus, by bringing "good genes" and enriched environments together, SGAM may increase inequality in the next generation.

Lastly, we contribute to a literature in economics that examines the relationship between genetic and economic vari-

ables. Benjamin et al. (2011) is an early review. Several more recent papers use polygenic scores, in particular polygenic scores for educational attainment (Barth, Papageorge, and Thom 2020; Papageorge and Thom 2020; Ronda et al. 2020). Barban et al. (2021) use PSEA as an instrument for education in a marital matching model. These papers – like the vast majority of the behavior genetics literature (see e.g. Plomin, DeFries, and McClearn 2008) – take genetic endowments as exogenous and examine how they affect individual outcomes, perhaps in interaction with the environment. We take a different approach by putting genetics on the "left hand side". Thus, our paper challenges the assumption, in economics and beyond, that genetic endowment is exogenous to economic characteristics. While this may be tenable in within-generation studies, it ceases to hold in intergenerational models. Social-genetic assortative mating is a causal mechanism going from socio-economic status to genes for heritable traits.

Also, our model shows that the strength of this mechanism is endogenous to social and economic institutions. When SES is highly transmissible across the generations, this has the long-run effect of increasing the association between SES and genetics. If so, institutional reforms that increase *intergenerational mobility*, like mass education or inheritance taxation, may in the long run affect not only economic but genetic inequality. Conversely, an increase in *economic meritocracy* increases the association between SES and genetics in the long run.[6] This poses the problem raised first by Young (1958), and more recently by Markovits (2019): meritocracy may be self-limiting or even self-undermining.

The observations behind SGAM are not new. That status and physical attractiveness assort in marriage markets is a commonplace and a perennial theme of literature. In the Iliad, powerful leaders fight over the beautiful slave-girl Bryseis. In Jane Austen's novels, wealth, attractiveness and "virtue" all make a good match. Marx (1844) wrote "the effect of ugliness, its repelling power, is destroyed by money." And Donald Trump claimed: "part of the beauty of me is that I am very rich." The literature on mate preference from evolutionary psychology (Buss and Barnes 1986; Buss 1989; Buss and Schmitt 2019) confirms that attractive mate characteristics include aspects of social status ("high earning capacity," "professional status") as well as traits that are partly under genetic influence ("intelligent," "tall," "kind," "physically attractive"). Despite this, to our knowledge, this is the first paper in either genetics or economics to formally analyse SGAM and its consequences.[7]

[6] See Proposition 4 below.

[7] Halsey (1958) showed in a two-class model that social mobility combined with assortative mating might increase the association between genetics and social class. Belsky et al. (2018) offer three reasons for the association between education-linked genetics and SES, but do not consider SGAM.

Model

People in the marriage market have two characteristics: $x = (x_1, x_2)$, drawn from a normal distribution

$$\mathcal{N}\left(\begin{array}{ccc} 0 & s^2 & \sigma \\ 0 & , & \\ & \sigma & S^2 \end{array}\right).$$

We interpret x_1 as a genetic measure, for example of genes predictive of height, physical attractiveness, health or intelligence. x_2 is a measure of socio-economic status, such as income or wealth, or social status more generally (we sometimes use "wealth" as a shorthand). The correlation between x_1 and x_2 is

$$Corr = \frac{\sigma}{sS} < 1.$$

People's attractiveness is given by

$$i\left(x\right) = ax_1 + \left(1 - a\right)x_2$$

where $a \in [0, 1]$ is a parameter reflecting the relative importance of genetics to wealth in the marriage market.[8] If $a = 0$, marriage markets are highly inegalitarian, such that only SES matters. If $a = 1$, marriage markets are economically egalitarian and only genetics matter. We expect realistic societies to fall between these extremes, with $0 < a < 1$. Then, both genes and SES matter to attractiveness, and as a result, social-genetic assortative mating (SGAM) takes place.

Attractiveness i is distributed $N(0, \sigma_I^2)$, where

$$\sigma_I^2 = a^2 s^2 + \left(1 - a\right)^2 S^2 + 2a\left(1 - a\right)\sigma.$$

People form matches with transferable utility, where the surplus for a match between x and y is $S(i(x), i(y))$ such that $\partial^2 S / \partial i \partial j > 0$, i.e. S is supermodular. As a result there is positive assortative mating on attractiveness: x matches with y only if they are at the same quantile of attractiveness, i.e. if $i(x_1, x_2) = i(y_1, y_2)$. We describe this as social-genetic assortative mating (SGAM).

We also consider random matching as a benchmark to compare against SGAM. Under random matching, the distribu-

[8] Note that since the variance of the shocks to x_1 and x_2 (see below) has been normalized to 1, a also reflects this variance. That is, a large variance of SES shocks (compared to genetic shocks) translates into a being large.

tion of couples' characteristics is normal with mean 0 and covariance matrix

$$\mathbb{C}\begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} = \begin{pmatrix} s^2 & \sigma & 0 & 0 \\ \sigma & S^2 & 0 & 0 \\ 0 & 0 & s^2 & \sigma \\ 0 & 0 & \sigma & S^2 \end{pmatrix}.$$

Our first proposition shows that if SGAM is taking place, i.e. if $0 < a < 1$, then there is a positive correlation between one partner's wealth and the other partner's genetics.

Proposition 1. *Under SGAM, the distribution of couples' characteristics is normal, with mean 0 and covariance matrix*

$$\mathbb{C}\begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} = \begin{pmatrix} s^2 & \sigma & A^2 & AC \\ \sigma & S^2 & AC & C^2 \\ A^2 & AC & s^2 & \sigma \\ AC & C^2 & \sigma & S^2 \end{pmatrix} \tag{1}$$

where:

$$A = \frac{as^2 + (1-a)\,\sigma}{\sqrt{a^2 s^2 + (1-a)^2 S^2 + 2a\,(1-a)\,\sigma}} = \frac{as^2 + (1-a)\,\sigma}{\sigma_I};$$
$$C = \frac{a\sigma + (1-a)\,S^2}{\sqrt{a^2 s^2 + (1-a)^2 S^2 + 2a\,(1-a)\,\sigma}} = \frac{a\sigma + (1-a)\,S^2}{\sigma_I}.$$

In particular, the covariance between x_2 and y_1, AC, is positive if either x_1 and x_2 are already correlated ($\sigma > 0$) or if they are uncorrelated ($\sigma = 0$) and the attractiveness parameter a is strictly between 0 and 1.

Proof. See Appendix. □

We consider the distribution of couples' wealth. Under random matching this has mean 0 and variance $2S^2$. Under SGAM, the variance is:

$$V(x_2 + y_2) = 2S^2 + 2C^2 > 2S^2$$

This is decreasing in a and equals $4S^2$ if $a = 0$. Thus, SGAM increases cross-sectional inequality, but less so than pure matching on wealth.

Children

All couples have the same number of children. A child's characteristics are given by:

$$\begin{aligned} x_1' &= \frac{\tau}{2}(x_1 + y_1) + \varepsilon \\ x_2' &= \frac{\theta}{2}(x_2 + y_2) + \eta \end{aligned} \tag{2}$$

where x and y are the child's parents, and ε and η are independent normal random shocks with mean 0 and variance 1.

Parameter $\tau \approx 1$ reflects genetic inheritance. Under standard biological assumptions $\tau = 1$ and characteristics show no regression to the mean. In our model this leads the variance of x_1 to grow without limit over generations. In reality, we expect $\tau < 1$ because very extreme characteristics are selected against, a process known as stabilizing selection (Schmalhausen 1949; Sanjak et al. 2018).

Parameter $\theta \in [0, 1]$ reflects inheritance of SES. Unlike τ it may vary between societies. θ is high when there is high intergenerational transmission of SES. Thus, θ captures social and economic institutions that affect this intergenerational transmission, from taxation and public education to hereditary nobility. If we interpret x_2 narrowly as wealth, $(1 - \theta)$ can be thought of as the rate of inheritance tax.

For the time being, we assume that a person's genetic endowment has no impact on their SES. Technically, thus, x_2' does not directly depend on x_1'. In a meritocratic society we would expect adult SES to partly depend on genetics. We show that even absent meritocracy, correlations between x_1' and x_2' can arise. In an extension below, we relax this assumption and allow meritocracy.

We can now calculate the covariance matrix for $x' = (x_1', x_2')$ under SGAM as:

$$\begin{aligned} \mathbb{C} &= \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} + \begin{pmatrix} \frac{\tau}{2} & 0 & \frac{\tau}{2} & 0 \\ 0 & \frac{\theta}{2} & 0 & \frac{\theta}{2} \end{pmatrix} \begin{pmatrix} s^2 & \sigma & A^2 & AC \\ \sigma & S^2 & AC & C^2 \\ A^2 & AC & s^2 & \sigma \\ AC & C^2 & \sigma & S^2 \end{pmatrix} \begin{pmatrix} \frac{1}{2}\tau & 0 \\ 0 & \frac{1}{2}\theta \\ \frac{1}{2}\tau & 0 \\ 0 & \frac{1}{2}\theta \end{pmatrix} \\ &= \begin{pmatrix} \frac{1}{2}A^2\tau^2 + \frac{1}{2}s^2\tau^2 + 1 & \frac{1}{2}\theta\sigma\tau + \frac{1}{2}AC\theta\tau \\ \frac{1}{2}\theta\sigma\tau + \frac{1}{2}AC\theta\tau & \frac{1}{2}C^2\theta^2 + \frac{1}{2}S^2\theta^2 + 1 \end{pmatrix} \end{aligned} \tag{3}$$

We now explore two issues. First, under SGAM, genetic characteristics are no longer exogenous; because of assortative matching, they are (partly) socially determined. In particular, even if genetics and SES are uncorrelated among parents, the expected genetic endowment of the child is positively related to parental SES. Second, as a result, in the long run a correlation appears between traits; that is, high SES people inherit genes that are attractive in marriage markets.

Regarding point 1, we compute the expected genetic characteristic of the child, conditional on parental wealth:

$$\mathbb{E}\left[\frac{\tau}{2}(x_1+y_1)+\varepsilon \mid x_2=v, y_2=w\right]$$

Given the symmetry of the model, this conditional expectation only depends on the parents' total wealth, i.e. $v+w$.

Claim 1. *Under random matching, the expected genetic endowment of the children is proportional to the parents' SES and to the covariance between SES and genetics for the parents. In particular, if $\sigma = 0$ (i.e. genetics and SES are uncorrelated for the parents), then the expected genetic endowment of the children does not depend on parental SES.*

Claim 2. *Under SGAM, if $\sigma = 0$ (i.e. genetics and SES are uncorrelated for the parents), then the expected genetic endowment of the children is linearly increasing in parental SES. The relationship increases with the ratio of genetic variance to SES variance, is zero for $a = 0$ or $a = 1$, and is highest for intermediate values of a.*

Next, we study the correlation between children's traits 1 and 2 as a function of σ, the covariance of parents' traits. We first consider the general case, then concentrate on $\sigma = 0$, i.e. when traits are initially uncorrelated.

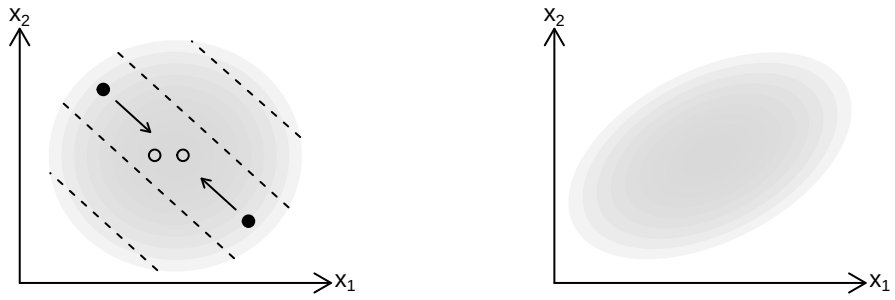
Claim 3. *Under random matching, the correlation between characteristics is smaller for children than for parents. In particular, if genetics and SES are uncorrelated for the parents, then they are uncorrelated for the children.*

Claim 4. *Under SGAM, if genetics and SES are uncorrelated for the parents, then they are positively correlated for the children so long as $0 < a < 1$. The correlation is increasing in θ.*

Whether characteristics are more or less correlated for children than for parents depends on whether the initial correlation between parents' characteristics is larger or smaller than the asymptotic one, derived below.

Figure 1 shows the intuition behind the model. Parents match on downward-sloping attractiveness isoquants given by $ax_1+(1-a)x_2 = u$. Their children are in between them on both dimensions. This compresses the distribution along the attractiveness isoquants, which leads to a positive correlation between genetics and SES. The correlation between x_1' and x_2' is 0 when $a = 0$ or $a = 1$, because then spouses don't trade off SES for genes. It is highest for intermediate values of a.

These results show that SGAM can lead to a genes-SES gradient, i.e. a positive correlation between genes and SES. Also, the strength of the genes-SES correlation is affected by economic institutions, as captured in θ. When θ is high,



Parents Children

Figure 1: Theory. The shaded area is the population distribution. Parents (solid circles) match along attractiveness isoquants (dotted lines). Children (hollow circles) are between them. As a result, in the children's generation, the distribution is squeezed along attractiveness isoquants.

the genes-SES correlation is high too.

We now consider the asymptotic distribution of x_1 and x_2 when the matching process is repeated over many generations. As we would expect, our main results continue to hold.

Proposition 2. *Under random matching, the dynamics converges to a stationary distribution that is normal with mean zero and covariance matrix*

$$\mathbb{C}\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} = \begin{pmatrix} \frac{2}{2-\tau^2} & 0 \\ 0 & \frac{2}{2-\theta^2} \end{pmatrix}$$

In particular, the traits are asymptotically uncorrelated and children's expected genetic endowment is independent of parents' wealth.

