

Economic Theory of Fertility Explains 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. Selection effects appear consistent over time. Polygenic scores which predict lower earnings and education are selected for. Effects are larger among low-income and less educated people, younger parents, and people not living with a partner. Overall effect sizes are substantially bigger when we correct for ascertainment bias. The direction of natural selection is reversed among older parents, or 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.

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 natural selection in contemporary populations. This paper uses data from UK Biobank to learn more. We test for natural selection on 33 different polygenic scores. We extend the analysis over two generations, using data on respondents' number of siblings as well as their number of children to reconstruct two generations of family size. This is interesting because consistent natural selection over multiple generations could lead to substantive effects in the long run. Most importantly, we examine the strength of natural selection in different subgroups of the population, in order to uncover patterns that can help illuminate the mechanisms involved.

We use data from UK Biobank. We compute 33 polygenic scores, normalized to mean 0, variance 1. Figure 1 plots mean polygenic scores 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.

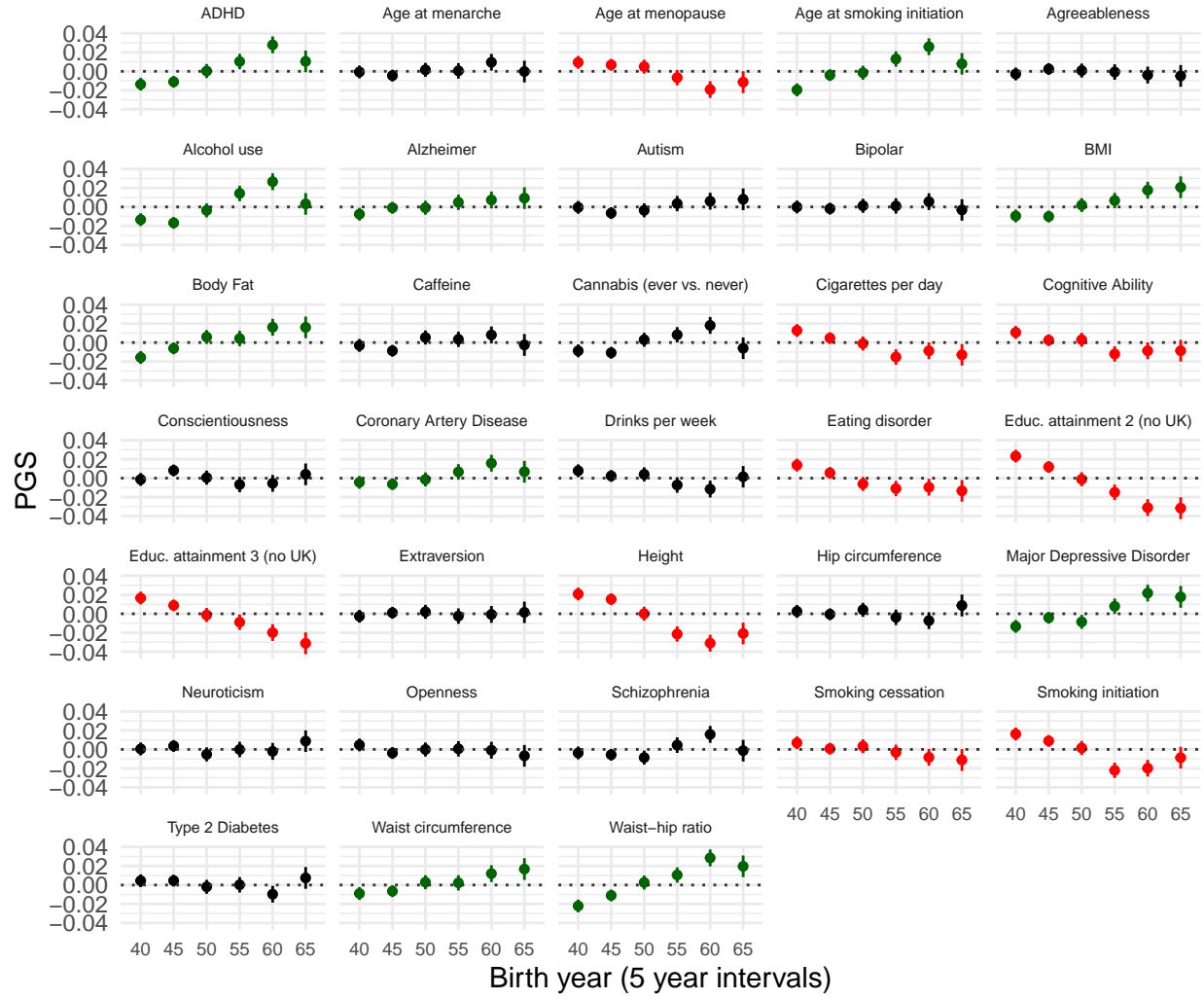


Figure 1: Mean polygenic scores by birth year in UK Biobank. Points are means for 5-year intervals. Lines are 95% confidence intervals. Green lines show a significant linear increase over time ($p < 0.05/33$). Red lines show a significant decrease.

These changes could reflect either natural selection within the UK population, or 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.

We next regress respondents’ polygenic scores on their number of children (y_i):

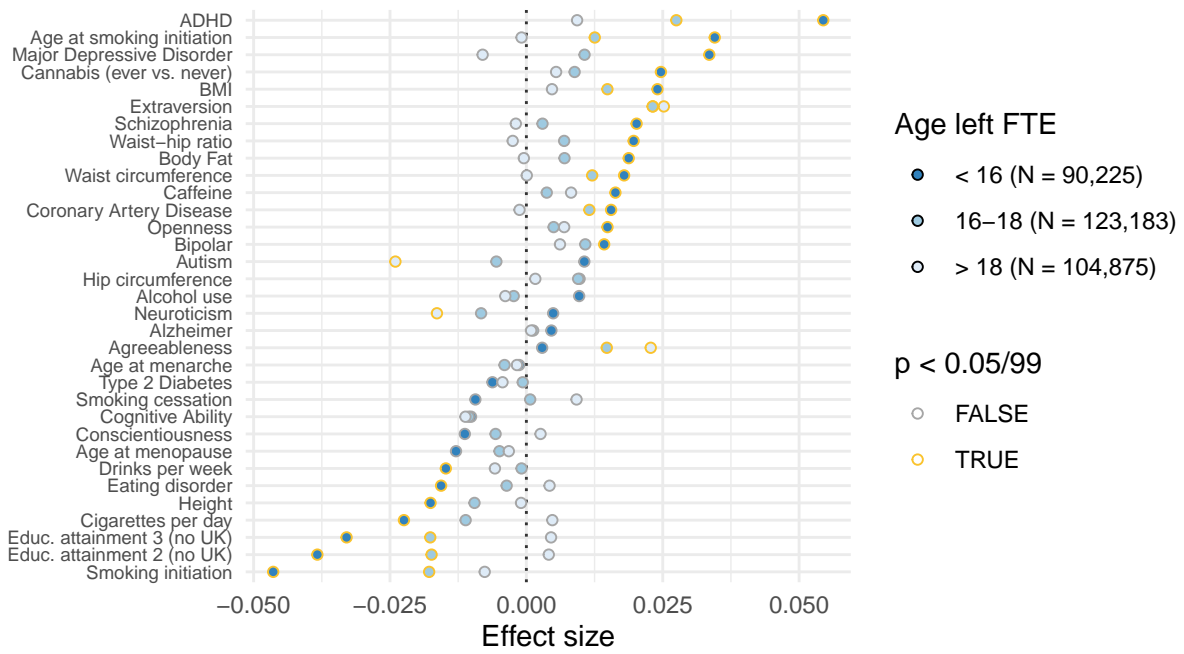
$$y_i = \alpha + \beta \text{PGS}_i + \varepsilon_i \tag{1}$$

The “selection effect”, β , measures the strength of natural selection within the sample. In fact, since polygenic scores are normalized to mean 0, β is the expected polygenic score among children of the sample (Beauchamp 2016). 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.

