

Human capital mediates natural selection in contemporary humans

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Abstract

Natural selection has been documented in contemporary humans, but little is known about the mechanisms behind it. We test for natural selection through the association between 33 polygenic scores and fertility, across two generations, using data from UK Biobank (N = 409,629 British subjects with European ancestry). Consistently over time, polygenic scores associated with lower (higher) earnings, education and health are selected for (against). Selection effects are concentrated among lower SES groups, younger parents, people with more lifetime sexual partners, and people not living with a partner. The direction of natural selection is reversed among older parents, or after controlling for age at first live birth. These patterns are in line with economic theories of fertility, in which earnings-increasing human capital may either increase or decrease fertility via income and substitution effects in the labour market. Studying natural selection can help us understand the genetic architecture of health outcomes: we find evidence in modern day Great Britain for multiple natural selection pressures that vary between subgroups in the direction and strength of their effects, that are strongly related to the socio-economic system, and that may contribute to health inequalities across income groups.

Footnote 2 in the main text states that “all our qualitative results are robust to using weights.” This version of the paper confirms that by using Age/Qualification weights throughout. The only substantive change is that two extra polygenic scores show statistically significant balancing/diversifying selection, see the Appendix.

I Introduction

Living organisms evolve through natural selection, in which allele frequencies change in the population through differential reproduction rates. Studying the mechanisms behind natural selection can help us better understand how individual differences in complex traits and disease risk arise (Benton et al. 2021). Recent work confirms that natural selection is taking place in modern human populations, using genome-wide analysis (Barban et al. 2016; Beauchamp 2016; Conley et al. 2016; Kong et al. 2017; Sanjak et al. 2018; Fieder and Huber 2022). In particular, genetic variants associated with higher educational attainment are being selected against, although effect sizes appear small.

As yet we know little about the social mechanisms behind natural selection. A potential explanation comes from the economic theory of fertility (Becker 1960). In this theory, higher potential earnings have two opposite effects on fertility: a fertility-increasing *income effect* (higher income makes children more affordable), and a fertility-lowering *substitution effect* (time spent on childrearing has a higher cost in foregone earnings). Thus, an individual’s *human capital* – skills and personality traits which are valuable in labour markets – can increase or decrease his or her fertility. This can lead to selection on inherited variants which are linked to human capital. Furthermore, the economic theory predicts systematic variation in the relative strength of the income and substitution effects across different social groups.

This study uses data from UK Biobank (Bycroft et al. 2018) to learn more about contemporary natural selection. We test for natural selection on 33 different polygenic scores by estimating their correlation with fertility. We extend the analysis over two generations, using data on respondents’ number of siblings as well as their number of children. This is interesting because consistent natural selection over multiple generations could lead to substantive effects in the long run. Next, we examine correlations with fertility in different subgroups. Across the board, selection effects are stronger in groups with lower income and less education, among younger parents, people not living with a partner, and people with more lifetime sexual partners. Outside these groups, effects are weaker and often statistically insignificant. In some subgroups, the direction of selection is even reversed.

We then show that a simple model of human capital, education and fertility choices can give rise to these empirical results. To test the theory, we show that polygenic scores’ correlation with fertility is associated with their correlation with education and earnings. Furthermore, a mediation analysis shows that part of the correlation with fertility is indeed *via* education. Thus, contemporary natural selection on polygenic scores can be explained by scores’ correlation with earnings-increasing human capital.

Lastly, we discuss the effects of natural selection. While measured effects on mean genetic scores are small, natural selection may substantially increase the correlation between genetic scores and income, i.e. the “genetic lottery” (Harden 2021).

2 Results

We created polygenic scores for 33 traits in 409,629 individuals of European descent, corrected for ancestry using 100 genetic principal components (see Materials and Methods). Figure 1 plots mean polygenic scores in the sample by 5-year birth intervals. Several scores show consistent increases or declines over this 30-year period, of the order of 5% of a standard deviation. These changes could reflect natural selection within the UK population, but also emigration, or ascertainment bias within the sample (Fry et al. 2017).

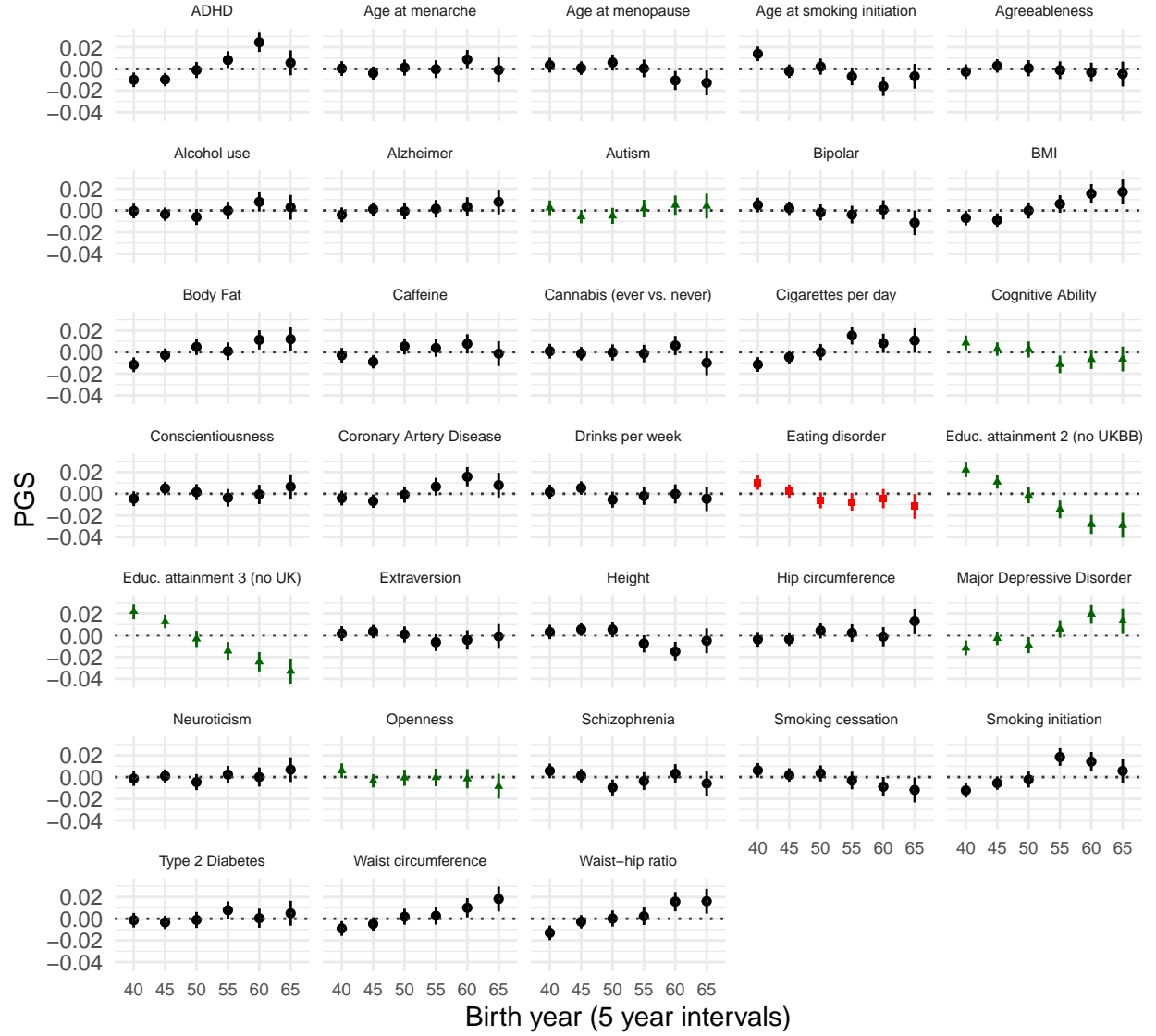


Figure 1: Mean polygenic scores (PGS) by birth year in UK Biobank. Points are means for 5-year intervals. Lines are 95% confidence intervals. Green triangles show a significant linear increase over time ($p < 0.05/33$). Red squares show a significant decrease.

To test more directly for natural selection, we regress respondents’ number of children (N_i) on their polygenic scores (PGS):

$$N_i = \alpha + \beta \text{PGS}_i + \varepsilon_i \quad (1)$$

The “selection effect,” β , reflects the strength of natural selection within the sample. In fact, since polygenic scores are normalized, β is the expected polygenic score among children of the sample (Beauchamp 2016).¹ Note that equation (1) does not control for many environmental and genetic factors that could affect fertility, and as a result, β is not an estimate of the causal effect of a polygenic score on fertility. However, natural selection is a correlational phenomenon: polygenic scores which correlate with high fertility are being selected for, whatever the underlying causal relationship.

To correct for ascertainment bias, we weight participants using population data. We try three alternative weighting schemes: (1) “Geographical” weighting, by geography, age and presence/absence of a partner; (2) age and highest educational qualification; and (3) for women only, age, highest qualification, and age at first live birth (AFLB). Figure 2 plots selection effects among the entire sample, estimated with the three weighting schemes, and also unweighted. Effects for 17 out of 33 polygenic scores are significant at $p < 0.05/33$ under all four weighting schemes, with 26 scores reaching significance under at least one scheme. Weighting makes a large difference to estimates: mean effect sizes across all polygenic scores are increased by a factor of 1.13 (geographical weighting), 1 (age/qualification) or 1.44 (age/qualification/AFLB). Estimates might be further affected by weighting on other demographic variables.²

We also check for balancing and diversifying selection by estimating (1) with a quadratic term.³ In particular, we find diversifying selection for educational attainment polygenic scores: at higher values of these scores, the negative effect on fertility is smaller (Appendix Figure 9).

To motivate our economic model, we split the sample by demographic and social variables, and re-estimate (1) within each subgroup. We examine socio-demographic variables including income, education and sex, and family structure variables including age at first live birth, presence of a partner, and lifetime number of sexual partners. We emphasize that these categories are not necessarily exogenous to the polygenic scores. For example, both in the data and in our theoretical model, age at first live birth is a choice variable, which is endogenous to human-capital-related polygenic scores (Appendix Figure 14). Nevertheless, differences in equation (1) across subgroups constrain the set of possible explanations for natural selection. As we show below, a simple economic model predicts the differences we find.

¹The selection effect β equals $Cov(N, PGS)/Var(PGS)$ where N is the number of children. Since PGS are normalized to variance 1 and mean 0, this reduces to $Cov(N, PGS) \equiv E(N PGS) - E(N)E(PGS) = E(N PGS)$. This is the polygenic score weighted by the number of children, which is the average polygenic score in the next generation.

²To make interpretation straightforward, we do not use weights in results below unless noted. However, all our qualitative results are robust to using weights.

³Balancing selection selects for intermediate values, reducing the variance of the trait under selection. Diversifying selection selects for extreme values and increases the variance.

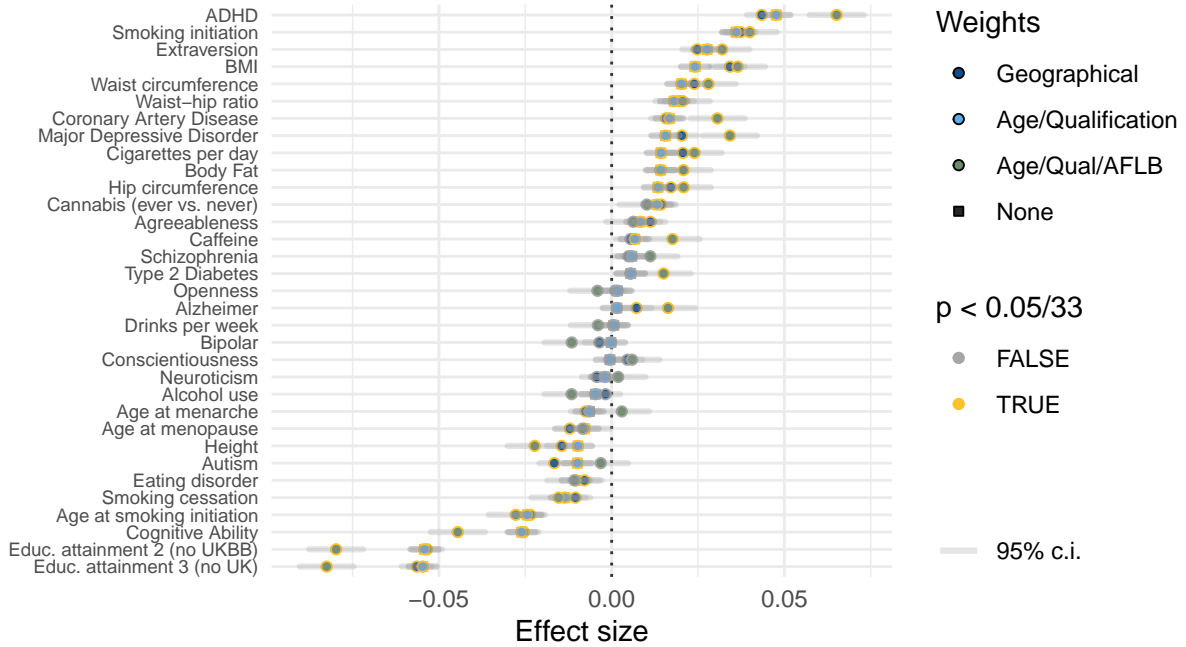


Figure 2: Selection effects: weighted and unweighted regressions. Each point represents a single bivariate regression of number of children on a polygenic score.

Figure 3 plots selection effects for each polygenic score, grouping respondents by age of completing full-time education, and by household income. Effects are larger and more significant for the lowest income category, and for the lowest education category. These results could be explained by age, if older respondents have lower income and are less educated, and also show more natural selection on polygenic scores. However, when we rerun the regressions, interacting the polygenic score with income category and also with a quadratic in age, the interaction with income remains significant at $p < 0.05/33$ for 20 out of 33 regressions. Similarly if we interact the PGS with age of leaving full time education and a quadratic in age, the interaction with age leaving full time education remains significant at $p < 0.05/33$ for 20 out of 33 regressions.

Selection effects are also different between men and women (Appendix Figure 8). Differences are particularly large for educational attainment, height and MDD. Several polygenic scores for mental illness and personality traits are more selected for (or less against) among women, including major depressive disorder (MDD), schizophrenia and neuroticism, while extraversion is more selected for among men. Scores for waist circumference and waist-hip ratio are less selected for among women. One possible reason for these sex differences is that polygenic scores may affect fertility via success in marriage markets, and men and women may value different characteristics in these.

We next focus on variables related to household type and reproductive strategy. We split males and females by lifetime number of sexual partners, at the median value of 3 (Figure 4a). For both sexes, selection effects are larger and more significant among those with more than 3 lifetime partners. Next we split respondents by whether they were living with

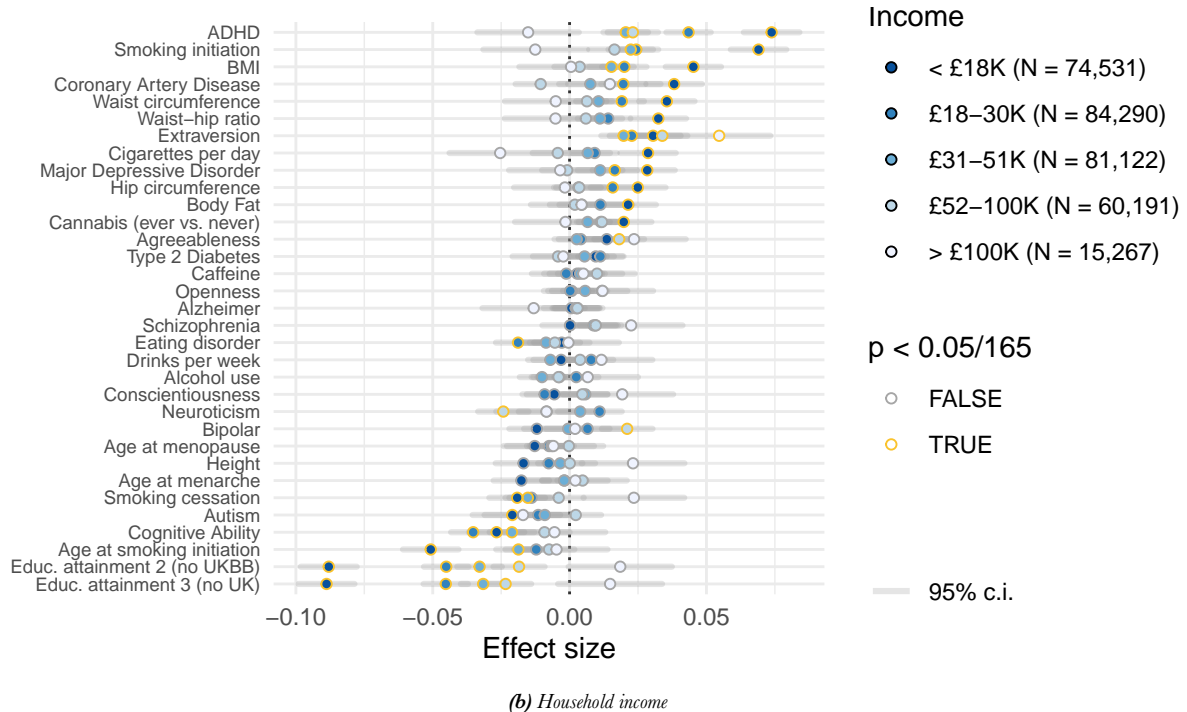
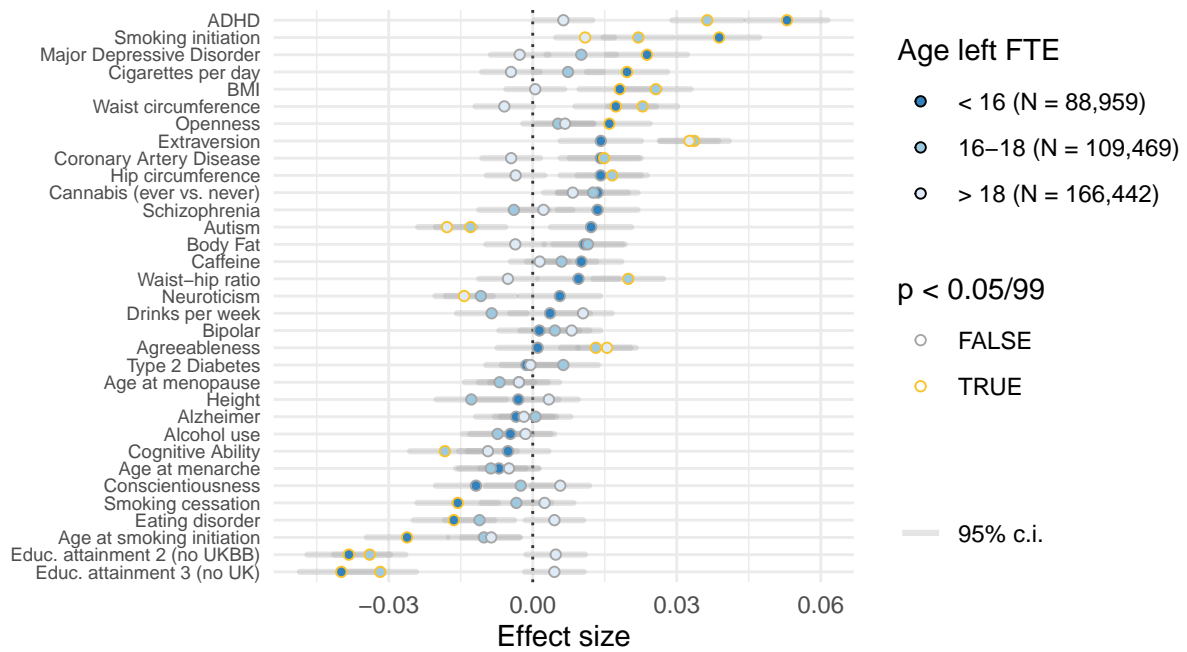


Figure 3: Selection effects by education and income.

a spouse or partner at the time of interview. Effects are larger among those not living with a spouse or partner (Figure 4b).

