Labor Market Income and Substitution Effects Explain Modern Natural Selection

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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 on 33 polygenic scores over two generations, using data from UK Biobank. Consistently over time, polygenic scores which predict lower (higher) earnings and education are selected for (against). Selection effects are concentrated among low-income and less educated people, younger parents, people with more lifetime sexual partners, and people not living with a partner. Effect sizes are substantially larger after correcting for ascertainment bias. The direction of natural selection is reversed among older parents, or after controlling for age at first live birth. These patterns can be explained by economic theories of fertility, in which higher earnings may either increase or decrease fertility via income and substitution effects in the labour market.

Living organisms evolve through natural selection, in which allele frequencies change in the population through differential reproduction rates. Geneticists have long hypothesized that natural selection is taking place in modern human populations. Recent work confirms this using genome-wide analysis (Barban et al. 2016; Beauchamp 2016; Kong et al. 2017; Sanjak et al. 2018). 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 mechanisms behind these effects. This study uses data from UK Biobank to learn more. 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. Most importantly, we examine reproductive rates in different subgroups of the population, in order to uncover patterns that can help illuminate the mechanisms behind modern natural selection.

We find selection effects on many polygenic scores. Effects are consistent across generations. The strength of natural selection on a polygenic score is associated with that score's correlation with education and earnings: scores that predict lower education and earnings are being selected for. Also, across the board, polygenic scores have stronger relationships with fertility among specific subgroups. Selection effects are stronger among people with lower income and less education, younger parents, people not living with a partner, and people with many lifetime sexual partners. Outside these groups, effects are weaker and often statistically insignificant. In fact, in some subgroups, the direction of selection is actually reversed: polygenic scores predicting higher education and earnings are associated with *higher* fertility.

These patterns can be explained by economic theories of fertility (Becker 1960). In these, 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). Our results suggest that the substitution effect dominates for single parents, while the income effect is stronger for couples.

Results

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 ascertainment bias within the sample. Respondents are higher income and better educated than the UK population, and they may also differ on other unobserved characteristics (Fry et al. 2017). Since richer and educated people also live longer, this bias might also increase with age.

To test directly for natural selection, we regress respondents'polygenic scores on their number of children (y_i):

$$y_i = \alpha + \beta \text{PGS}_i + \varepsilon_i \tag{1}$$

The "selection effect", β, measures 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] To learn more about the underlying mechanisms, we split the sample, starting with basic demographic variables including education, income and sex. These are all potential sources of ascertainment bias: as well as the ascertainment for income and education, mentioned above, the sample sex ratio skews 54.05% female.

Figure 2 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. Note that the overall effect is not a simple average of the effect among the different

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

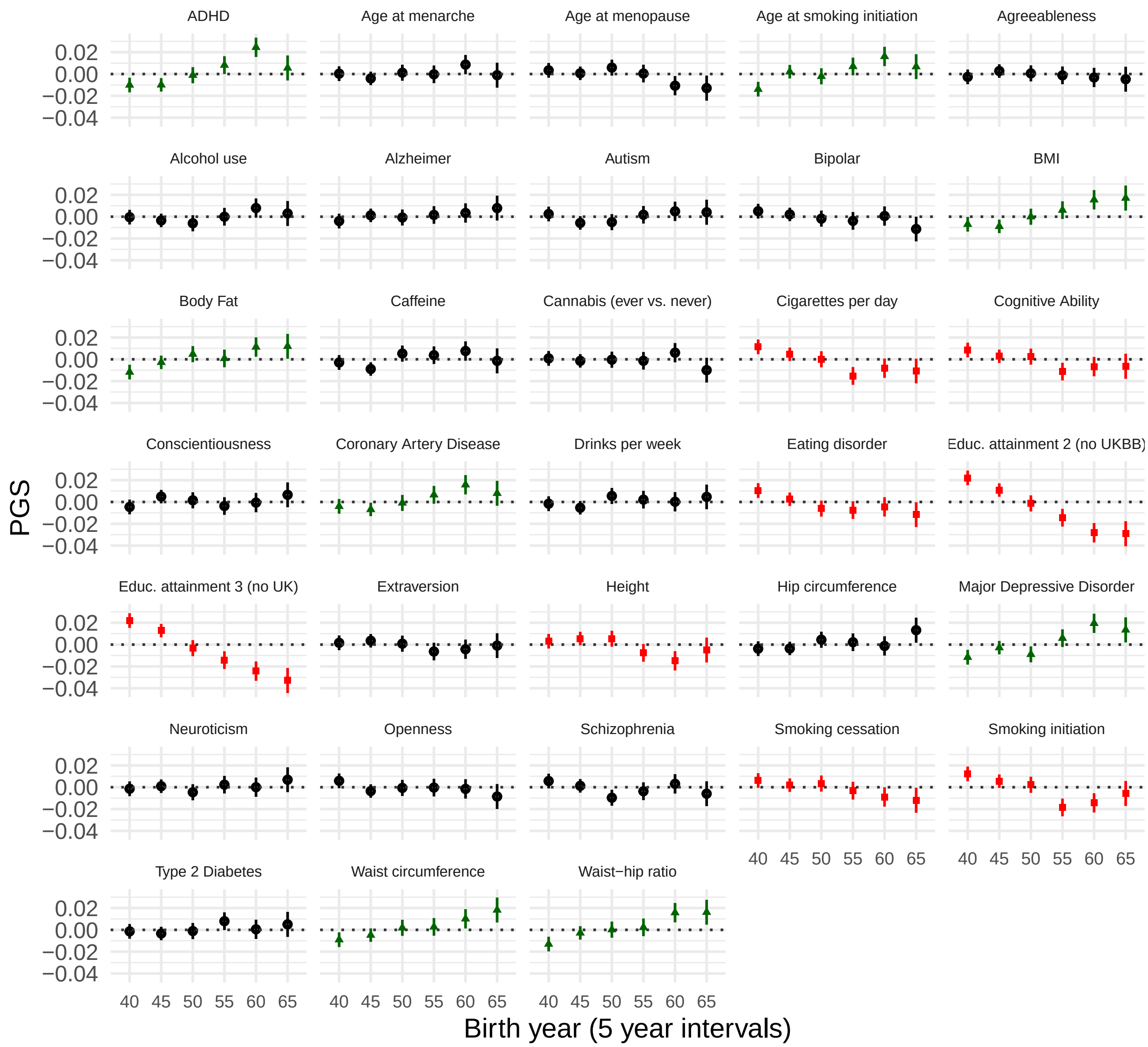


Figure 1: Mean polygenic scores by birth year in UK Biobank. Points are means for 5-year intervals. Lines are 95% confidence intervals. Green triangles show a significant linear increase over time ($p < 0.05/33$). Red squares show a significant decrease.

subgroups, because polygenic scores may also shift respondents between the subgroups. For example, a high PGS for educational attainment may predict having fewer children among early school leavers, but it may also increase the age at which a respondent leaves school.

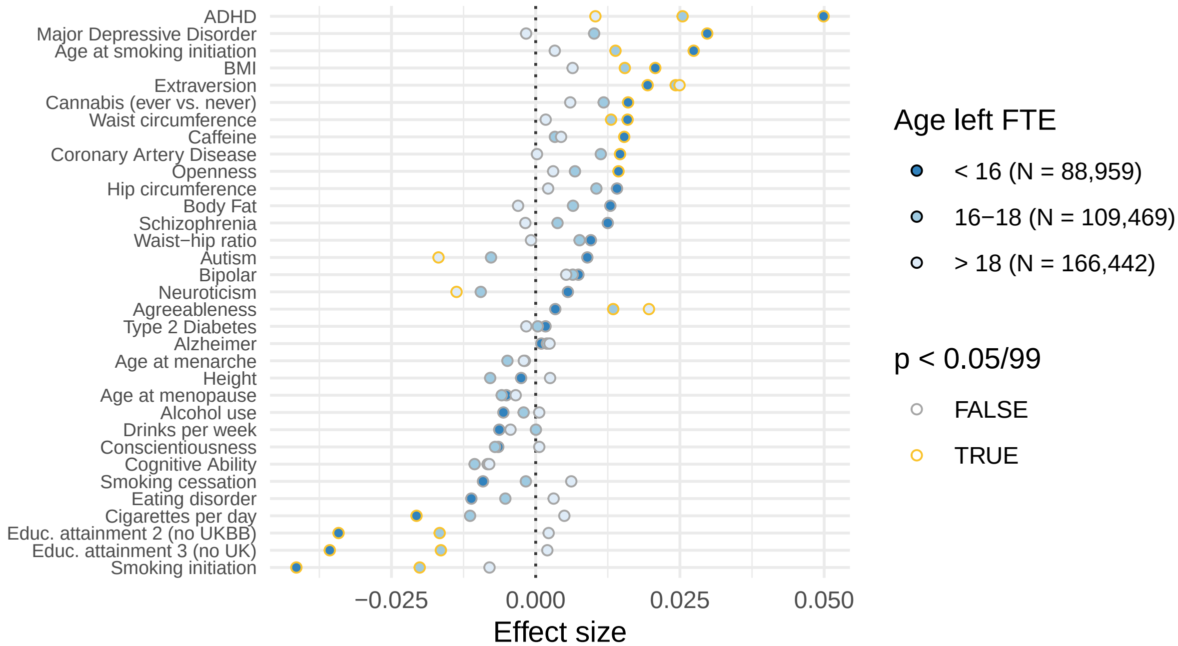
These results could be driven by age, if older respondents are poorer and less educated, and also more subject to selection on polygenic scores. However, if we rerun the regressions, interacting the polygenic score with income category and also with a quadratic in age, the interaction with income remains significant at 0.05/33 for 16 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 FTE remains significant at 0.05/33 for 12 out of 33 regressions.