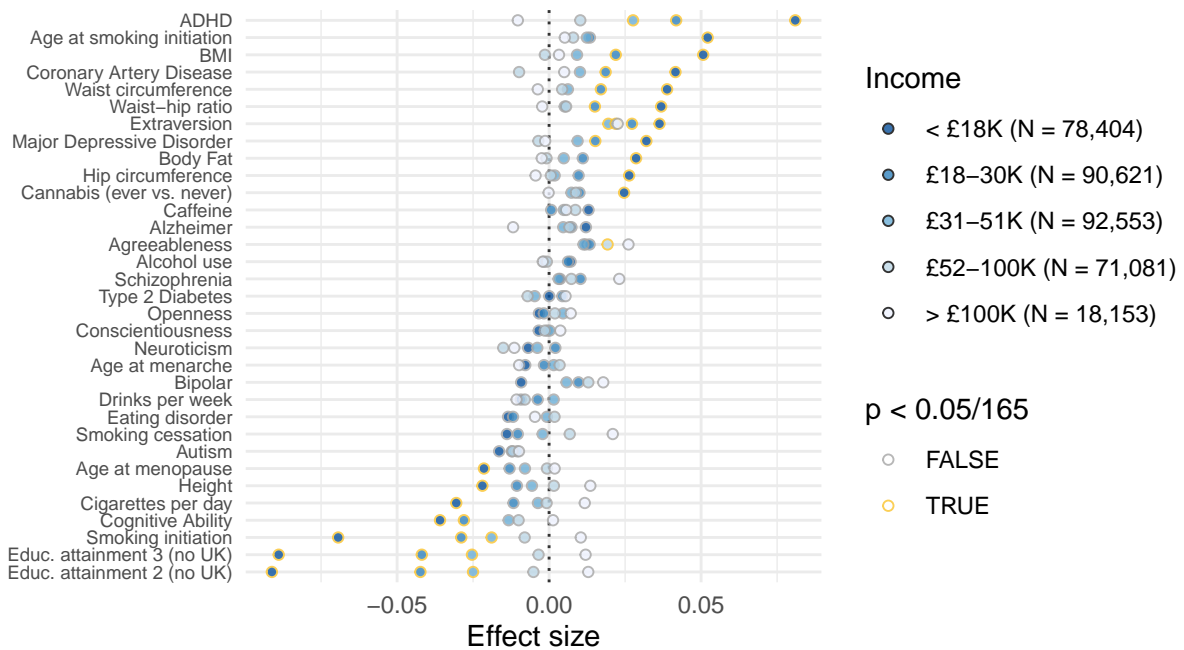
Figure 2 plots selection effects for each polygenic score, grouping respondents by age of completing full-time education, and by household income. Effects are larger and more significant for the lowest income category, and for the lowest education category. Note that the overall effect is not a simple average of the effect among the different subgroups, because polygenic scores may also shift respondents between the subgroups. For example, a high PGS for educational attainment may correlate with fewer children among early school leavers, but 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 21 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 17 out of 33 regressions.

Selection effects are also different between men and women (Appendix Figure 5). 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. Waist circumference and waist-hip ratio are less selected for among women, and educational attainment is more selected against, though the difference is only significant for EA3.



(a) Age left full-time education



(b) Household income

Figure 2: Selection effects by education and income

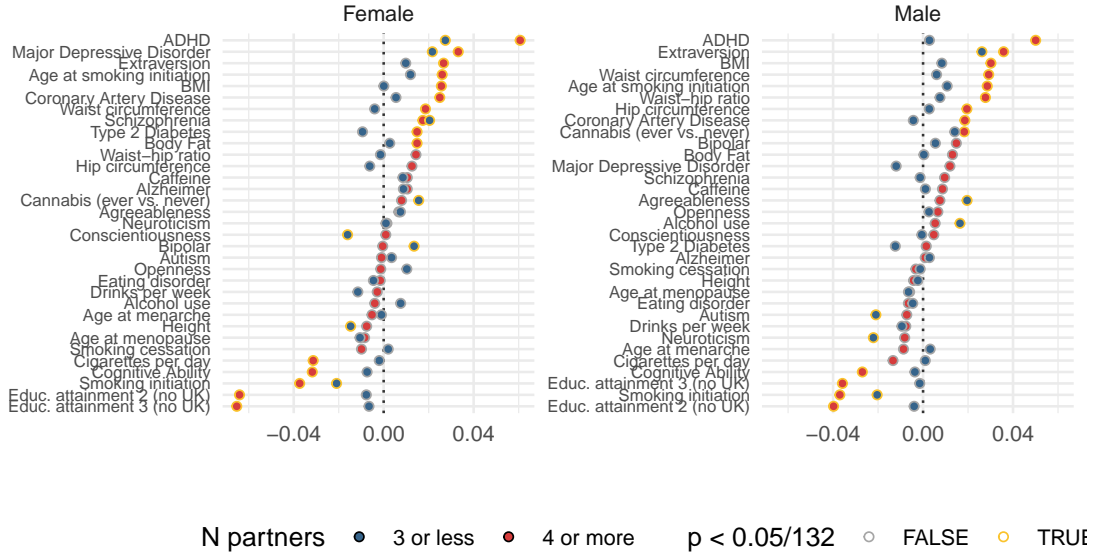
We next focus on variables related to mate search and household formation. We split males and females by lifetime number of sexual partners, at the median value of 3 (Figure 3a). For both sexes, selection effects are larger and more significant among those with more than 3 partners. Results are unchanged if we control for respondents' age (appendix Figure 11). Splitting 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).¹ 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 -0.54. As before, recall that PGS may have two effects: changing the number of children within categories, and shifting respondents between categories.

To investigate this further, we regress number of children on PGS *controlling* for AFLB (appendix Figure 6). 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 -0.75. Selection effects appear to come through two opposing channels: an effect on AFLB, and an opposite-signed effect on number of children controlling for AFLB.

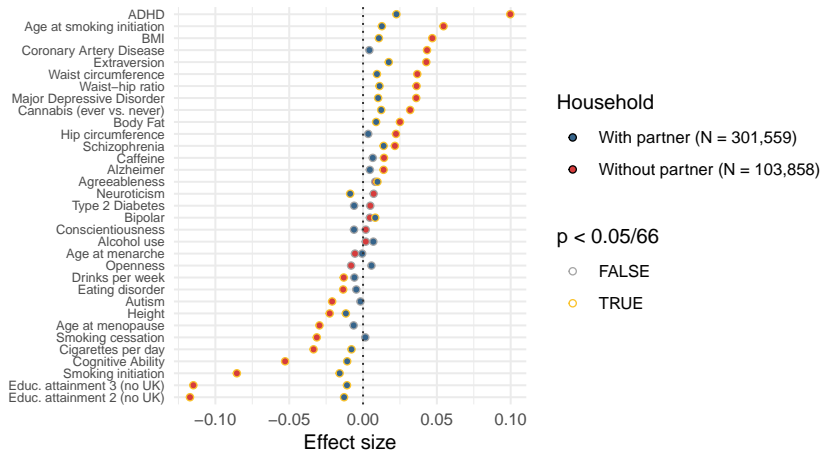
Since UK Biobank subjects are not representative of the wider population, our results suggest that naïve estimates of natural selection are at risk of ascertainment bias. To correct for this, we weight participants using data from the 2011 Census and from the 2006 General Household Survey (GHS). We try three alternative weighting schemes: geographical weighting by census MSOA, 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. Mean effect sizes across all polygenic scores are increased by a factor of 1.4 (geographical weighting), 1.21 (age/qualification) or 1.82 (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.