Proposition 3. *Under SGAM, for $\theta < 1$ and $\tau < 1$, the dynamics converge to a stationary distribution that is normal with mean zero and covariance matrix*

$$\mathbb{C}\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} = \begin{pmatrix} \bar{s}^2 & \bar{\sigma} \\ \bar{\sigma} & \bar{S}^2 \end{pmatrix}$$

Moreover, the asymptotic correlation between characteristics, $corr = \bar{\sigma}/\bar{s}\bar{S}$, is non-negative, positive for $0 < a < 1$, increasing in θ and increasing then decreasing in a. The coefficient of parents' wealth on children's genetics is also positive for $0 < a < 1$.

For $\theta = 1$, the dynamics diverge and $\bar{S}^2$ goes to $+\infty$; for $\tau = 1$, the dynamics diverges and $\bar{s}^2$ goes to $+\infty$.

Figure 2 plots the asymptotic correlation between x_1 and x_2 for $\tau = 0.95$.

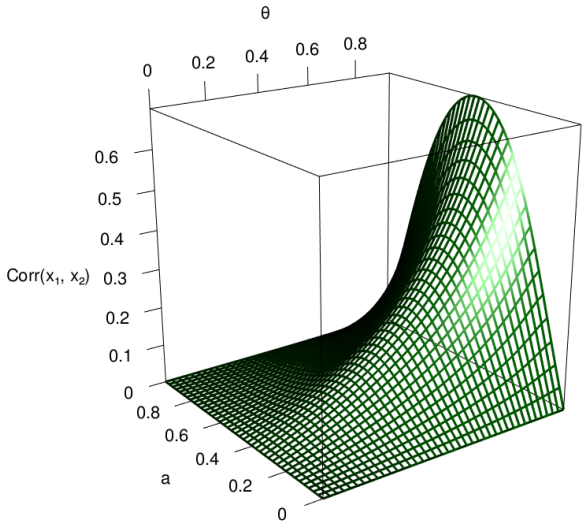


Figure 2: Long-run correlation between genetics x_1 and SES x_2, by weight of genetics in matching a and SES inheritance θ. $\tau = 0.95$.

Note that both $\bar{S}^2$ and $\bar{\sigma}$, as well as the correlation between characteristics and the conditional expectation of genetics given wealth, are increasing in θ, i.e. decreasing in the tax rate. Higher taxation reduces the asymptotic variance of wealth (not surprisingly), but also the correlation between genetics and wealth.

Extensions

We consider three extensions. First, the relative attractiveness of genes and SES might differ for men and women. Our basic result extends to this setup.

Claim 5. *Suppose that men's and women's attractiveness is given by*

$$i(x) = ax_1 + (1-a)x_2,$$
$$j(y) = by_1 + (1-b)y_2$$

respectively, with $0 \leq a \leq 1$, $0 \leq b \leq 1$. *Then if* $\sigma = 0$, *children's characteristics* x_1' *and* x_2' *will be positively correlated unless* $a = b = 0$ *or* $a = b = 1$. *The correlation is increasing in* θ.

Interestingly, the x_1-x_2 correlation is highest when a and b are most different from each other. So gender differences in what counts as attractive make the effects of SGAM stronger. Intuitively, if one sex only assorts on SES while the other sex only assorts on genetics, this induces a very reliable correlation between genes and SES in couples, since

(e.g.) every high-SES male is matched for sure with a high-genetics female.

Second, in modern meritocracies, people's adult SES depends not just on their parents' social status and on chance, but also on their own effort and skills, which might be related to their genetics. So, let

$$x_1' = \tau \frac{x_1 + y_1}{2} + \varepsilon$$
$$x_2' = \gamma x_1' + \theta \frac{x_2 + y_2}{2} + \eta \tag{4}$$

where $\gamma > 0$ represents the effect of own genetics on own SES. The basic result continues to hold, and also, the degree of meritocracy γ increases the correlation between genes and SES; a highly meritocratic society may in the long run lead to a highly unfair genes-SES gradient.

Proposition 4. *Under SGAM and equation (4), if genetics and SES are uncorrelated for the parents, then they are positively correlated for the children so long as* $0 < a < 1$ *or* $\gamma > 0$. *The correlation is increasing in* γ. *Also, so long as* $\gamma > 0$ *and either* $0 < a < 1$ *or* $\sigma > 0$, *the coefficient of parents' wealth on children's wealth exceeds* θ.

Surprisingly, in this case, the children's genes-SES correlation is not always increasing in θ. The reason is that when γ is high, a higher θ decreases the proportion of x_2' that comes via γ from own genetics, and increases the proportion that comes from parents' SES, which may be less strongly correlated with own genetics. Figure 3 plots the correlation for $\gamma = 0.15$, by a and θ. However, computing the asymptotic correlation shows that it is increasing in θ for all but values of θ very close to 1.

Third, we consider non-normal distributions of x_1 and x_2, non-normal shocks ε and η, and non-linear attractiveness functions. Suppose

$$i(x) = f(ax_1, (1-a)x_2) \tag{5}$$

with f strictly increasing in both its arguments. Our sole condition on the distribution of x is that a positive measure of the population has attractiveness $i(x) = i$ where the distribution of $(x_1, x_2)|i$ is non-degenerate, i.e. not everybody with attractiveness i is both genetically and socially identical. In particular, this allows for discrete distributions, like some kinds of social status.

Proposition 5. *Let attractiveness be given by (5). Let* (x_1, x_2) *have any distribution such that a positive measure of the population has* $i(x) = i$ *where the conditional distribution of* $(x_1, x_2)|i(x) = i$ *is non-degenerate. Let* η *and* ε *be mean 0 and independent of* x *and each other. If genetics and SES are uncorrelated for the parents, then the correlation among children is non-negative, and strictly positive if* $0 < a < 1$.

Other extensions are possible. We assumed that all couples have the same number of children. If fertility increased

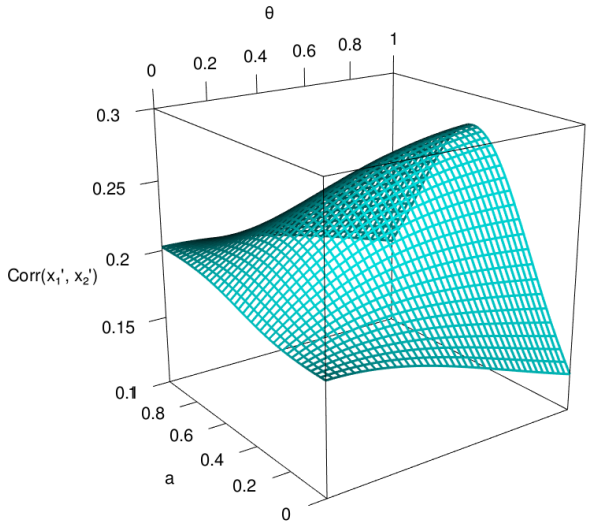


Figure 3: Correlation between children's genetics x'_1 and children's SES x'_2, by weight of genetics in matching a and SES inheritance θ, for meritocracy $\gamma = 0.15$. Other parameters: $\tau = 0.95$, $s = S = 1$, $\sigma = 0$.

with x_1 or x_2, we would expect this to reduce the variance of traits in the children's generation and possibly also their covariance. Here, matching preferences, as summarized in the a parameter, are exogenous. It would be natural to model a as an equilibrium outcome. For example, if parents care about their children's wealth, a might decrease in θ and increase in γ. Lastly, a gene-environment *interaction* (e.g. $x'_2 = \gamma x'_1 + \theta \frac{x_2+y_2}{2} + \zeta x'_1 \frac{x_2+y_2}{2}$) might increase the gene-environment correlation some more.

Discussion

The meanings of both social status, and "good genes" in the marriage market, are likely to vary across societies. Social status could encompass variables like social class or caste; ethnic identity in "ranked" ethnic systems; or in modern societies, SES, including wealth, income and occupation. Regarding genetics, standards of physical attractiveness, and other genetically-influenced characteristics which make someone a "good match", vary across societies and over time. The central prediction of the model is that whatever those characteristics, in the long run they will become correlated with SES.

Recent empirical work shows high persistence of SES over time, in particular at the top. Clark (2021) argues that this is due to unobserved genetic variation. Proposition 4 shows that if genes affect own wealth directly, under assortative mating, the regression coefficient of parents' wealth on own wealth exceeds the "direct" coefficient θ, because parents' wealth correlates with parents' genetics and via that with own wealth. Thus, regressions of wealth on wealth may

include the effect of unobserved genetic variation. This may be a confound due to pre-existing gene-SES correlation (if $\sigma > 0$). But under SGAM it can also be a genuine cause, since changes in someone's wealth may indeed affect the identity of their spouse, and therefore the genetics of their offspring.

The converse also holds: regressions of children's characteristics on their genetics alone risk overestimating the effect of genetics, by confounding it with the effects of correlated socio-economic status. Recent work in genetics has shown this. Polygenic scores for educational attainment have smaller effects in between-sibling regressions, where between-family variation in SES is partialled out and where genetic variants are guaranteed to be randomly allocated, than in regressions which pool the whole sample (Howe et al. 2021). Parents' genetic variants which are *not* passed on to children predict children's characteristics, via environmental effects (Kong et al. 2018).

The model predicts variation in the strength of SGAM. In particular, in "caste societies" where there is complete endogamy within social status groups, there is no scope for SGAM, because marriage partners do not trade off genetics for social status. Also, SGAM is increased by the institutional variable θ, which captures intergenerational persistence of SES. This implies that policy has long-run effects on the social structure: reducing θ not only increases intergenerational mobility, but reduces the correlation of genes with SES, and hence the unfairness of what Harden (2021) calls the "genetic lottery".

Data and methods

In modern societies, both SGAM and meritocratic mobility may be at play. Genetic variants that cause higher SES, e.g. higher income and wealth, will be passed down along with that status. At the same time, higher SES and "good genes" will assort in the marriage market. To test this, we look at correlations among spouses between one partner's SES and the other partner's genetics.

We use data from the UK Biobank, a study of about 500,000 individuals born between 1935 and 1970 (Bycroft et al. 2018). The Biobank contains information on respondents' genetics, derived from DNA microarrays, along with questionnaire data on health and social outcomes. The Biobank does not contain explicit information on spouse pairs. We categorize respondents as pairs if they:

- had the same home postcode on at least one occasion;[9]
- both reported the same homeownership/renting status, length of time at the address, and number of children;
- attended the same UK Biobank assessment center on the same day;
- both reported living with their spouse ("husband, wife or partner");
- consisted of one male and one female.

[9] A typical UK postcode contains about 15 properties.

We also eliminate all pairs where either spouse appeared more than once in the data. This leaves a total of 35,682 pairs. Some of these could be false positives, i.e. people who are not each others' spouse but simply live in the same postcode. To validate the accuracy of our pairs, we use genetic relationships. Some respondents in the UK Biobank sample have a child (inferred from genetic data) who is also in the sample. Among our spouse pairs, 511 have a genetic child of at least one partner in the sample. For 441 of these, the child is the genetic child of both partners. If this subsample is representative, then at least 86% of the pairs who have had a child, have had a child together. This is a lower bound, because those who had a child with someone else may also have had a child with their partner who is not in the UK Biobank sample. As a point of comparison, 11% of families with dependent children included a stepchild in England and Wales in 2011 (National Statistics 2014).

It is still possible that some pairs in our data may not be actual spouses. In the appendix, to sign any possible bias in our estimates resulting from this, we use a dataset of "known fake" pairs. We show that estimated coefficients of interest are closer to zero among these fake pairs than among our candidate "real pairs". Because of this, any fake pairs remaining in our data are likely to bias our coefficients towards zero.

Our key dependent variable is spouse's *Polygenic Score for Educational Attainment* (PSEA). A polygenic score is a DNA-derived summary measure of genetic risk or propensity for a particular outcome, created from summing small effects of many common genetic variants, known as Single Nucleotide Polymorphisms (SNPs). We focus on PSEA rather than other polygenic scores for two reasons. First, educational attainment plays a key role in human mate search. People are attracted to educated potential partners (Buss and Barnes 1986; Belot and Francesconi 2013); spouse pairs often have similar levels of educational attainment, as well as similar PSEA (Vandenberg 1972; Schwartz and Mare 2005; Greenwood et al. 2014; Hugh-Jones et al. 2016). Second, PSEA predicts a set of important socioeconomic variables, including not only education but also social and geographic mobility, IQ, future income and wealth (Belsky et al. 2016; Barth, Papageorge, and Thom 2020; Papageorge and Thom 2020).[10]

We calculate PSEA using per-SNP summary statistics from Lee et al. (2018), re-estimated excluding UK Biobank participants.[11] We normalize the score to have mean 0 and variance 1. Because polygenic scores are created from estimates of many presumably tiny effects, they contain a large amount of noise relative to the true best estimator that could be derived from genetic data. For instance, PSEA explains only 11–13% of variance in educational attainment (out of sample, Lee et al. 2018), whereas the true proportion explained by genetic variation – the heritability – is estimated from twin studies to be about 40% (Branigan, McCallum, and Freese 2013). In addition, polygenic scores are no more guaranteed to be causal than any other independent variable. For example, social stratification by ancestry

[10] See Papageorge and Thom (2020) for a detailed discussion of polygenic scores, aimed at economists.

[11] PSEA was computed by summing the alleles across ~1.3 million genetic variants weighted by their effect sizes as estimated in genome-wide association studies (GWASs) that excluded UK Biobank. PSEA was then residualized on the first 100 principal components of the SNP array data. Further details can be found in Abdellaoui et al. (2019).

may lead genes to be associated with educational attainment even if they play no causal role (Selzam et al. 2019).