Lastly, we split female respondents by age at first live birth (AFLB).⁴ There is evidence for genetic effects on AFLB (Barban et al. 2016), and there is a close link between this variable and number of children born. Figure 5 shows effect sizes estimated separately for each tercile of AFLB. Several effects are strikingly different across terciles. ADHD and MDD are selected for amongst the youngest third of mothers, but selected against among the oldest two-thirds. Educational attainment is selected for among the oldest two-thirds of mothers, but is not significantly selected among the youngest third. Similarly, several polygenic scores for body measurements are selected against only among older mothers. The correlation between effect sizes for the youngest and oldest terciles is -0.54 .

To investigate this further, we estimate equation (1) among females, *controlling* for AFLB. In 25 out of 33 cases, effects change sign when controls are added. The correlation between effect sizes controlling for AFLB, and raw effect sizes, is -0.75 . Thus, selection effects seem to come through two opposing channels: an effect on AFLB, and an opposite-signed effect on number of children after AFLB is controlled for.

We also examine selection effects among respondents' parents, using information on respondents' number of siblings. Effect sizes of polygenic scores are highly correlated across the two generations. Median-splitting respondents by year of birth, we find little evidence of change in effect sizes among either the parents' or the respondents' generation. We also check whether selection effects vary by socio-economic status and AFLB in the parents' generation. We have no information about parents' income, so we use the 1971 Townsend deprivation score of respondents' birthplace as a proxy (Townsend 1987). Results show the same pattern as for respondents. Effect sizes are larger and more often significant in the most deprived areas (Appendix Figure 11). Effects are larger among younger parents, and change sign when controlling for AFLB, among both mothers and fathers (Appendix Figures 12, 13). Lastly, we check for a "quantity-quality tradeoff" between parents' number of children and number of grandchildren. We do not find any: in fact, the correlation between *number of siblings* and *number of children* is positive ($\rho = 0.1$, $p < 2 \times 10^{-16}$).

⁴This information is unavailable for men.

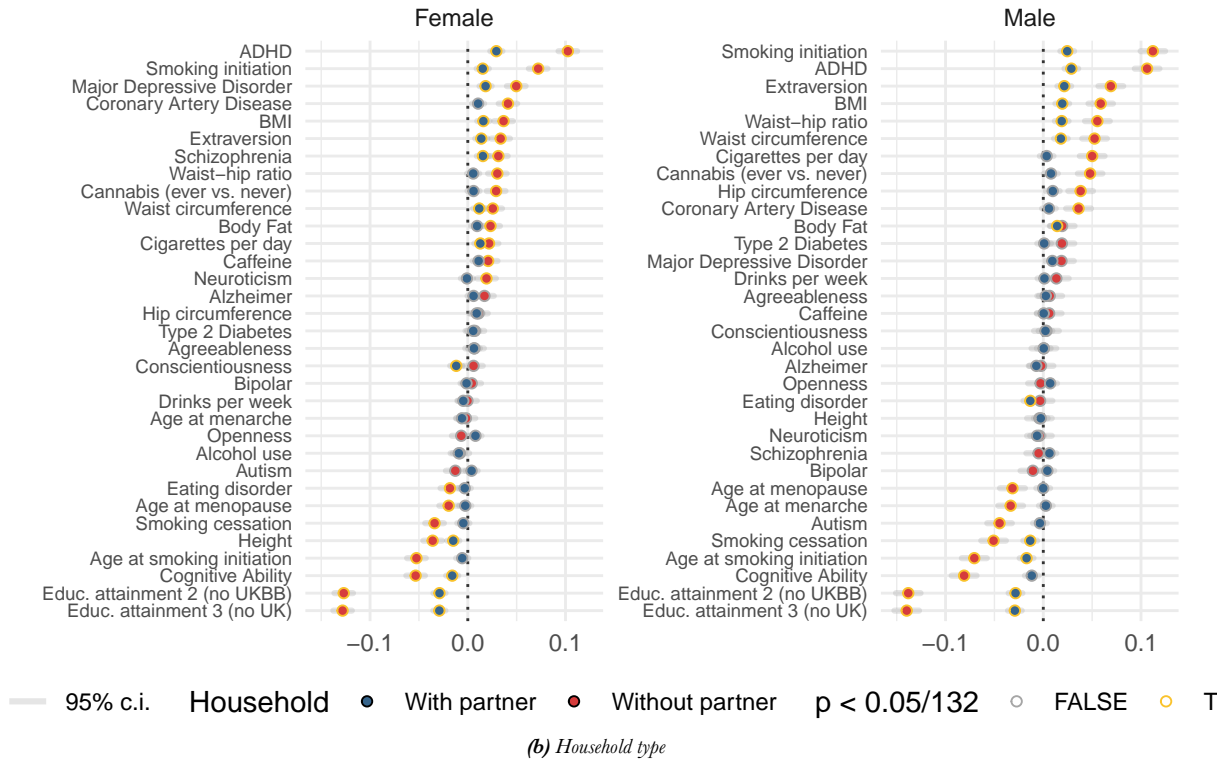
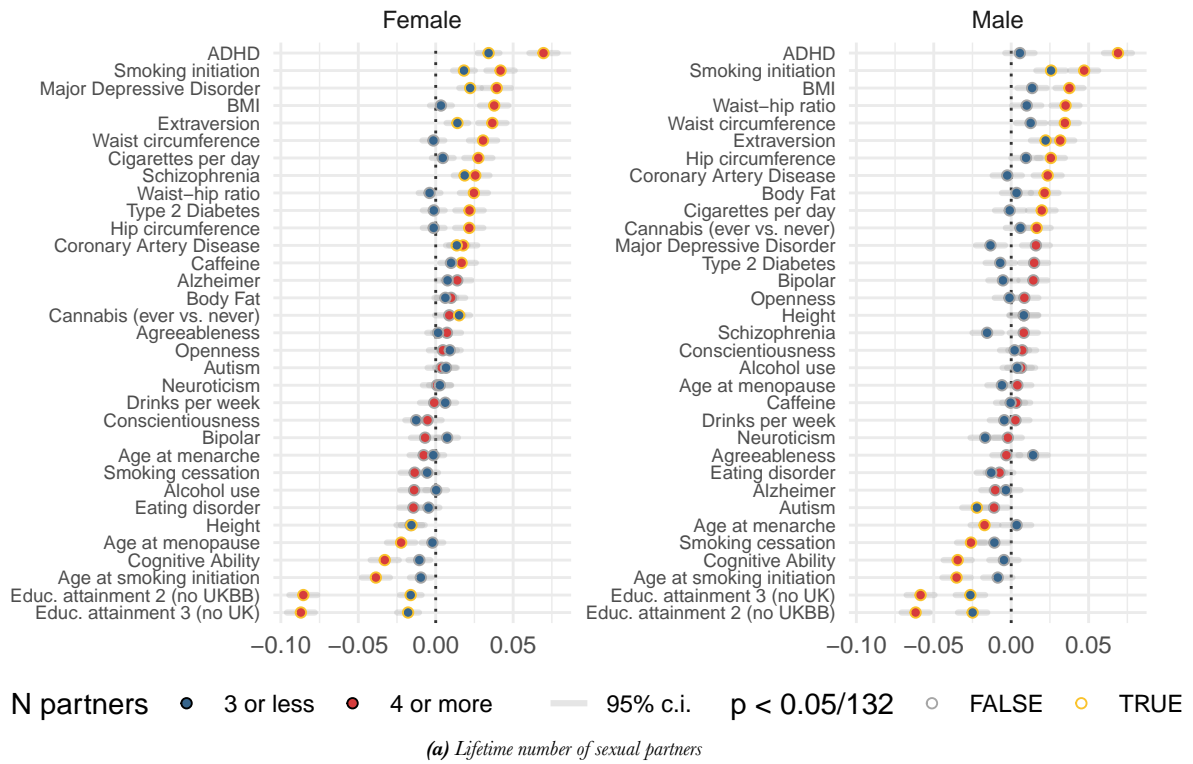


Figure 4: Selection effects by number of sexual partners and household type.

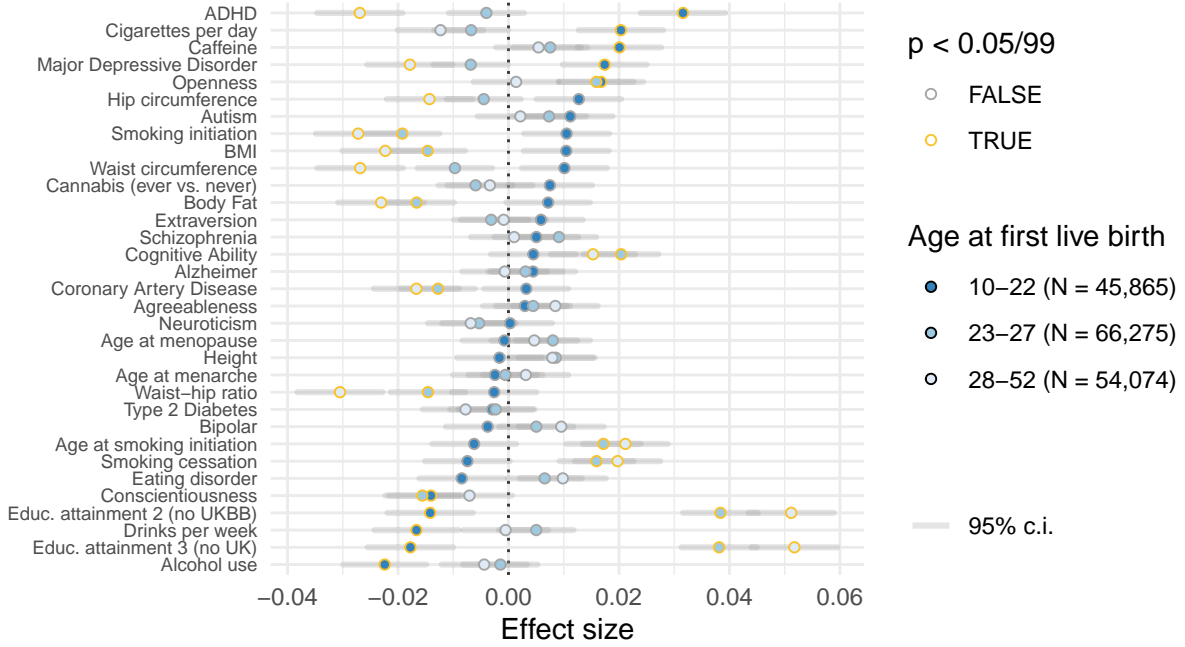


Figure 5: Selection effects by age at first live birth terciles (women only).

3 Human capital and natural selection

These results show that selection effects are weaker, absent, or even reversed among some subgroups of the population. A possible explanation for this comes from the economic theory of fertility (Becker 1960; Willis 1973; Becker and Tomes 1976). According to this theory, increases in a person's wage affect their fertility via two opposing channels. There is an *income effect* by which children become more affordable, like any other good. There is also a *substitution effect*: since childrearing has a cost in time, the opportunity cost of childrearing increases if one's market wage is higher. The income effect leads higher earners to have more children. The substitution effect leads them to have fewer.

Suppose that certain genetic variants correlate with human capital – skills or other characteristics that affect an individual's earnings in the labour market (Mincer 1958; Becker 1964). These variants may then be associated with opposing effects on fertility. The income effect will lead to natural selection in favour of earnings-increasing variants (or variants that are merely associated with higher earnings). The substitution effect will do the reverse.

To show this, we write down a simple model of fertility choices. h is an individual's level of human capital. For now, we simply identify this with his or her wage W . Raising a child takes time b . People maximize utility U from the number of children N and from income $Y \equiv (1 - bN)W$:

$$U = u(Y) + aN.$$

Here a captures the strength of preference for children. $u(\cdot)$ captures the taste for income, and is increasing and concave. We treat N as continuous, in line with the literature: this can be thought of as the expected number of children among similar people. The marginal benefit of an extra child is $\frac{dU}{dN} = -bWu'(Y) + a$. The effect of an increase in human capital on this marginal benefit is

$$\frac{d^2U}{dNdh} = \underbrace{-bu'(Y)}_{\text{Substitution effect}} \underbrace{-bYu''(Y)}_{\text{Income effect}}.$$

The *substitution effect* is negative and reflects that when wages increase, time devoted to childcare costs more in foregone income. The positive *income effect* depends on the curvature of the utility function, and reflects that when income is higher, the marginal loss of income from children is less painful.

To examine education and fertility timing, we extend the model to two periods. For convenience we ignore time discounting, and assume that credit markets are imperfect so that agents cannot borrow. Write

$$U(N_1, N_2) = u(Y_1) + u(Y_2) + aN_1 + aN_2 \quad (2)$$

Instead of identifying human capital with wages, we now allow individuals to choose a level of education $s \in [0, 1]$, which has a time cost in period 1. Education is complementary to human capital $h > 0$, and increases period 2 wages, which take the simple functional form $w(s, h) = sh$. We normalize period 1 wages to 1. Letting $u(\cdot)$ take the constant relative risk aversion form $u(y) = \frac{y^{1-\sigma}-1}{1-\sigma}$, where $\sigma > 0$ captures risk aversion in income, we examine total fertility $N^* = N_1^* + N_2^*$ and the *fertility-human capital relationship*, $\frac{dN^*}{dh}$. For $\sigma < 1$ in a neighbourhood of $\sigma = 1$, Table 1 shows five theoretical predictions, along with our corresponding empirical results for the correlation between polygenic scores and fertility.⁵ The key insight of the model is that for middling levels of risk aversion, the substitution effect dominates at low income levels, but is balanced by the income effect as income increases.

Thus, a simple economic model can explain many of our results. Other empirical work in economics also supports our mechanism. Caucutt, Guner, and Knowles (2002) and Monstad, Proper, and Salvanes (2008) show that education and skills affect age at first birth and fertility. Income decreases fertility at low income levels, but increases it at higher income levels (Cohen, Dehejia, and Romanov 2013). US fertility decreases faster with education among single mothers than married mothers (Baudin, De La Croix, and Gobbi 2015), in line with our prediction 3 and as predicted by Becker (1981).

⁵Predictions 1-3 also hold in the one-period model with constant relative risk aversion. Our empirical results are actually stronger than prediction 5, in that correlations with fertility are *reversed* at higher AFLB. This prediction can be accommodated in the model if children have a money cost as well as a time cost (Appendix Figure 21).

Table 1: Predictions from the theoretical model and corresponding empirical results.

	Theory	Empirical results
1.	The fertility-human capital relationship is negative: $\frac{dN^*}{dh} < 0$.	Figures 1 and 2.
2.	The fertility-human capital relationship is weaker (closer to zero) at higher wages and/or levels of human capital.	Figure 3a. Selection effects are also weaker at higher polygenic scores for educational attainment (Appendix Figure 9).
3.	The relationship is more negative when the time burden of children, b , is larger.	Stronger effects for single parents (Figure 4).
4.	The relationship is weaker at higher levels of education s .	Figure 3b.
5.	The relationship is weaker among those who start fertility in period 2 ($N_1^* = 0$) than among those who start fertility in period 1 ($N_1^* > 0$).	Effects weaker among those starting fertility later (Figure 5).