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

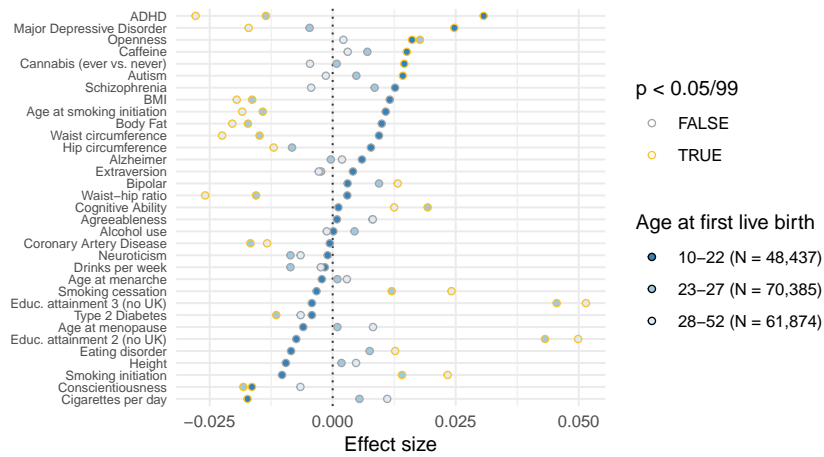
¹This information is unavailable for men.



(a) Number of partners



(b) Household type



(c) Age at first live birth

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

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 8). 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. After we residualize polygenic scores on the first 100 principal components of the array data, selection effects on *number of siblings* are reduced by about ten percent on average, whereas effects on *number of children* barely change (appendix Figure 9). 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. Another difference is that in the parents' generation, raw selection effects and effects controlling for AFLB are positively correlated, whereas they are negatively correlated in the respondents' generation.² These differences suggest that the mechanisms of natural selection may have changed over time, even though the outcomes are broadly the same.

Our empirical results can 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 predicts that genetic variants will be selected for (or against) in proportion to their effect on earnings. Figure 4 plots selection effects on each PGS against that PGS's correlation with earnings in

²Educational attainment is an exception. It is selected against in the parents' generation, but selected for if we control for AFLB, which is the same pattern as for respondents.

a respondent's first job³, and against its correlation with educational attainment, a predictor 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, in accordance with the widespread association between income and lower fertility (Becker 1960; Jones and Tertilt 2006).

We also re-estimate equation (1) for each PGS, controlling for earnings in first job and education levels. Effect sizes are generally reduced (appendix Figure 15), and only 4 out of 33 PGS are significant at $p = 0.05/33$, suggesting that earnings and education indeed mediate the effect of PGS on fertility.

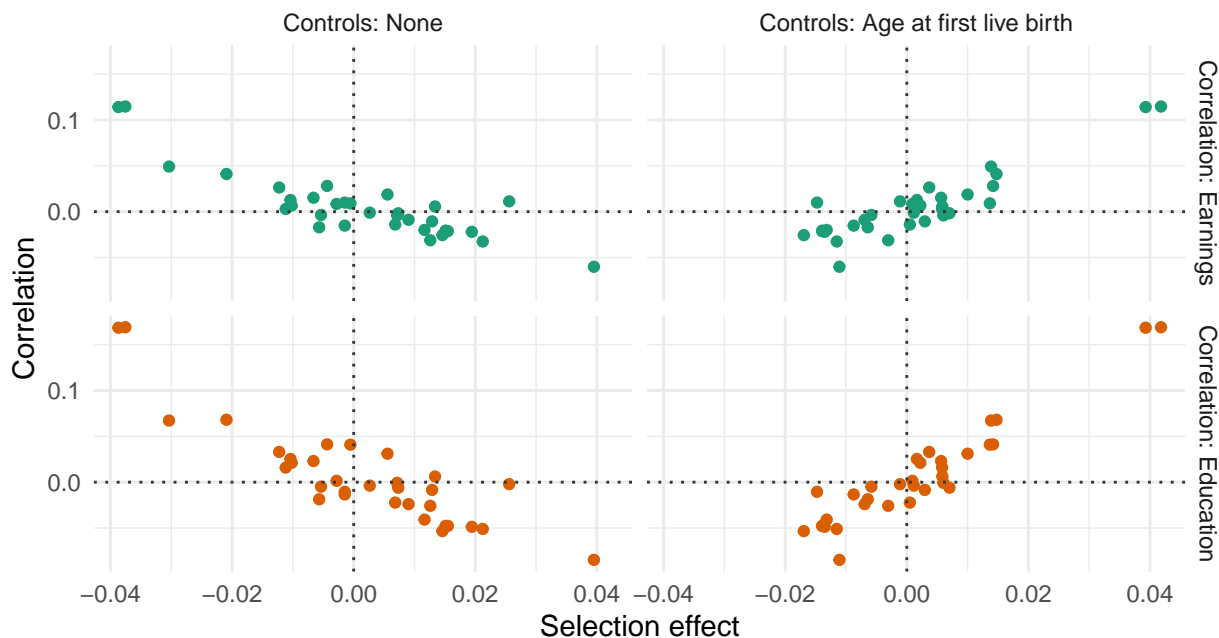


Figure 4: Selection effects, with and without controls for age at first live birth, by correlations with earnings and educational attainment. Each point represents one PGS.

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, and that education and innate human capital are complements. They 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.⁴ Whilst in the labour market, higher earners will have more children (i.e. income

³Earnings are estimated from the 2007 Annual Survey of Hours and Earnings, using the SOC 2000 job code.

⁴This argument assumes that capital market imperfections hinder people from borrowing against future income.

effects dominate). These assumptions will lead to the pattern we observe: earnings-increasing variants lead to later first births (and fewer children overall), but more children for any given AFLB.

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.

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). If so, then earnings-increasing genetic variants may decrease fertility more among single parents than among couples, as in our data. The same idea could explain our results for lifetime number of sexual partners, if this is associated with relationship stability.

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 natural selection effects 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 correlates with the association between a PGS and labour market earnings. The economic theory of fertility provides a parsimonious explanation for this.

An alternative theory is that traits selected for are linked to externalizing behaviour 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 change sign among older parents. Overall, we believe that the economic theory is the leading explanation.

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 would be seen as undesirable to have by many people. 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 these traits. Potentially, this could increase the health burden on modern societies, 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). Nevertheless, 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.

In addition, we do not know whether estimated effect sizes of natural selection will increase or decrease as more accurate polygenic scores are produced. We are also unsure whether genetic variants underlying other phenotypes will show a similar pattern of natural selection as these ones. In short, it is probably too early to tell whether modern natural selection is substantively important or not.

Research on natural selection is controversial. It brings to mind eugenics, which has been used to justify mass murder and human rights violations, in the past and possibly the present (Mao 1998; Sleeboom-Faulkner 2011; Zenz 2020). Whilst we claim scientific legitimacy for our empirical results, we have no special authority to speak on how society should react to them. This is a matter for open debate, in which scientists take part as equals among many. We therefore choose not to discuss policy implications here.