Despite these points, PSEA has non-trivial estimated effects on educational attainment. PSEA correlates with measures of education, including university attendance and years of full-time education; within-siblings regressions, where PSEA is randomly assigned by the "lottery of meiosis", confirm this correlation is at least partly causal (Lee et al. 2018). We recheck these facts within the UK Biobank sample. In a simple linear regression (N = 408,524) of university attendance on PSEA, a one-standard-deviation increase in PSEA was associated with a 9.2 percentage point increase in the probability of university attendance ($p < 2 \times 10^{-16}$). In a within-siblings regression among genetic full siblings (N = 36,748), the increase was 4.5 percentage points ($p < 2 \times 10^{-16}$). This suggests that about half of the raw correlation of PSEA with university attendance is down to confounds like good environments or parental nurture, while the remainder is causal. Still, the causal effect remains substantial: for a rough comparison, the (ITT) effect on college attendance of the Moving To Opportunity experiment in the US was 2.5 percentage points (Chetty, Hendren, and Katz 2016).

We use two measures of socio-economic status: income, and university attendance. Income is a direct measure of SES. University attendance is a predictor of income over the whole life course, and a form of SES in itself. The UK Biobank data only contains a direct measure of current household income, which is inappropriate for our purposes because it includes income from both spouses and is measured after marriage. Instead, we estimate income in £000s in the respondent's first job, by matching the job's Standard Occupational Classification (SOC) code with average earnings by SOC from National Statistics (2007). Job codes are only available for a subset of 7681 respondents among our pairs.

Figure 4 illustrates the core idea of SGAM within our pair data. The X axis shows a measure of one partner's socio-economic status: university attendance or income. The Y axis plots the other partner's mean PSEA. Both males and females who went to university had spouses with higher PSEA. So did males and females with higher income in their first job. Since DNA is inherited, these people's children will also have higher PSEA.

These figures do not prove that SGAM 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 genetic assortative mating (GAM) alone (Hugh-Jones et al. 2016). Indeed, recent studies show much higher levels of GAM than could be explained by matching on the observed education phenotype alone (Okbay et al. 2022). So, to demonstrate SGAM, we need a source of social status which is exogenous to genetics. Also, the link between social status and spouse genetics is likely to be noisy, for three reasons: first, polygenic scores contain a large amount of error, as discussed above; second, causal mechanisms behind variation in social status are likely to be noisy; third, to paraphrase Shakespeare (1595), the spouse matching process is highly unpredictable. So, we need a large N to give us sufficient power. This rules out time-limited shocks

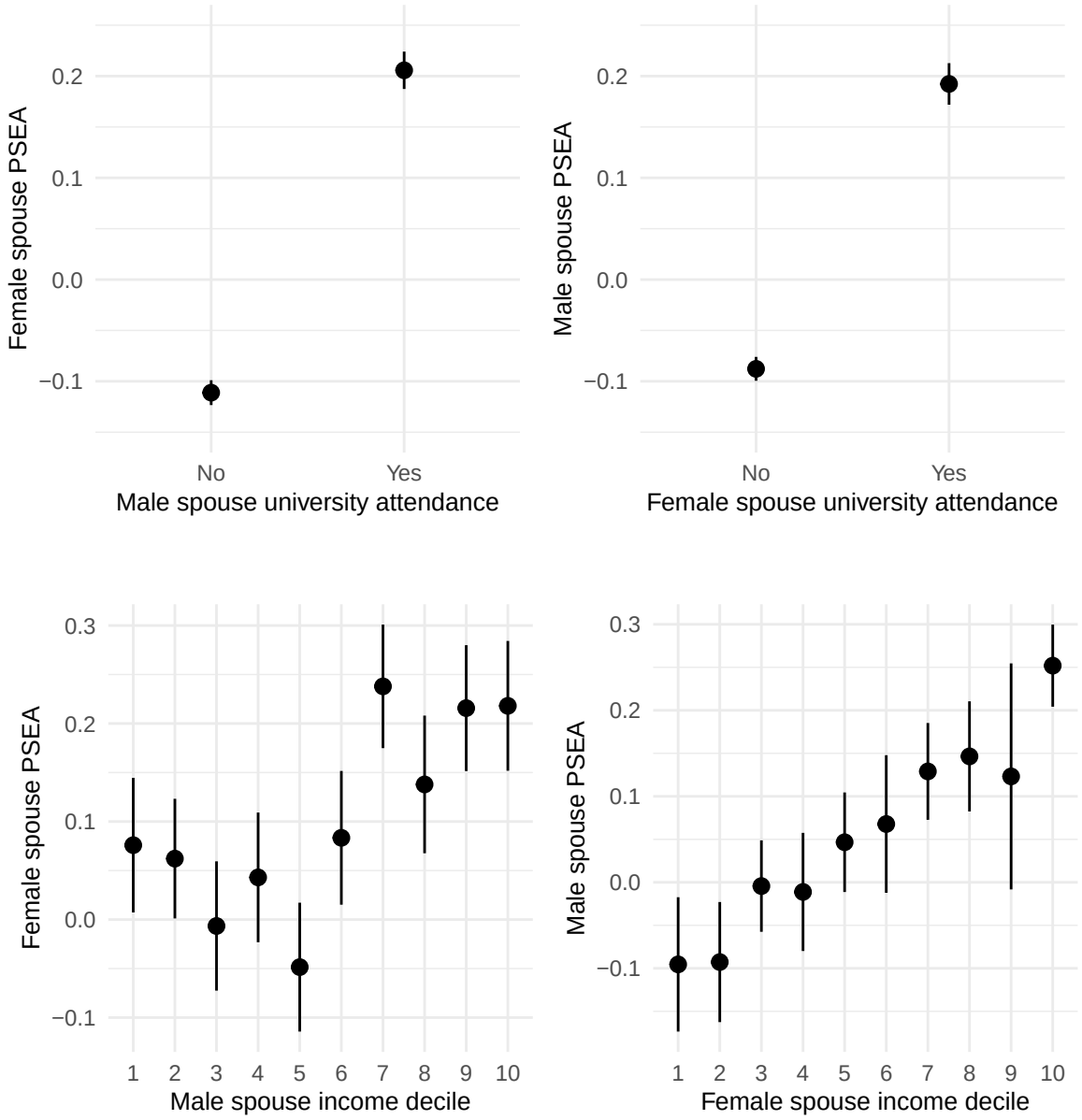


Figure 4: Spouse PSEA against own university attendance and own income in first job. Lines show 95% confidence intervals.

such as changes to the school leaving age (Davies et al. 2018).

We use *birth order*. It is known that earlier-born children receive more parental care and have better life outcomes, including measures of SES such as educational attainment and occupational status (Lindahl 2008; Booth and Kee 2009; Black, Devereux, and Salvanes 2011). On the other hand, all full siblings have the same *ex ante* expected genetic endowment from their parents, irrespective of their birth order. This is guaranteed by the biological mechanism of meiosis, which ensures that any gene is transmitted from either the mother or the father to the child, with independent 50% probability (Mendel 1865; Lawlor et al. 2008). For example, siblings' expected polygenic score is equal to the mean of their parents' polygenic scores.[12] We can therefore use birth order as a "shock" to social status. We put "shock" in quotes because we do not claim that birth order is exogenous to all other variables. For example, it naturally correlates with parental age, and it may also correlate with household SES at the time of birth. We only claim that birth order is exogenous to genetic variation.

Our main independent variable is respondents' birth order, i.e. their number of elder siblings plus one. For controls we use family size, i.e. respondents' total number of siblings including themselves; month of birth; age at interview; respondents' own PSEA; and their father's and/or mother's age at their birth (calculated from parent's current age, only available if the parent was still alive). For most regressions, we use only respondents with between 1 and 5 siblings, i.e. with a family size of 2-6.

Decomposing the birth order effect on spouse genetics

Ideally, we might prefer to use birth order as an instrument for SES. However, our measures of social status are noisy and incomplete. For example, we know whether subjects attended university, but not which university. Birth order likely affects both measured and unmeasured aspects of SES. So, an instrumental variables approach would be likely to fall foul of the exclusion restriction.

Instead, we conduct a mediation analysis, following the strategy of Heckman, Pinto, and Savelyev (2013). We first confirm statistically that birth order affects our measures of respondents' SES (income and education). Then, we regress spouse's PSEA on birth order, with and without controlling for SES. 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 SES is controlled for, that is evidence that SES mediates the effect of birth order.

We follow Heckman, Pinto, and Savelyev (2013) to decompose the aggregate treatment effect into components due to

[12] Although genetic variation is randomly assigned to children at birth, genetics and birth order could be dependent if parents' choice of whether to have more children is endogenous to the genetic endowment of their earlier children. We check for this below. Isungset et al. (2021) also find that birth order differences in education are not genetic.

observed and unobserved proximate channels affected by the treatment. Our aim is to estimate the effect of SES (as an effect of birth order) on spouse PSEA.

Assume B is a multivalued variable indicating birth order. Let Y_b be the counterfactual outcome (spouse PSEA) for the first-born, second-born etc. Given b, spouse PSEA is assumed to be independent across observations conditional on some predetermined controls which are assumed not to be affected by B.

Let m_b be a set of mediators, i.e. proximate outcomes determined by b, which account (at least in part) for the b treatment effect on spouse PSEA. We can think of m_b as all the effects on attractiveness, such as increments to SES, health, cognitive and non-cognitive skills, that individuals receive due to their birth rank. We can split the mediators in m_b into a set J_m of measured mediators, including university attendance and income in first job, and a set J_u of mediators that we cannot measure.

Our linear model is:

$$Y_b = \kappa_b + \sum_{j \in J_m} \alpha_b^j m_b^j + \sum_{j \in J_u} \alpha_b^j m_b^j + \mathbf{X}'\boldsymbol{\beta}_\mathbf{b} + \tilde{\varepsilon}_b = \tau_b + \sum_{j \in J_m} \alpha_b^j m_b^j + \mathbf{X}'\boldsymbol{\beta}_\mathbf{b} + \varepsilon_b \tag{6}$$

where $\tilde{\varepsilon}_b$ is a mean-zero residual assumed independent of m_b and $\mathbf{X}$; $\tau_b = \kappa_b + \sum_{j \in J_u} \alpha_b^j E(m_b^j)$; and $\varepsilon_b = \tilde{\varepsilon}_b + \sum_{j \in J_u} (m_b^j - E(m_b^j))$. We simplify by assuming that $\beta_b = \beta$ and $\alpha_b = \alpha$ for all b, i.e. that the effects of $\mathbf{X}$ and m_B don't differ by birth order.[13] We assume differences in unmeasured investments due to b are independent of $\mathbf{X}$.

We use a linear model for each observed mediator variable:

$$m_b^j = \mu_{0,j} + \mathbf{X}'\boldsymbol{\mu}_{\mathbf{1,j}} + \mu_{2,j} \cdot b + \eta_j, j \in J_m \tag{7}$$

where η_j is a mean-zero residual. We also assume the treatment-specific intercepts are linear in b:

$$\tau_b = \tau_0 + \tau b. \tag{8}$$

With the simplifying assumptions above and substituting (7) and (8) into (6) we obtain:

$$Y_b = \tau_0 + \tau b + \sum_{j \in J_m} \alpha^j (\mu_{0,j} + \mathbf{X}'\boldsymbol{\mu}_{\mathbf{1,j}} + \mu_{2,j} \cdot b + \eta_j) + \mathbf{X}'\boldsymbol{\beta} + \varepsilon_b \tag{9}$$

[13] Under the assumption that measured and unmeasured mediators are uncorrelated, we can test these assumptions by running an OLS regression of an extended model (11) where we interact the measured mediators and controls with the treatment B, and test the significance of the coefficients on the interaction terms ($\boldsymbol{\alpha}_\mathbf{b} = 0$ and $\boldsymbol{\beta}_\mathbf{b} = 0$). See Heckman, Pinto, and Savelyev (2013) and Fagereng, Mogstad, and Rønning (2021) for details and different applications. When we run the model with interactions, only one interaction is significant after Bonferroni correction at $p < 0.05/34$: the interaction of income in first job with the dummy for birth order 6. So overall, the uninteracted model seems a good enough approximation.

Using equation (9), we can decompose the average treatment effect of a change from birth order b to b' into the effect of measured mediators m^j and unmeasured mediators on the outcome:

$$E(Y'_b - Y_b) = \tau(b' - b) + \sum_{j \in J_m} \alpha^j E(m^j_{b'} - m^j_b) = \underbrace{\tau(b' - b)}_{\text{Effect of unmeasured mediators}} + \underbrace{\sum_{j \in J_m} \alpha^j \mu_{2,j}(b' - b)}_{\text{Effect of measured mediators}} \tag{10}$$

We are primarily interested in estimating the effect of SES on spouse PSEA, amongst the measured mediators, and furthermore we would like to measure the relative importance of SES compared to other factors in predicting spouse PSEA.

We therefore estimate:

$$Y = \tau_0 + \tau B + \sum_{j \in J_m} \alpha^j m^j_b + \mathbf{X}'\boldsymbol{\beta} + \varepsilon \tag{11}$$

Estimating the above by OLS will generate unbiased estimates of α^j if m^j is measured without error and is uncorrelated with the error term ε. Since ε contains both individual disturbances and differences in unmeasured investments due to birth order, there are two identifying assumptions that need to hold for unbiased OLS estimates: (a) the measured investments (specifically SES) should be independent of unmeasured investments generated by birth order. Failing this, the estimates of α^j will be conflated with the effects of unmeasured investments. Second, (b) the measured investments should be uncorrelated with other shocks $\tilde{\varepsilon}_b$.

By running a least square regression of (11), we can estimate τ and α^j. If assumption (a) holds, the part of the birth order treatment effect on spouse PSEA that is due to measured mediators, including SES, can be constructed using the estimated α^j and the effects of birth order on measured mediators. We can estimate these effects in a second step, from OLS regressions based on equation (7) for each measured mediator (in particular university attendance and income) on $\mathbf{X}$ and B. The part of the birth order effect that is due to university attendance (or income) on spouse PSEA will be the coefficient of university/income in the regression of spouse PSEA in equation (11), multiplied by the coefficient of birth order on university/income from equation (7).