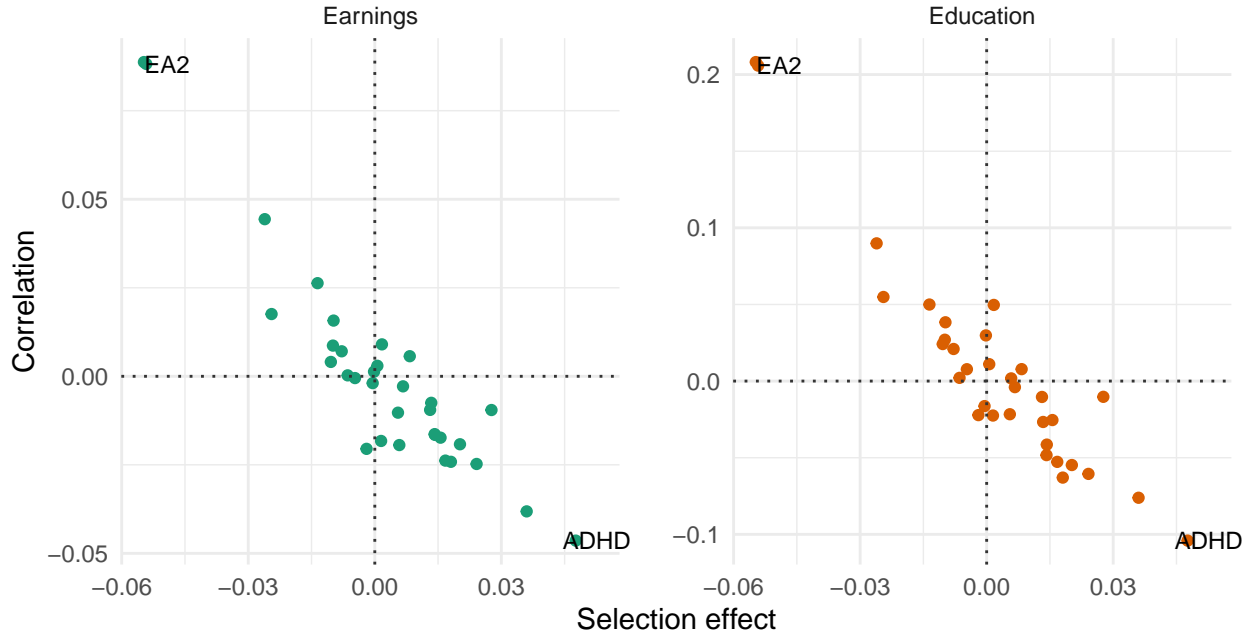


Figure 6: Selection effects by correlations with earnings and educational attainment. Each point represents one polygenic score.

4 Testing the theory

We test this explanation in two ways. First, the economic theory predicts that genetic variants will be selected for (or against) in proportion to their correlation with human capital. Figure 6 plots selection effects on each polygenic score against that score’s correlation with two measures of human capital: earnings in a respondent’s first job, and educational attainment. The relationships are strongly negative. Thus, human capital appears to be relevant to natural selection. The negative relationship suggests that substitution effects dominate income effects, which fits the known negative association between income and fertility (Becker 1960; Jones and Tertilt 2006). The correlations reverse when we control for age at first live birth, suggesting that within AFLB categories, the income effect dominates. (See also the Supplementary Animations for the uncorrected effects per AFLB group, which show the correlation reversing after age 22.)

Second, we run a mediation analysis to directly test whether the correlation between each polygenic score and fertility is mediated by educational attainment (Appendix Table 5). For 24 scores, the indirect effect of the score on fertility *via* educational attainment is significantly different from zero ($p < 0.05/33$). Among these scores, the median proportion of the total effect explained by the indirect effect is 22.24%. The educational attainment variable is a relatively crude measure of human capital: more accurate measures would likely explain more of the total effect.

These results support our theory that natural selection on polygenic scores is driven by their *correlation* with human capital. We also test whether polygenic scores *cause* fertility by running within-siblings regressions (Appendix Figure 16). With a reduced sample size, all within-sibling effects are insignificant after Bonferroni correction. However, effect sizes are positively correlated with effect sizes from the pooled model, and about 50% smaller (regressing within-sibling on pooled effect sizes, $b = 0.43$). This attenuation is in line with the ~50% decrease in heritability in within-sibling GWASs on age at first birth and educational attainment (Howe et al. 2021). We see these results as providing tentative evidence that polygenic scores cause fertility, with effects being partly driven by correlations with environmental variation in human capital. We also reran within-siblings regressions adding a control for education. Most effect sizes barely change, suggesting that our measure of education does not in general mediate differences in fertility among siblings. However, for the educational attainment polygenic score (EA3), effect sizes are reduced by 23.49%.

We consider three alternative theories that might explain our results. First, welfare benefits which incentivize child-bearing might be taken up more among low-income individuals. However, most effect sizes appear unchanged over a large span of twentieth-century history (Appendix Tables 3 and 4), during which government spending on child-related benefits varied considerably (Social Security Committee 1999). In general, there is only weak evidence that welfare benefits affect fertility (Gauthier 2007). A second alternative theory is that polygenic scores correlate with the

motivation to have children, i.e. parameter a in the model (cf. Jones, Schoonbroodt, and Tertilt 2008). This theory would not explain why selection effects are smaller at higher incomes and education levels. (Indeed, in the model, the effect of a on fertility becomes stronger at higher levels of human capital.) A third alternative is that traits under selection are linked to externalizing behaviour and risk-seeking. This might be partially captured by our risk-aversion parameter σ , which affects fertility by changing the marginal utility of income, but a more direct channel is risky sexual behaviour (Mills et al. 2021). The data here provide some support for this: scores which might plausibly be linked to externalizing behaviour, like ADHD and younger age at smoking initiation, are selected for. However, this theory is less good at explaining variation in selection across the full range of scores, including physical measures such as waist-hip ratio and BMI. We test this theory directly by re-estimating equation (1) controlling for a measure of risk attitude (UK Biobank field 2040). The median ratio of effect sizes between regressions with and without controls is 0.99; all scores which are significant at $p < 0.05/33$ in uncontrolled regressions remain so when controlling for risk attitude. This non-result could simply reflect the imprecision of the risk attitude measure, which is a single yes/no question. However, this measure *does* predict the overall number of children, highly significantly ($p < 2 \times 10^{-16}$ in 33 out of 33 regressions). Given that, and the statistical power we get from our sample size, we believe that the non-result is real: while risk attitude does predict fertility in the sample, it is not an important channel for natural selection.

5 Discussion

Previous work has documented natural selection in modern populations on variants underlying polygenic traits (Beauchamp 2016; Kong et al. 2017; Sanjak et al. 2018). We show that correlations between polygenic scores and fertility are highly concentrated among specific subgroups of the population, including people with lower income, lower education, younger first parenthood, and more lifetime sexual partners. Among mothers aged 22+, selection effects are reversed. Furthermore, the size of selection effects on a polygenic score correlates with that score’s association with labour market earnings. The economic theory of fertility provides a parsimonious explanation for these findings. Because of the substitution effect of earnings on fertility, scores are selected for when they correlate with low human capital, and this effect is stronger at lower levels of income and education.

Polygenic scores which correlate with high (low) earnings and more (less) education are being selected against (selected for). In addition, many of the phenotypes under positive selection are linked to disease risk. Many people would probably prefer to have high educational attainment, a low risk of ADHD and major depressive disorder, and a low risk of coronary artery disease, but natural selection is pushing against genes associated with these traits. Potentially, this could increase the health burden on modern populations, but that depends on effect sizes. Our results suggest that naïve estimates can be affected by sample ascertainment bias. This problem may be less serious in surveys which aim to

be representative (as the UK Biobank does not). However, there is still scope for bias, since not all respondents consent to the collection of genetic data. For instance, completion rates for genotype data in the US Health and Retirement Study were around 80-85% (HRS 2020). Researchers should be aware of the risks of ascertainment when studying modern natural selection.

We also do not know how estimated effect sizes of natural selection will change as more accurate measures of genetic variation are produced, or whether genetic variants underlying other phenotypes will show a similar pattern to those studied here. In addition, effects of polygenic scores may be inflated in population-based samples, because of indirect genetic effects, gene-environment correlations, and/or assortative mating (Lee et al. 2018; Selzam et al. 2019; Kong et al. 2018; Howe et al. 2021), although we do not expect that this should impact their association with number of offspring, or the resulting changes in allele frequencies. In short, it is probably too early to tell whether modern natural selection has a substantively important effect on population means of phenotypes under selection. Nevertheless, we note that selection effects on our measured polygenic scores are still relatively small, even after reweighting to account for ascertainment bias.

Because selection effects are concentrated in lower-income groups, they may also increase inequality with respect to polygenic scores. For example, Figure 7 graphs mean polygenic scores for educational attainment (EA3) among children from households of different income groups. The blue bars show the actual means, i.e. parents' mean polygenic score weighted by number of children. The grey bars show the hypothetical means if all households had equal numbers of children. Natural selection against genes associated with educational attainment, which lowers the mean, is stronger at the bottom of the income distribution, and this increases the differences between groups. Overall, natural selection increases the correlation of polygenic scores with income for 28 out of 33 polygenic scores, with a median percentage increase of 13.53% (Appendix Table 6).

If inequalities in polygenic scores are important for understanding social structure and mobility (Belsky et al. 2018; Rimfeld et al. 2018; Harden 2021), then these increases are substantive. A back-of-the-envelope calculation, using estimated heritability for educational attainment of 0.4 (Branigan, McCallum, and Freese 2013), gives the proportion of variance in household income attributable to the “true” polygenic score for educational attainment as 15.5% among respondents. Natural selection would raise this proportion to 19.4% among respondents' children, counterfactually holding environmental conditions constant. Similarly, since many polygenic scores are predictive of disease risk, they could potentially increase health inequalities. In general, the evolutionary history of anatomically modern humans is related to disease risk (Benton et al. 2021); understanding the role of contemporary natural selection may aid in mapping the genetic architecture of current health disparities.

Existing evidence on human natural selection has led some to “biocosmic pessimism” (Sarraf, Feltham, et al. 2019).

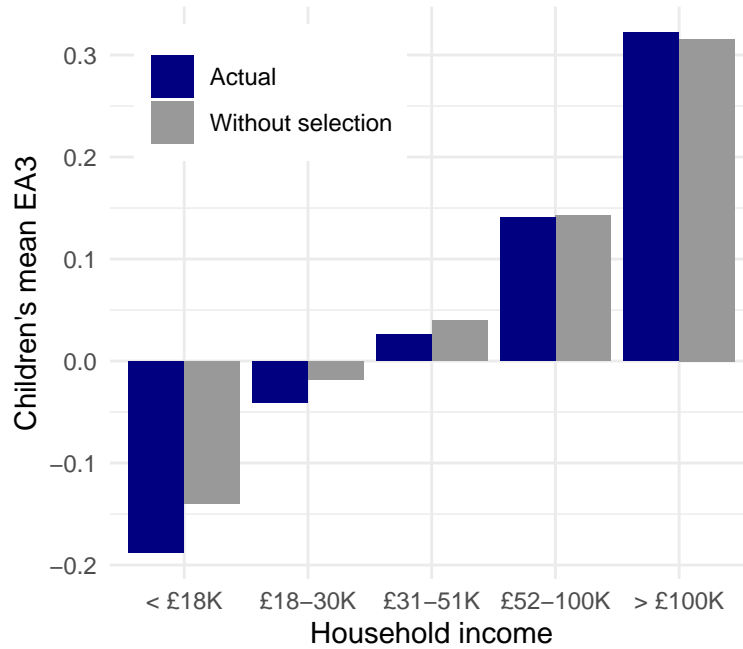


Figure 7: Mean polygenic score for educational attainment (EA3) of children by household income group. Blue is actual. Grey is hypothetical in the absence of selection effects. Respondents weighted by Age/Qualification.

Others are more sanguine, and argue that natural selection's effects are outweighed by environmental improvements, such as those underlying the Flynn effect (Flynn 1987). The evidence here may add some nuance to this debate. Patterns of natural selection have been relatively consistent across the past two generations, but they are not the outcome of a universal, society-wide phenomenon. Instead they result from opposing forces, operating in different parts of society and pulling in different directions.

Any model of fertility is implicitly a model of natural selection, but so far, the economic and human genetics literatures have developed in parallel. Integrating the two could deepen our understanding of natural selection in modern societies. Economics possesses a range of theoretical models on the effects of skills, education and income (see Hotz, Klerman, and Willis 1997; Lundberg and Pollak 2007). One perennial problem is how to test these theories in a world where education, labour and marriage markets all interact. Genetic data, such as polygenic scores, could help to pin down the direction of causality, for example via Mendelian randomization (Davey Smith and Ebrahim 2003). Conversely, theory and empirical results from economics can shine a light on the mechanisms behind natural selection, and thereby on the nature of individual differences in complex traits and disease risk.

6 Materials and methods

We use participant data from UK Biobank (Bycroft et al. 2018), which has received ethical approval from the National Health Service North West Centre for Research Ethics Committee (reference: 11/NW/0382). We limit the sample to white British participants of European descent, as defined by genetic estimated ancestry and self-identified ethnic group, giving a sample size of 409,629. For regressions on number of children we use participants over 45, since most fertility is completed by this age. This gives a sample size of 371,088.

Polygenic scores were chosen so as to cover a reasonably broad range of traits, and based on the availability of a large and powerful GWAS which did not include UK Biobank. Scores were computed by summing the alleles across ~ 1.3 million genetic variants weighted by their effect sizes as estimated in 33 genome-wide association studies (GWASs) that excluded UK Biobank. To control for population stratification, we corrected the polygenic scores for 100 principal components (PCs).

Earnings in first job are estimated from mean earnings in the 2007 Annual Survey of Hours and Earnings, using the SOC 2000 job code (Biobank field 22617).

Population data for weighting is taken from the 2011 UK Census and the 2006 General Household Survey (GHS). Weighting for Age/Qualification and Age/Qualification/AFLB weights was done using marginal totals from a linear model, using the `calibrate()` function in the R “survey” package (Lumley 2020). Geographical weighting was done with iterative post-stratification using the `rake()` function, on Census Middle Layer Super Output Areas, sex and presence/absence of a partner.

7 Appendix

Contents

7.1	Natural selection by sex	18
7.2	Weighted regressions	19
7.3	Balancing and diversifying selection	20
7.4	Parents' generation	21
7.4.1	Selection effects	21
7.4.2	Area deprivation	24
7.4.3	Age at first live birth	25
7.5	Effects of polygenic scores on age at first live birth	28
7.6	Mediation analysis	30
7.7	Within-siblings regressions	32
7.8	Effects on inequality	33
7.9	Estimating the effect of natural selection on the contribution of PSEA to inequality	34
7.9.1	Proof of (5)	35
7.10	Further results	37
7.10.1	Selection effects on raw polygenic scores	37
7.10.2	Genetic correlations with EA3	39
7.11	Model proofs	40

7.1 Natural selection by sex

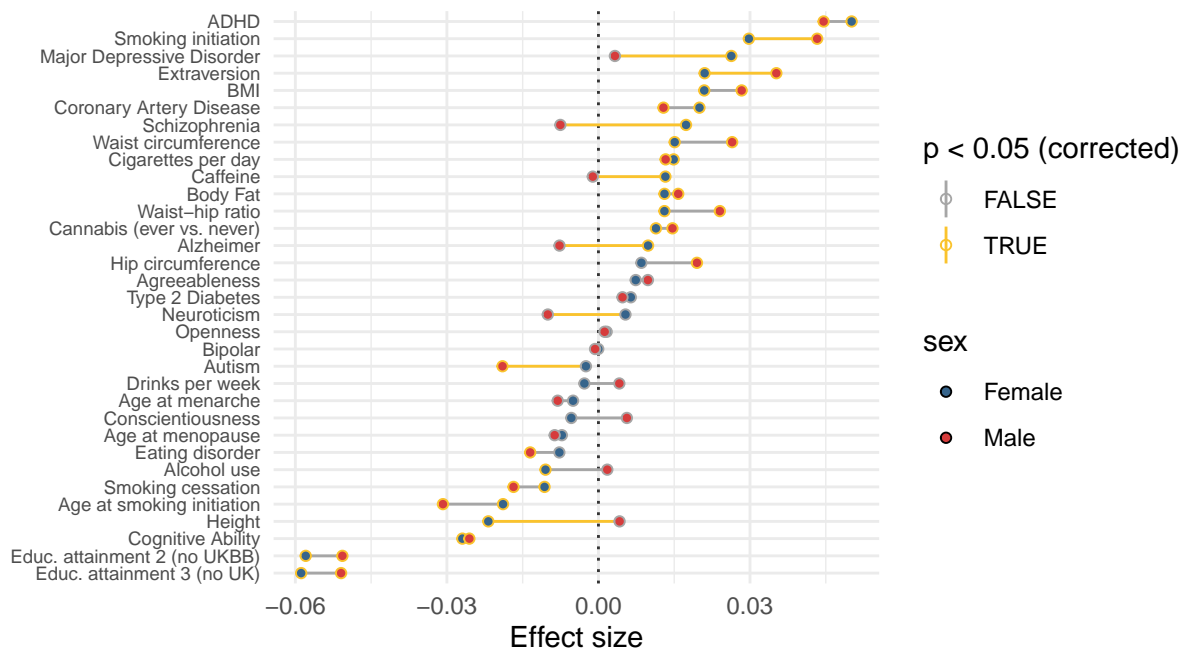


Figure 8: Selection effects by sex. Highlighted lines are significant differences at $p < 0.05/33$. Highlighted points are significantly different from 0 at $p < 0.05/66$.

7.2 Weighted regressions

Table 2 gives effect sizes as a proportion of the unweighted effect size, for all polygenic scores which are consistently signed and which are significantly different from zero in unweighted regressions.