Appendix

Natural selection by sex

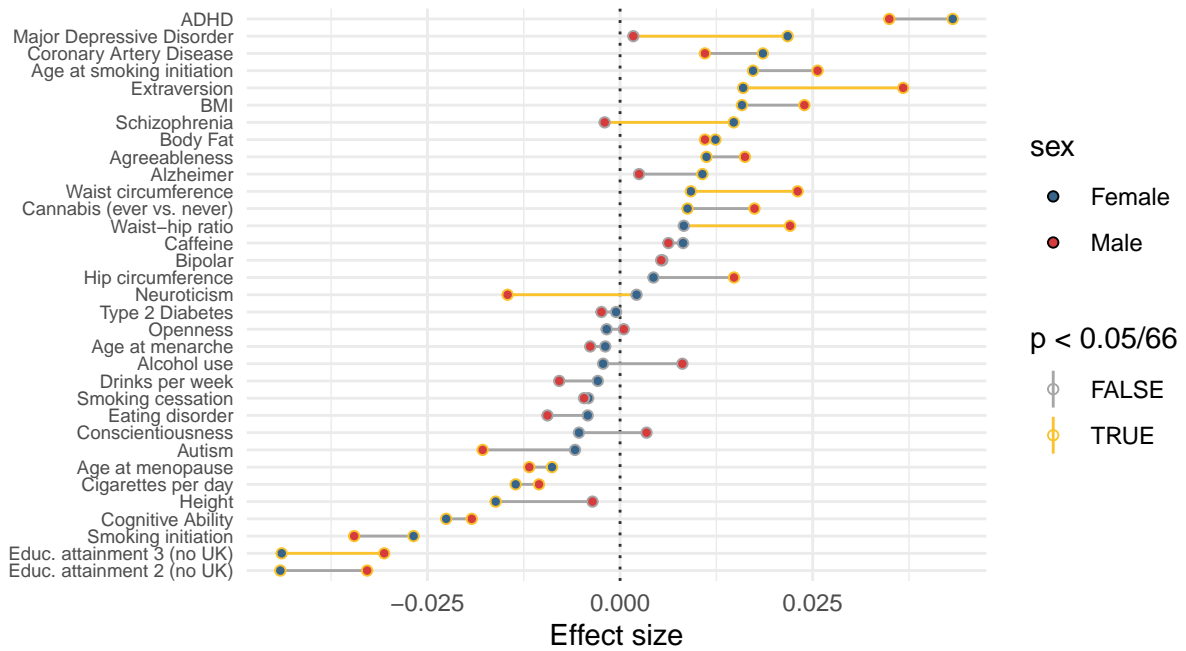


Figure 5: Selection effects by sex. Lines show significant differences.

Controlling for AFLB

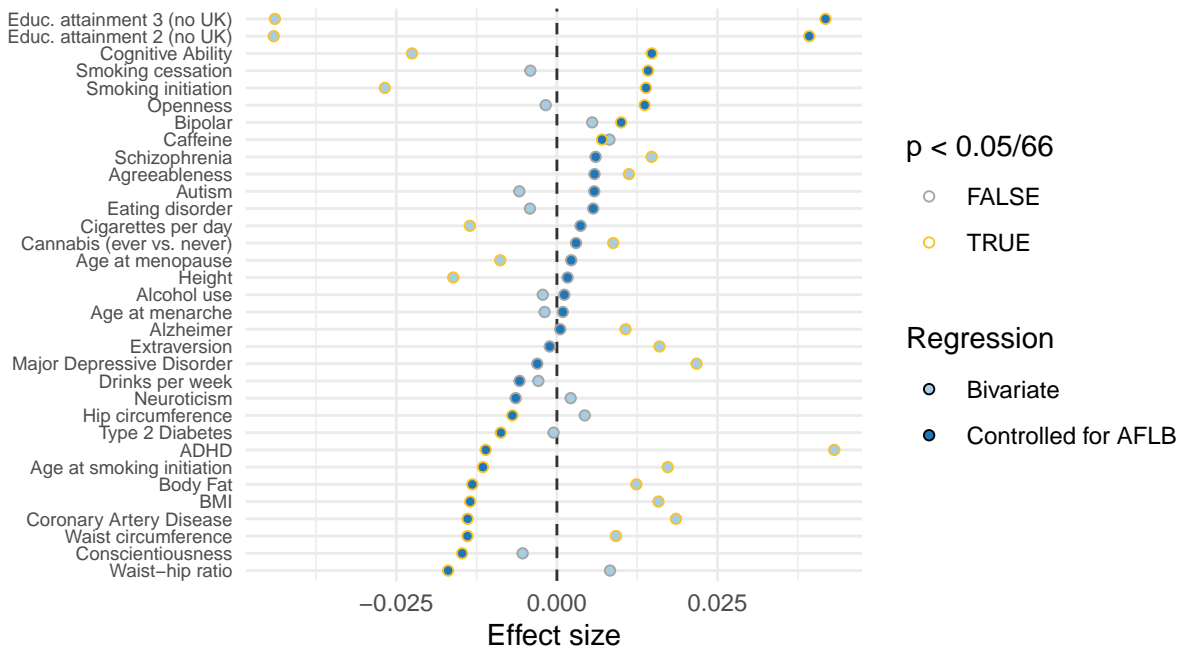


Figure 6: Selection effects controlling for age at first live birth (women only). Effect sizes for women without controls are shown for comparison

Weighted regressions

Figure 7 plots effect sizes among the entire sample, using our three weighting schemes. We include unweighted regressions for comparison. Table 1 gives effect sizes as a proportion of the unweighted effect size.

Table 1: Weighted effect sizes as a proportion of unweighted

PGS	Weighting		
	Geographical	Age/Qualification	Age/Qual/AFLB
Height	1.71	1.25	1.32
BMI	1.71	1.18	2.03
Age at menopause	1.66	1.06	1.27
Waist-hip ratio	1.65	1.46	2.47
Educ. attainment 3 (no UK)	1.6	1.53	2.04
Waist circumference	1.57	1.31	2.52
Cigarettes per day	1.54	1.25	2.12
Hip circumference	1.52	1.16	2.96
Educ. attainment 2 (no UK)	1.51	1.52	2.02
Major Depressive Disorder	1.49	0.891	1.35
Eating disorder	1.48	1.67	2.15
Body Fat	1.47	1.47	2.18
Autism	1.41	1.05	1.94
Schizophrenia	1.39	1.18	0.744
Alzheimer	1.36	0.429	1.4
Coronary Artery Disease	1.3	1.2	2.04
Cognitive Ability	1.28	1.34	2.03
Smoking initiation	1.26	1.21	1.62
Cannabis (ever vs. never)	1.19	1.15	0.761
Extraversion	1.17	1.15	1.87
ADHD	1.17	1.28	1.63
Caffeine	1.11	1.18	2.15
Age at smoking initiation	1.08	1.26	1.86
Agreeableness	1.01	0.868	1.09
<i>Mean</i>	1.4	1.21	1.82
<i>Median</i>	1.44	1.21	1.98

Only consistent and significant (when unweighted) estimates are shown.

Age/Qual/AFLB as a proportion of unweighted regressions for females only.