Results

We first regress our measures of socio-economic status, university attendance and income from first job, on birth order in our sample. We also do the same for four non-SES mediators that could be affected by birth order: fluid IQ, height, body mass index (BMI) and a measure of self-reported health. We control for respondent's own PSEA and their parents' age at birth (see below). Table 1 shows that birth order significantly predicts all these variables. Effect sizes are quite

Table 1: Regressions of mediators on birth order

	University	Income	Fluid IQ	Height	BMI	Health
Birth order	−0.0790 ***	−1.0899 *	−0.2733 ***	−0.7012 ***	0.1907 **	−0.0430 ***
	(0.0067)	(0.4264)	(0.0304)	(0.1355)	(0.0662)	(0.0103)
PSEA	0.0889 ***	1.5144 ***	0.3180 ***	0.1970 *	−0.4281 ***	0.0533 ***
	(0.0046)	(0.3307)	(0.0200)	(0.0921)	(0.0456)	(0.0068)
Parents' age at birth	0.0163 ***	0.2623 ***	0.0588 ***	0.1514 ***	−0.0989 ***	0.0110 ***
	(0.0012)	(0.0722)	(0.0053)	(0.0241)	(0.0117)	(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

Estimates from OLS regressions with the mediators (university attendance, income, fluid IQ, height, BMI, self-reported health) as dependent variables, and own birth order as the main independent variable. PSEA is the polygenic score for educational attainment, which is normalized with mean 0 and standard deviation 1. We include parents' age at birth (the mean of parents' ages) and further controls to ensure the balance of covariates across birth order. All data is from the UK Biobank for a sample of UK individuals born between 1935 and 1970. *** p $<$ 0.001; ** p $<$ 0.01; * p $<$ 0.05; + p $<$ 0.1. Standard errors: robust.

substantial: on average, one extra elder sibling reduces the chance of attending university by about 7.9 percentage points, income by about £1,089, fluid IQ by about 0.27 points on a 13 point test, height by about 0.7 centimeters, and self reported health by 0.043 points on a 4-point scale; and increases BMI by 0.19.

Next we run regressions of spouse PSEA on birth order, within our dataset of spouse pairs. Table 2 reports the results. Column 1 reports results controlling only for family size (using dummies). As expected, higher birth order is negatively associated with spouse's PSEA, though the estimated effect size is small and insignificant. Column 2 reports results controlling for the respondent's own PSEA, as well as dummies for birth year to control for cohort effects, and dummies for birth month to control for seasonal effects. The effect size of birth order is not much changed.

Column 3 reports results controlling for parents' age at birth. Within a family, later children have older parents by definition. Older parents have more life experience and may have higher income, which would presumably help later

children.[14] Including parents' age means we can separate the effect of parental age from birth order. This reduces the N by a lot, since only respondents with a live parent reported the necessary data. However, the effect of birth order jumps in size and becomes significant at the 5 per cent level. Meanwhile, parents' age has a positive effect. This suggests that estimates in columns 1-2 mixed two opposite-signed effects: having older parents versus being later in birth order.

Table 2: Regressions of spouse PSEA on birth order

	(1)	(2)	(3)
Birth order	−0.0091	−0.0075	−0.0314 *
	(0.0074)	(0.0074)	(0.0146)
Own PSEA		0.0650 ***	0.0573 ***
		(0.0065)	(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
R2	0.003	0.010	0.013

Estimates from OLS regressions with spouse PSEA as dependent variable, and own birth order as the main independent variable. PSEA is the polygenic score for educational attainment, which is normalized with mean 0 and standard deviation 1. We include own PSEA, parents' age at birth (the mean of parents' ages), and further controls (family size, birth year, and birth month dummies) in columns 2−3 to ensure the balance of covariates across birth order. All data is from the UK Biobank for a sample of UK individuals born between 1935 and 1970. *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Having tested that birth order affects spouse's PSEA, we now look for potential mediators of this effect. Despite the lower N, we continue to control for respondents' parents' age, since this removes a confound which would bias our results towards zero.[15]

Table 3 shows the results. Column 1 shows the effect of birth order, using the same specification as column 3 of the previous table. The remaining columns add potential mediators of birth order effects. Column 2 controls for our first measure of socio-economic status: university attendance. We also include potential non-SES mediators, which are

[14] We often only have data only for one parent. We use this, or take the mean if we have both. There are also potential genetic effects from parental age, though recent research has rejected these in favour of "social" explanations (Kristensen and Bjerkedal 2007; Black, Devereux, and Salvanes 2011). Cochran and Harpending (2013) report that mutational load is approximately linear in father's age, while it is constant in mother's age. We observe very similar results if we control only for father's age at respondent's birth.

[15] The appendix reports results without controlling for parents' age.

affected by birth order and might affect spouse matching: fluid IQ, height, BMI and self-reported health. Column 3 adds our second measure of socio-economic status, income in first job. Column 4 includes both.

When we add university attendance and other mediators (column 2), the effect of birth order drops and becomes insignificant, while the coefficient for university is positive and highly significant. Fluid IQ, height and BMI are also positive and significant, while self-reported health has the right sign but is insignificant. Controlling for income instead of university attendance (column 3), again the effect of birth order shrinks and becomes insignificant, while income has a positive and highly significant effect. Lastly, the same pattern holds when we control for both university and income (column 4).

Under the assumptions discussed above, we can estimate the proportion of the birth order effect that is mediated by these variables. Table 4 reports this for each model in columns 2-4. Each estimate is the coefficient of birth order on the mediator, times the coefficient of the mediator on spouse PSEA, divided by the coefficient of birth order on spouse PSEA estimated from column 1, i.e. without mediators. Education explains about 38-55 percent of the effect, much more than all the other mediators. Income, fluid IQ, height and BMI all explain between 6 and 17 percent of the effect, depending on the specification.

These results provide evidence that birth order affects spouse PSEA via education and income, with education being especially important.

Our next regressions split up the data into subsets. Cultural stereotypes often assume that the link between status and genes is not symmetric across the genders, for example, that males with high SES are particularly likely to marry attractive spouses. Claim 5 showed that these differences would strengthen the effects of SGAM. To test for this, we separately regress female spouses' PSEA on male birth order, and male spouses' PSEA on female birth order. We also rerun regressions among the subset of individuals who had children. A significant result here will confirm that the association between status and genetics is carried over into the next generation.

Table 5 shows the results. Columns 1 and 2 present results using birth order of male respondents to predict female spouses' PSEA. Column 1 shows the regression of birth order plus controls; in column 2, we add university attendance and non-SES mediators (here, we exclude first job income so as to keep our N large). Columns 3 and 4 repeat the exercise for female respondents, using their birth order to predict male spouses' PSEA. The effect of birth order is imprecisely estimated in these subsets due to the lower sample size. However, the pattern of coefficient sizes is the same as in the main regression: the coefficient of birth order is about -0.3, and adding university attendance reduces the absolute size of the birth order effect. Columns 5 and 6 show results from regressions on the subsample of couples with children. Here, birth order is significant in the base specification, and again, university attendance still seems to mediate the birth order effect.

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
R2	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

Estimates from OLS regressions with spouse PSEA as dependent variable, and own birth order and mediators (university attendance and income) as the main independent variables. Columns 2−4 correspond to model 7. PSEA is the polygenic score for educational attainment, which is normalized with mean 0 and standard deviation 1. We include own PSEA, mean of parents' ages at birth, potential non-SES mediators (fluid IQ, height, BMI, self-reported health) and further controls (family size, birth year, and birth month dummies) to ensure the balance of covariates across birth order. All data is from the UK Biobank for a sample of UK individuals born between 1935 and 1970. *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

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

Percentage of the effects of birth order in Table 3, columns 2 to 4, explained by by each mediating variable.

Table 5: Regressions of spouse PSEA on birth order: subsets

	Male respondents	Male respondents	Female respondents	Female respondents	With children	With children
Birth order	−0.030	−0.001	−0.031	−0.009	−0.035 *	−0.007
	(0.022)	(0.022)	(0.019)	(0.019)	(0.015)	(0.015)
University		0.272 ***		0.169 ***		0.217 ***
		(0.033)		(0.031)		(0.024)
Fluid IQ		0.019 *		0.015 *		0.022 ***
		(0.008)		(0.007)		(0.006)
Height		0.004		0.004 +		0.002 *
		(0.002)		(0.002)		(0.001)
BMI		−0.008 *		−0.012 ***		−0.011 ***
		(0.004)		(0.003)		(0.002)
Self-reported health		0.028		0.010		0.022
		(0.022)		(0.021)		(0.016)
Own PSEA	0.059 ***	0.022	0.057 ***	0.030 *	0.062 ***	0.029 **
	(0.015)	(0.015)	(0.014)	(0.014)	(0.011)	(0.011)
Parents' age at birth	0.013 **	0.005	0.011 **	0.005	0.013 ***	0.006 *
	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
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	4675	4675	5531	5531	9127	9127
R2	0.017	0.043	0.017	0.031	0.015	0.035

Estimates from OLS regressions corresponding to columns 1 and 2 in Table 3, separately for males, females and respondents with children. Spouse PSEA is the dependent variable, and own birth order and university attendance are the main independent variables. PSEA is the polygenic score for educational attainment, which is normalized with mean 0 and standard deviation 1. We include own PSEA, parents' age at birth (the mean of parent's ages) and further controls (family size, birth year, and birth month dummies) to ensure the balance of covariates across birth order. All data is from the UK Biobank for a sample of UK individuals born between 1935 and 1970. *** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Robustness

Although all children of the same parents have the same polygenic scores in expectation, it might still be possible that genetics correlates with birth order within the sample. This could happen in three ways. First, siblings with high birth order will typically come from larger families than those with low birth order, and parents of different-sized families are likely to differ systematically on many dimensions, including genetics. We controlled for this by including a full set of family size dummies in the regression. Second, there could be selection bias. For example, if later siblings with high PSEA, and earlier siblings with low PSEA, are more likely to enter the sample, then this would bias our results. Thirdly, parents might choose family size in a way related to genetics. For example, suppose that when the first child has a phenotype reflecting a high PSEA, parents are more likely to have a second child. Then within the subset of two-child families, first children would have higher-than-average PSEA, while second children would not.

To check for the latter two problems, we run balance tests on 33 different polygenic scores.[16] We regress each score on own birth order, controlling for family size. No scores were significant at $p < 0.10/33$. Four scores were significant at $p < 0.10$, all with effect sizes of less than 0.02 per standard deviation. Table 9 in the appendix reports regressions controlling for these scores. Results are almost unchanged. To test whether polygenic scores might vary across birth orders within a particular family size, we also regress each score on a full set of birth order dummies, interacted with a full set of family size dummies. None of the 495 birth order coefficients were significant at $p < 0.001$. However, among families of size 3, there is a marginally significant positive correlation of birth order with own PSEA (effect size $0.0277, p = 0.06$). Table 11 in the appendix therefore reports regressions with families of size 3 excluded. Results are substantially unchanged. Of course, there could still be unmeasured genetic variants which correlate with birth order in our sample. Nevertheless, a wide set of polygenic scores shows no large or significant correlation. This makes us more confident that birth order is indeed exogenous to genetics.

Another concern is that our chosen SES mediators might not be exogenous. We have already seen that birth order affects intelligence, height, BMI and health. So there might be other unobserved variables which mediate the effect of birth order on spousal PSEA, and which correlate with education or income, but which do not themselves capture SES. If so, that would threaten our claim that education and income are important mediators. However, the effects of education on spouse PSEA in Table 3, and of birth order on education in Table 1, are both large and highly significant. In other literature on spouse matching, education is a common, robust and significant predictor. For these admittedly informal reasons, we think that our results are unlikely to be driven wholly by other unobserved mediators.

[16] Polygenic scores were residualized on the first 100 principal components of the genetic data. Scores were for: ADHD, age at menarche, age at menopause, agreeableness, age at smoking initiation, alcohol use, Alzheimer's, autism, bipolarity, BMI, body fat, caffeine consumption, cannabis (ever vs. never), cognitive ability, conscientiousness, coronary artery disease, smoking (cigarettes per day), type II diabetes, drinks per week, educational attainment (EA2 and EA3), anorexia, extraversion, height, hip circumference, major depressive disorder, neuroticism, openness, smoking cessation, schizophrenia, smoking initiation, waist circumference, and waist-to-hip ratio. For full details of score construction, see Abdellaoui et al. (2019).

The appendix reports other robustness checks, including a specification with separate dummies for each value of birth order, and replacing university attendance with age of leaving full-time education. The basic pattern that birth order coefficients shrink after adding SES mediators appears robust across our variations.

Conclusion

Our empirical analysis shows that in a contemporary developed society, individuals with fewer elder siblings had spouses with higher PSEA. We also provide evidence that these effects are mediated by socio-economic status, specifically income and education. We interpret this as evidence of social-genetic assortative mating (SGAM).

There are multiple mechanisms by which advantage is transmitted across generations. High-income parents may invest more in their children's human capital, transfer wealth via gifts and bequests, model valuable skills, or provide them with advantageous social networks. They may also pass on causally relevant genetic variants. This channel has been proposed as a reason for the surprising persistence of inequality over generations (Clark and Cummins 2015; Clark 2021). One problem with this theory is that in the absence of assortative mating, genetic variation regresses swiftly to the mean, with coefficient $r = 0.5$ per generation. Thus to explain long-run persistence, the genetic theory seems to require very high levels of genetic assortative mating. SGAM may help to solve this puzzle. Persistence will be increased if, in addition to genetic assortative mating, high SES itself attracts "good genes". At the same time, SGAM changes the interpretation of genetics. As our model shows, genetic variation is not an exogenous input into the social system, but an endogenous outcome – not a confound for wealth, but a mediator.