Table 2: *Weighted effect sizes as a proportion of unweighted effect sizes.*

PGS	Weighting		
	Geographical	Age/Qualification	Age/Qual/AFLB
Autism	1.68	1.00	1.27
Age at menopause	1.55	1.00	1.16
Height	1.48	1.00	1.02
Cigarettes per day	1.45	1.00	1.61
BMI	1.42	1.00	1.74
Agreeableness	1.35	1.00	0.85
Major Depressive Disorder	1.30	1.00	1.30
Hip circumference	1.28	1.00	2.45
Waist circumference	1.18	1.00	1.86
Cannabis (ever vs. never)	1.09	1.00	0.89
Waist-hip ratio	1.09	1.00	1.57
Smoking initiation	1.04	1.00	1.34
Educ. attainment 3 (no UK)	1.03	1.00	1.40
Body Fat	0.99	1.00	1.59
Educ. attainment 2 (no UKBB)	0.99	1.00	1.38
Cognitive Ability	0.97	1.00	1.65
Age at smoking initiation	0.96	1.00	1.47
Coronary Artery Disease	0.94	1.00	1.53
ADHD	0.91	1.00	1.30
Extraversion	0.90	1.00	1.52
Caffeine	0.82	1.00	1.33
Smoking cessation	0.77	1.00	1.44
Eating disorder	0.76	1.00	1.39
<i>Mean</i>	1.13	1.00	1.44
<i>Median</i>	1.04	1.00	1.40

Only consistently-signed and significant (when unweighted) estimates are shown.
Age/Qual/AFLB as a proportion of unweighted regressions including females only.

7.3 Balancing and diversifying selection

Balancing selection reduces variance in the trait under selection, while diversifying selection increases variance. To check for these, we rerun equation (1), adding a quadratic term in PGS_i , and using our age/qualification weights. Scores for hip circumference show significant balancing selection ($p < 0.05/33$, negative coefficient on quadratic term). Scores for educational attainment (EA2 and EA3) show significant diversifying selection ($p < 0.05/33$, positive coefficient), which reduces the strength of selection against educational attainment at very high levels of the PGS. Figure 9 plots predicted number of children against polygenic score from these regressions.

We also checked for balancing selection in the parents' generation, using age/qualification weights multiplied by the inverse of number of siblings. Scores for EA2 and EA3 again show significant diversifying selection ($p < 0.05/33$, positive coefficient). Other scores including hip circumference were not significant.

Weighted regressions only: in addition to the above, major depressive disorder shows diversifying selection and drinks per week show balancing selection.

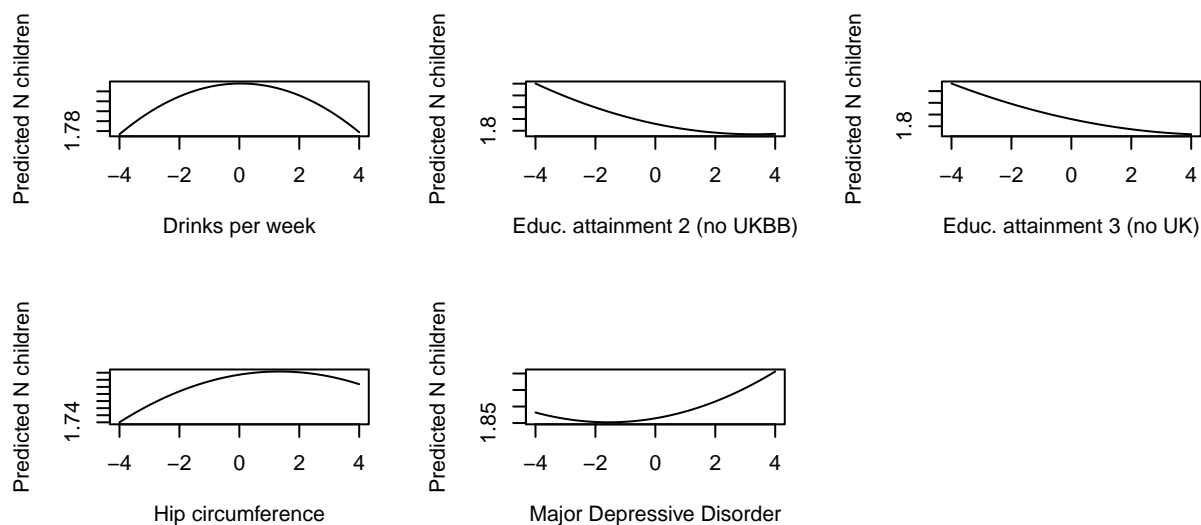


Figure 9: Purifying/diversifying selection: predicted number of children by polygenic score.

7.4 Parents' generation

7.4.1 Selection effects

The UK Biobank data contains information on respondents' number of siblings, i.e. their parents' number of children. Since respondents' polygenic scores are equal in expectation to the mean scores of their parents, we can use this to look at selection effects in the parents' generation. We estimate equation (1) using *number of siblings* (including the respondent) as the dependent variable. The parents' generation has an additional source of ascertainment bias: sampling parents of respondents overweights parents who have many children. For instance, parents of three children will have, on average, three times more children represented in UK Biobank than parents of one child. Parents of no children will by definition not be represented. To compensate, we reweight our preferred weightings (Age/Qualification) by the inverse of *number of siblings*.

Figure 10 shows regressions of *number of siblings* on polygenic scores. For a clean comparison with the respondents' generation, we rerun regressions on *number of children* excluding those with no children, and show results in the figure. Selection effects are highly correlated across the two generations, and most share the same sign. Absolute effect size estimates are larger for the parents' generation. We treat this result cautiously, because when we split respondents up by year of birth, we find few differences in effect sizes between early- and late-born respondents, for either generation. In other words, since estimated effect sizes change across "generations," but do not change over time within either generation, the change may be due to remaining ascertainment bias within the sample or other effects. In particular, effect sizes in both generations may depend on polygenic scores' correlation with childlessness, and we cannot estimate this for the parents' generation.

Excluding childless people in the parents' generation could bias our estimates. To learn about this, we compare effect sizes excluding and including childless people in the *current* generation. The correlation between the two sets of effect sizes is 0.92. So, patterns across different scores are broadly similar whether the childless are counted or not. However, absolute effect sizes are smaller when the childless are excluded, for 26 out of 33 scores; the median percentage change is -51.49%.

The fact that childless people have such a strong effect on estimates makes it hard to compare total effect sizes across generations. In particular, since the parents' generation has a different distribution of numbers of children, childless people may have had more or less effect in that generation. Another issue is that we are estimating parents' polygenic scores by the scores of their children. This introduces noise into our independent variable, which might lead to errors-in-variables and bias coefficients towards zero.

As an alternative approach, we run regressions interacting polygenic scores with birth year, median split at 1950 ("early

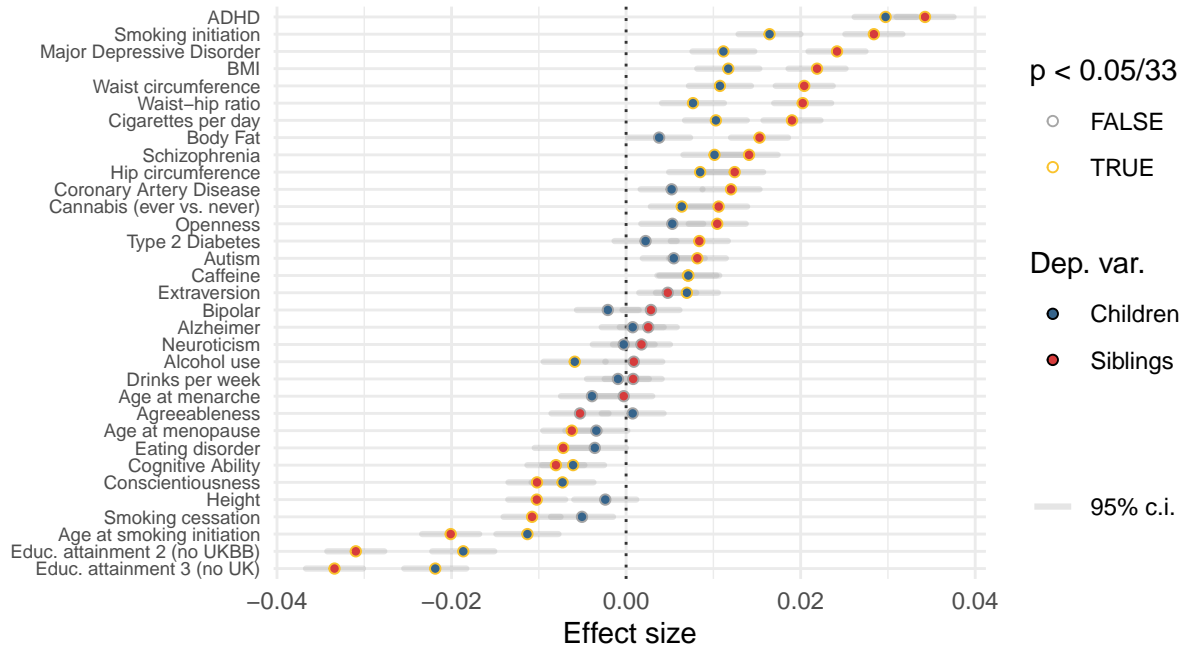


Figure 10: Selection effects, respondents’ parents vs. respondents. Age/qualification weights. Parental generation weights multiplied by 1/number of siblings. Respondents’ regression excludes childless respondents.

born” versus “late born”). We use both *number of children* and *number of siblings* as a dependent variable. We weight using age/qualification cells, and further adjust for selection in the parents’ generation (see above).

Tables 3 and 4 summarize the results. There is no evidence for changes in selection effects within the parents’ generation. In the respondents’ generation, effect sizes were significantly larger in absolute size among the later-born for four polygenic scores: cognitive ability, EA2, EA3 and extraversion. These changes are inconsistent with the intergenerational change, where estimated effect sizes were larger among the earlier, parents’ generation. One score, conscientiousness, showed a significant change in sign, from negative to positive effects on fertility.

Overall, there is weak evidence for change over time. The clearest results are that (a) the direction of selection, and (b) the pattern of relative effect sizes across scores, are broadly consistent over time.

Table 3: Change in selection effects between parents of early and late born respondents (regressions on number of siblings). Weights: Age/Qualification times 1/number of siblings.

Change	Number of scores
Insignificant	33
Significance is measured at $p < 0.05/66$	

Table 4: *Change in selection effects between early and late born respondents (regressions on number of children). Weights: Age/Qualification.*

Change	Number of scores
Change sign	1
Insignificant	28
Size increasing	4
Significance is measured at $p < 0.05/66$	

7.4.2 Area deprivation

Figure 11 plots effects on *number of siblings* by Townsend deprivation quintile of birth area.

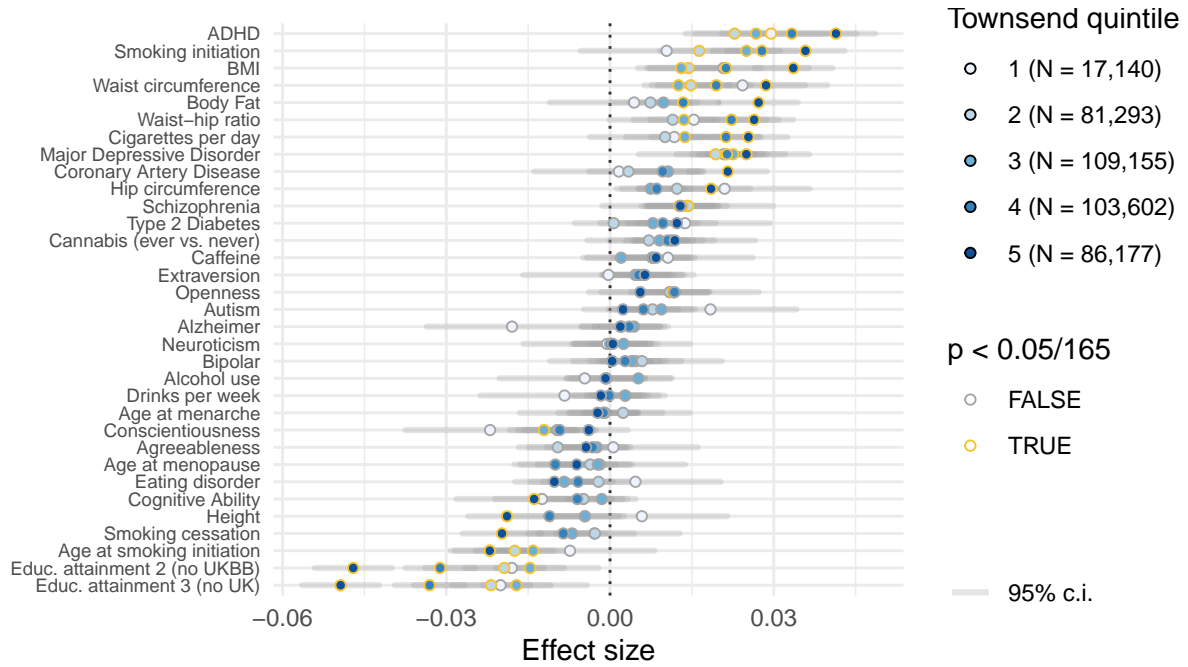
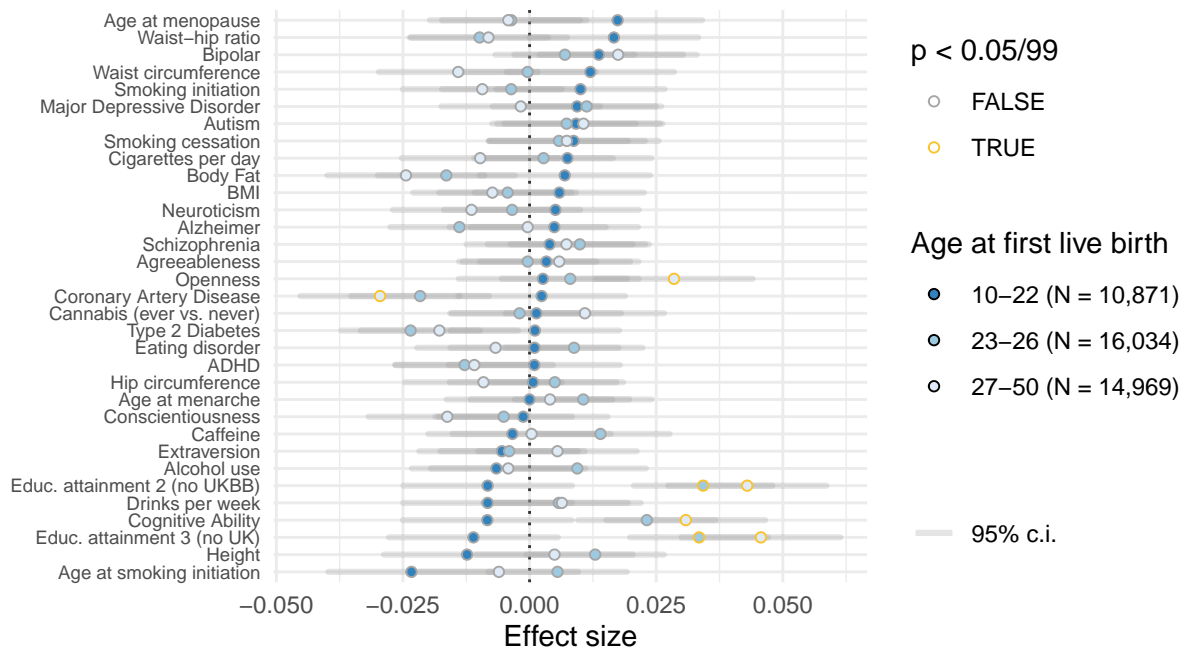


Figure 11: Selection effects in the parents' generation by Townsend deprivation quintile of birth area. Higher = more deprived. Estimates weighted by 1/number of siblings.

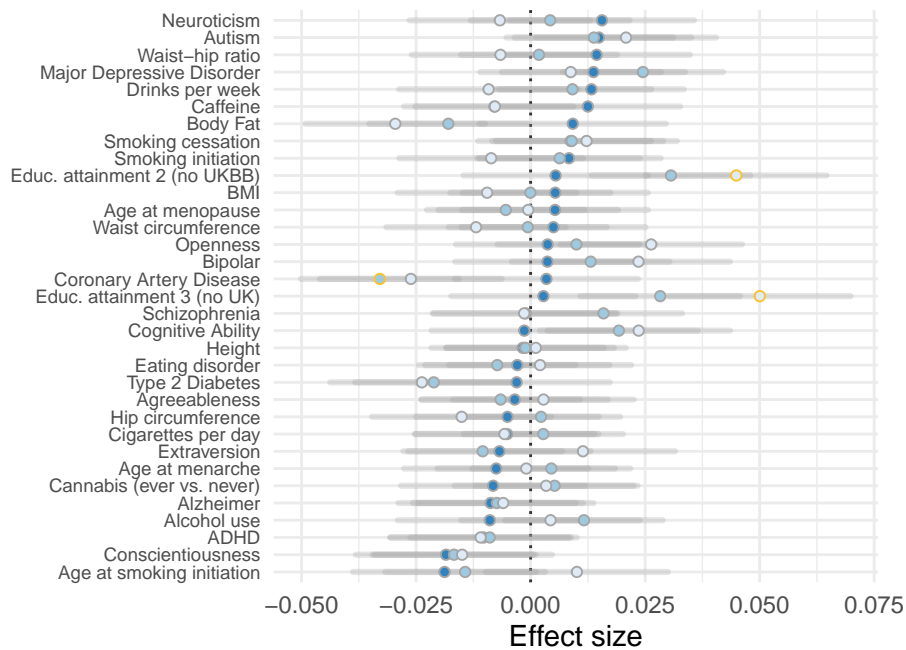
7.4.3 Age at first live birth

Among the parents' generation, we can control for age at first live birth using the subsets of respondents who reported their mother's or father's age, and who had no elder siblings. We run regressions on *number of siblings* on these subsets. Figure 12 shows selection effects by terciles of age at first live birth, for mothers and fathers. As in the respondents' generation, effect sizes are smaller, or even oppositely signed, for older parents. Importantly, this holds for both sexes.