Selection effects are also different between men and women (Appendix Figure 7). Differences are particularly large for educational attainment, height and MDD. Several PGS 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. PGS for waist circumference and waist-hip ratio are less selected for among women, and PGS for educational attainment are more selected against, though the difference is only significant for EA3. 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.

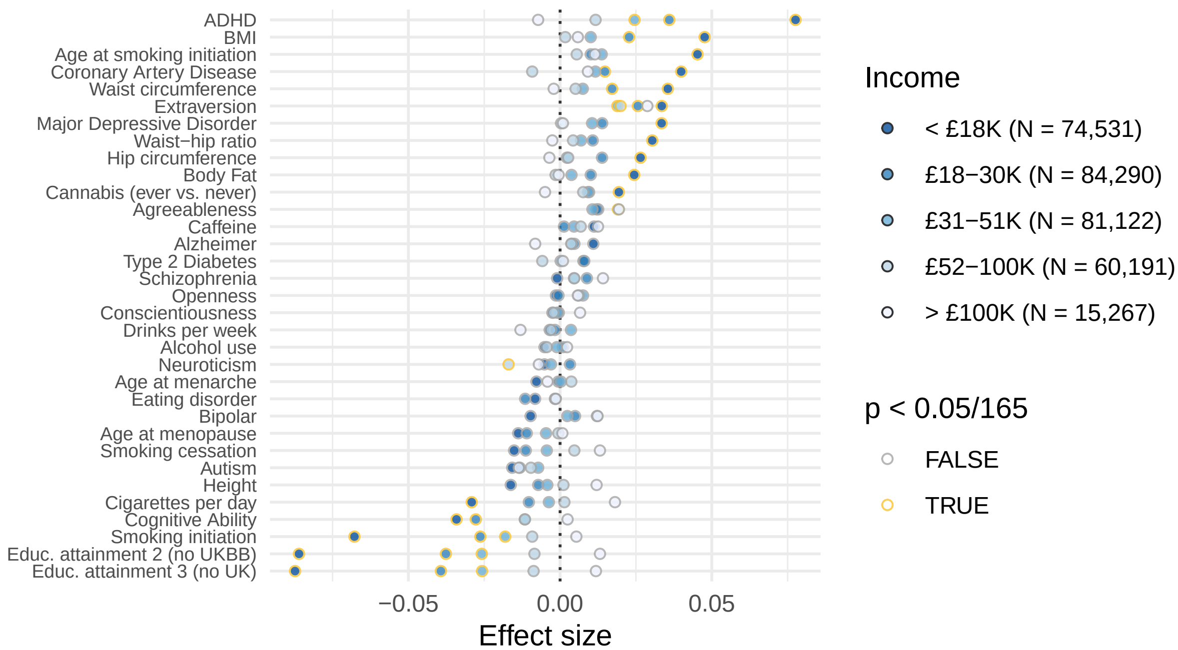
We next focus on variables related to household type and reproductive strategy. We split males and females by lifetime number of sexual partners, at the median value of 3 (Figure 3a). For both sexes, selection effects are larger and more significant among those with more than 3 partners. Next we split respondents by whether they were living with a spouse or partner at the time of interview. Effects are larger among those not living with a spouse or partner (Figure 3b). Lastly, we split female respondents by age at first live birth (AFLB).[2] 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 3c 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 PGS of body measurements are selected against only among older mothers. The correlation between effect sizes for the youngest and oldest terciles is -0.46. As before, recall that PGS may have two effects: changing the number of children within each category, and changing the number of children by shifting respondents between categories.

To investigate this further, we regress number of children on PGS *controlling* for AFLB (appendix Figure 8). In 24 out of 33 cases, effects change sign when controls are added. The correlation between effect sizes controlling for AFLB, and raw effect sizes, is -0.78. Thus, selection effects seem to come through two opposing channels: an effect on AFLB,

[2]This information is unavailable for men.