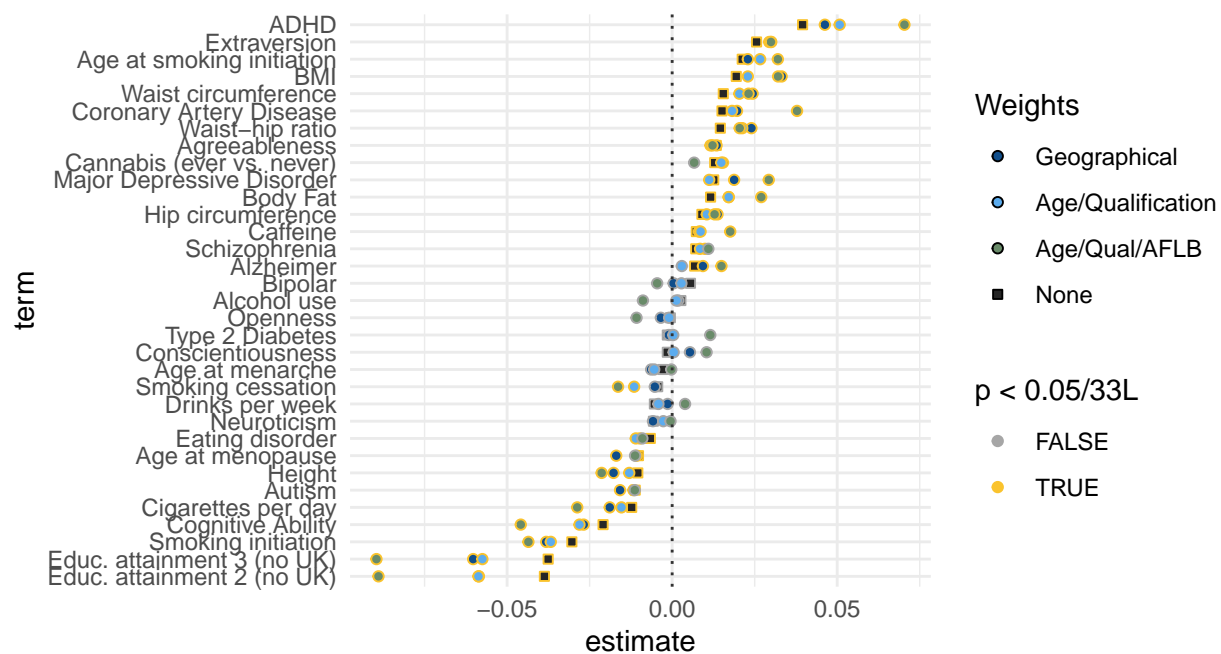


Figure 7: Effects of polygenic scores on fertility. Weighted regressions.

Parents' generation

Figure 8 shows regressions of *number of siblings* on polygenic scores. For comparison, we show regressions on the respondents' generation (*number of children*), excluding respondents with no children.

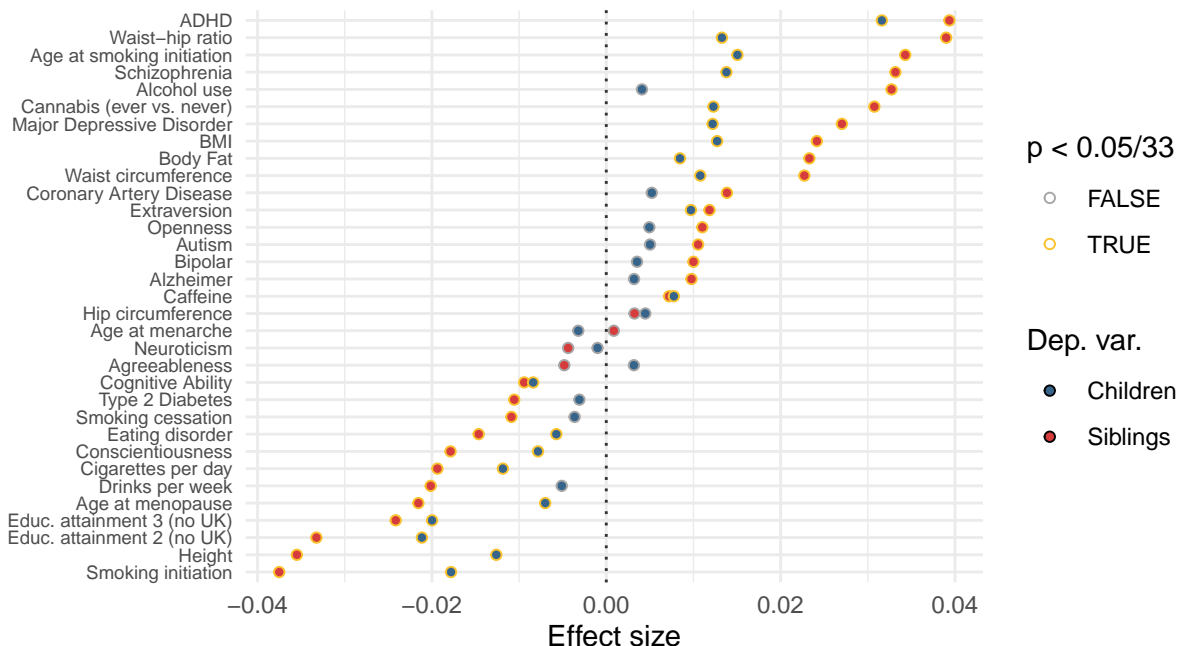


Figure 8: Selection effects, respondents' parents vs. respondents

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 five PGS: autism, cognitive ability, EA2, EA3 and extraversion. These changes are inconsistent with the intergenerational change, where 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 effect sizes 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 effect sizes between early and late born respondents (regressions on number of children)

Change	Number of scores
Change sign	3
Insignificant	25
Size increasing	5

Significance is measured at $p < 0.05/66$

Controlling for principal components

Polygenic scores could capture effects that are really due to population stratification, although would not change our results for natural selection of the scores. Figure 9 shows selection effects on polygenic scores residualized for the top 100 principal components of the genetic data, calculated within the UK Biobank population.

In siblings regressions, effect sizes are smaller when residualizing for principal components – sometimes much smaller, as in the case of height. 25 out of 33 “controlled” effect sizes have a smaller absolute value than the corresponding “raw” effect size. The median proportion between raw and controlled effect sizes is 0.89. 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).

To get a further insight into this we regress *siblings* and *children* on individual principal components. As Figure 10 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 by number of partners, controlling for age

Figure 11 shows selection effects by lifetime number of sexual partners. Age, age squared, and interactions of age and age squared with PGS are controlled for.

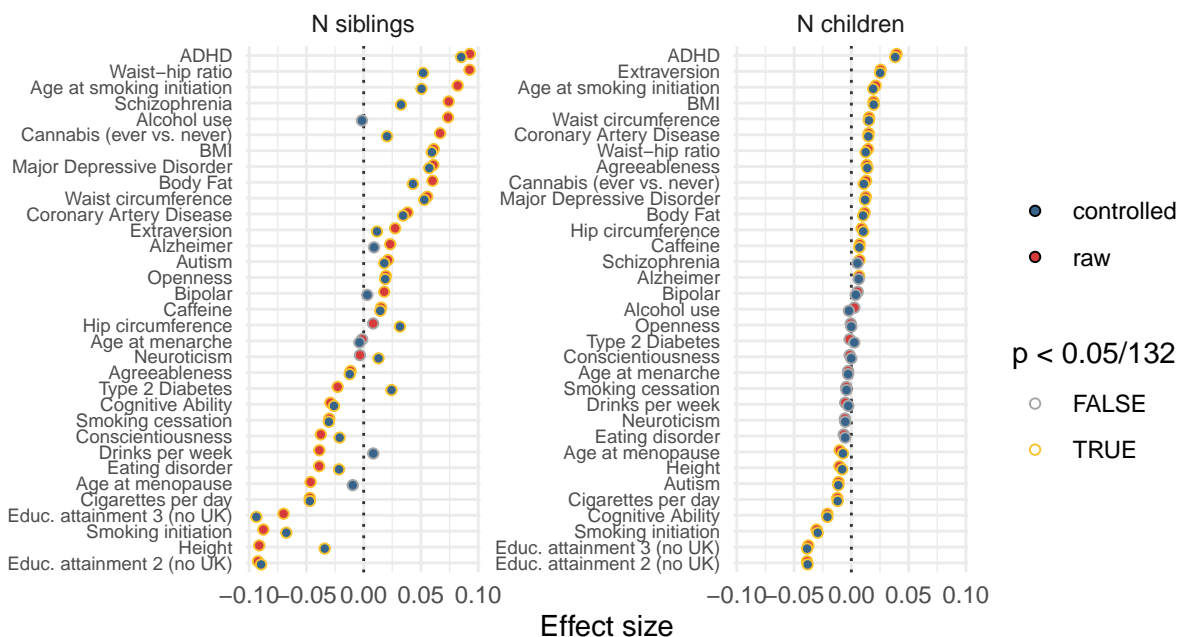


Figure 9: Effects of residualized polygenic scores on number of siblings/children.