Figure 13 shows the regressions controlling for either parent's age at their birth. Effect sizes are very similar, whether controlling for father's or mother's age. As in the respondents' generation, effect sizes are negatively correlated with the effect sizes from bivariate regressions without the age at birth control (father's age at birth: $\rho -0.42$; mother's age at birth: $\rho -0.59$).



(a) Mothers



(b) Fathers

Figure 12: Selection effects (parents' generation) by age at first live birth terciles. Estimates weighted by 1/number of siblings.

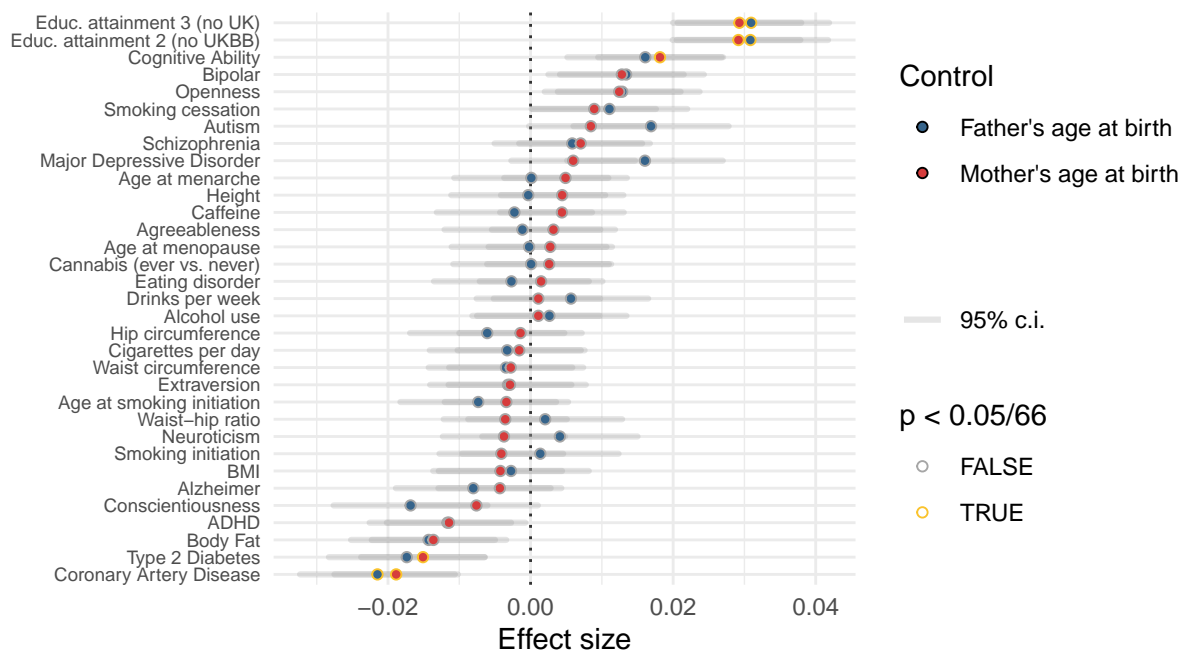


Figure 13: Selection effects (parents' generation) among eldest siblings, controlling for parents' age at birth. Estimates weighted by $1/\text{number of siblings}$.

7.5 Effects of polygenic scores on age at first live birth

Our results suggest that polygenic scores may directly correlate with age at first live birth. Figure 14 plots estimated effect sizes from bivariate regressions for respondents. Figure 15 does the same for their parents, using only eldest siblings.⁶ Effect sizes are reasonably large. They are also highly correlated across generations. Effect sizes of polygenic scores on father's age at own birth, and on own age at first live birth, have a correlation of 0.98; for mother's age and own age it is 0.98.

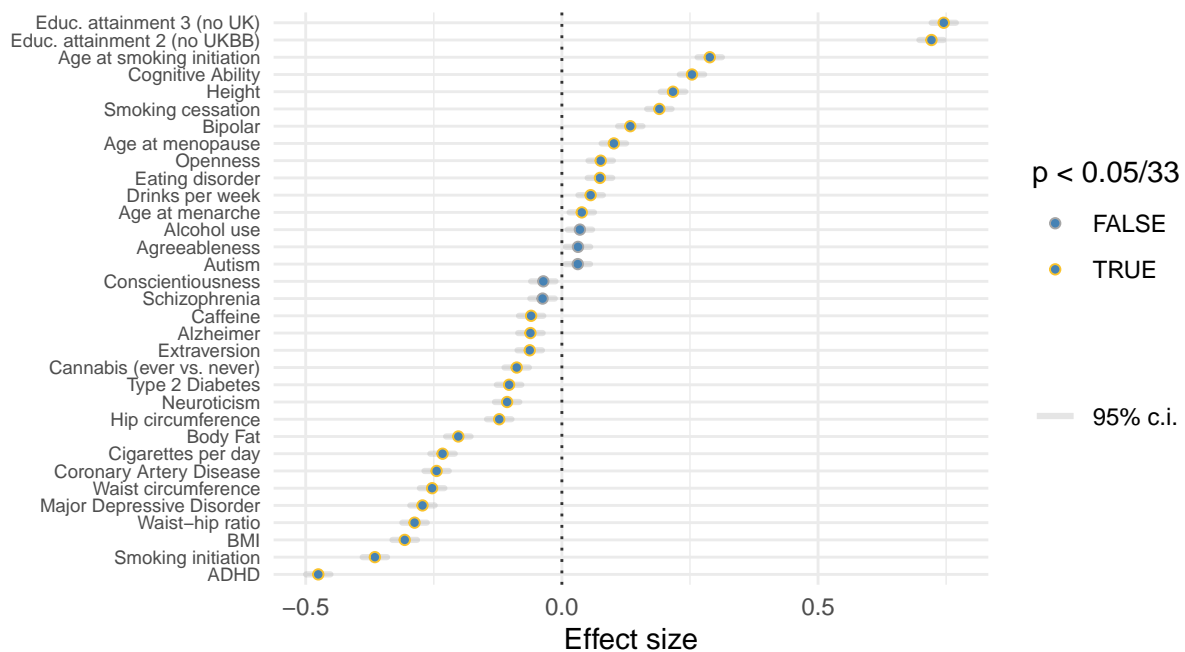


Figure 14: Effects of polygenic scores on age at first live birth.

⁶Parental AFLB can only be calculated for this group.

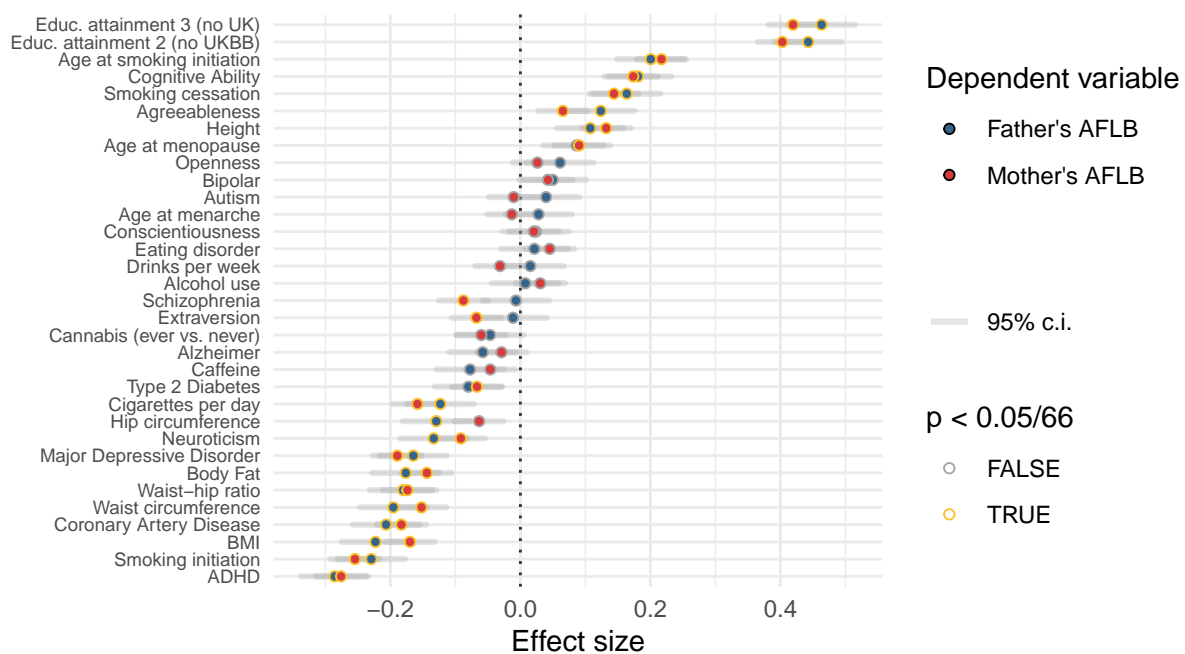


Figure 15: Effects of polygenic scores on parents' age at respondent's birth, eldest siblings. Estimates weighted by 1/number of siblings.

7.6 Mediation analysis

We run a standard mediation analysis in the framework of Baron and Kenny (1986). We estimate

$$N_i = \alpha + \beta PGS_i + \gamma EA_i + X_i\mu + \varepsilon_i \quad (3)$$

$$EA_i = \delta + \zeta PGS_i + X_i\mu + \eta_i \quad (4)$$

where N_i is fertility, PGS_i is each polygenic score, EA_i is educational attainment (age of leaving fulltime education), and X_i is a vector of controls. The total effect of PGS on number of children is $\beta + \gamma\zeta$. The “indirect effect” mediated by EA is $\gamma\zeta$. The standard error of the indirect effect can be calculated as

$$\sqrt{\hat{\gamma}^2 \hat{\sigma}_\zeta^2 + \hat{\zeta}^2 \hat{\sigma}_\gamma^2}$$

where $\hat{\sigma}_\zeta$ is the standard error of $\hat{\zeta}$, etc.

We are not concerned about endogeneity of polygenic scores, since our theory requires only that these correlate with human capital. However, endogeneity of educational attainment is a concern, i.e., educational attainment might correlate with unobserved characteristics that affect one’s fertility (and which are not related to human capital). To partially address this, we include controls for age, sex, fluid IQ, height and risk attitude in X .

Table 5 shows results. For 24 scores, the indirect effect on fertility via human capital is significantly greater than 0 at $p = 0.05/33$.

Table 5: *Mediation analysis*

PGS	Total effect	Indirect effect	95% c.i. Indirect/total		
ADHD	0.0389	0.0055	[0.0049, 0.0061]	14.1%	*
Smoking initiation	0.0290	0.0048	[0.0042, 0.0054]	16.4%	*
Extraversion	0.0268	−0.0001	[−0.0005, 0.0003]	−0.3%	
BMI	0.0256	0.0034	[0.0029, 0.0039]	13.3%	*
Waist circumference	0.0191	0.0042	[0.0037, 0.0047]	22.0%	*
Cannabis (ever vs. never)	0.0169	0.0007	[0.0003, 0.0011]	4.0%	*
Hip circumference	0.0147	0.0032	[0.0027, 0.0037]	21.6%	*
Major Depressive Disorder	0.0136	0.0018	[0.0014, 0.0022]	13.3%	*
Waist-hip ratio	0.0134	0.0036	[0.0031, 0.0041]	26.7%	*
Cigarettes per day	0.0115	0.0028	[0.0024, 0.0033]	24.6%	*
Body Fat	0.0086	0.0032	[0.0027, 0.0037]	37.4%	*
Caffeine	0.0060	0.0006	[0.0002, 0.0010]	10.1%	
Agreeableness	0.0057	−0.0009	[−0.0013, −0.0005]	−16.1%	*
Coronary Artery Disease	0.0039	0.0028	[0.0023, 0.0033]	71.7%	*
Age at menarche	0.0039	−0.0001	[−0.0005, 0.0003]	−2.5%	
Conscientiousness	0.0035	0.0001	[−0.0003, 0.0005]	4.1%	
Alzheimer	0.0030	0.0004	[0.0000, 0.0008]	14.3%	
Height	0.0021	0.0019	[0.0014, 0.0024]	92.9%	*
Type 2 Diabetes	0.0018	0.0012	[0.0008, 0.0017]	69.4%	*
Bipolar	0.0011	−0.0028	[−0.0032, −0.0023]	−258.8%	*
Drinks per week	−0.0000	−0.0007	[−0.0011, −0.0003]	1563.4%	*
Neuroticism	−0.0041	0.0010	[0.0006, 0.0014]	−24.7%	*
Alcohol use	−0.0041	0.0002	[−0.0002, 0.0006]	−3.9%	
Schizophrenia	−0.0043	−0.0024	[−0.0029, −0.0020]	55.9%	*
Openness	−0.0075	−0.0035	[−0.0040, −0.0030]	47.1%	*
Autism	−0.0102	−0.0012	[−0.0016, −0.0008]	12.1%	*
Smoking cessation	−0.0128	−0.0029	[−0.0033, −0.0024]	22.5%	*
Age at menopause	−0.0128	−0.0015	[−0.0019, −0.0011]	11.8%	*
Eating disorder	−0.0141	−0.0021	[−0.0026, −0.0017]	15.1%	*
Cognitive Ability	−0.0172	−0.0036	[−0.0041, −0.0031]	20.9%	*
Age at smoking initiation	−0.0188	−0.0036	[−0.0041, −0.0031]	18.9%	*
Educ. attainment 3 (no UK)	−0.0368	−0.0115	[−0.0127, −0.0104]	31.3%	*
Educ. attainment 2 (no UKBB)	−0.0391	−0.0114	[−0.0125, −0.0102]	29.1%	*

* indirect effect $p < 0.05/33$

7.7 Within-siblings regressions

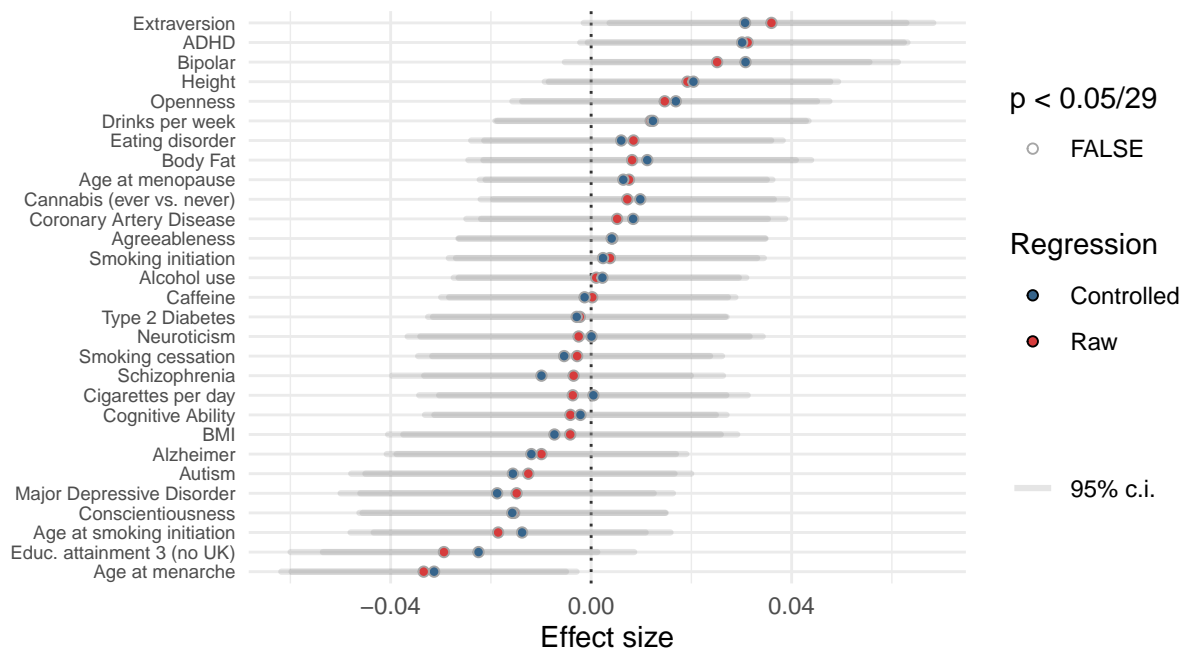


Figure 16: Selection effects controlling for sibling-group fixed effects, with and without a control for education (left education before 16, 16-18, or after 18). Each set of 29 results is from a single regression of n children on 29 polygenic scores. Standard errors clustered by sibling group.

We run a single regression on 29 polygenic scores within 17801 sibling groups ($N = 34030$). Thus, we control both for environmental confounds (since scores are randomly allocated within sib-groups by meiosis), and for genetic confounds captured by our polygenic scores. We remove four scores which correlate highly with other scores (educational attainment 2, hip circumference, waist circumference and waist-hip ratio). Figure 16 shows the results.

7.8 Effects on inequality

Table 6 shows correlations between children’s polygenic scores and household income (UKB data field 738). Column “With selection” uses respondents’ scores weighted by Age/Qualification, times number of children. Column “Without selection” uses scores weighted by Age/Qualification only, i.e. the counterfactual correlation if all respondents had the same number of children.