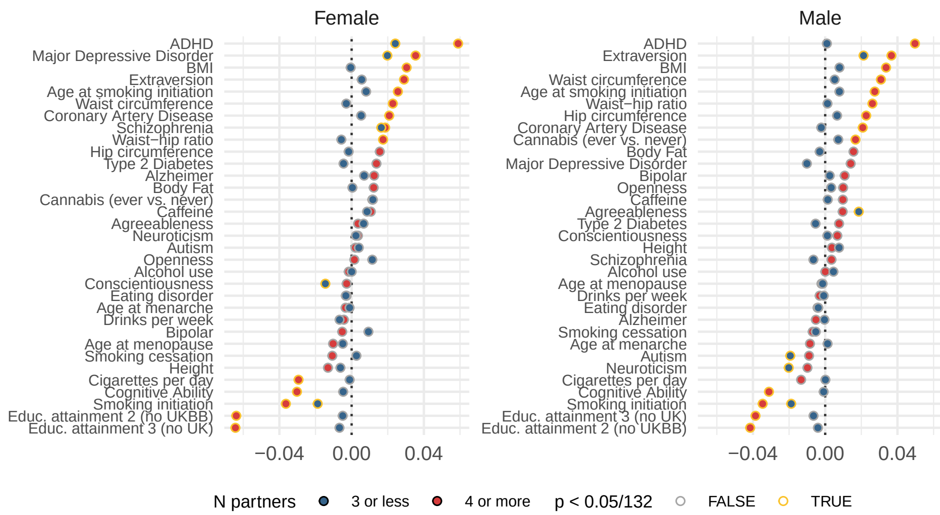


(a) Age left full-time education

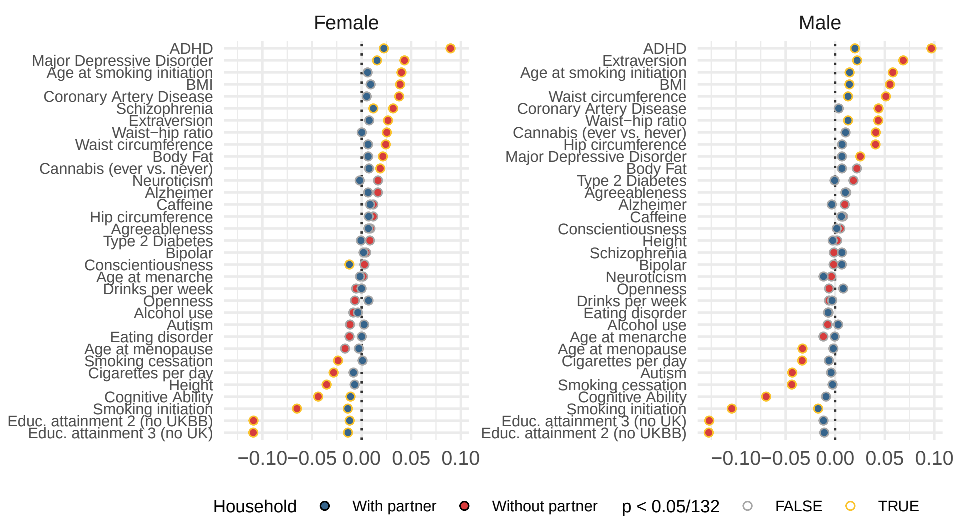


(b) Household income

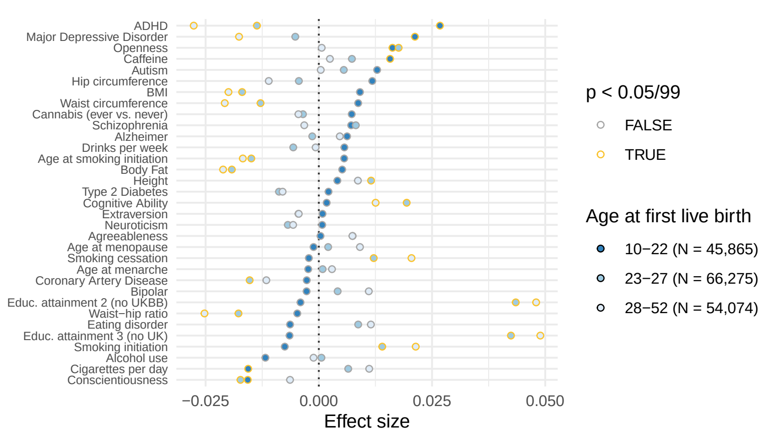
Figure 2: Selection effects by education and income. Each point represents a single bivariate regression of number of children on a PGS



(a) Lifetime number of sexual partners



(b) Household type



(c) Age at first live birth, females

Figure 3: Selection effects by number of sexual partners, household type, and age at first live birth

and an opposite-signed effect on number of children controlling for AFLB.

Correcting for sampling bias

Since UK Biobank subjects are not representative of the wider population, our results suggest that naïve estimates of natural selection are at risk of ascertainment bias. To correct for this, we weight participants using population data. We try three alternative weighting schemes: weighting by geography, age and presence/absence of a partner; weighting by age and highest educational qualification; and age, highest qualification, and age at first live birth, for women only. Figure 4 plots selection effects among the entire sample, estimated with the three weighting schemes. Mean effect sizes across all polygenic scores are increased by a factor of 1.4 (geographical weighting), 1.22 (age/qualification) or 1.89 (age/qualification/AFLB). Estimates might be further affected by weighting on other demographic variables. Since the Biobank sample seems to be ascertained in ways that shrink estimates of selection effects, we suspect that our weighted estimates are still conservative.

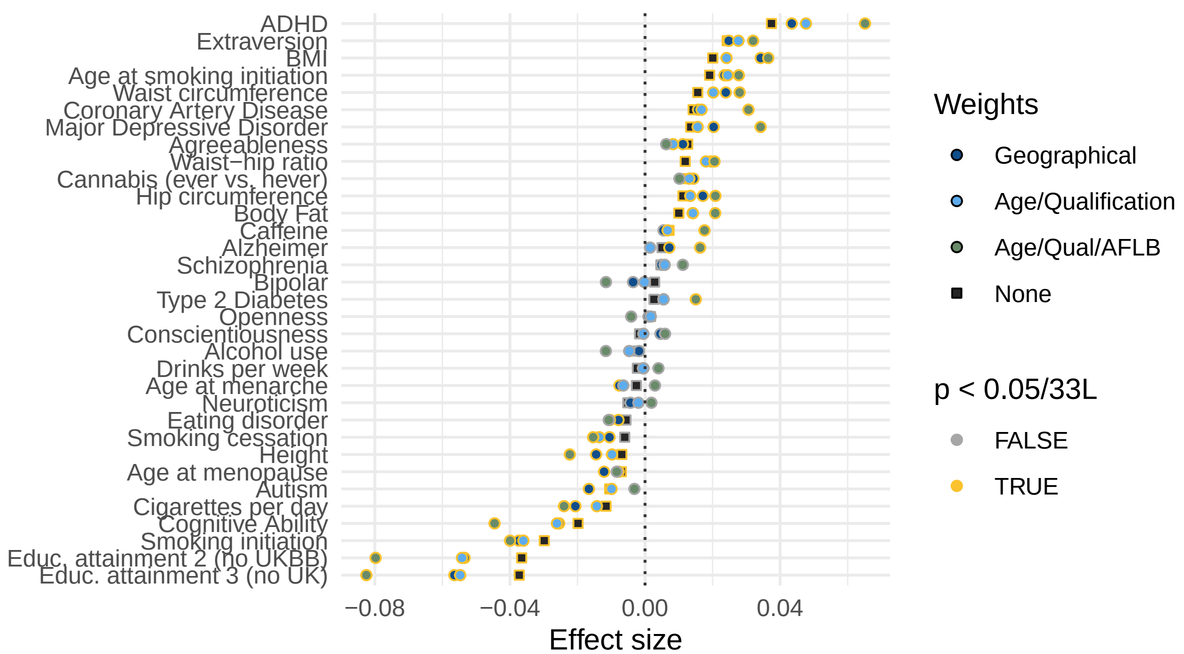


Figure 4: Selection effects: weighted regressions. Unweighted estimates are shown for comparison.

Selection in the parents' generation

The UK Biobank data contains information on respondents' number of siblings. 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 a further 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*.

Selection effects are highly correlated across the two generations, and most share the same sign (appendix Figure 9). 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 when we change the dependent variable, but do not change over time within either dependent variable, this may be due to remaining ascertainment bias within the sample.

Although the direction of selection effects does not change between the generations, there are other differences. Compared to residualized polygenic scores, selection effects on unresidualized scores are about ten percent higher on average for *n siblings*, whereas effects for *number of children* barely change (appendix Figure 11). This could be because earlier fertility is driven more by geographically clustered deprivation (e.g. via an insurance motive, Rendall and Bahchieva 1998), which may correlate with the broad-scale genetic variation captured by principal components.

We also check whether selection effects differ by socio-economic status 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 (appendix Figure 10) show the same pattern as for respondents: effect sizes are larger and more often significant in the most deprived areas.

Lastly, the siblings data lets us check for a "quantity-quality tradeoff" between number of children and number of grandchildren (for the parents' generation). We do not find any: in fact, the correlation between *number of siblings* and *number of children* is positive ($\rho = 0.1$).

Economic fertility theory and natural selection

These results show that selection effects are weaker, absent, or even reversed among some subgroups of the population. This can potentially be explained by the economic theory of fertility (Becker 1960; Willis 1973; Becker and Tomes 1976). According to this, 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 would lead higher earners to have more children. The substitution effect would lead higher earners to have fewer children. If so, then genetic variants which affect earnings potential in the labour market may cause opposing effects on fertility. The income effect will cause natural selection in favour of earnings-increasing variants. The substitution effect will do the reverse.

This theory can explain why selection effects have opposite signs when AFLB is controlled for. Suppose that people are aware of their endowments of human capital, i.e. skills and characteristics that are valuable in the labour market. Suppose also that education and innate human capital are complements. Individuals choose how long to stay in education; after leaving education they enter the labour market and form families. If so, then people with less human capital will leave education earlier and have their first child earlier (cf. Caucutt, Guner, and Knowles 2002; Monstad, Propper, and Salvanes 2008). This is a pure substitution effect: the income effect plays no role in the decision to leave education, since people are not yet earning wages.[3] Whilst in the labour market, higher earners will have more children (i.e. income effects dominate). These assumptions will lead to the pattern we observe: earnings-increasing variants will predict later first births (and fewer children overall), but they will also predict more children within any given age at first live birth.

The theory can also explain why natural selection is weaker among higher-income parents. Becker and Tomes (1976) show that if parents care about child quality as well as quantity, under certain assumptions the income effect will be stronger at higher income levels; this can lead to a U-shaped relationship between income and fertility. Empirically, Cohen, Dehejia, and Romanov (2013) find that income decreases fertility at low income levels but increases it at higher income levels. If, in our data, at high income levels income and substitution effects are roughly balanced , then income-linked polygenic scores will be neither selected for nor against.

Lastly, it is often assumed that the substitution effect will dominate for lone parents, or those in unstable relationships, since they have less opportunity to share childcare responsibilities, while the income effect will dominate for couples who are able to reap the gains from specialization. Indeed, US fertility decreases faster with education among single mothers than married mothers (Baudin, De La Croix, and Gobbi 2015). This can explain why earnings-increasing genetic variants decrease fertility more among single parents than among couples. The same logic could explain our results for lifetime number of sexual partners, if this is associated with relationship stability.