SGAM also provides a new explanation for the observed association of genes with SES. Unlike meritocratic social mobility, the leading alternative explanation, SGAM may apply to a historically wide range of societies. Whilst a degree of meritocracy exists in modern capitalist economies, opportunities to earn status have been far more limited in most societies throughout history (Smelser and Lipset 1966). On the other hand, assortative mating is likely to be a cultural universal (Buss 1989). Thus, SGAM predicts that genetic differences across social status should exist in all stratified societies. In fact, people in many societies have believed that innate traits do vary by social status. The ancient Greeks described the social elite as *καλοί κἀγαθοί* ("fine and good"), while the Roman nobility were the *optimates* ("best").[17] This belief has been explained by the tendency to believe in a just world (Furnham 1993), or as an ideology promoted by the dominant class (e.g. Gramsci 1971). However, it may also simply have been a recognition of (social) reality. In other words, the belief that elites are taller, stronger, better-looking, etc. is not much different from the belief that elites are richer and more powerful, and may be held for similar reasons. In future, it may be possible to directly test for genetic differences across social status in ancient DNA samples.

[17] The appendix contains a selection of relevant historical quotations.

In our model, the association between SES and genetic variation depends on economic and social institutions. Institutions that make wealth more persistent across generations also increase the correlation between SES and genetics. If so, then institutional differences may have long run effects over generations by altering the genes-SES gradient. There could be hysteresis, with initial social differences cumulating over time via their effect on genetic inequality. On the other hand, increases in the level of meritocracy paradoxically also strengthen the genes-SES gradient, suggesting a deep conflict between meritocracy and egalitarianism. Lastly, the structure of marriage markets also affects the gradient. We have not been able to test these comparative statics here: doing so across societies is a challenge for future work.

The broadest message of this paper is that genetics are a social outcome. Both popular and scientific discourse often parse genetics as "nature", in opposition to "nurture" or "environment" (e.g. Chakravarti and Little 2003; Plomin 2019). This reflects the fact that our individual genetic endowment is fixed at birth, affects our body and brain through proximate biological mechanisms, and cannot be changed by our social environment (though genes always interact with the environment to cause individual outcomes). But the idea that human genetics are natural can be highly misleading. Humans inherit their genes from their parents, along with other forms of inheritance such as economic and cultural capital. Human parents, in turn, form spouse pairs and bear children within social institutions. A person's genetic inheritance is a social and historical fact about them, not just a fact of nature. As Marx (1844) wrote, "History is the true natural history of man". Genetic endowments can even be viewed as another form of capital, alongside human, social and cultural capital: a resource to be sought, accumulated and competed over. The analysis of this kind of capital is an exciting area for further research, which will require the contributions of both economists and geneticists.

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Appendix

Contents

Proofs

Proof of Proposition 1. By a change of variable, rewrite:

$$\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} \to \begin{pmatrix} x_1 \\ u \end{pmatrix} \text{ where } u = \frac{ax_1 + (1-a)\, x_2}{\sqrt{a^2 s^2 + (1-a)^2 S^2 + 2a\,(1-a)\,\sigma}} = \frac{ax_1 + (1-a)\, x_2}{\sigma_I}$$

is the attractiveness rescaled to $\mathcal{N}(0,1)$. Thus,

$$\begin{pmatrix} x_1 \\ u \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ a/\sigma_I & (1-a)/\sigma_I \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \end{pmatrix}.$$

Note that the means are still zero, but the covariance of (x_1, u) is:

$$\begin{aligned} \mathbb{C}\begin{pmatrix} x_1 \\ u \end{pmatrix} &= \begin{pmatrix} 1 & 0 \\ a/\sigma_I & (1-a)/\sigma_I \end{pmatrix} \begin{pmatrix} s^2 & \sigma \\ \sigma & S^2 \end{pmatrix} \begin{pmatrix} 1 & a/\sigma_I \\ 0 & (1-a)/\sigma_I \end{pmatrix} \\ &= \begin{pmatrix} s^2 & A \\ A & 1 \end{pmatrix} \end{aligned}$$

where

$$A = \frac{as^2 + (1-a)\,\sigma}{\sqrt{a^2 s^2 + (1-a)^2 S^2 + 2a\,(1-a)\,\sigma}} = \frac{as^2 + (1-a)\,\sigma}{\sigma_I}.$$

Under SGAM, individual $\begin{pmatrix} x_1 \\ u \end{pmatrix}$ is matched with $\begin{pmatrix} y_1 \\ v \end{pmatrix}$ such that $u = v = t$.

The distribution of t is $\mathcal{N}(0,1)$. Therefore the vector $\begin{pmatrix} x_1 \\ y_1 \\ t \end{pmatrix}$ is normally distributed, with mean 0, and covariance

$$\Sigma = \begin{pmatrix} s^2 & A^2 & A \\ A^2 & s^2 & A \\ A & A & 1 \end{pmatrix}$$

Finally, we are interested in

$$\begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ -\frac{a}{1-a} & 0 & \frac{\sigma_I}{1-a} \\ 0 & 1 & 0 \\ 0 & -\frac{a}{1-a} & \frac{\sigma_I}{1-a} \end{pmatrix} \begin{pmatrix} x_1 \\ y_1 \\ t \end{pmatrix}$$

therefore again the means are 0 and

$$\begin{aligned} \mathbb{C} \begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} &= \begin{pmatrix} 1 & 0 & 0 \\ -\frac{a}{1-a} & 0 & \frac{\sigma_I}{1-a} \\ 0 & 1 & 0 \\ 0 & -\frac{a}{1-a} & \frac{\sigma_I}{1-a} \end{pmatrix} \Sigma \begin{pmatrix} 1 & 0 & 0 \\ -\frac{a}{1-a} & 0 & \frac{\sigma_I}{1-a} \\ 0 & 1 & 0 \\ 0 & -\frac{a}{1-a} & \frac{\sigma_I}{1-a} \end{pmatrix}^T \\ &= \begin{pmatrix} s^2 & \sigma & A^2 & AC \\ \sigma & S^2 & AC & C^2 \\ A^2 & AC & s^2 & \sigma \\ AC & C^2 & \sigma & S^2 \end{pmatrix} \end{aligned}$$

where:

$$\begin{aligned} A &= \frac{as^2 + (1-a)\,\sigma}{\sigma_I} \text{ and} \\ C &= \frac{a\sigma + (1-a)\,S^2}{\sigma_I}. \end{aligned}$$

□

Lemma 1. *$C^2 \leq S^2$, with strict inequality if $a > 0$ and $\sigma < 1$.*

Proof. Write

$$\begin{aligned} C^2 &= \frac{a^2\sigma^2 + (1-a)^2 S^4 + 2a(1-a)\sigma S^2}{a^2 s^2 + (1-a)^2 S^2 + 2a(1-a)\sigma} \\ &\leq \frac{a^2 s^2 S^2 + (1-a)^2 S^4 + 2a(1-a)\sigma S^2}{a^2 s^2 + (1-a)^2 S^2 + 2a(1-a)\sigma}, \text{since } \sigma/sS = Corr(x_1, x_2) \leq 1 \\ &= S^2 \end{aligned}$$

and observe that the inequality is strict if $a > 0$ and $\sigma < sS$. □

Proof of Claim 1. Under random matching, the joint distribution of $\left(\frac{\tau}{2}(x_1+y_1)+\varepsilon, x_2, y_2\right)$ is normal with mean $(0,0,0)$ and covariance

$$\mathbb{C} = \begin{pmatrix} \frac{\tau^2}{2}(s^2+\sigma)+1 & \frac{\tau}{2}\sigma & \frac{\tau}{2}\sigma \\ \frac{\tau}{2}\sigma & S^2 & 0 \\ \frac{\tau}{2}\sigma & 0 & S^2 \end{pmatrix}$$

Using the matrix formula for the conditional mean of normal variables,

$$\begin{aligned} \mathbb{E}\left[\frac{\tau}{2}(x_1+y_1)+\varepsilon \mid x_2=v, y_2=w\right] &= \begin{pmatrix} \frac{\tau}{2}\sigma & \frac{\tau}{2}\sigma \end{pmatrix} \begin{pmatrix} S^2 & 0 \\ 0 & S^2 \end{pmatrix}^{-1} \begin{pmatrix} v \\ w \end{pmatrix} \\ &= \frac{\sigma\tau}{2S^2}(v+w) \end{aligned}$$

In particular, if $\sigma = 0$, this expectation is equal to 0. □

Proof of Claim 2. From (3), the joint distribution of $\left(\frac{\tau}{2}(x_1+y_1)+\varepsilon, x_2, y_2\right)$ is normal with mean $(0,0,0)$ and covariance

$$\Sigma = \begin{pmatrix} \frac{1}{2}\tau^2(A^2+s^2)+1 & \frac{\tau}{2}(\sigma+AC) & \frac{\tau}{2}(\sigma+AC) \\ \frac{\tau}{2}(\sigma+AC) & S^2 & C^2 \\ \frac{\tau}{2}(\sigma+AC) & C^2 & S^2 \end{pmatrix}$$

Therefore

$$\begin{aligned} \mathbb{E}\left[\frac{\tau}{2}(x_1+y_1)+\varepsilon \mid x_2=v, y_2=w\right] &= \begin{pmatrix} \frac{\tau}{2}(\sigma+AC) & \frac{\tau}{2}(\sigma+AC) \end{pmatrix} \begin{pmatrix} S^2 & C^2 \\ C^2 & S^2 \end{pmatrix}^{-1} \begin{pmatrix} v \\ w \end{pmatrix} \\ &= \frac{1}{2}\tau\frac{\sigma+AC}{C^2+S^2}(v+w) \end{aligned} \tag{12}$$

In particular, if $\sigma = 0$, we have

$$\begin{aligned} A &= \frac{as^2}{\sqrt{a^2s^2+(1-a)^2S^2}} \text{ and} \\ C &= \frac{(1-a)S^2}{\sqrt{a^2s^2+(1-a)^2S^2}}, \end{aligned}$$

and (12) becomes

$$\begin{aligned}\mathbb{E}\left[\frac{\tau}{2}\left(x_1+y_1\right)+\varepsilon \mid x_2=v, y_2=w\right] &= \frac{1}{2}\tau\frac{a\left(1-a\right)s^2}{a^2s^2+2\left(1-a\right)^2S^2}\left(v+w\right)\\ &= \frac{1}{2}\tau\frac{a\left(1-a\right)\lambda}{a^2\lambda+2\left(1-a\right)^2}\left(v+w\right)\end{aligned}$$

where $\lambda = s^2/S^2$ is the ratio of genetic variance to wealth variance. The coefficient $\frac{a(1-a)\lambda}{a^2\lambda+2(1-a)^2}$ is increasing, then decreasing in a and is 0 for $a=0$ or $a=1$. □

Proof of Claim 3. Under random matching, the covariance matrix for children's characteristics is:

$$\begin{aligned}\mathbb{C} &= \begin{pmatrix}1&0\\0&1\end{pmatrix}+\begin{pmatrix}\frac{\tau}{2}&0&\frac{\tau}{2}&0\\0&\frac{\theta}{2}&0&\frac{\theta}{2}\end{pmatrix}\begin{pmatrix}s^2&\sigma&0&0\\\sigma&S^2&0&0\\0&0&s^2&\sigma\\0&0&\sigma&S^2\end{pmatrix}\begin{pmatrix}\frac{1}{2}\tau&0\\0&\frac{1}{2}\theta\\\frac{1}{2}\tau&0\\0&\frac{1}{2}\theta\end{pmatrix}\\ &= \begin{pmatrix}\frac{1}{2}s^2\tau^2+1&\frac{1}{2}\theta\sigma\tau\\\frac{1}{2}\theta\sigma\tau&\frac{1}{2}S^2\theta^2+1\end{pmatrix}\end{aligned}$$

so that the correlation between characteristics for children is:

$$Corr\left(x_1',x_2'\right)=\frac{\frac{1}{2}\theta\sigma\tau}{\sqrt{\frac{1}{2}\tau^2s^2+1}\sqrt{\frac{1}{2}\theta^2S^2+1}}$$

Note that $\sigma=0$ gives a zero correlation for children as well. Also, because $\theta<1$ and $\tau<1$, the correlation is less than the parents' correlation of σ/sS.

□

Proof of Claim 4. Again applying (3), under SGAM, the correlation between children's traits is:

$$Corr\left(x_1',x_2'\right)=\frac{\frac{1}{2}\theta\tau\left(\sigma+AC\right)}{\sqrt{\frac{1}{2}\tau^2\left(A^2+s^2\right)+1}\sqrt{\frac{1}{2}\theta^2\left(C^2+S^2\right)+1}}$$

This is positive if $\sigma=0$ so long as $AC>0$ i.e. $0<a<1$. To show it is increasing in θ, strip out constant terms and take the derivative of

$$\frac{\theta}{\sqrt{\frac{1}{2}\theta^2(C^2+S^2)+1}}$$

The derivative is signed by

$$\begin{aligned}
&\left(\frac{1}{2}\theta^2(C^2+S^2)+1\right)^{0.5} - \frac{1}{2}\theta^2(C^2+S^2)\left(\frac{1}{2}\theta^2(C^2+S^2)+1\right)^{-0.5} \\
&> \left(\frac{1}{2}\theta^2(C^2+S^2)+1\right)^{0.5} - \left(\frac{1}{2}\theta^2(C^2+S^2)+1\right)\left(\frac{1}{2}\theta^2(C^2+S^2)+1\right)^{-0.5} \\
&= 0.
\end{aligned}$$

□

Proof of Proposition 2. The fixed point condition on the covariance matrix is

$$\begin{pmatrix} s^2 & \sigma \\ \sigma & S^2 \end{pmatrix} = \begin{pmatrix} \frac{1}{2}s^2\tau^2+1 & \frac{1}{2}\theta\sigma\tau \\ \frac{1}{2}\theta\sigma\tau & \frac{1}{2}S^2\theta^2+1 \end{pmatrix}$$

which gives

$$s^2 = \frac{2}{2-\tau^2}, S^2 = \frac{2}{2-\theta^2}, \sigma = 0.$$

The asymptotic conditional expectation of children's genetics given parental SES is:

$$\mathbb{E}\left[\frac{\tau}{2}(x_1+y_1)+\varepsilon \mid x_2 = v, y_2 = w\right] = 0$$

since the traits x_1, x_2, y_1, y_2 are uncorrelated.