Table 6: *Correlations of polygenic scores with income group.*

PGS	Cor. with selection	Cor. without selection	Ratio
Educ. attainment 3 (no UK)	0.148	0.133	1.12
Educ. attainment 2 (no UKBB)	0.146	0.130	1.12
Cognitive Ability	0.059	0.054	1.08
Height	0.041	0.036	1.14
Smoking cessation	0.041	0.036	1.12
Age at smoking initiation	0.039	0.031	1.25
Openness	0.022	0.019	1.14
Bipolar	0.020	0.016	1.31
Agreeableness	0.017	0.016	1.07
Extraversion	0.016	0.015	1.13
Eating disorder	0.014	0.014	1.03
Drinks per week	0.013	0.012	1.10
Age at menopause	0.012	0.010	1.21
Age at menarche	0.008	0.004	1.94
Alcohol use	0.008	0.008	0.99
Autism	0.001	−0.003	−0.20
Conscientiousness	−0.001	−0.004	0.20
Caffeine	−0.006	−0.007	0.83
Alzheimer	−0.012	−0.011	1.04
Cannabis (ever vs. never)	−0.012	−0.010	1.26
Schizophrenia	−0.016	−0.018	0.88
Type 2 Diabetes	−0.020	−0.017	1.15
Hip circumference	−0.025	−0.020	1.26
Neuroticism	−0.028	−0.026	1.10
Cigarettes per day	−0.037	−0.029	1.25
Body Fat	−0.039	−0.035	1.12
Coronary Artery Disease	−0.040	−0.031	1.27
Major Depressive Disorder	−0.042	−0.036	1.17
Waist circumference	−0.045	−0.038	1.18
Waist-hip ratio	−0.049	−0.043	1.14
BMI	−0.051	−0.042	1.20
Smoking initiation	−0.058	−0.047	1.25
ADHD	−0.078	−0.066	1.19

7.9 Estimating the effect of natural selection on the contribution of PSEA to inequality

From the meta-analysis of twin studies in Branigan, McCallum, and Freese (2013), we take the heritability of educational attainment to be around 0.4. This is the variance of educational attainment explained by the “true” polygenic score for educational attainment p^* , i.e. the best linear unbiased predictor of educational attainment from genetic data. We write the measured polygenic score of educational attainment, “EA3” from Lee et al. (2018), as

$$p = p^* + \eta.$$

For realized educational attainment EA , write:

$$EA = \alpha + \beta p^* + \varepsilon = \alpha + \beta(p - \eta) + \varepsilon$$

We assume the classical errors-in-variables model, i.e. that η is independent of both p and ε . Under these conditions, the ratio between the estimated R^2 in a regression of p on EA , and the true R^2 of p^* on EA , satisfies:

$$\frac{\text{plim} \hat{R}^2}{R^2} = \frac{\sigma_p^2 - \sigma_\eta^2}{\sigma_p^2} = \frac{\sigma_{p^*}^2}{\sigma_p^2} \equiv \lambda, \quad (5)$$

as we show below. The right hand side is the “reliability ratio.” We regress educational attainment on EA3 in our sample, weighting by our Age/Qualification weights, to give $\text{plim} \hat{R}^2 = 0.0453$. Plugging this into the above, with $R^2 = h^2 \approx 0.4$, gives $\lambda = 0.113$.

We now consider the R^2 of p on income, that is, the proportion of variance in income that is explained by the true polygenic score of educational attainment. Applying (5) again gives that

$$\frac{\text{plim} \hat{R}_{\text{income}}^2}{R_{\text{income}}^2} = \lambda. \quad (6)$$

We compute $\hat{R}_{\text{income}}^2$ by regressing income group on EA3, first weighting by age/qualification, and then by age/qualification weights times respondent’s number of children. The estimated R^2 are 0.0176 and 0.022 respectively. Applying (6) to both estimates gives the proportion of variance in income explained by true polygenic score for education attainment (a) among respondents as 0.155; (b) accounting for natural selection as 0.194. The second figure gives the counterfactual estimate of R_{income}^2 after one generation of natural selection, holding environmental conditions unchanged.

The errors-in-variables conditions are quite restrictive. In particular, they rule out that the unmeasured part of PSEA

(η) correlates with the error term ε , e.g. with environmental causes of educational attainment. If unmeasured PSEA correlates positively with ε , then the reliability coefficient λ will be lower than calculated. However, the proportional effect of natural selection on the PSEA-income relationship will remain the same, so long as λ does not differ between the two sets of weighted regressions.

The estimate above uses a pooled regression of income on EA3, which may include confounding environmental variation such as genetic nurture. Within-siblings regression would give a smaller estimate of the effect of EA3. However, this would also affect the reliability of the regression (Griliches and Hausman 1986), making it hard to back out estimates of the true R^2 . Nevertheless, we can calculate the proportional increase in R^2 due to natural selection, again so long as the reliability coefficient is unaffected by the weighting scheme. Running within-siblings regressions of EA3 on income group gives within- R^2 estimates of 0.0014 (weighted by age and qualification) and 0.00339 (multiplying weights by number of children). Thus, this procedure estimates the proportional change in R_{income}^2 due to natural selection as 142.6%.

7.9.1 Proof of (5)

Write

$$\hat{R}^2 = 1 - \frac{\sum_{i=1}^N (\hat{\alpha} + \hat{\beta}p_i - EA_i)^2}{\sum_{i=1}^N (EA_i - \bar{EA})^2} \quad (7)$$

Using the fact that $\hat{\alpha} = \bar{EA} - \hat{\beta}\bar{p}$, where bars signify sample averages, we can simplify the numerator in the above:

$$\sum_{i=1}^N (\hat{\alpha} + \hat{\beta}p_i - EA_i)^2 = \sum_{i=1}^N (\bar{EA} - \hat{\beta}\bar{p} + \hat{\beta}p_i - EA_i)^2 = \sum_{i=1}^N (\hat{\beta}(p_i - \bar{p}) - (EA_i - \bar{EA}))^2$$

Now, we can compute the following probability limit:

$$\begin{aligned} & \text{plim} \frac{\sum_{i=1}^N (\hat{\beta}(p_i - \bar{p}) - (EA_i - \bar{EA}))^2}{N} \\ &= \text{plim} \frac{\sum_{i=1}^N \hat{\beta}^2(p_i - \bar{p})^2 + (EA_i - \bar{EA})^2 - 2\hat{\beta}(p_i - \bar{p})(EA_i - \bar{EA})}{N} \\ &= \beta^2 \lambda^2 \sigma_p^2 + \sigma_{EA}^2 - 2\beta \lambda \sigma_{p,EA}, \end{aligned} \quad (8)$$

using the standard errors-in-variables formula $\text{plim} \hat{\beta} = \lambda\beta$, and where $\sigma_{p,EA}$ is the covariance between p and EA .

Write

$$\begin{aligned}
\sigma_{p,EA} &= \text{Cov}(p^* + \eta, \alpha + \beta p^* + \varepsilon) \\
&= 0 + \beta \sigma_{p^*}^2 + \beta \text{Cov}(\eta, p^*) + \text{Cov}(\eta, \varepsilon) \\
&= \beta \sigma_{p^*}^2
\end{aligned}$$

using the errors-in-variables assumption that η is independent of p^* and ε . Plug this into (8) to give

$$\begin{aligned}
&\text{plim} \frac{\sum_{i=1}^N (\hat{\beta}(p_i - \bar{p}) - (EA_i - \bar{EA}))^2}{N} \\
&= \beta^2 \lambda^2 \sigma_p^2 + \sigma_{EA}^2 - 2\beta^2 \lambda \sigma_{p^*}^2 \\
&= \beta^2 \lambda \sigma_{p^*}^2 + \sigma_{EA}^2 - 2\beta^2 \lambda \sigma_{p^*}^2, \text{ using } \sigma_{p^*}^2 = \lambda \sigma_p^2, \\
&= \sigma_{EA}^2 - \beta^2 \lambda \sigma_{p^*}^2
\end{aligned}$$

Inserting this into (7) gives

$$\begin{aligned}
\text{plim} \hat{R}^2 &= 1 - \frac{\sigma_{EA}^2}{\sigma_{EA}^2} + \frac{\beta^2 \lambda \sigma_{p^*}^2}{\sigma_{EA}^2} \\
&= \lambda \frac{\beta^2 \sigma_{p^*}^2}{\sigma_{EA}^2} \\
&= \lambda R^2
\end{aligned}$$

since for bivariate regression R^2 is the square of the correlation between EA and p^* , which can be written $r = \beta \frac{\sigma_{p^*}^2}{\sigma_{EA} \sigma_{p^*}}$.

We thank an anonymous commenter on <https://stats.stackexchange.com/questions/238878/how-do-errors-in-variables-affect-the-r2> for much of this proof.

7.10 Further results

7.10.1 Selection effects on raw polygenic scores

Figure 17 compares selection effects on polygenic scores residualized for the top 100 principal components of the genetic data, to selection effects on raw, unresidualized polygenic scores. In siblings regressions, effect sizes are larger for raw scores – sometimes much larger, as in the case of height. 30 out of 33 “raw” effect sizes have a larger absolute value than the corresponding “residualized” effect size. The median proportion between raw and controlled effect sizes is 0.76. Among the children regressions, this no longer holds. Effect sizes are barely affected by controlling for principal components.

Overall, 78.79 per cent of effect sizes are consistently signed across all four regressions (on children and siblings, and with and without residualization).

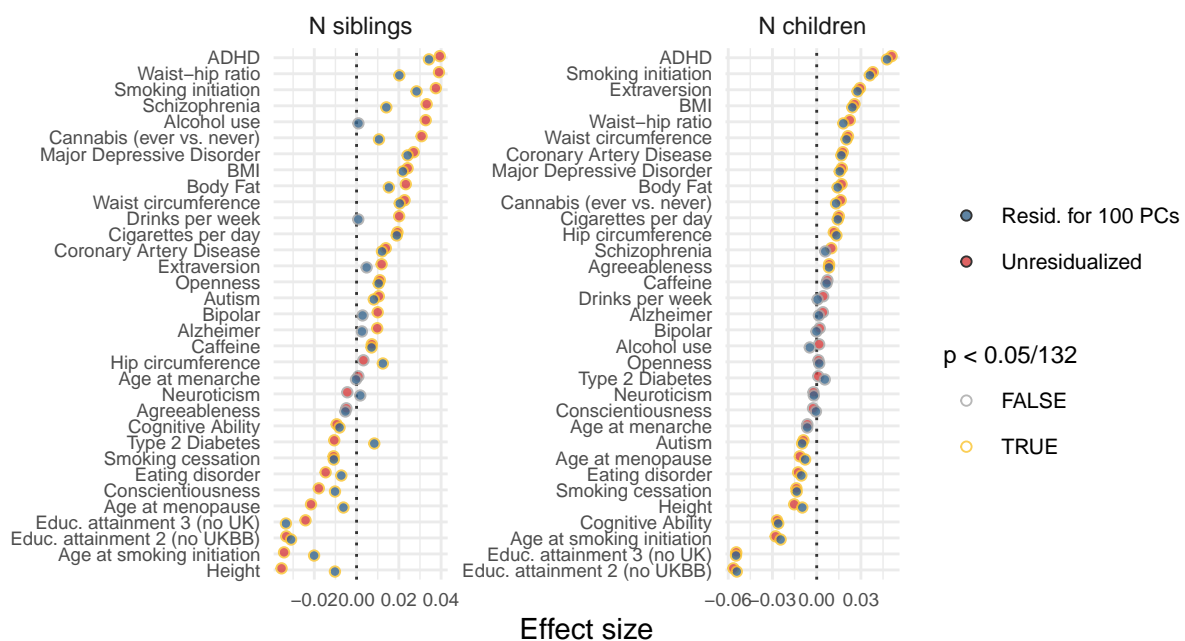


Figure 17: Selection effects using unresidualized polygenic scores on number of siblings/children. Sibling estimates weighted by 1/number of siblings.

To get a further insight into this we regress n siblings and n children on individual principal components. Figure 18 shows the results. Labels show the top principal components. These have larger effect sizes in siblings regressions. One possibility is that the parents’ generation was less geographically mobile, and so geographic patterns of childrearing were more correlated with principal components, which partly capture the location of people’s ancestors.

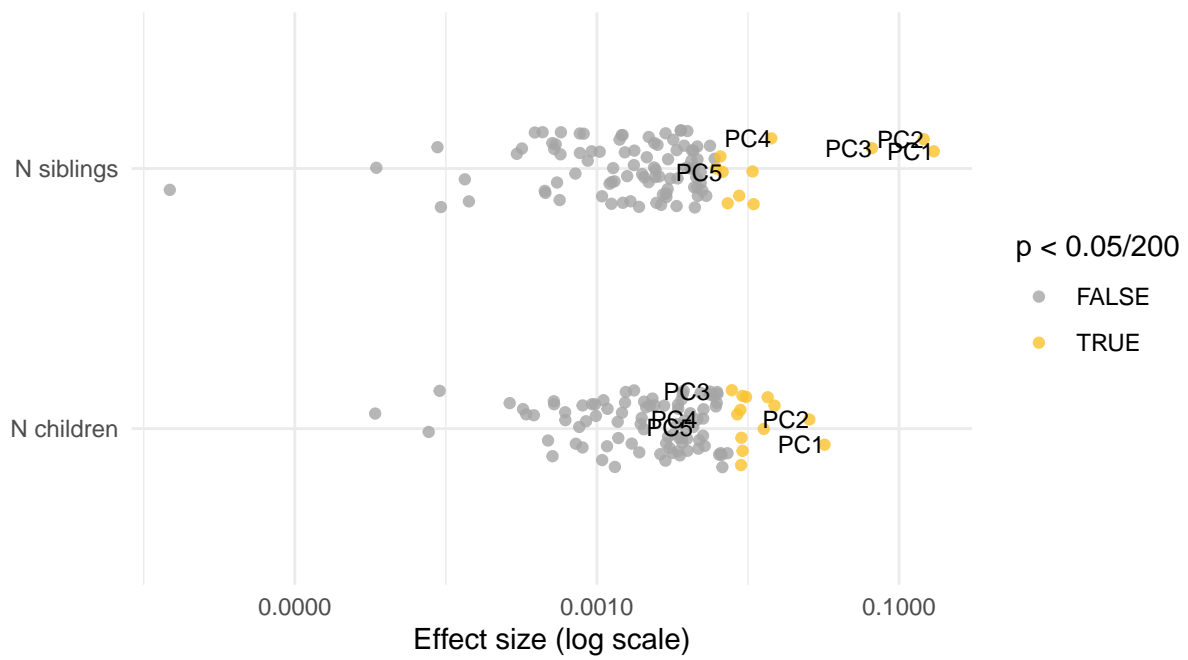


Figure 18: Selection effects of 100 principal components of genetic data. Each dot represents one bivariate regression. Sibling estimates weighted by $1/\text{number of siblings}$. Absolute effect sizes are plotted. Points are jittered on the Y axis. Top principal components are labelled.

7.10.2 Genetic correlations with EA3

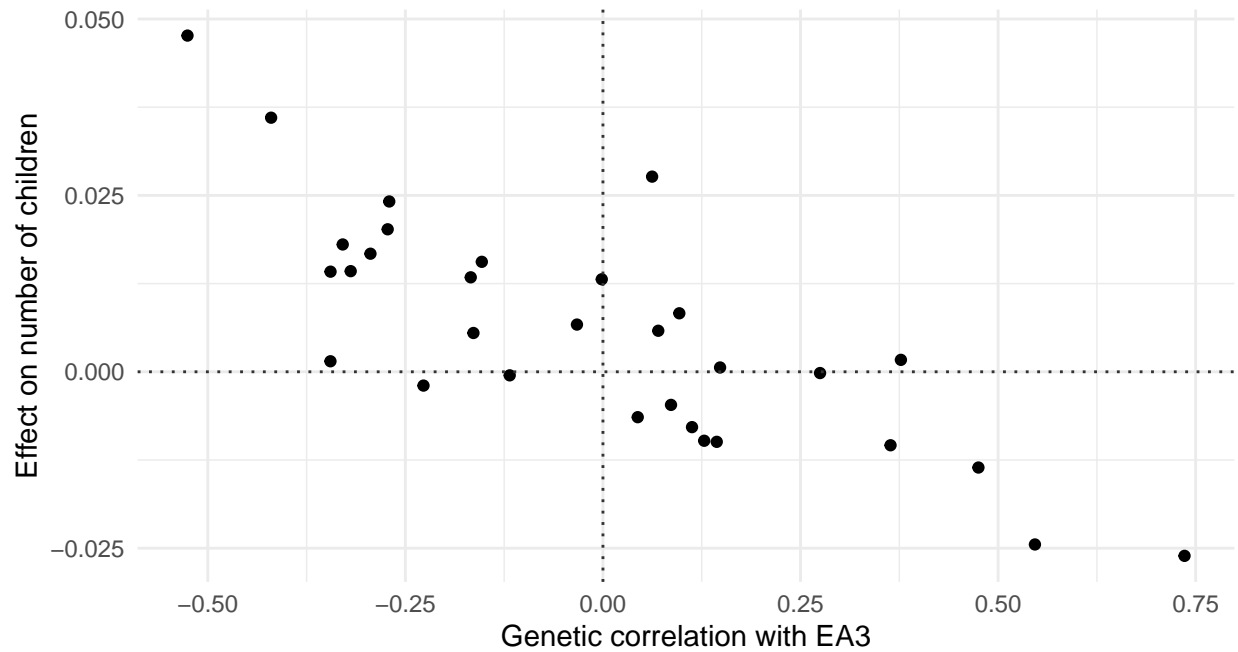


Figure 19: Selection effects plotted against genetic correlation with EA3.