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 effect on earnings. Figure 5 plots selection effects on each PGS against that PGS's correlation with earnings in a respondent's first job, and against its correlation with educational attainment, a predictor

[3] This argument assumes that capital market imperfections hinder people from borrowing against future income.

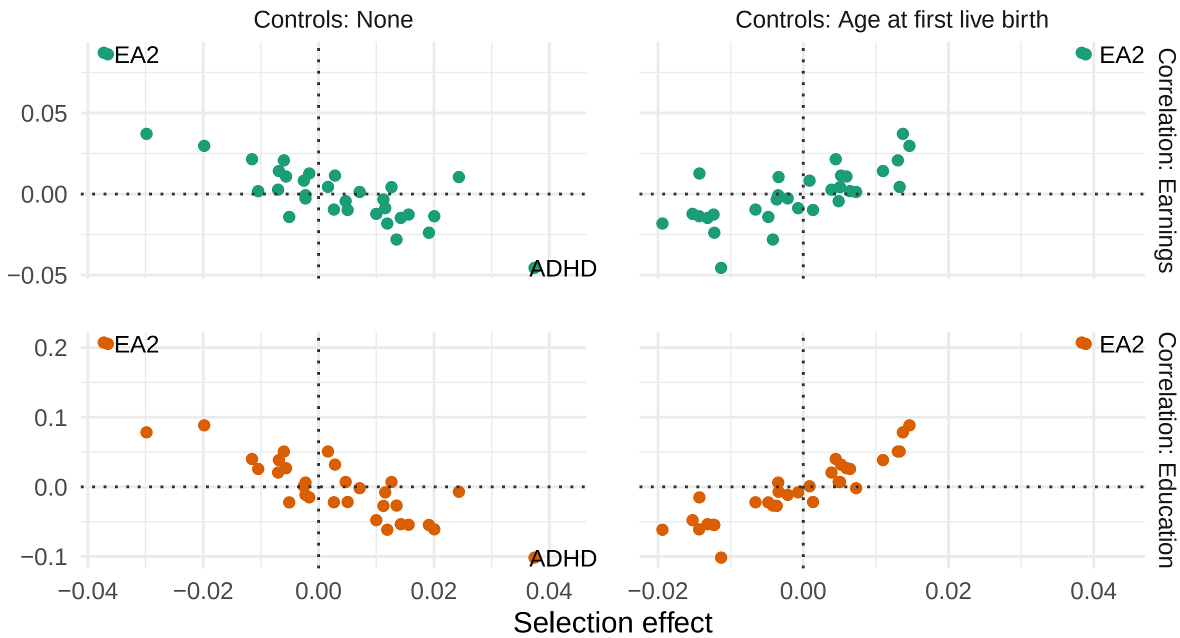


Figure 5: Selection effects, with and without controls for age at first live birth, by correlations with earnings and educational attainment. Each point represents one PGS.

of lifetime earnings. The raw relationships (left column) are strongly negative. If we plot selection effects controlling for age at first live birth (right column), the effect reverses and becomes positive. Thus, the labour market appears to play an important role in natural selection. Substitution effects dominate income effects overall, which fits the known association between income and lower fertility (Becker 1960; Jones and Tertilt 2006).

Second, we re-estimate equation (1) for each PGS, controlling for earnings in first job and education levels. Effect sizes are generally reduced (appendix Figure 16), and only 4 out of 33 PGS are significant at $p = 0.05/33$. This suggests that earnings and education indeed mediate the effect of PGS on fertility.

An alternative theory is that traits selected for are linked to externalizing behaviour, risk-seeking and low time discount rates, via the channel of early sexual behaviour (Mills et al. 2020). The data here provide some support for this: scores which might plausibly be linked to externalizing behaviour, like ADHD and 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, e.g. waist-hip ratio and BMI. Externalizing behaviour also does not explain why selection effects should work in the opposite direction among older parents, whereas the economic theory can explain this via income effects, as described above. We test the alternative theory directly by re-estimating equation (1) controlling for a measure of risk attitude (Biobank field 2040). While risk attitude is always a highly significant predictor of number of children, it has little impact on the effect size or significance of the PGS. The median ratio of PGS effect sizes between regressions with and without controls is 0.97; all PGS which are significant at $p \leq 0.05/33$ in uncontrolled regressions remain so when controlling for risk attitude. Thus, although risk attitude does predict number of children, it appears not to mediate selection effects. Overall, we believe that the economic theory is the most likely explanation.

Discussion

Previous work has documented natural selection in modern populations on variants underlying many polygenic scores (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 poorer people, less educated people, younger parents and those with more lifetime sexual partners. Indeed, among older parents selection effects are actually 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 this.

PGS 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 poor health, or are what many people would see as undesirable to have. For example, most people would probably prefer to have high educational attainment, a low risk of major depressive disorder, and a low risk of coronary artery disease, but natural selection is pushing against genetics linked to 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. And we are unsure whether genetic variants underlying other phenotypes will show a similar pattern of natural selection to those studied here. In short, it is probably too early to tell whether modern natural selection has a substantively important effect on population genetics. Nevertheless, 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 poorer groups, they also increase inequality with respect to polygenic scores. For example, Figure 6 graphs mean polygenic scores for educational attainment (EA3), for children in 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 EA3, which lowers the mean, is stronger at the bottom of the income distribution, and this increases the differences between groups.

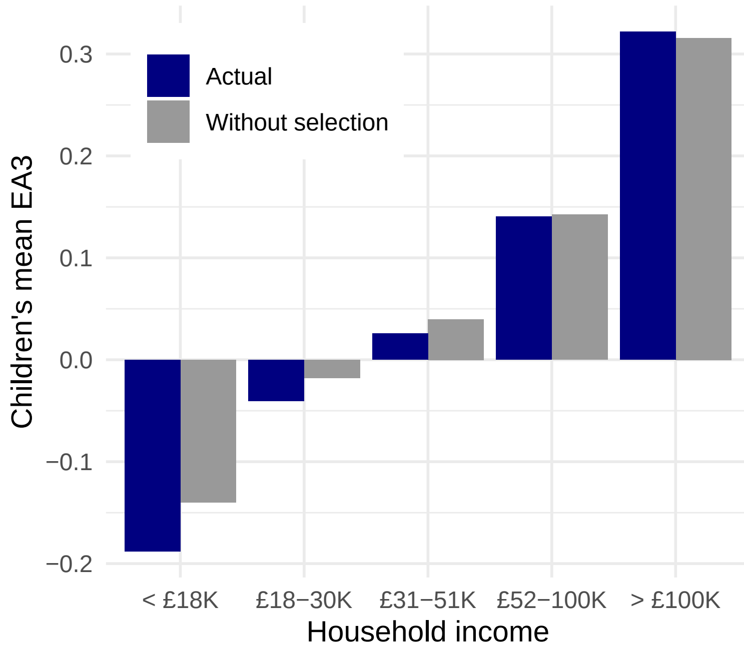


Figure 6: Mean PGS for educational attainment (EA3) of children by household income group. Blue is actual. Grey is hypothetical in the absence of selection effects.

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 modern natural selection. Economics possesses a range of theoretical models on the effects of skills, education and income (see Hotz, Klerman, and Willis 1997, @lundberg2007american). 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 causality. Conversely, theory and empirical results from economics can shine a light on the mechanisms behind natural selection.

Materials and methods

We use participant data from UK Biobank (Sudlow et al. 2015), 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, 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.

We computed 33 polygenic scores. The effect size estimates were obtained from GWASs that were chosen to not have included the UK Biobank dataset, to avoid overestimation of the genetic predisposition of a trait. The polygenic scores were computed using the summary-data based best linear unbiased prediction (SBLUP) approach. As a reference sample for the linkage disequilibrium, we used a random sample of 10,000 unrelated individuals from UK Biobank, imputed using the Haplotype Reference Consortium panel. Scores were normalized to mean 0, variance 1, and residualized on the first 100 principal components of array data, which were calculated as in Abdellaoui et al. (2019).

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.