□

Proof of Proposition 3. Start by characterizing the invariant distribution. This must satisfy:

$$\begin{pmatrix} s^2 & \sigma \\ \sigma & S^2 \end{pmatrix} = \begin{pmatrix} \frac{1}{2}\tau^2\left(A^2+s^2\right)+1 & \frac{1}{2}\theta\tau\left(\sigma+AC\right) \\ \frac{1}{2}\theta\tau\left(\sigma+AC\right) & \frac{1}{2}\theta^2\left(C^2+S^2\right)+1 \end{pmatrix}$$

where

$$\begin{aligned}
A &= \frac{as^2+(1-a)\sigma}{\sqrt{a^2s^2+(1-a)^2S^2+2a(1-a)\bar{\sigma}}} \text{ and} \\
C &= \frac{a\sigma+(1-a)S^2}{\sqrt{a^2s^2+(1-a)^2S^2+2a(1-a)\bar{\sigma}}},
\end{aligned}$$

Note that if the distribution converges, s^2 and S^2 must be above 1. Also, for A and C to have a real-valued solution, it must be that $a^2s^2 + (1-a)^2S^2 + 2a(1-a)\sigma > 0$; using this,

$$AC = \frac{a^2s^2\sigma + (1-a)^2S^2\sigma + a(1-a)(\sigma^2 + S^2s^2)}{a^2s^2 + (1-a)^2S^2 + 2a(1-a)\sigma} > \sigma, \text{by } \sigma^2 < S^2s^2.$$

Since $AC > \sigma$, $\frac{\theta\tau}{2}(\sigma + AC) > \theta\tau\sigma$. If $\sigma < 0$ then $\sigma = \frac{\theta\tau}{2}(\sigma + AC) > \theta\tau\sigma > \sigma$, a contradiction. Thus $\sigma \geq 0$. Also if $\sigma = 0$ then

$$\sigma = \frac{\theta\tau}{2}\left(\frac{a(1-a)(S^2s^2)}{a^2s^2 + (1-a)^2S^2}\right)$$

which implies $a = 0$ or $a = 1$. This proves that σ is non-negative, and positive if $a \in (0,1)$, so long as the distribution converges.

From the invariant distribution, first:

$$\sigma\left(1 - \frac{1}{2}\theta\tau\right) = \frac{\theta\tau}{2}\frac{\left(as^2 + (1-a)\,\sigma\right)\left(a\sigma + (1-a)\,S^2\right)}{a^2s^2 + (1-a)^2\,S^2 + 2a\,(1-a)\,\sigma}$$

or

$$\mu\left(1 - \frac{1}{2}\theta\tau\right) = \frac{\theta\tau}{2}\frac{\left(a\lambda + (1-a)\,\mu\right)\left(a\mu + (1-a)\right)}{a^2\lambda + (1-a)^2 + 2a\,(1-a)\,\mu}$$

where

$$\lambda = s^2/S^2 \text{ and } \mu = \sigma/S^2.$$

Solving for λ gives

$$\lambda = \frac{1-a}{a}\mu\frac{(-2a + 4a\mu - 2\theta\tau + 2a\theta\tau - 3a\theta\tau\mu + 2)}{(1-a)\,\theta\tau - 2a\mu\,(1-\theta\tau)} \tag{13}$$

Then

$$s^2\left(1 - \frac{1}{2}\tau^2\right) = \frac{1}{2}A^2\tau^2 + 1$$
$$S^2\left(1 - \frac{1}{2}\theta^2\right) = \frac{1}{2}C^2\theta^2 + 1$$

give

$$s^2\left(1 - \frac{1}{2}\tau^2\right) - \frac{1}{2}A^2\tau^2 = S^2\left(1 - \frac{1}{2}\theta^2\right) - \frac{1}{2}C^2\theta^2 \text{ therefore}$$
$$\lambda\left(1 - \frac{1}{2}\tau^2\right) - \frac{1}{2}\frac{A^2}{S^2}\tau^2 = \left(1 - \frac{1}{2}\theta^2\right) - \frac{1}{2}\frac{C^2}{S^2}\theta^2$$

Here

$$\frac{A^2}{S^2} = \frac{\left(a\lambda + (1-a)\,\mu\right)^2}{D} \text{ and}$$
$$\frac{C^2}{S^2} = \frac{\left(a\mu + (1-a)\right)^2}{D},$$
$$D = a^2\lambda + (1-a)^2 + 2a\,(1-a)\,\mu$$

which give a quadratic equation in μ:

$$\lambda\left(1 - \frac{1}{2}\tau^2\right)D - \frac{1}{2}\left(a\lambda + (1-a)\,\mu\right)^2\tau^2 - \left(1 - \frac{1}{2}\theta^2\right)D + \frac{1}{2}\left(a\mu + (1-a)\right)^2\theta^2 = 0$$

Plugging in λ given by (13), this can be rewritten to

$$F(\mu) = \frac{(1 - a + a\mu)^2}{a}\frac{N(\mu)}{D(\mu)} = 0$$

where

$$N(\mu) = X\mu^2 + Y\mu + Z, \text{ with } X, Y, Z \text{ polynomials in } a, \theta, \tau$$

and

$$D(\mu) = \left(\theta\tau\,(1-a) - 2a\mu\,(1-\theta\tau)\right)^2$$

One can check that the discriminant is always positive. Therefore this has two solutions (not shown), of which only one is acceptable (it goes to the exact solution when the coefficient of μ^2 goes to 0). Writing

$$\mu = \phi_1(a, \theta, \tau)$$

for this solution:

$$\lambda = \psi(a, \theta, \tau) = \frac{1-a}{a}\phi_1(a, \theta, \tau)\,\frac{(-2a + 4a\phi_1(a, \theta, \tau) - 2\theta\tau + 2a\theta\tau - 3a\theta\tau\phi_1(a, \theta, \tau) + 2)}{(1-a)\,\theta\tau - 2a\phi_1(a, \theta, \tau)\,(1-\theta\tau)}.$$

Finally

$$S^2 = \frac{1}{1 - \frac{1}{2}\theta^2 - \frac{1}{2}\frac{C^2}{S^2}\theta^2} \text{ where}$$
$$\frac{C^2}{S^2} = \frac{\left(a\phi_1(a, \theta, \tau) + (1-a)\right)^2}{a^2\psi(a, \theta, \tau) + (1-a)^2 + 2a\,(1-a)\,\phi_1(a, \theta, \tau)}$$

and

$$s^2 = \lambda S^2 = \frac{\psi(a,\theta,\tau)}{1 - \frac{1}{2}\theta^2 - \frac{1}{2}\frac{C^2}{S^2}\theta^2};$$
$$\sigma = \mu S^2 = \frac{\phi_1(a,\theta,\tau)}{1 - \frac{1}{2}\theta^2 - \frac{1}{2}\frac{C^2}{S^2}\theta^2}.$$

Conditional expectations of children's genetics given parents' wealth under SGAM are calculated using the same formula as before, plugging in moments of the asymptotic distribution:

$$\mathbb{E}\left[\frac{\tau}{2}(x_1 + y_1) + \varepsilon \mid x_2 = v, y_2 = w\right] = \frac{1}{2}\tau\frac{\sigma + AC}{C^2 + S^2}(v + w)$$

□

Proof of Claim 5. Since men and women have different distributions of attractiveness, we have to match them by quantiles of their respective distributions. Men's and women's attractiveness are distributed

$$N(0, \sigma_I^2) \text{ where } \sigma_I = \sqrt{a^2 s^2 + (1-a)^2 S^2 + 2a(1-a)\sigma};$$
$$N(0, \sigma_J^2) \text{ where } \sigma_J = \sqrt{b^2 s^2 + (1-b)^2 S^2 + 2b(1-b)\sigma}.$$

Thus, men with normalized attractiveness $i(x)/\sigma_I$ match women with normalized attractiveness $j(y)/\sigma_J$.

Change variables so that

$$\begin{pmatrix} x_1 \\ x_2 \end{pmatrix} \to \begin{pmatrix} x_1 \\ u \end{pmatrix} \text{ where } u = \frac{ax_1 + (1-a)x_2}{\sigma_I};$$
$$\begin{pmatrix} y_1 \\ y_2 \end{pmatrix} \to \begin{pmatrix} y_1 \\ v \end{pmatrix} \text{ where } v = \frac{by_1 + (1-b)y_2}{\sigma_J}.$$

Thus

$$\begin{pmatrix} x_1 \\ u \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ a/\sigma_I & (1-a)/\sigma_I \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \end{pmatrix};$$
$$\begin{pmatrix} y_1 \\ v \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ b/\sigma_J & (1-b)/\sigma_J \end{pmatrix} \begin{pmatrix} y_1 \\ y_2 \end{pmatrix}.$$

and their respective covariance matrices are

$$\mathbb{C}\begin{pmatrix} x_1 \\ u \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ a/\sigma_I & (1-a)/\sigma_I \end{pmatrix}\begin{pmatrix} s^2 & \sigma \\ \sigma & S^2 \end{pmatrix}\begin{pmatrix} 1 & a/\sigma_I \\ 0 & (1-a)/\sigma_I \end{pmatrix}$$
$$= \begin{pmatrix} s^2 & A \\ A & 1 \end{pmatrix}, \text{ where } A = \frac{as^2 + (1-a)\sigma}{\sigma_I};$$

similarly

$$\mathbb{C}\begin{pmatrix} y_1 \\ v \end{pmatrix} = \begin{pmatrix} s^2 & B \\ B & 1 \end{pmatrix}, \text{ where } B = \frac{bs^2 + (1-b)\sigma}{\sigma_J}.$$

Under SGAM, couples have characteristics $\begin{pmatrix} x_1 \\ t \\ y_1 \\ t \end{pmatrix}$, where $\begin{pmatrix} x_1 \\ y_1 \\ t \end{pmatrix}$ is trivariate normal with mean 0 and covariance matrix

$$\Sigma = \begin{pmatrix} s^2 & AB & A \\ AB & s^2 & B \\ A & B & 1 \end{pmatrix}$$

Lastly, we calculate the covariance matrix of couples' original characteristics. Since

$$\begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ \frac{-a}{1-a} & 0 & \frac{\sigma_I}{1-a} \\ 0 & 1 & 0 \\ 0 & \frac{-b}{1-b} & \frac{\sigma_J}{1-b} \end{pmatrix}\begin{pmatrix} x_1 \\ y_1 \\ t \end{pmatrix}$$

we have that the mean is again 0 and the covariance matrix is

$$\mathbb{C}\begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix} = \begin{pmatrix} 1 & 0 & 0 \\ \frac{-a}{1-a} & 0 & \frac{\sigma_I}{1-a} \\ 0 & 1 & 0 \\ 0 & \frac{-b}{1-b} & \frac{\sigma_J}{1-b} \end{pmatrix} \Sigma \begin{pmatrix} 1 & \frac{-a}{1-a} & 0 & 0 \\ 0 & 0 & 1 & \frac{-b}{1-b} \\ 0 & \frac{\sigma_I}{1-a} & 0 & \frac{\sigma_J}{1-b} \end{pmatrix}$$
$$= \begin{pmatrix} s^2 & \sigma & AB & AD \\ \sigma & S^2 & BC & CD \\ AB & BC & s^2 & \sigma \\ AD & CD & \sigma & S^2 \end{pmatrix}$$

where

$$C = \frac{a\sigma + (1-a)S^2}{\sigma_I}; D = \frac{b\sigma + (1-b)S^2}{\sigma_J}.$$

From the above and (2) we can calculate the covariance matrix of children's characteristics as

$$\mathbb{C}\begin{pmatrix} x_1' \\ x_2' \end{pmatrix} = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix} + \begin{pmatrix} \frac{\tau}{2} & 0 & \frac{\tau}{2} & 0 \\ 0 & \frac{\theta}{2} & 0 & \frac{\theta}{2} \end{pmatrix} \begin{pmatrix} s^2 & \sigma & AB & AD \\ \sigma & S^2 & BC & CD \\ AB & BC & s^2 & \sigma \\ AD & CD & \sigma & S^2 \end{pmatrix} \begin{pmatrix} \frac{\tau}{2} & 0 \\ 0 & \frac{\theta}{2} \\ \frac{\tau}{2} & 0 \\ 0 & \frac{\theta}{2} \end{pmatrix}$$
$$= \begin{pmatrix} \frac{\tau^2}{2}(s^2 + AB) + 1 & \frac{\tau\theta}{4}(2\sigma + AD + BC) \\ \frac{\tau\theta}{4}(2\sigma + AD + BC) & \frac{\theta^2}{2}(S^2 + CD) + 1 \end{pmatrix}.$$

Thus x_1' and x_2' will be positively correlated if $2\sigma + AD + BC > 0$. This is always positive if $\sigma > 0$; if $\sigma = 0$ it reduces to

$$\frac{(a + b - 2ab)s^2S^2}{\sigma_I\sigma_J}$$

which is positive unless $a = b = 0$ or $a = b = 1$. The correlation is

$$\frac{\frac{\tau\theta}{4}(2\sigma + AD + BC)}{\sqrt{\frac{\tau^2}{2}(s^2 + AB) + 1}\sqrt{\frac{\theta^2}{2}(S^2 + CD) + 1}}$$

and taking the derivative shows it is increasing in θ, as in the proof for Claim 4. □