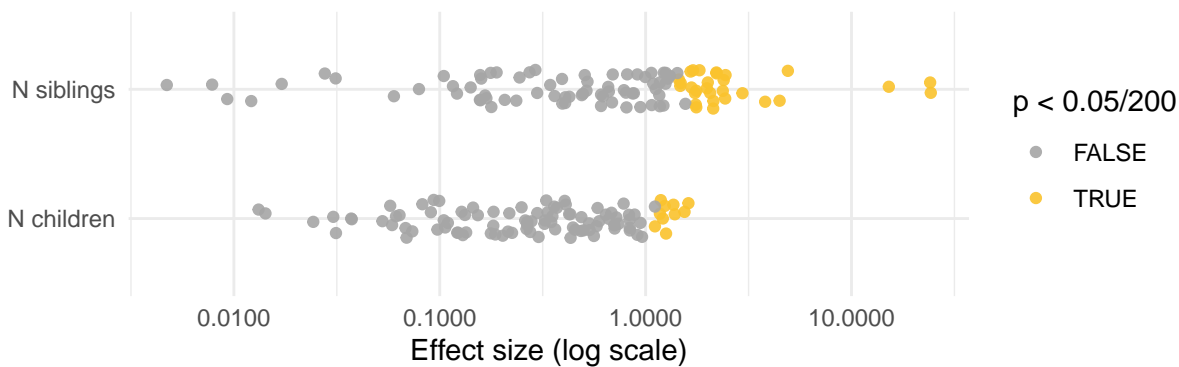


Figure 10: Effect of principal components of genetic data on number of siblings/children. Absolute effect sizes are plotted. Each dot represents one bivariate regression. Points are jittered on the Y axis.

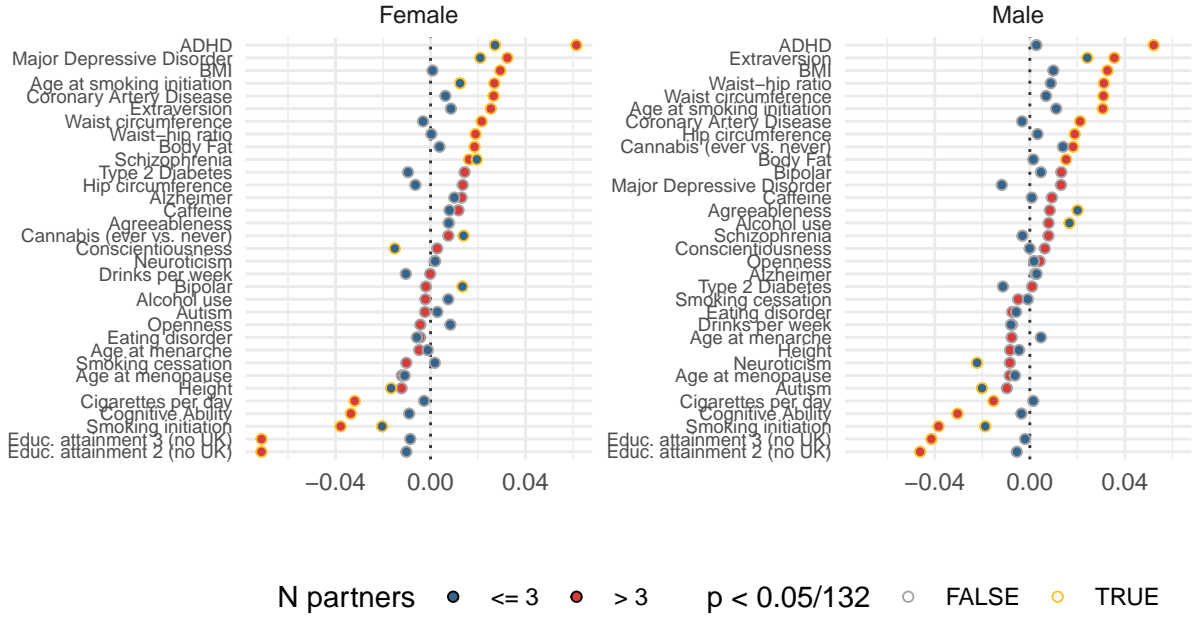


Figure 11: Effect sizes on number of siblings interacted with number of sexual partners (median split at 3), respondent's age and age squared.

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, controlling for either parent's age at their birth. Figure 12 shows the results. Effect sizes are very similar, whether controlling for father's or mother's age. Unlike for the respondents' generation, effect sizes are positively correlated with the effect sizes from bivariate regressions without the age at birth control (father's age at birth: ρ 0.51; mother's age at birth: ρ 0.57).

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 13 plots estimated effect sizes from bivariate regressions for respondents, and Figure 14 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

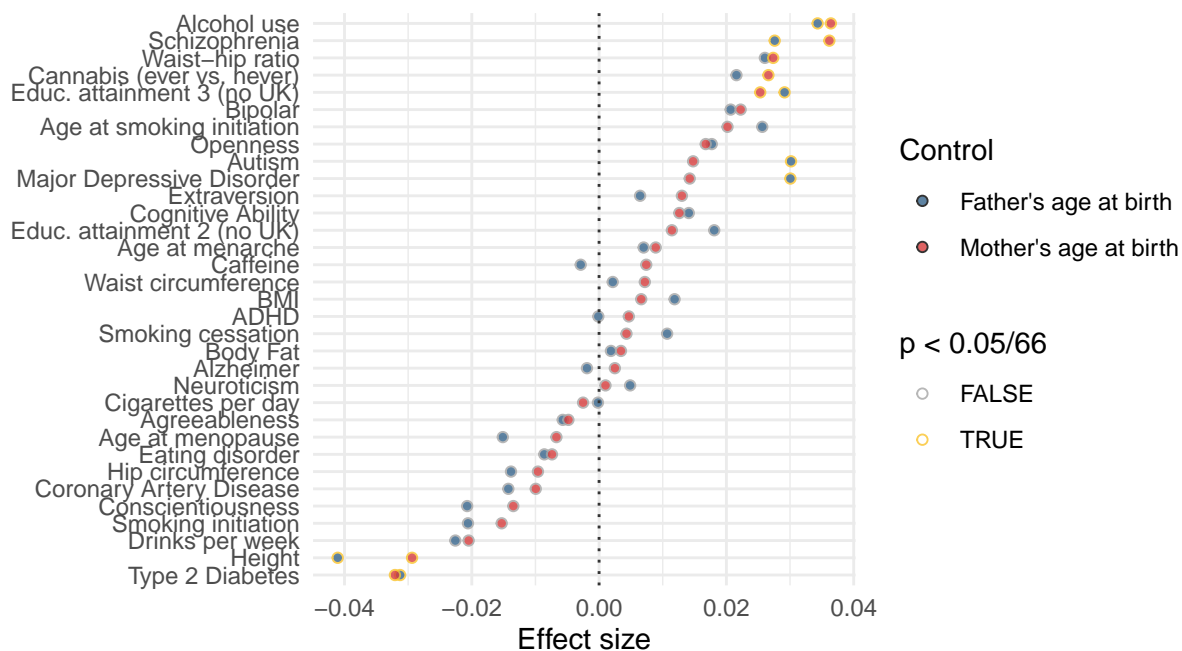


Figure 12: Selection effects controlling for parents' age at birth, eldest siblings