Appendix

Natural selection by sex

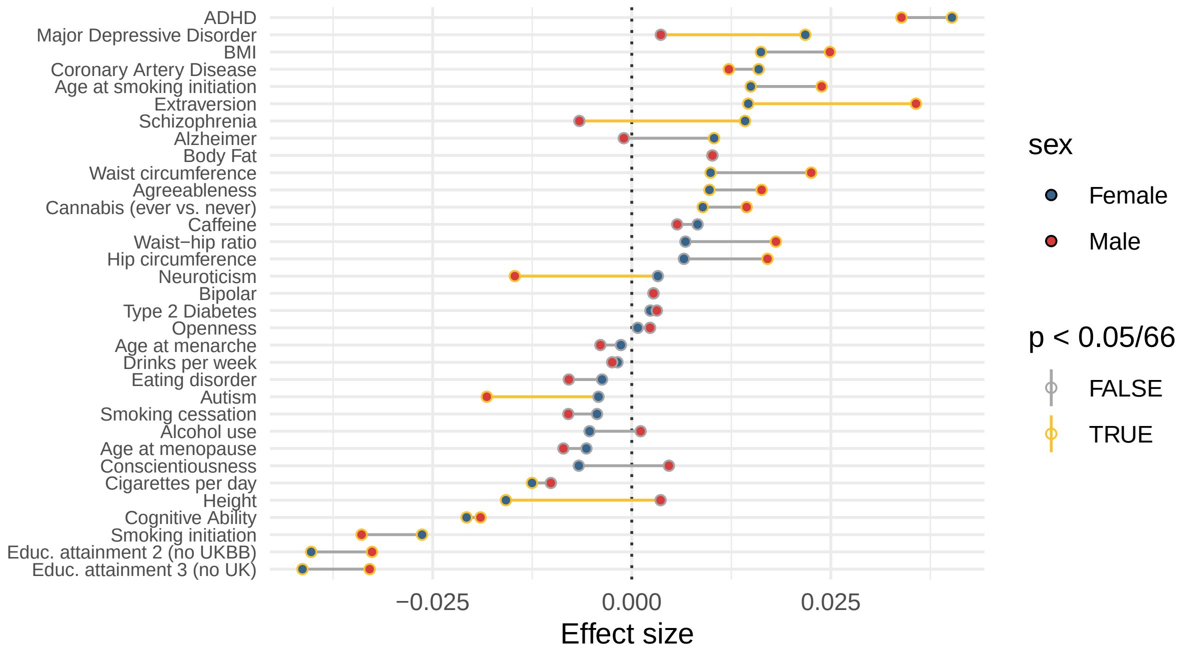


Figure 7: Selection effects by sex. Lines show significant differences.

Controlling for AFLB

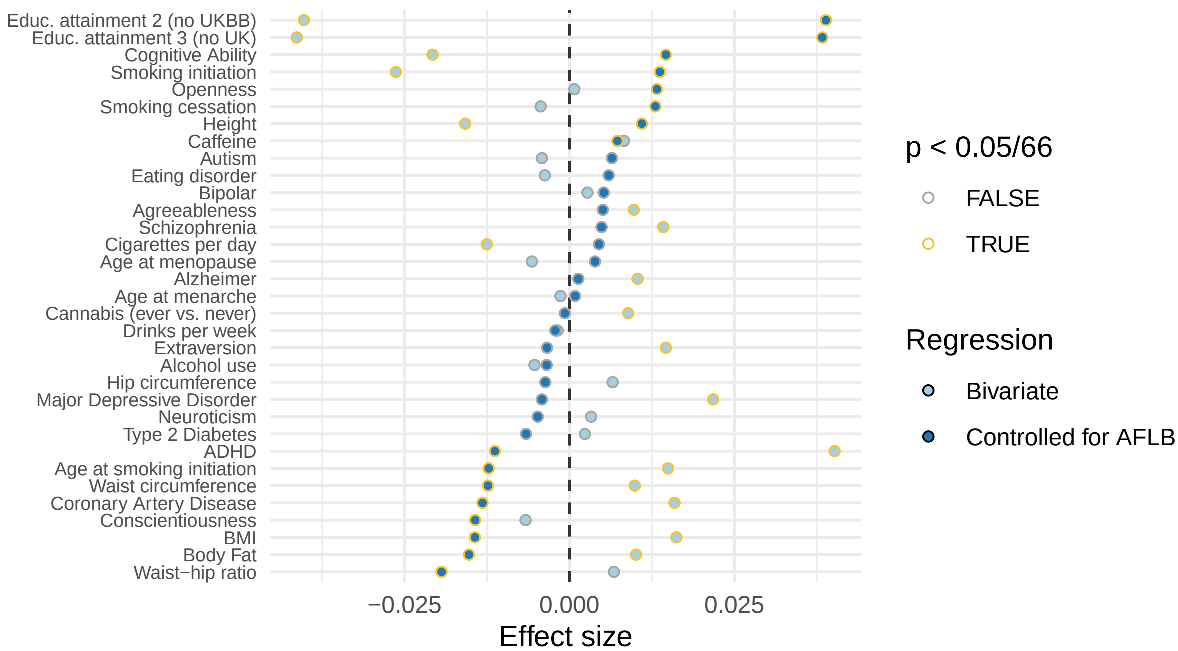


Figure 8: Selection effects controlling for age at first live birth (women only). Effect sizes for women only without controls are shown for comparison.

Weighted regressions

Table 1 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 1: Weighted effect sizes as a proportion of unweighted effect sizes.

	Weighting		
PGS	**Geographical**	**Age/Qualification**	**Age/Qual/AFLB**
Height	2.10	1.42	1.41
Cigarettes per day	1.79	1.24	1.91
Age at menopause	1.73	1.12	1.47
BMI	1.71	1.20	2.25
Waist-hip ratio	1.65	1.52	3.05
Autism	1.59	0.95	0.75
Waist circumference	1.53	1.29	2.83
Hip circumference	1.52	1.19	3.18
Educ. attainment 3 (no UK)	1.52	1.47	2.00
Major Depressive Disorder	1.50	1.15	1.57
Educ. attainment 2 (no UKBB)	1.46	1.48	1.98
Body Fat	1.41	1.42	2.06
Cognitive Ability	1.28	1.32	2.15
Smoking initiation	1.25	1.21	1.52
Cannabis (ever vs. never)	1.24	1.14	1.14
Age at smoking initiation	1.23	1.28	1.86
ADHD	1.16	1.27	1.62
Coronary Artery Disease	1.10	1.17	1.92
Extraversion	1.02	1.14	2.19
Agreeableness	0.89	0.66	0.64
Caffeine	0.77	0.94	2.13
Mean	1.40	1.22	1.89
Median	1.46	1.21	1.92

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

Parents' generation

Figure 9 shows regressions of *number of siblings* on polygenic scores. By definition, people who had no children cannot be included in this data. For a cleaner comparison, we rerun regressions on the respondents' generation (*number of children*) after excluding respondents with no children.

To check whether effect sizes were changing over time, we ran regressions interacting PGS with birth year, median split at 1950 ("early 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 2 and 3 summarize the results. There is no change in natural selection within the parents' generation. In the respondents' generation, effect sizes were significantly larger in absolute size among the late-born for four PGS: cognitive ability, EA2, EA3 and extraversion. These changes are inconsistent with the intergenerational change, where

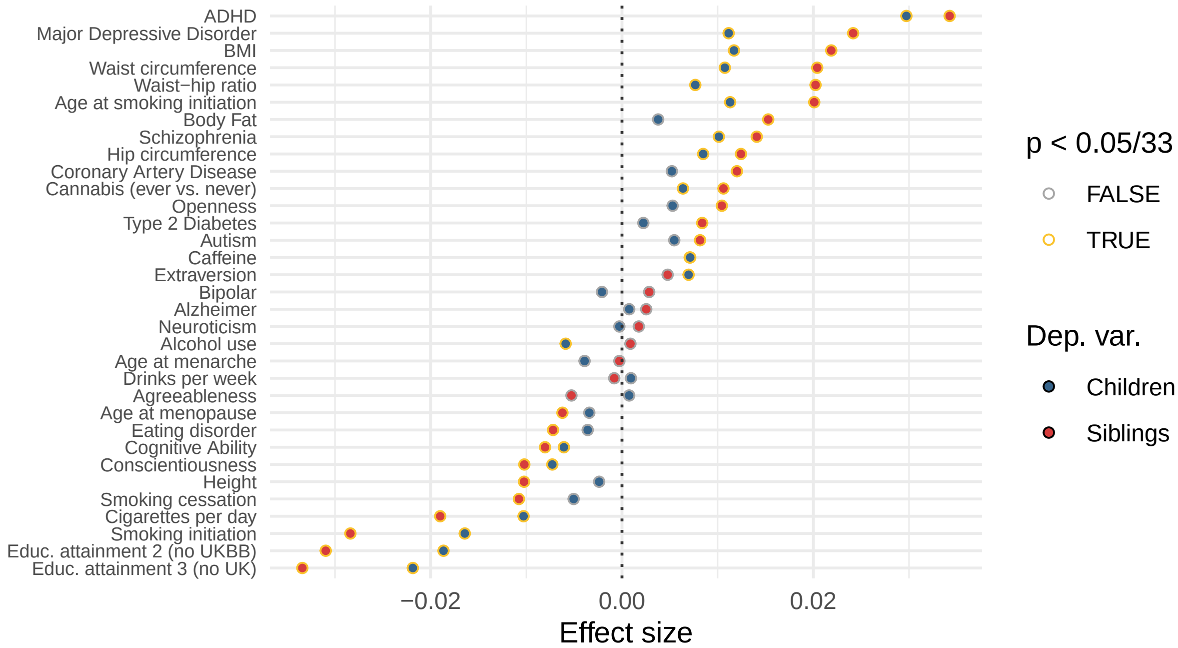


Figure 9: Selection effects, respondents' parents vs. respondents.