Proof of Proposition 4. Write

$$\begin{aligned} x_1' &= \tau\frac{x_1+y_1}{2} + \varepsilon \\ x_2' &= \gamma x_1' + \theta\frac{x_2+y_2}{2} + \eta \\ &= \gamma\tau\frac{x_1+y_1}{2} + \theta\frac{x_2+y_2}{2} + \eta + \gamma\varepsilon \end{aligned}$$

Since

$$\begin{pmatrix} \tau\frac{x_1+y_1}{2} \\ \gamma\tau\frac{x_1+y_1}{2} + \theta\frac{x_2+y_2}{2} \end{pmatrix} = \begin{pmatrix} \frac{\tau}{2} & 0 & \frac{\tau}{2} & 0 \\ \frac{\gamma\tau}{2} & \frac{\theta}{2} & \frac{\gamma\tau}{2} & \frac{\theta}{2} \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix}$$

we can use (1) to derive the covariance matrix for children:

$$\begin{aligned} \mathbb{C} &= \begin{pmatrix} 1 & \gamma \\ \gamma & 1+\gamma^2 \end{pmatrix} + \begin{pmatrix} \frac{\tau}{2} & 0 & \frac{\tau}{2} & 0 \\ \frac{\gamma\tau}{2} & \frac{\theta}{2} & \frac{\gamma\tau}{2} & \frac{\theta}{2} \end{pmatrix} \begin{pmatrix} s^2 & \sigma & A^2 & AC \\ \sigma & S^2 & AC & C^2 \\ A^2 & AC & s^2 & \sigma \\ AC & C^2 & \sigma & S^2 \end{pmatrix} \begin{pmatrix} \frac{\tau}{2} & \frac{\gamma\tau}{2} \\ 0 & \frac{\theta}{2} \\ \frac{\tau}{2} & \frac{\gamma\tau}{2} \\ 0 & \frac{\theta}{2} \end{pmatrix} \\ &= \begin{pmatrix} \frac{\tau^2}{2}(s^2+A^2)+1 & \frac{\gamma\tau^2}{2}(s^2+A^2)+\frac{\tau\theta}{2}(\sigma+AC)+\gamma \\ \frac{\gamma\tau^2}{2}(s^2+A^2)+\frac{\tau\theta}{2}(\sigma+AC)+\gamma & \frac{\gamma^2\tau^2}{2}(s^2+A^2)+\gamma\tau\theta(\sigma+AC)+\frac{\theta^2}{2}(S^2+C^2)+1+\gamma^2 \end{pmatrix} \end{aligned}$$

The first claim in the proof follows from the covariance:

$$\frac{\gamma\tau^2}{2}(s^2+A^2)+\frac{\tau\theta}{2}(\sigma+AC)+\gamma$$

This is increasing in γ, and positive if any of $\sigma > 0, \gamma > 0$, or $AC > 0$ (which holds if $0 < a < 1$ when $\sigma = 0$).

The correlation $Cov(x_1', x_2')/\sqrt{Var(x_1')Var(x_2')}$ is proportional to

$$\frac{\gamma p + q}{\sqrt{\gamma^2 p + \gamma 2q + r}}$$

where

$$p = \tau^2(s^2 + A^2) + 2;$$
$$q = \tau\theta(\sigma + AC);$$
$$r = \theta^2(S^2 + C^2) + 2.$$

The derivative of this with respect to γ is signed by $pr - q^2$, which equals

$$\begin{aligned}
&[\tau^2(s^2 + A^2) + 2][\theta^2(S^2 + C^2) + 2] - [\tau\theta(\sigma + AC)]^2 \\
=&\tau^2\theta^2(s^2S^2 + A^2S^2 + s^2C^2 - 2\sigma AC - \sigma^2) + 2[\theta^2(S^2 + C^2) + \tau^2(s^2 + A^2)] + 4
\end{aligned}$$

The last two terms are positive. In the first term, $\sigma^2 < s^2S^2$, and

$$\begin{aligned}
0 &< (AS - Cs)^2 \\
&= A^2S^2 + C^2s^2 - 2ACSs \\
&< A^2S^2 + C^2s^2 - 2\sigma AC \text{ , again using } \sigma < sS.
\end{aligned}$$

Hence the whole sum is positive.

Now we can calculate

$$\mathbb{E}[x_2'|x_2 + y_2]$$

using

$$\begin{pmatrix} \gamma\tau\frac{x_1+y_1}{2} + \theta\frac{x_2+y_2}{2} \\ x_2 \\ y_2 \end{pmatrix} = \begin{pmatrix} \frac{\gamma\tau}{2}0 & \frac{\theta}{2}1 & \frac{\gamma\tau}{2}0 & \frac{\theta}{2}0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix} \begin{pmatrix} x_1 \\ x_2 \\ y_1 \\ y_2 \end{pmatrix}$$

to give

$$\mathbb{C}\begin{pmatrix} x_2' \\ x_2 \\ y_2 \end{pmatrix} = \begin{pmatrix} \frac{\gamma\tau}{2} & \frac{\theta}{2} & \frac{\gamma\tau}{2} & \frac{\theta}{2} \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix} \begin{pmatrix} s^2 & \sigma & A^2 & AC \\ \sigma & S^2 & AC & C^2 \\ A^2 & AC & s^2 & \sigma \\ AC & C^2 & \sigma & S^2 \end{pmatrix} \begin{pmatrix} \frac{\gamma\tau}{2} & 0 & 0 \\ \frac{\theta}{2} & 1 & 0 \\ \frac{\gamma\tau}{2} & 0 & 0 \\ \frac{\theta}{2} & 0 & 1 \end{pmatrix} + \begin{pmatrix} 1+\gamma^2 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$= \begin{pmatrix} \ldots & \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) & \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) \\ \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) & S^2 & C^2 \\ \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) & C^2 & S^2 \end{pmatrix}.$$

Next

$$\mathbb{E}[x_2'|x_2, y_2] = \begin{pmatrix} \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) & \frac{\gamma\tau}{2}(\sigma + AC) + \frac{\theta}{2}(S^2 + C^2) \end{pmatrix} \begin{pmatrix} S^2 & C^2 \\ C^2 & S^2 \end{pmatrix}^{-1} \begin{pmatrix} x_2 \\ y_2 \end{pmatrix}$$

$$= \left(\gamma\tau \frac{\sigma + AC}{S^2 + C^2} + \theta\right) \frac{x_2 + y_2}{2}$$

So long as $0 < a < 1$ or $\sigma > 0$, the coefficient on parents' wealth is thus higher than θ.

□

Proof of Proposition 5. Without loss of generality let $Ex_1 = Ex_2 = 0$. The correlation is signed by the covariance. Write K for the set of couples in the parents' generation with typical member $k = (x, y)$. Without loss of generality let $x_1 \geq y_1$. Then, since the iso-attractiveness curves defined by f are downward-sloping, $x_2 \leq y_2$. (If $a = 1$ then $x_1 = y_1$; pick x so that $x_2 \leq y_2$.) Also, for $a \in (0, 1)$, if $x_1 > y_1$ then $x_2 < y_2$.

Since $Ex_1 = Ex_2 = 0$, the covariance among the parents' generation is

$$\int_K (x_1 x_2 + y_1 y_2)/2 \, dk$$

Write

$$x_1' = \tau x_1^* + \varepsilon \qquad \text{where } x_1^* = (x_1 + y_1)/2$$

$$x_2' = \theta x_2^* + \eta \qquad \text{where } x_2^* = (x_2 + y_2)/2$$

and write the children's covariance as

$$Cov(x_1', x_2') = Cov(\tau x_1^*, \theta x_2^*) + Cov(\tau x_1^*, \eta) + Cov(\varepsilon, \theta x_2^*) + Cov(\varepsilon, \eta).$$

By independence of the shocks, the last 3 terms are zero. So we need to show that

$$Cov(\tau x_1^*, \theta x_2^*) = \tau\theta Cov(x_1^*, x_2^*) > 0$$

Write

$$Cov(x_1^*, x_2^*) = \int_K x_1^* x_2^* \, dk$$

using that $Ex_1^* = Ex_2^* = 0$.

Take a typical parent, and write

$$x_1 x_2 = (x_1^* - \Delta_1)(x_2^* - \Delta_2)$$
$$y_1 y_2 = (x_1^* + \Delta_1)(x_2^* + \Delta_2)$$

where

$$\Delta_1 = (x_1 - y_1)/2; \ \Delta_2 = (x_2 - y_2)/2.$$

By assumption $\Delta_1 \geq 0$ and $\Delta_2 \leq 0$. Furthermore, if $a \in (0, 1)$, then for a set of positive measure, $\Delta_1 > 0$ and $\Delta_2 < 0$, by our assumption that not all matching couples are identical.

Taking the average of the parents gives

$$(x_1 x_2 + y_1 y_2)/2 = x_1^* x_2^* + \Delta_1 \Delta_2$$

and if $a \in (0, 1)$, this is strictly less than $x_1^* x_2^*$ for a set of positive measure. Plugging this into the integral gives

$$Cov(x_1, x_2) \leq Cov(x_1^*, x_2^*) = \int_K x_1^* x_2^* \, dk$$

with strict inequality if $a \in (0, 1)$. Since the parental covariance was 0 by assumption, this completes the proof.

□

Robustness checks

Table 6 reruns our central regressions, dropping the control for parents' age at birth. Results show the same pattern as in the main text: the coefficient for birth order is negative, but changes sign when university attendance is added as a potential mediator. However, the birth order effect is smaller overall, and is never significant. We also ran regressions using father's age only; results are similar to those in the main text.

Table 7 reruns our central regressions but includes a separate coefficient for each position in the birth order (with firstborn as the baseline). The basic pattern of our main result holds: birth order coefficients are generally negative; adding mediators causes them to increase towards zero or to change sign. Birth order effects appear largest for birth order 2-3. However, effects for later birth orders are also imprecisely estimated (since fewer respondents come from large families).

Notably when we add income, dummies for birth order 5 and 6 become large and positive. This could be (for instance) because being the last born has advantages after effects on SES have been netted out. Table 8 runs the same exercise for different subsets: male respondents, female respondents, and couples with children. The basic pattern that birth order coefficients shrink after adding mediators is quite robust. Note however that here, the estimates of effects for birth order 2-3 are larger for females.

We also ran a specification with separate birth order dummies within each family size. Figure 5 shows 95% confidence intervals for the birth order coefficients, from the column 2 specification including height and IQ controls but no mediators. Not surprisingly, coefficients are imprecisely estimated. But most birth order coefficients are negative compared to the baseline for firstborns.

Table 9 reruns our regressions controlling for several polygenic scores. Results are very close to those in the main text.

Table 10 reruns relevant columns of Table 3 using age of leaving full-time education as a measure of educational SES, instead of the university attendance dummy. Results are similar to those in the main text: controlling for age of leaving full-time education shrinks the effect of birth order and makes it insignificant.

Table 11 reruns Table 3 excluding families of size 3. Results are very similar to those in the main text.

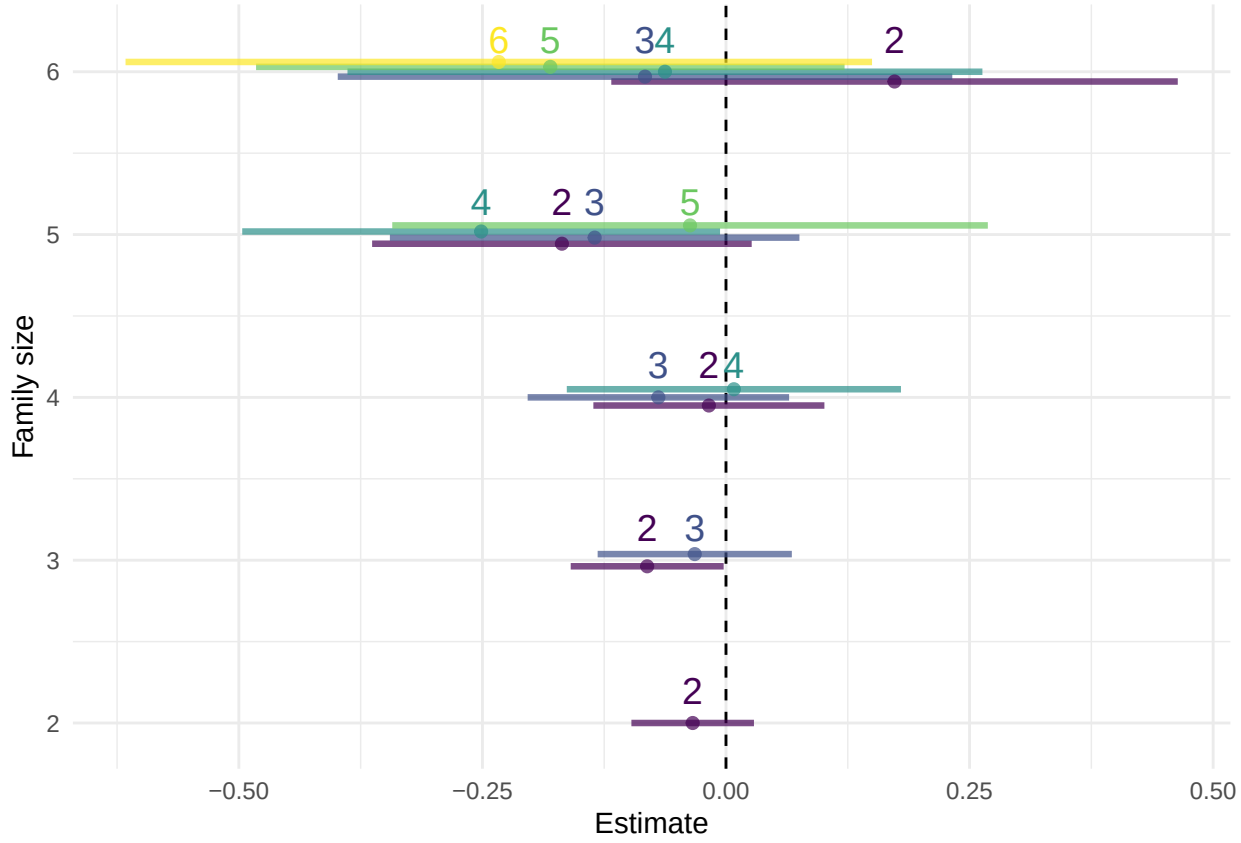

Figure 5: Regressions of spouse PSEA: birth order dummies within different family sizes. Labels show birth order. Lines are 95 per cent confidence intervals. The omitted category is birth order 1.