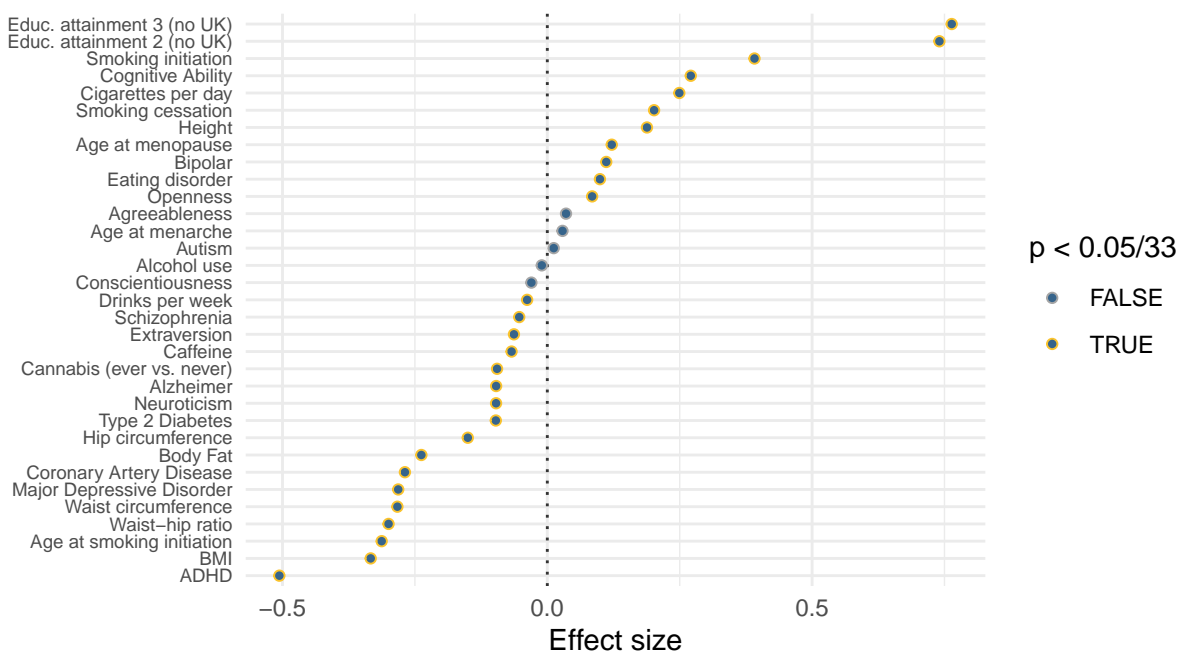


Figure 13: Effects of polygenic scores on age at first live birth

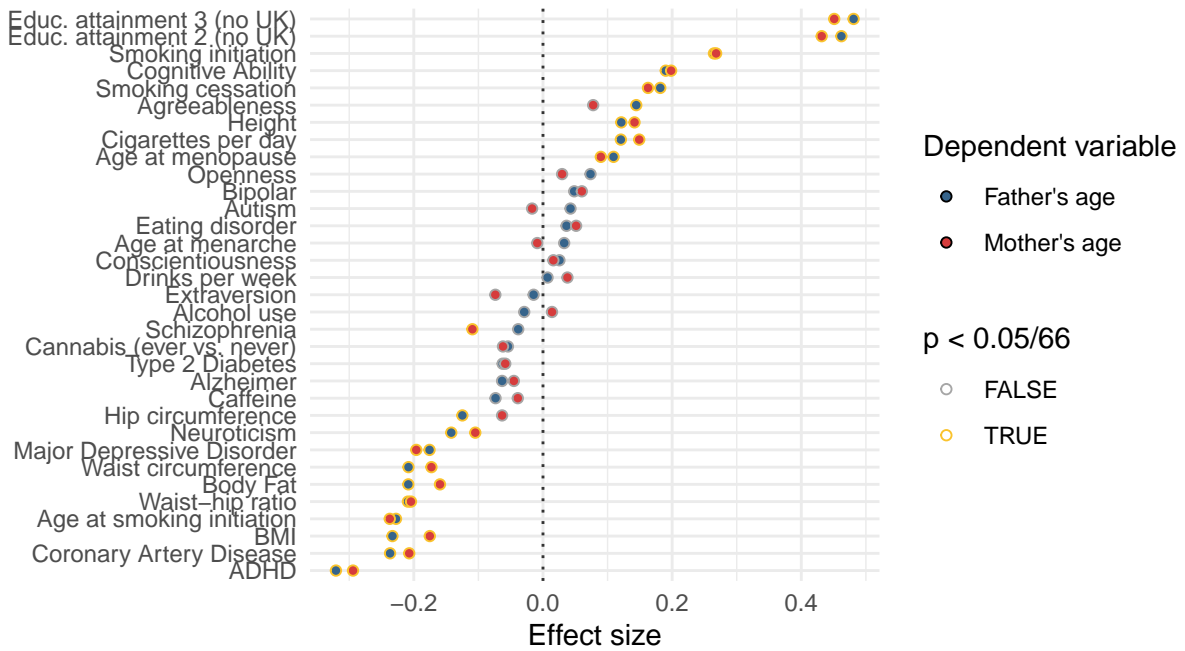


Figure 14: Effects of polygenic scores on parents' age at own birth, eldest siblings