effect sizes were if anything larger among the earlier, parents' generation. Three PGS showed significant changes in sign: alcohol use (positive to negative); conscientiousness (negative to positive); and type II diabetes (negative to positive). Overall, there is weak evidence for change over time, and the clearest result is that the direction of selection is broadly consistent.

Table 2: Change in selection effects between parents of early and late born respondents (regressions on number of siblings).

Change	**Number of scores**
Insignificant	33

Significance is measured at $p < 0.05/66$

Table 3: Change in selection effects between early and late born respondents (regressions on number of children).

Change	**Number of scores**
Change sign	1
Insignificant	28
Size increasing	4

Significance is measured at $p < 0.05/66$

Figure 10 plots effects on *n siblings* by Townsend deprivation quintile of birth area.

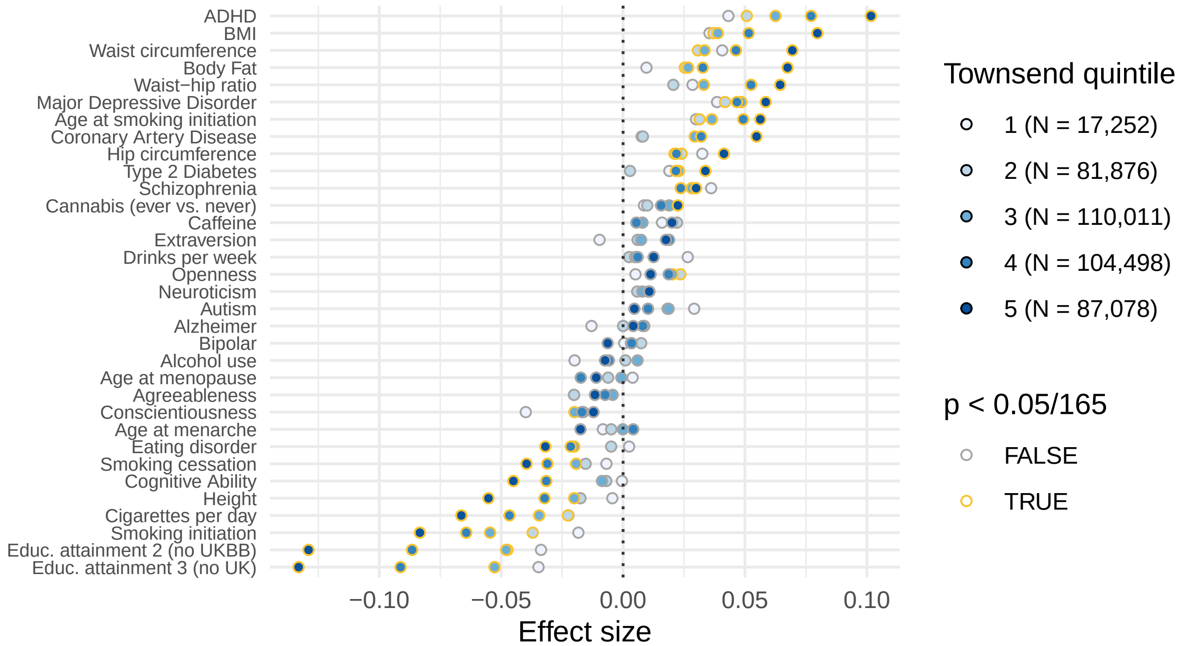


Figure 10: Selection effects in the parents' generation by Townsend deprivation quintile of birth area

Selection effects on raw polygenic scores

Figure 11 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 principal components – sometimes much larger, as in the case of height. 28 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 0.87. Among the children regressions, this no longer holds. Effect sizes are barely affected by controlling for principal components.

Overall, 81.82 per cent of effect sizes are consistently signed across all four regressions (on children and siblings, and with and without residualization).

To get a further insight into this we regress *n siblings* and *n children* on individual principal components. As Figure 12 shows, effects are larger and more significant in siblings regressions. 29 principal components significantly predicted number of siblings, while only 10 significantly predicted number of children.

Selection controlling for age at first live birth: respondents' parents

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,

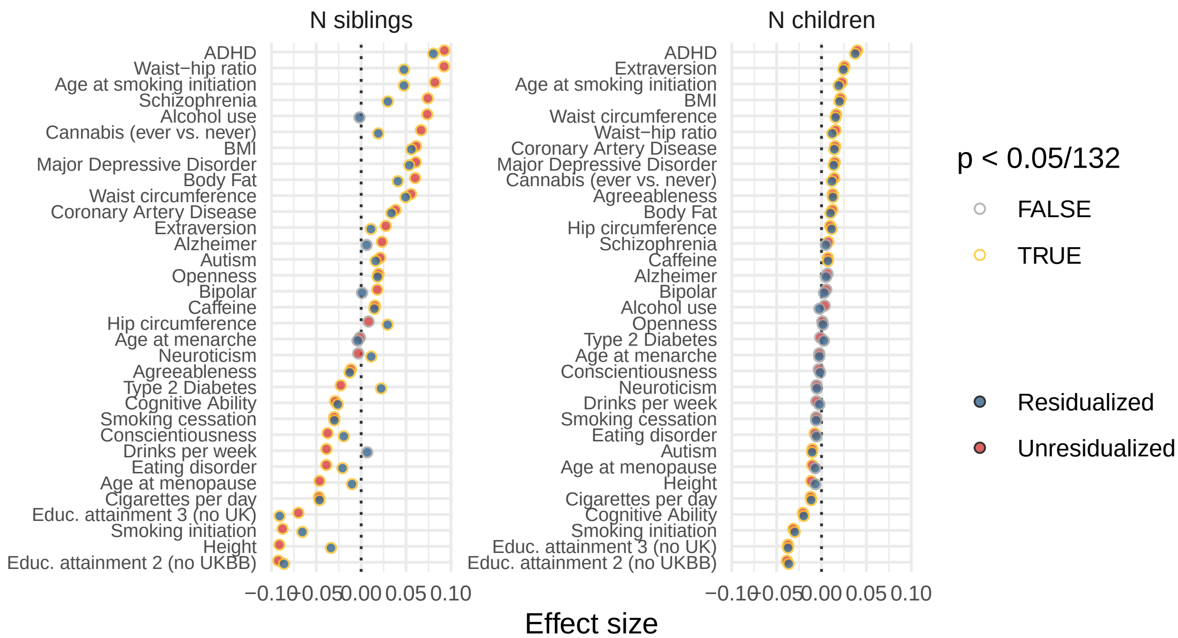


Figure 11: Selection effects using unresidualized polygenic scores on number of siblings/children.