Table 6: Regressions of spouse PSEA, without controls for parents' age at respondent's birth

	(1)	(2)	(3)	(4)
Birth order	−0.0075	0.0027	−0.0024	0.0019
	(0.0074)	(0.0073)	(0.0137)	(0.0137)
University		0.2386 ***		0.1965 ***
		(0.0149)		(0.0250)
Income			0.0034 ***	0.0023 **
			(0.0008)	(0.0008)
Fluid IQ		0.0156 ***	0.0167 **	0.0053
		(0.0034)	(0.0061)	(0.0062)
Height		0.0019 **	0.0036 **	0.0031 *
		(0.0007)	(0.0013)	(0.0013)
BMI		−0.0115 ***	−0.0150 ***	−0.0139 ***
		(0.0015)	(0.0027)	(0.0027)
Self-reported health		0.0184 +	0.0138	0.0051
		(0.0097)	(0.0179)	(0.0178)
Own PSEA	0.0650 ***	0.0316 ***	0.0393 **	0.0273 *
	(0.0065)	(0.0066)	(0.0120)	(0.0120)
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
N	23797	23797	7658	7658
R2	0.010	0.031	0.021	0.029
logLik	−33426.686	−33179.643	−10735.387	−10703.995
AIC	66953.372	66469.287	21578.773	21517.989

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Table 7: Regressions of spouse PSEA, separate birth order dummies

	(1)	(2)	(3)	(4)
Birth order 2	−0.0500 *	−0.0204	−0.0464	−0.0443
	(0.0232)	(0.0231)	(0.0411)	(0.0410)
Birth order 3	−0.0557	0.0013	−0.0174	0.0020
	(0.0376)	(0.0374)	(0.0673)	(0.0673)
Birth order 4	−0.0736	0.0099	−0.0162	−0.0004
	(0.0655)	(0.0651)	(0.1270)	(0.1268)
Birth order 5	−0.0801	0.0022	0.1038	0.1207
	(0.1190)	(0.1181)	(0.2294)	(0.2289)
Birth order 6	−0.2746	−0.1997	0.1632	0.2067
	(0.2371)	(0.2349)	(0.5966)	(0.5953)
University		0.2182 ***		0.1553 ***
		(0.0221)		(0.0375)
Income			0.0037 ***	0.0030 **
			(0.0010)	(0.0010)
Own PSEA	0.0574 ***	0.0263 **	0.0219	0.0117
	(0.0099)	(0.0101)	(0.0180)	(0.0181)
Parents' age at birth	0.0116 ***	0.0052 *	0.0092 +	0.0079 +
	(0.0026)	(0.0026)	(0.0047)	(0.0047)
Wald p-value, birth order	0.2452	0.7917	0.8285	0.7867
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
Other mediators (IQ, height, BMI, s.-r. health)	No	Yes	Yes	Yes
N	10206	10206	3407	3407
R2	0.013	0.032	0.030	0.035
logLik	−14296.681	−14196.868	−4810.108	−4801.412
AIC	28703.362	28513.735	9740.215	9724.824

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.
Grey background: coefficients are higher than column 1.

Table 8: Regressions of spouse PSEA, separate birth order dummies: subsets

	Males	Males	Females	Females	With children	With children
Birth order 2	−0.0260	0.0048	−0.0701 *	−0.0452	−0.0520 *	−0.0220
	(0.0357)	(0.0353)	(0.0309)	(0.0310)	(0.0246)	(0.0245)
Birth order 3	−0.0231	0.0384	−0.0818	−0.0336	−0.0625	−0.0033
	(0.0579)	(0.0570)	(0.0508)	(0.0507)	(0.0406)	(0.0403)
Birth order 4	−0.1011	−0.0030	−0.0481	0.0197	−0.0805	0.0056
	(0.1001)	(0.0996)	(0.0855)	(0.0851)	(0.0688)	(0.0685)
Birth order 5	−0.2165	−0.1531	0.0243	0.1060	−0.1358	−0.0526
	(0.1458)	(0.1469)	(0.1472)	(0.1474)	(0.1071)	(0.1076)
Birth order 6	−0.6127 *	−0.4881 *	−0.0480	−0.0175	−0.1888	−0.0954
	(0.2582)	(0.2266)	(0.2250)	(0.2328)	(0.1892)	(0.1843)
University		0.2730 ***		0.1684 ***		0.2169 ***
		(0.0332)		(0.0307)		(0.0239)
Own PSEA	0.0591 ***	0.0219	0.0569 ***	0.0307 *	0.0622 ***	0.0291 **
	(0.0148)	(0.0150)	(0.0135)	(0.0137)	(0.0106)	(0.0107)
Parents' age at birth	0.0125 **	0.0050	0.0107 **	0.0055	0.0125 ***	0.0061 *
	(0.0040)	(0.0040)	(0.0034)	(0.0035)	(0.0028)	(0.0028)
Wald p-value, birth order	0.2187	0.2549	0.2844	0.6257	0.3039	0.9304
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
Other mediators (IQ, height, BMI, s.-r. health)	No	Yes	No	Yes	No	Yes
N	4675	4675	5531	5531	9127	9127
R2	0.018	0.044	0.018	0.032	0.015	0.035
logLik	−6587.036	−6525.476	−7680.051	−7641.553	−12771.914	−12678.456
AIC	13282.073	13168.953	15466.102	15399.106	25651.828	25474.913

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.
Grey background: birth dummy coefficient is higher than corresponding coefficient without mediators.

Table 9: Regressions of spouse PSEA with controls for polygenic scores

	(1)	(2)	(3)	(4)
Birth order	−0.0313	−0.0047	−0.0113	−0.0049
	(0.0183)	(0.0177)	(0.0311)	(0.0308)
University		0.2178 ***		0.1534 ***
		(0.0245)		(0.0232)
Income			0.0037 ***	0.0030 ***
			(0.0007)	(0.0007)
Fluid IQ		0.0168 *	0.0198	0.0110
		(0.0066)	(0.0116)	(0.0120)
Height		0.0029 *	0.0046 *	0.0042 *
		(0.0011)	(0.0018)	(0.0018)
BMI		−0.0109 ***	−0.0112 **	−0.0107 *
		(0.0023)	(0.0039)	(0.0039)
Self-reported health		0.0173	0.0138	0.0071
		(0.0199)	(0.0341)	(0.0339)
Own PSEA	0.0519 ***	0.0231 +	0.0178	0.0084
	(0.0111)	(0.0115)	(0.0245)	(0.0243)
Parents' age at birth	0.0114 ***	0.0052 +	0.0093 *	0.0080 +
	(0.0028)	(0.0028)	(0.0040)	(0.0041)
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
Polygenic score controls	Yes	Yes	Yes	Yes
N	10206	10206	3407	3407
R2	0.013	0.032	0.030	0.035

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.
Polygenic scores: alzheimer's, cognitive ability, neuroticism, substance use.

Table 10: Regressions of spouse PSEA using age of leaving full-time education

	(1)	(2)	(3)
Birth order	−0.0314 *	0.0022	0.0036
	(0.0146)	(0.0147)	(0.0270)
Age left full-time educ.		0.0475 ***	0.0403 ***
		(0.0044)	(0.0078)
Income			0.0029 *
			(0.0011)
Fluid IQ		0.0144 **	0.0077
		(0.0053)	(0.0098)
Height		0.0029 **	0.0042 *
		(0.0011)	(0.0019)
BMI		−0.0105 ***	−0.0106 **
		(0.0022)	(0.0040)
Self-reported health		0.0148	0.0088
		(0.0151)	(0.0270)
Own PSEA	0.0573 ***	0.0252 *	0.0127
	(0.0100)	(0.0101)	(0.0185)
Parents' age at birth	0.0116 ***	0.0041	0.0064
	(0.0026)	(0.0026)	(0.0047)
Family size dummies	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes
N	10206	10156	3400
R2	0.013	0.035	0.037
logLik	−14297.465	−14116.670	−4788.765
AIC	28694.930	28343.341	9689.529

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Table 11: Regressions of spouse PSEA, excluding family size 3

	(1)	(2)	(3)	(4)
Birth order	−0.0360 *	−0.0126	−0.0228	−0.0204
	(0.0170)	(0.0170)	(0.0330)	(0.0329)
University		0.2056 ***		0.1622 ***
		(0.0271)		(0.0463)
Income			0.0018	0.0012
			(0.0015)	(0.0015)
Fluid IQ		0.0198 **	0.0114	0.0030
		(0.0062)	(0.0113)	(0.0117)
Height		0.0025 *	0.0044 +	0.0037
		(0.0013)	(0.0024)	(0.0023)
BMI		−0.0124 ***	−0.0130 **	−0.0126 *
		(0.0026)	(0.0049)	(0.0049)
Self-reported health		0.0121	0.0071	−0.0015
		(0.0182)	(0.0335)	(0.0335)
Own PSEA	0.0524 ***	0.0210 +	0.0126	−0.0001
	(0.0121)	(0.0122)	(0.0230)	(0.0232)
Parents' age at birth	0.0125 ***	0.0065 *	0.0076	0.0064
	(0.0032)	(0.0032)	(0.0056)	(0.0056)
Family size dummies	Yes	Yes	Yes	Yes
Birth month dummies	Yes	Yes	Yes	Yes
Birth year dummies	Yes	Yes	Yes	Yes
N	6959	6959	2286	2286
R2	0.016	0.034	0.033	0.039
logLik	−9723.678	−9656.955	−3227.692	−3221.305
AIC	19545.356	19421.909	6561.383	6550.611

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Regressions with "fake pairs"

Our dataset of pairs could still contain pairs who live in the same postcode but are not spouses. These pairs might still show a relationship between one partner's phenotype and the other's genotype. For example, maybe early-born children grow up to live in richer postcodes, along with people who have higher PSEA scores (Abdellaoui et al. 2019). This could then bias the results. If the coefficient for "fake pairs" is absolutely larger (smaller) than for real pairs, then our results will be biased away from zero (towards zero).

To sign the bias, we create a dataset of "known fake pairs". These are opposite-sexed pairs who live in the same postcode, but do not share all the characteristics listed for the real pairs. Specifically, from the list of characteristics used to create our real pairs (same homeownership status, same length of time at address, same number of children, attended same assessment center, attended on same day, husband reported living with spouse, wife reported living with spouse) the fake pairs ticked exactly 5 out of 7 boxes.

We again use genetic children to confirm that the fake pairs are "real fakes". Out of 817 genetic children of the fake pairs, only 33 were children of both parents. Thus, the vast majority of fake pairs do not appear to be spouses. Table 12 reruns the regressions of Table 2 using the fake pairs. Although the coefficients on birth order are always negative, and significant when controlling for parent's age, they are always absolutely smaller than the corresponding coefficient in the main text. This suggests that any fake pairs remaining in our data will have the effect of biasing our results towards zero.

Table 12: Regressions of PSEA on birth order: fake pairs

	(1)	(2)	(3)
Birth order	−0.0074	−0.0061	−0.0273 +
	(0.0080)	(0.0080)	(0.0144)
Own PSEA		0.0510 ***	0.0514 ***
		(0.0068)	(0.0099)
Parents' age at birth			0.0096 ***
			(0.0025)
Family size dummies	Yes	Yes	Yes
Birth month dummies	No	Yes	Yes
Birth year dummies	No	Yes	Yes
N	21550	21508	10400
R2	0.001	0.007	0.011

*** $p < 0.001$; ** $p < 0.01$; * $p < 0.05$; + $p < 0.1$. Standard errors: robust.

Quotations on natural inequality

…your face and figure have nothing of the slave about them, and proclaim you of noble birth.

– *Odyssey*, Odysseus to Laertes

Citizens, we shall say to them in our tale, you are brothers, yet God has framed you differently. Some of you have the power of command, and in the composition of these he has mingled gold, wherefore also they have the greatest honour; others he has made of silver, to be auxiliaries; others again who are to be husbandmen and craftsmen he has composed of brass and iron; and the species will generally be preserved in the children. But as all are of the same original stock, a golden parent will sometimes have a silver son, or a silver parent a golden son.

– Plato *Republic*

Nature would like to distinguish between the bodies of freemen and slaves, making the one strong for servile labor, the other upright, and although useless for such services, useful for political life in the arts both of war and peace. But the opposite often happens – that some have the souls and others have the bodies of freemen.

– Aristotle *Politics*

Sons have no richer endowment than the quality

A noble and brave father gives in their begetting.

– Euripides *Heracleidae*

His looks are full of peaceful majesty,

His head by nature fram'd to wear a crown,

His hands to wield a sceptre….

– Shakespeare *Henry VI Part 3*

A daughter of a green Grocer, walks the Streets in London dayly with a baskett of Cabbage Sprouts, Dandelions and Spinage on her head. She is observed by the Painters to have a beautiful Face, an elegant figure, a graceful Step and a debonair. They hire her to Sitt. She complies, and is painted by forty Artists, in a Circle around her. The Scientific Sir William Hamilton outbids the Painters, Sends her to Schools for a genteel Education and Marries her. This Lady not only causes the Tryumphs of the Nile of Copenhagen and Trafalgar, but Seperates Naples from France and finally banishes the King and Queen from Sicilly. Such is the Aristocracy of the natural Talent of Beauty.

– John Adams to Thomas Jefferson, on Emma Hamilton

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