Another way to examine the “earnings” theory of natural selection is to compare selection effects of polygenic scores with their genetic correlation with educational attainment (EA3). Since EA3 strongly predicts earnings, if earnings drives differences in fertility, we’d expect a correlation between the two sets of results. Figure 19 shows this is so: the correlation, after excluding EA2, is -0.8 . Genetic correlations were calculated using LD score regression from GWAS summary statistics.

7.1.1 Model proofs

Solution for the one-period model

Differentiating and setting $\frac{dU}{dN} = 0$ gives the first order condition for an optimal choice of children $N^* > 0$:

$$\frac{bW}{(W(1 - bN^*))^\sigma} \geq a, \text{ with equality if } N^* > 0.$$

Rearranging gives

$$N^* = \max \left\{ \frac{1}{b} \left(1 - \left(\frac{b}{a} \right)^{1/\sigma} W^{(1-\sigma)/\sigma} \right), 0 \right\}. \quad (9)$$

Note that when $\sigma < 1$, for high enough W , $N^* = 0$. Differentiating gives the effect of wages on fertility for $N^* > 0$.

This is also the fertility-human capital relationship:

$$\frac{dN^*}{dW} = \frac{dN^*}{dW} = -\frac{1}{b} \left(\frac{b}{a} \right)^{1/\sigma} \frac{1-\sigma}{\sigma} W^{(1-2\sigma)/\sigma}. \quad (10)$$

This is negative if $\sigma < 1$. Also,

$$\frac{d^2 N^*}{dW^2} = -\frac{1}{b} \left(\frac{b}{a} \right)^{1/\sigma} \frac{1-\sigma}{\sigma} \frac{1-2\sigma}{\sigma} W^{(1-3\sigma)/\sigma}$$

For $0.5 < \sigma < 1$, this is positive, so the effect of fertility on wages shrinks towards zero as wages increase (and becomes 0 when $N^* = 0$). Next, we consider the time cost of children b :

$$\frac{d^2 N^*}{dWdb} = -\left(\frac{1}{a} \right)^{1/\sigma} \left(\frac{1-\sigma}{\sigma} \right)^2 (Wb)^{(1-2\sigma)/\sigma} < 0.$$

Lastly we consider the effect of a . From (9), N^* is increasing in a . Differentiating (10) by a gives

$$\frac{d^2 N^*}{dadW} = b^{1/\sigma-1} \frac{1-\sigma}{\sigma^2} W^{(1-2\sigma)/\sigma} a^{-1/\sigma-1}$$

which is positive for $\sigma < 1$.

Solution for the two-period model

Period 1 and period 2 income are:

$$Y_1 = 1 - s - bN_1 \quad (11)$$

$$Y_2 = w(s, h)(1 - bN_2) \quad (12)$$

Write the Lagrangian of utility U (2) as

$$\mathcal{L}(N_1, N_2, s) = u(Y_1) + u(Y_2) + a(N_1 + N_2) + \lambda_1 N_1 + \lambda_2 N_2 + \lambda_3 \left(\frac{1}{b} - N_2\right) + \mu s$$

Lemma 5 below shows that if $\sigma > 0.5$, this problem is globally concave, guaranteeing that the first order conditions identify a unique solution. We assume $\sigma > 0.5$ from here on.

Plugging (11) and (12) into the above, we can derive the Karush-Kuhn-Tucker conditions for an optimum (N_1^*, N_2^*, s^*) as:

$$\frac{d\mathcal{L}}{dN_1} = -bY_1^{-\sigma} + a + \lambda_1 = 0, \text{ with } \lambda_1 = 0 \text{ if } N_1^* > 0; \quad (13)$$

$$\frac{d\mathcal{L}}{dN_2} = -bs^*hY_2^{-\sigma} + a + \lambda_2 - \lambda_3 = 0, \text{ with } \lambda_2 = 0 \text{ if } N_2^* > 0, \lambda_3 = 0 \text{ if } N_2^* < \frac{1}{b}; \quad (14)$$

$$\frac{d\mathcal{L}}{ds} = -Y_1^{-\sigma} + h(1 - bN_2^*)Y_2^{-\sigma} + \mu = 0; \quad (15)$$

$$N_1^*, N_2^*, s^*, \lambda_1, \lambda_2, \lambda_3, \mu \geq 0; N_2^* \leq \frac{1}{b}. \quad (16)$$

Note that the Inada condition ($\lim_{x \rightarrow 0} u'(x) = \infty$) for period 1 rules out $s^* = 1$ and $N_1 = 1/b$, so we need not impose these constraints explicitly. Also, so long as $N_2^* < 1/b$, the same condition rules out $s^* = 0$. We consider four cases, of which only three can occur.

Case 1: $N_1^* > 0, N_2^* > 0$

Rearranging (13), (14) and (15) gives:

$$N_1^* = \frac{1}{b} \left(1 - s^* - \left(\frac{b}{a} \right)^{1/\sigma} \right); \quad (17)$$

$$N_2^* = \frac{1}{b} \left(1 - \left(\frac{b}{a} \right)^{1/\sigma} (s^* h)^{(1-\sigma)/\sigma} \right); \quad (18)$$

$$s^* = \frac{1 - bN_1^*}{1 + ((1 - bN_2^*)h)^{1-1/\sigma}}. \quad (19)$$

Plugging the expressions for N_1^* and N_2^* into s^* gives

$$s^* = \frac{s^* + \left(\frac{b}{a} \right)^{1/\sigma}}{1 + \left(\left(\frac{b}{a} \right)^{1/\sigma} s^{*(1-\sigma)/\sigma} h^{1/\sigma} \right)^{1-1/\sigma}}$$

which simplifies to

$$s^* = \left(\frac{b}{a} \right)^{1/(2\sigma-1)} h^{(1-\sigma)/(2\sigma-1)}. \quad (20)$$

Plugging the above into (17) and (18) gives:

$$N_1^* = \frac{1}{b} \left(1 - \left(\frac{b}{a} \right)^{1/(2\sigma-1)} h^{(1-\sigma)/(2\sigma-1)} - \left(\frac{b}{a} \right)^{1/\sigma} \right);$$

$$N_2^* = \frac{1}{b} \left(1 - \left(\frac{b}{a} \right)^{1/(2\sigma-1)} h^{(1-\sigma)/(2\sigma-1)} \right).$$

Note that that $N_1^* < N_2^*$. For these both to be positive requires low values of h if $\sigma < 1$ and high values of h if $\sigma > 1$.

Also:

$$w(s^*, h) \equiv s^* h = \left(\frac{b}{a} \right)^{1/(2\sigma-1)} h^{\sigma/(2\sigma-1)}.$$

Observe that $w(s^*, h)$ is increasing in h for $\sigma > 0.5$, and convex iff $0.5 < \sigma < 1$.

While N_1^* and N_2^* are positive, they have the same derivative with respect to h :

$$\frac{dN_t^*}{dh} = -\frac{1}{b} \left(\frac{b}{a} \right)^{1/(2\sigma-1)} \frac{1-\sigma}{2\sigma-1} h^{(1-\sigma)/(2\sigma-1)-1} \quad (21)$$

Examining this and expression (20) gives:

Lemma 1. For $\sigma < 1$, case 1 holds for h low enough, and in case 1, N_1^* and N_2^* decrease in h , while s^* increases in h .

For $\sigma > 1$, case 1 holds for h high enough, and in case 1 N_1^* and N_2^* increase in h , while s^* decreases in h .

N_t^* is convex in h for $\sigma > 2/3$, and concave otherwise. s^* is convex in h if $\sigma < 2/3$, and concave otherwise.

Case 2: $N_1^* = 0, N_2^* > 0$

Replace $N_1^* = 0$ into the first order condition for s^* from (15), and rearrange to give:

$$s^* = \frac{1}{1 + ((1 - bN_2)h)^{1-1/\sigma}}.$$

Now since $N_2^* > 0$, we can rearrange (14) to give

$$N_2^* = \frac{1}{b} \left(1 - \left(\frac{b}{a} \right)^{1/\sigma} (s^*h)^{(1-\sigma)/\sigma} \right). \quad (22)$$

Plugging this into s^* gives

$$s^* = \frac{1}{1 + \left(\frac{bh}{a} \right)^{(\sigma-1)/\sigma^2} (s^*)^{-(1-\sigma)^2/\sigma^2}}$$

which can be rearranged to

$$(1 - s^*)(s^*)^{(1-2\sigma)/\sigma^2} = \left(\frac{a}{bh} \right)^{(1-\sigma)/\sigma^2}. \quad (23)$$

Differentiate the left hand side of the above to get

$$\begin{aligned} & \frac{1-2\sigma}{\sigma^2} (1 - s^*)(s^*)^{(1-2\sigma)/\sigma^2-1} - (s^*)^{(1-2\sigma)/\sigma^2} \\ &= \frac{1-2\sigma}{\sigma^2} (s^*)^{(1-2\sigma)/\sigma^2-1} - \frac{\sigma^2 + 1 - 2\sigma}{\sigma^2} (s^*)^{(1-2\sigma)/\sigma^2} \\ &= \frac{1-2\sigma}{\sigma^2} (s^*)^{(1-2\sigma)/\sigma^2-1} - \frac{(1-\sigma)^2}{\sigma^2} (s^*)^{(1-2\sigma)/\sigma^2}. \end{aligned} \quad (24)$$

This is negative if and only if

$$s^* > \frac{1-2\sigma}{(1-\sigma)^2}$$

which is always true since $\sigma > 0.5$. Note also that since $\sigma > 0.5$, then the left hand side of (23) approaches infinity as $s^* \rightarrow 0$ and approaches 0 as $s^* \rightarrow 1$. Thus, (23) implicitly defines the unique solution for s^* .

To find how s^* changes with h , note that the right hand side of the above decreases in h for $\sigma < 1$, and increases in h for $\sigma > 1$. Putting these facts together: for $\sigma < 1$, when h increases the RHS of (23) decreases, hence the LHS decreases

and s^* increases, i.e. s^* is increasing in h . For $\sigma > 1$, s^* is decreasing in h .

To find how N_2^* changes with h , we differentiate (22):

$$\frac{dN_2^*}{dh} = -\frac{1}{b} \left(\frac{b}{a}\right)^{1/\sigma} \frac{1-\sigma}{\sigma} (s^*h)^{(1-2\sigma)/\sigma} \left(s^* + h \frac{ds^*}{dh}\right) \quad (25)$$

which is negative for $\sigma < 1$, since $\frac{ds^*}{dh} > 0$ in this case.

Differentiating again:

$$\begin{aligned} \frac{d^2N_2}{dh^2} &= -X \left[\frac{1-2\sigma}{\sigma} (s^*h)^{(1-3\sigma)/\sigma} \left(s^* + h \frac{ds^*}{dh}\right)^2 + (s^*h)^{(1-2\sigma)/\sigma} \left(2 \frac{ds^*}{dh} + h \frac{d^2s^*}{dh^2}\right) \right] \\ &= X (s^*h)^{(1-3\sigma)/\sigma} \left[\frac{2\sigma-1}{\sigma} \left(s^* + h \frac{ds^*}{dh}\right)^2 - (s^*h) \left(2 \frac{ds^*}{dh} + h \frac{d^2s^*}{dh^2}\right) \right] \end{aligned}$$

where $X = \frac{1}{b} \left(\frac{b}{a}\right)^{1/\sigma} \frac{1-\sigma}{\sigma} > 0$. Note that $\frac{d^2N_2}{dh^2}$ is continuous in σ around $\sigma = 1$. Note also from (23) that for $\sigma = 1$, s^* becomes constant in σ . The term in square brackets then reduces to $(s^*)^2 > 0$. Putting these facts together, for σ sufficiently close to 1, $\frac{d^2N_2^*}{dh^2} > 0$, i.e. N_2^* is convex in h .

Conditions (13) and (14) give the bounds for this case. When $\sigma < 1$, (13) puts a minimum on h and (14) puts a maximum on h . The situation is reversed for $\sigma > 1$.

Summarizing:

Lemma 2. *Case 2 holds for intermediate values of h . In case 2: for $\sigma < 1$, s^* is increasing in h and N_2^* is decreasing in h . For $\sigma > 1$, s^* is decreasing in h . For σ close enough to 1, N_2^* is convex in h .*

Case 3: $N_1^* = 0, N_2^* = 0$

We can solve for s^* by substituting values of Y_1 and Y_2 into (15):

$$-(1-s^*)^{-\sigma} + h(s^*h)^{-\sigma} = 0$$

which rearranges to

$$s^* = \frac{1}{1 + h^{(\sigma-1)/\sigma}}. \quad (26)$$

Conditions (13) and (14) become:

$$-b(1 - s^*)^{-\sigma} + a \leq 0$$

$$-bs^*h(s^*h)^{-\sigma} + a \leq 0$$

equivalently

$$\frac{a}{b} \leq (1 - s^*)^{-\sigma}$$

$$\frac{a}{b} \leq s^*h(s^*h)^{-\sigma}$$

which can both be satisfied for a/b close enough to zero. Note from (26) that as $h \rightarrow \infty$, s^* increases towards 1 for $\sigma < 1$, and decreases towards 0 for $\sigma > 1$. Note also that the right hand side of the first inequality above approaches infinity as $s^* \rightarrow 1$, therefore also as $h \rightarrow \infty$ for $\sigma < 1$. Rewrite the second inequality as

$$\frac{a}{b} < (s^*h)^{1-\sigma} = \left(\frac{h}{1 + h^{(\sigma-1)/\sigma}} \right)^{1-\sigma} = (h^{-1} + h^{-1/\sigma})^{\sigma-1}$$

and note that again, as $h \rightarrow \infty$, the RHS increases towards infinity for $\sigma < 1$, and decreases towards zero otherwise. Thus, for $\sigma < 1$, both equations will be satisfied for h high enough. For $\sigma > 1$, they will be satisfied for h low enough. Summarizing

Lemma 3. *For $\sigma < 1$, case 3 holds for h high enough, and in case 3, s^* increases in h . For $\sigma > 1$, case 3 holds for h low enough and s^* decreases in h .*

Case 4: $N_1^* > 0, N_2^* = 0$

Rearranging the first order conditions (13) and (14) for N_1^* and N_2^* gives

$$\frac{a}{b} = (1 - s^* - bN_1^*)^{-\sigma}$$

$$\frac{a}{b} \leq s^*hY_2^{-\sigma}$$

hence

$$\begin{aligned}
(1 - s^* - bN_1^*)^{-\sigma} &\leq s^* h Y_2^{-\sigma} = (s^* h)^{1-\sigma} \\
\Leftrightarrow (1 - s^* - bN_1^*)^\sigma &\geq (s^* h)^{\sigma-1} \\
\Leftrightarrow 1 - s^* - bN_1^* &\geq (s^* h)^{1-1/\sigma}
\end{aligned}$$

Now rearrange the first order condition for s^* from (15), noting that since $N_2^* = 0$, $s^* > 0$ by the Inada condition.

$$\begin{aligned}
h^{1/\sigma-1}(1 - s^* - bN_1^*) &= s^* \\
1 - s^* - bN_1^* &= s^* h^{1-1/\sigma}
\end{aligned}$$

This, combined with the previous inequality, implies

$$\begin{aligned}
(s^* h)^{1-1/\sigma} &\leq s^* h^{1-1/\sigma} \\
\Leftrightarrow (s^*)^{-1/\sigma} &\leq 1
\end{aligned}$$

which cannot hold since $0 < s^* < 1$.

Comparative statics

We can now examine how the fertility-human capital relationship

$$\frac{dN^*}{dh}, \text{ where } N^* \equiv N_1^* + N_2^*,$$

changes with respect to other parameters. We focus on the case $\sigma < 1$, since it gives the closest match to our observations, and since it also generates “reasonable” predictions in other areas, e.g. that education levels increase with human capital. Figure 20 shows how N^* changes with h for $a = 0.4, b = 0.25, \sigma = 0.7$.

Lemma 4. *For $\sigma < 1$ in a neighbourhood of 1, N^* is globally convex in h .*

Proof. From Lemmas 1, 2 and 3, as h increases we move from $N_1^*, N_2^* > 0$ to $N_1^* = 0, N_2^* > 0$ to $N_1^* = N_2^* = 0$. Furthermore, for $\sigma > 2/3$, N_1^* and N_2^* are convex in h when they are both positive, and for σ close enough to 1, N_2^* is convex in h when $N_1^* = 0$. All that remains is to check that the derivative is increasing around the points where these 3 regions meet. That is trivially satisfied where N_2^* becomes 0, since thereafter $\frac{dN^*}{dh}$ is zero. The derivative as N_1^*

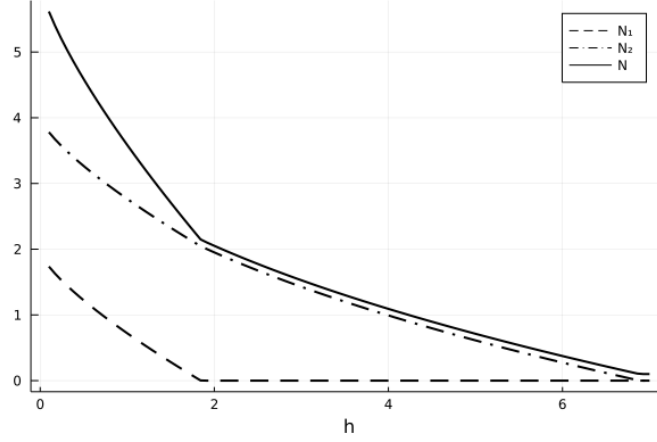


Figure 20: Fertility vs. human capital in the two-period model with $a = 0.4, b = 0.25, \sigma = 0.7$.

approaches zero is twice the expression in (21):

$$-\frac{2}{b} \left(\frac{b}{a}\right)^{1/(2\sigma-1)} \frac{1-\sigma}{2\sigma-1} h^{(1-\sigma)/(2\sigma-1)-1} \quad (27)$$

and the derivative to the right of this point is given by (25):

$$-\frac{1}{b} \left(\frac{b}{a}\right)^{1/\sigma} \frac{1-\sigma}{\sigma} (s^*h)^{(1-2\sigma)/\sigma} (s^* + h \frac{ds^*}{dh}) \quad (28)$$

We want to prove that the former is larger in magnitude (i.e. more negative). Dividing (27) by (28) gives

$$2 \frac{\sigma}{2\sigma-1} \left(\frac{b}{a}\right)^{(1-\sigma)/(\sigma(2\sigma-1))} \frac{h^{(1-\sigma)^2/(\sigma(2\sigma-1))}}{s^*(s^* + h \frac{ds^*}{dh})}$$

Examining (23) shows that as $\sigma \rightarrow 1$, $s^* \rightarrow 0.5$ and $\frac{ds^*}{dh} \rightarrow 0$, and therefore the above approaches

$$2 \frac{1}{(0.5)^2} = 8.$$

□

We can now gather the theoretical predictions stated in Table 1.