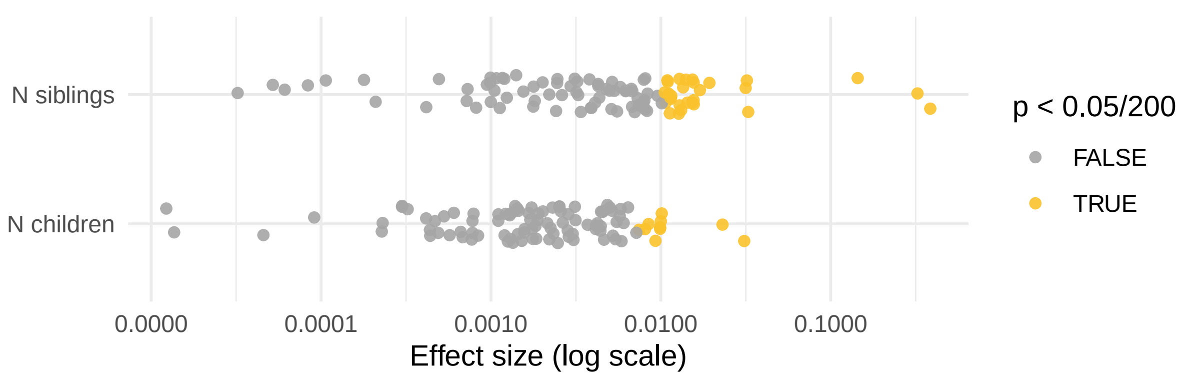


Figure 12: Selection effects of principal components of genetic data. Absolute effect sizes are plotted. Each dot represents one bivariate regression. Points are jittered on the Y axis.

controlling for either parent's age at their birth. Figure 13 shows the results. 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, though the correlation is smaller (father's age at birth: ρ -0.59; mother's age at birth: ρ -0.69).

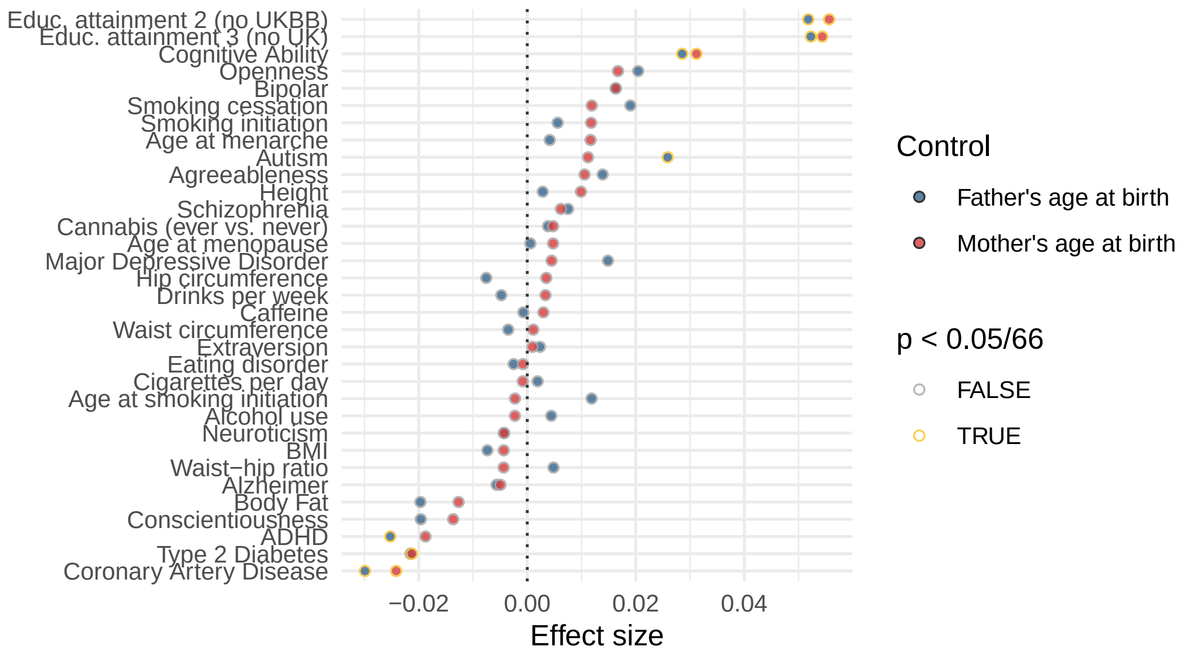


Figure 13: Selection effects (parents' generation) among eldest siblings, controlling for parents' age at birth.

Effects of PGS 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, and Figure 15 does the same for their parents. Effect sizes are reasonably large. They are also very highly correlated across generations. Effect sizes of PGS 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.

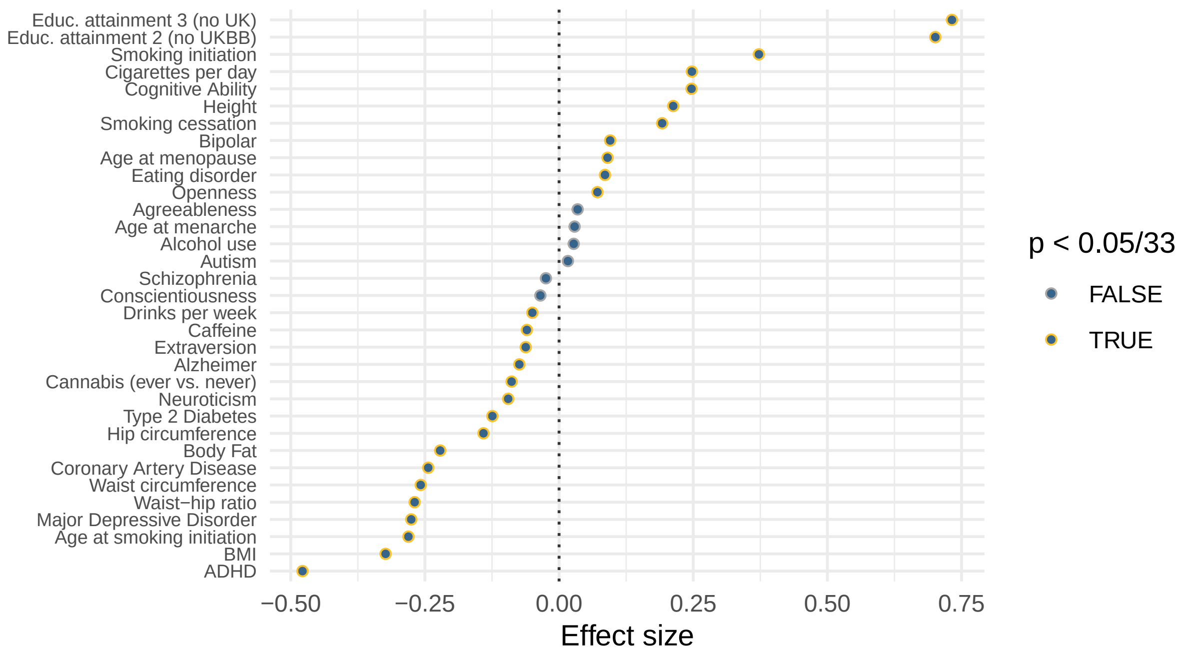


Figure 14: Effects of polygenic scores on age at first live birth.

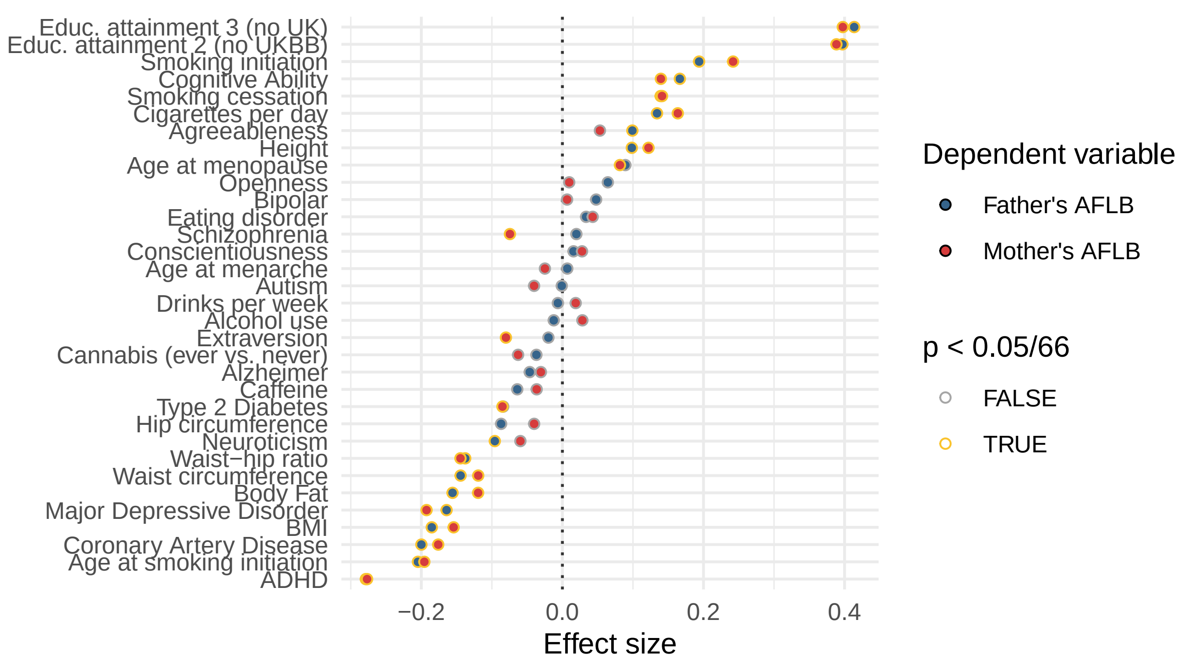


Figure 15: Effects of polygenic scores on parents' age at respondent's birth, eldest siblings.

Controlling for earnings and education

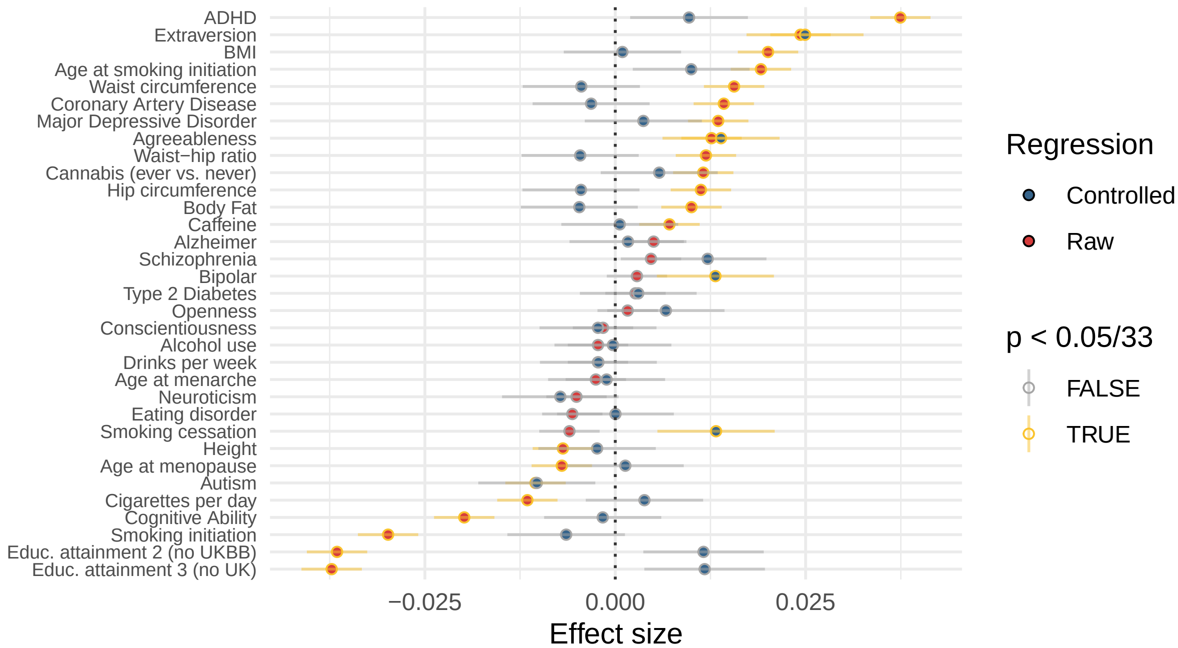


Figure 16: Selection effects controlling for earnings in first job (estimated by mean earnings from ASHE 2007 for the SOC2000 job code) and education (left education before 16, 16-18, or after 18). Raw effects are shown for comparison. Lines are 95% confidence intervals uncorrected for multiple testing.

Genetic correlations with EA3

Another way to examine the "earnings" theory of natural selection is to compare selection effects of PGS 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 17 shows this is so.

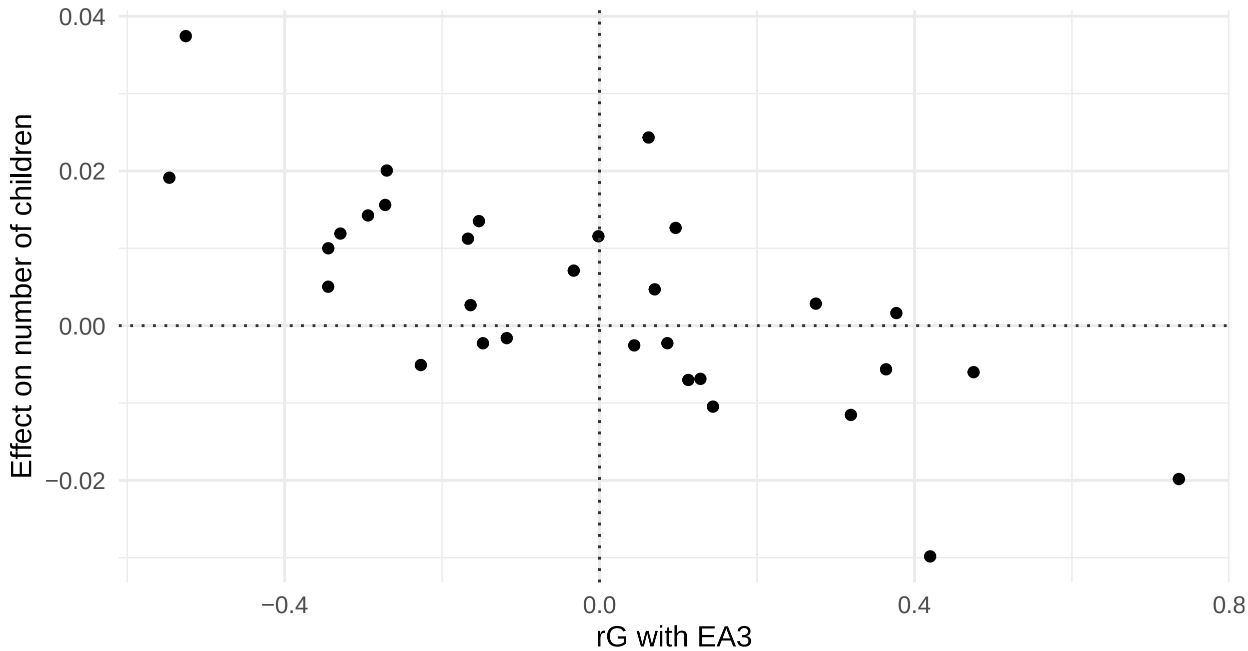


Figure 17: Selection effects plotted against genetic correlation with EA3.

Effects on inequality

Table 4 estimates differences in children's mean polygenic scores between the highest and lowest income groups. Column "Actual" shows respondents' scores weighted by Age/Qualification times number of children. Column "Without selection" shows scores weighted by Age/Qualification only, i.e. if all couples had the same number of children.

Table 4: Differences in polygenic scores between highest and lowest income group.

PGS	With selection	Without selection
Educ. attainment 3 (no UK)	0.542	0.487
Educ. attainment 2 (no UKBB)	0.533	0.477
Smoking initiation	0.210	0.166
Cognitive Ability	0.191	0.185
Height	0.161	0.140
Smoking cessation	0.156	0.132
Cigarettes per day	0.105	0.084
Extraversion	0.102	0.095
Openness	0.100	0.087
Bipolar	0.081	0.064
Eating disorder	0.057	0.056
Agreeableness	0.055	0.045
Age at menopause	0.039	0.036
Alcohol use	0.034	0.034
Age at menarche	0.020	0.014
Conscientiousness	0.001	-0.013
Autism	-0.016	-0.020
Schizophrenia	-0.025	-0.050
Caffeine	-0.027	-0.021
Cannabis (ever vs. never)	-0.042	-0.043
Type 2 Diabetes	-0.046	-0.039
Alzheimer	-0.047	-0.036
Drinks per week	-0.068	-0.059
Hip circumference	-0.086	-0.072
Neuroticism	-0.125	-0.119
Coronary Artery Disease	-0.125	-0.109
Body Fat	-0.135	-0.125
Age at smoking initiation	-0.137	-0.109
Major Depressive Disorder	-0.159	-0.142
Waist circumference	-0.160	-0.137
Waist-hip ratio	-0.165	-0.146
BMI	-0.176	-0.156
ADHD	-0.281	-0.230

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