Prediction 1: for $\sigma < 1$, total fertility $N^* \equiv N_1^* + N_2^*$ is decreasing in human capital h .

Furthermore, for σ close enough to 1, fertility is convex in human capital, i.e.

Prediction 2 part 1: the fertility-human capital relationship is closer to 0 at high levels of h .

For $\sigma < 1$, education levels s^* increase in h , and so therefore do equilibrium wages $w(s^*, h)$. This, plus fact 1, gives:

Prediction 2 part 2: for $\sigma < 1$ and close to 1, the fertility-human capital relationship is weaker among higher earners.

Prediction 4: for $\sigma < 1$ and close to 1, the fertility-human capital relationship is weaker at high levels of education.

Next, we compare people who start fertility early ($N_1^* > 0$) versus those who start fertility late ($N_1^* = 0$). Again, for $\sigma < 1$ the former group have lower h than the latter group. Thus we have:

Prediction 5: for $\sigma < 1$ and close to 1, the fertility-human capital relationship is weaker among those who start fertility late.

Lastly, we prove prediction 3. Differentiating dN_t^*/dh in (21) with respect to b , for when $N_1^* > 0$ gives:

$$\frac{d^2 N_t^*}{dhdb} = \frac{2\sigma - 2}{2\sigma - 1} b^{(3-4\sigma)/(2\sigma-1)} \left(\frac{1}{a}\right)^{1/(2\sigma-1)} \frac{1-\sigma}{2\sigma-1} h^{(\sigma-1)^2/(\sigma(2\sigma-1))}$$

which is negative for $0.5 < \sigma < 1$. When $N_1^* = 0$, differentiating dN_2^*/dh in (25) gives:

$$\frac{d^2 N_2^*}{dhdb} = -\frac{1-\sigma}{\sigma} b^{(1-2\sigma)/\sigma} \left(\frac{1}{a}\right)^{1/\sigma} \frac{1-\sigma}{\sigma} (s^* h)^{(1-2\sigma)/\sigma} (s^* + h \frac{ds^*}{dh})$$

which again is negative for $\sigma < 1$. Therefore:

Prediction 3: for $\sigma < 1$, the fertility-human capital relationship is more negative when the burden of childcare b is larger.

Including a money cost

The model can be extended by adding a money cost m per child. Utility is then

$$U = u(1 - s - bN_1 - mN_1) + u(w(s, h)(1 - bN_2) - mN_2) + a(N_1 + N_2)$$

Figure 21 shows a computed example with $a = 0.4, b = 0.175, \sigma = 0.7, m = 0.075$. Fertility first declines steeply with human capital, then rises. In addition, for parents with low AFLB ($N_1 > 0$), the fertility-human capital relationship is negative, while for parents with higher AFLB ($N_1 = 0$) it is positive.

Concavity

Lemma 5. For $\sigma > 0.5$, U in equation (2) is concave in N_1, N_2 and s .

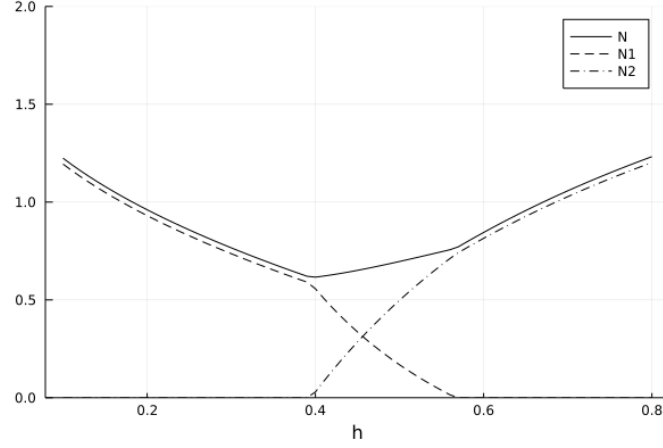


Figure 21: Fertility vs. human capital in the two-period model with money costs of children. $a = 0.4, b = 0.175, \sigma = 0.7, m = 0.075$.

Proof. We examine the Hessian matrix of utility in each period. Note that period 1 utility is constant in N_2 and period 2 utility is constant in N_1 . For period 1 the Hessian with respect to N_1 and s is:

$$\begin{bmatrix} d^2u/dN_1^2 & d^2u/dsdN_1 \\ d^2u/dsdN_1 & d^2u/ds^2 \end{bmatrix} = \begin{bmatrix} -\sigma b^2 & -\sigma b \\ -\sigma b & -\sigma \end{bmatrix} Y_1^{-\sigma-1}$$

with determinant

$$(\sigma^2 b^2 - \sigma^2 b^2) Y_1^{-2\sigma-2} = 0.$$

Thus, first period utility is weakly concave. For period 2 with respect to N_2 and s , the Hessian is:

$$\begin{bmatrix} d^2u/dN_2^2 & d^2u/dsdN_2 \\ d^2u/dsdN_2 & d^2u/ds^2dN_2 \end{bmatrix} = \begin{bmatrix} -\sigma(bsh)^2 Y_2^{-\sigma-1} & -(1-\sigma)bhY_2^{-\sigma} \\ -(1-\sigma)bhY_2^{-\sigma} & -\sigma[h(1-bN_2^*)]^2 Y_2^{-\sigma-1} \end{bmatrix}$$

with determinant

$$\begin{aligned} & (-\sigma(bsh)^2 Y_2^{-\sigma-1})(-\sigma[h(1-bN_2^*)]^2 Y_2^{-\sigma-1}) - (-(1-\sigma)bhY_2^{-\sigma})^2 \\ &= \sigma^2(bsh)^2[h(1-bN_2^*)]^2 Y_2^{-2\sigma-2} - (1-\sigma)^2(bh)^2 Y_2^{-2\sigma} \\ &= \sigma^2(bh)^2 Y_2^{-2\sigma} - (1-\sigma)^2(bh)^2 Y_2^{-2\sigma}, \text{ using that } Y_2 = (sh)(1-bN) \\ &= (bh)^2 Y_2^{-2\sigma}(\sigma^2 - (1-\sigma)^2) \end{aligned}$$

which is positive if and only if $\sigma > 0.5$. Thus, if $\sigma > 0.5$ then the Hessian is negative definite and thus utility is concave; this combined with weak concavity of period 1, and linearity of $a(N_1 + N_2)$, shows that (2) is concave. \square

Effect of a

Lemma 6. For $\sigma < 1$, $d^2 N^*/dadh > 0$, i.e. the effect of a increases at higher levels of human capital.

Proof. Differentiating (21) with respect to a gives

$$\frac{d^2 N_t^*}{dadh} = -\frac{1}{b} \frac{1}{1-2\sigma} a^{-1} \left(\frac{a}{b}\right)^{1/(1-2\sigma)} \frac{1-\sigma}{2\sigma-1} h^{(1-\sigma)/(2\sigma-1)-1}$$

for $t = 1, 2$ when $N_1^*, N_2^* > 0$. For $\sigma > 0.5$ this is positive.

When $N_1^* = 0, N_2^* > 0$, differentiating (25) with respect to a gives

$$\frac{d^2 N_2^*}{dadh} = \frac{1}{b} \frac{1}{\sigma} a^{-1} \left(\frac{a}{b}\right)^{-1/\sigma} \frac{1-\sigma}{\sigma} (s^* h)^{(1-2\sigma)/\sigma} \left(s^* + h \frac{ds^*}{dh}\right)$$

which is $-a^{-1}/\sigma$ times (25) and hence is positive.

□

References

- Barban, Nicola, and Rick Jansen, Ronald de Vlaming, Ahmad Vaez, Jornt J Mandemakers, Felix C Tropf, Xia Shen, et al. 2016. "Genome-Wide Analysis Identifies 12 Loci Influencing Human Reproductive Behavior." *Nature Genetics* 48 (12): 1462–72. <https://doi.org/10.1038/ng.3698>.
- Baron, Reuben M, and David A Kenny. 1986. "The Moderator–Mediator Variable Distinction in Social Psychological Research: Conceptual, Strategic, and Statistical Considerations." *Journal of Personality and Social Psychology* 51 (6): 1173.
- Baudin, Thomas, David De La Croix, and Paula E Gobbi. 2015. "Fertility and Childlessness in the United States." *American Economic Review* 105 (6): 1852–82.
- Beauchamp, Jonathan P. 2016. "Genetic Evidence for Natural Selection in Humans in the Contemporary United States." *Proceedings of the National Academy of Sciences* 113 (28): 7774–79.
- Becker, Gary S. 1960. "An Economic Analysis of Fertility." *National Bureau Committee for Economic Research* 209.
- . 1964. "Human Capital."
- . 1981. "A Treatise on the Family." *NBER Books*.
- Becker, Gary S, and Nigel Tomes. 1976. "Child Endowments and the Quantity and Quality of Children." *Journal of Political Economy* 84 (4, Part 2): S143–62.
- Belsky, Daniel W, Benjamin W Domingue, Robbee Wedow, Louise Arseneault, Jason D Boardman, Avshalom Caspi, Dalton Conley, et al. 2018. "Genetic Analysis of Social-Class Mobility in Five Longitudinal Studies." *Proceedings of the National Academy of Sciences* 115 (31): E7275–84.
- Benton, Mary Lauren, Abin Abraham, Abigail L. LaBella, Patrick Abbot, Antonis Rokas, and John A. Capra. 2021. "The Influence of Evolutionary History on Human Health and Disease." *Nature Reviews Genetics*.
- Branigan, Amelia R, Kenneth J McCallum, and Jeremy Freese. 2013. "Variation in the Heritability of Educational Attainment: An International Meta-Analysis." *Social Forces* 92 (1): 109–40.
- Bycroft, Clare, Colin Freeman, Desislava Petkova, Gavin Band, Lloyd T Elliott, Kevin Sharp, Allan Motyer, et al. 2018. "The UK Biobank Resource with Deep Phenotyping and Genomic Data." *Nature* 562 (7726): 203–9.
- Caucutt, Elizabeth M, Nezih Guner, and John Knowles. 2002. "Why Do Women Wait? Matching, Wage Inequality, and the Incentives for Fertility Delay." *Review of Economic Dynamics* 5 (4): 815–55.
- Cohen, Alma, Rajeev Dehejia, and Dmitri Romanov. 2013. "Financial Incentives and Fertility." *Review of Economics and Statistics* 95 (1): 1–20.
- Conley, Dalton, Thomas Laidley, Daniel W Belsky, Jason M Fletcher, Jason D Boardman, and Benjamin W Domingue. 2016. "Assortative Mating and Differential Fertility by Phenotype and Genotype Across the 20th Century." *Proceedings of the National Academy of Sciences* 113 (24): 6647–52.

- Davey Smith, George, and Shah Ebrahim. 2003. “‘Mendelian Randomization’: Can Genetic Epidemiology Contribute to Understanding Environmental Determinants of Disease?” *International Journal of Epidemiology* 32 (1): 1–22.
- Fieder, Martin, and Susanne Huber. 2022. “Contemporary Selection Pressures in Modern Societies? Which Factors Best Explain Variance in Human Reproduction and Mating?” *Evolution and Human Behavior* 43 (1): 16–25. <https://doi.org/https://doi.org/10.1016/j.evolhumbehav.2021.08.001>.
- Flynn, James R. 1987. “Massive IQ Gains in 14 Nations: What IQ Tests Really Measure.” *Psychological Bulletin* 101 (2): 171.
- Fry, Anna, Thomas J Littlejohns, Cathie Sudlow, Nicola Doherty, Ligia Adamska, Tim Sprosen, Rory Collins, and Naomi E Allen. 2017. “Comparison of Sociodemographic and Health-Related Characteristics of UK Biobank Participants With Those of the General Population.” *American Journal of Epidemiology* 186 (9): 1026–34. <https://doi.org/10.1093/aje/kwx246>.
- Gauthier, Anne H. 2007. “The Impact of Family Policies on Fertility in Industrialized Countries: A Review of the Literature.” *Population Research and Policy Review* 26 (3): 323–46.
- Griliches, Zvi, and Jerry A Hausman. 1986. “Errors in Variables in Panel Data.” *Journal of Econometrics* 31 (1): 93–118.
- Harden, Kathryn Paige. 2021. *The Genetic Lottery: Why DNA Matters for Social Equality*. Princeton University Press.
- Hotz, V Joseph, Jacob Alex Klerman, and Robert J Willis. 1997. “The Economics of Fertility in Developed Countries.” *Handbook of Population and Family Economics* 1 (Part A): 275–347.
- Howe, Laurence J, Michel G Nivard, Tim T Morris, Ailin F Hansen, Humaira Rasheed, Yoonsu Cho, Geetha Chittoor, et al. 2021. “Within-Sibship GWAS Improve Estimates of Direct Genetic Effects.” *bioRxiv*. <https://doi.org/10.1101/2021.03.05.433935>.
- HRS. 2020. https://hrs.isr.umich.edu/data-products/genetic-data/products?_ga=2.248005447.1729788711.1602771602-1571093872.1602771602.
- Jones, Larry E, Alice Schoonbroodt, and Michele Tertilt. 2008. “Fertility Theories: Can They Explain the Negative Fertility-Income Relationship?”
- Jones, Larry E, and Michèle Tertilt. 2006. “An Economic History of Fertility in the US: 1826-1960.” *NBER Working Paper*, no. w12796.
- Kong, Augustine, Michael L Frigge, Gudmar Thorleifsson, Hreinn Stefansson, Alexander I Young, Florian Zink, Gudrun A Jonsdottir, et al. 2017. “Selection Against Variants in the Genome Associated with Educational Attainment.” *Proceedings of the National Academy of Sciences* 114 (5): E727–32.
- Kong, Augustine, Gudmar Thorleifsson, Michael L Frigge, Bjarni J Vilhjalmsen, Alexander I Young, Thorgeir E Thorgeirsson, Stefania Benonisdottir, et al. 2018. “The Nature of Nurture: Effects of Parental Genotypes.” *Science* 359 (6374): 424–28.
- Lee, James J, Robbee Wedow, Aysu Okbay, Edward Kong, Omeed Maghzian, Meghan Zacher, Tuan Anh Nguyen-Viet,

- et al. 2018. “Gene Discovery and Polygenic Prediction from a Genome-Wide Association Study of Educational Attainment in 1.1 Million Individuals.” *Nature Genetics* 50 (8): 1112–21.
- Lumley, Thomas. 2020. “Survey: Analysis of Complex Survey Samples.”
- Lundberg, Shelly, and Robert A Pollak. 2007. “The American Family and Family Economics.” *Journal of Economic Perspectives* 21 (2): 3–26.
- Mills, Melinda C, Felix C Tropf, David M Brazel, Natalie van Zuydam, Ahmad Vaez, Tune H Pers, Harold Snieder, et al. 2021. “Identification of 371 Genetic Variants for Age at First Sex and Birth Linked to Externalising Behaviour.” *Nature Human Behavior*.
- Mincer, Jacob. 1958. “Investment in Human Capital and Personal Income Distribution.” *Journal of Political Economy* 66 (4): 281–302.
- Monstad, Karin, Carol Propper, and Kjell G Salvanes. 2008. “Education and Fertility: Evidence from a Natural Experiment.” *Scandinavian Journal of Economics* 110 (4): 827–52.
- Rimfeld, Kaili, Eva Krapohl, Maciej Trzaskowski, Jonathan R. I. Coleman, Saskia Selzam, Philip S. Dale, Tonu Esko, Andres Metspalu, and Robert Plomin. 2018. “Genetic Influence on Social Outcomes During and After the Soviet Era in Estonia.” *Nature Human Behaviour* 2 (4): 269–75. <https://doi.org/10.1038/s41562-018-0332-5>.
- Sanjak, Jaleal S, Julia Sidorenko, Matthew R Robinson, Kevin R Thornton, and Peter M Visscher. 2018. “Evidence of Directional and Stabilizing Selection in Contemporary Humans.” *Proceedings of the National Academy of Sciences* 115 (1): 151–56.
- Sarraf, Matthew Alexandar, Colin Feltham, et al. 2019. *Modernity and Cultural Decline: A Biobehavioral Perspective*. Springer Nature.
- Selzam, Saskia, Stuart J Ritchie, Jean-Baptiste Pingault, Chandra A Reynolds, Paul F O’Reilly, and Robert Plomin. 2019. “Comparing Within-and Between-Family Polygenic Score Prediction.” *The American Journal of Human Genetics* 105 (2): 351–63.
- Social Security Committee. 1999. “Fourth Report.”
- Townsend, Peter. 1987. “Deprivation.” *Journal of Social Policy* 16 (2): 125–46.
- Willis, Robert J. 1973. “A New Approach to the Economic Theory of Fertility Behavior.” *Journal of Political Economy* 81 (2, Part 2): S14–64.