Controlling for earnings and education

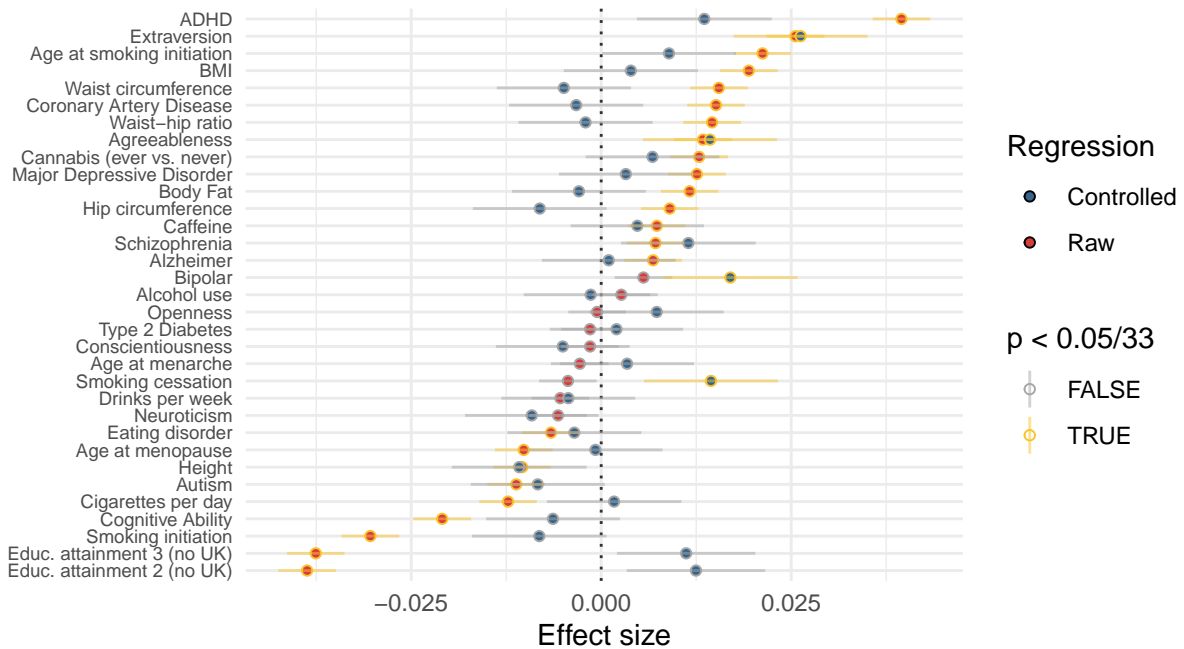


Figure 15: Selection effects controlling for earnings in first job (estimated by median earnings from ASHE 2007 for the SOC2000 job code) and education (left education before 16, 16-18, or after 18). Raw effects are shown for comparison. Lines are 95% confidence intervals uncorrected for multiple testing.

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 16 shows this is so.

TODO: ref source of data.

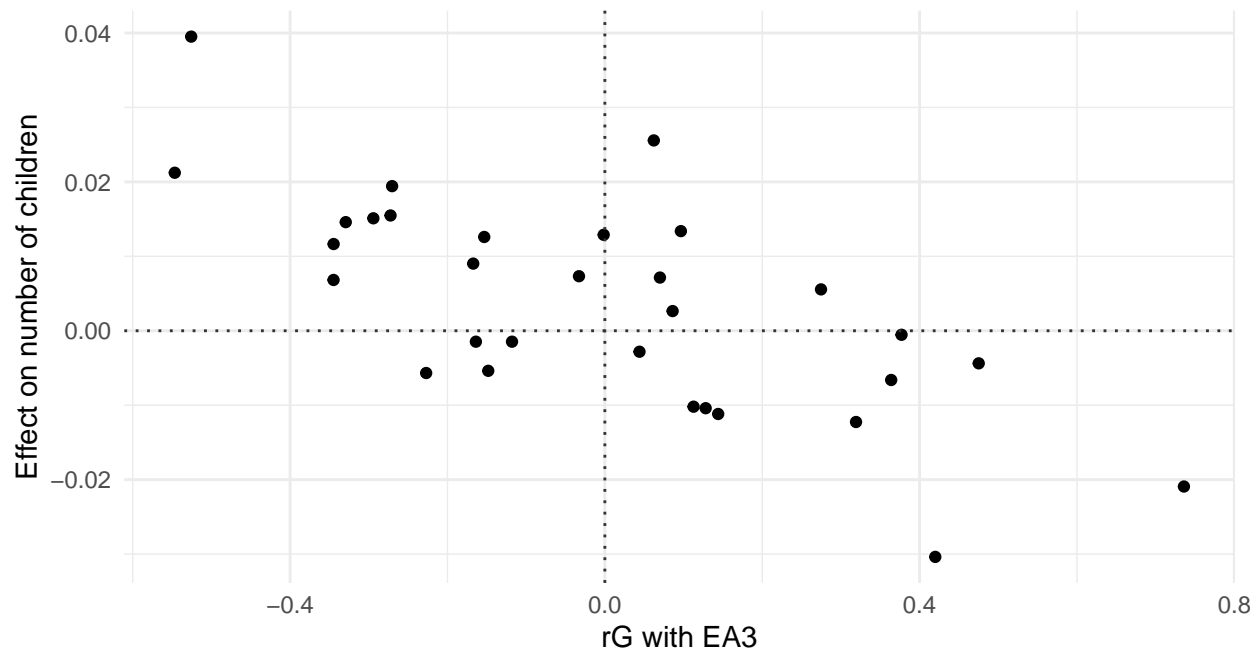


Figure 16: Effect size on n siblings by genetic correlation with EA3

Number of children

Figure 17 shows the full distribution of number of children born for different ventiles of the EA3 polygenic score. The strongest relationship seems to be for having 0 children versus 1 or more.

Causality

Different polygenic scores are correlated. Table 4 shows the top correlations in the sample. Because of this, bivariate correlations between PGS and number of children might be driven by other genetic scores. To explore which polygenic scores are driving negative selection, we run a single omnibus regression of *number of children* on all the PGS. We exclude EA2, waist-hip ratio, waist-circumference, and “Hip combined” since they are highly correlated with other scores, which could make our estimates unstable. Figure 18 shows the results. Interestingly, several PGS remain independently significant, although effect sizes are reduced.

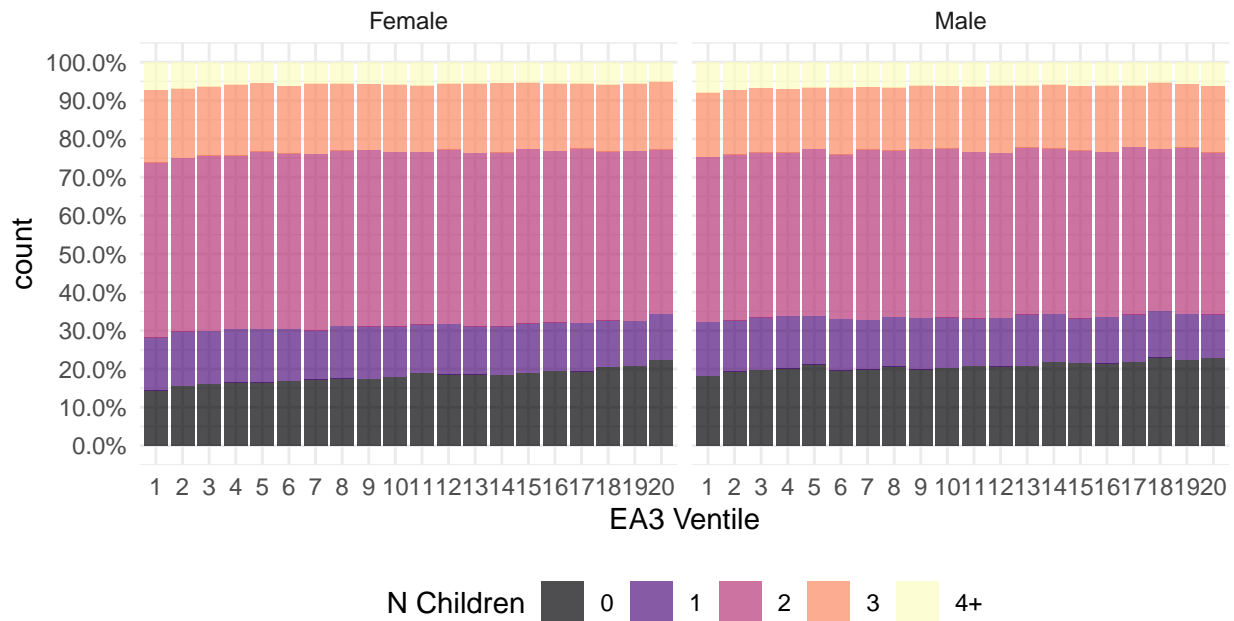


Figure 17: Number of children by ventiles of EA3 PGS

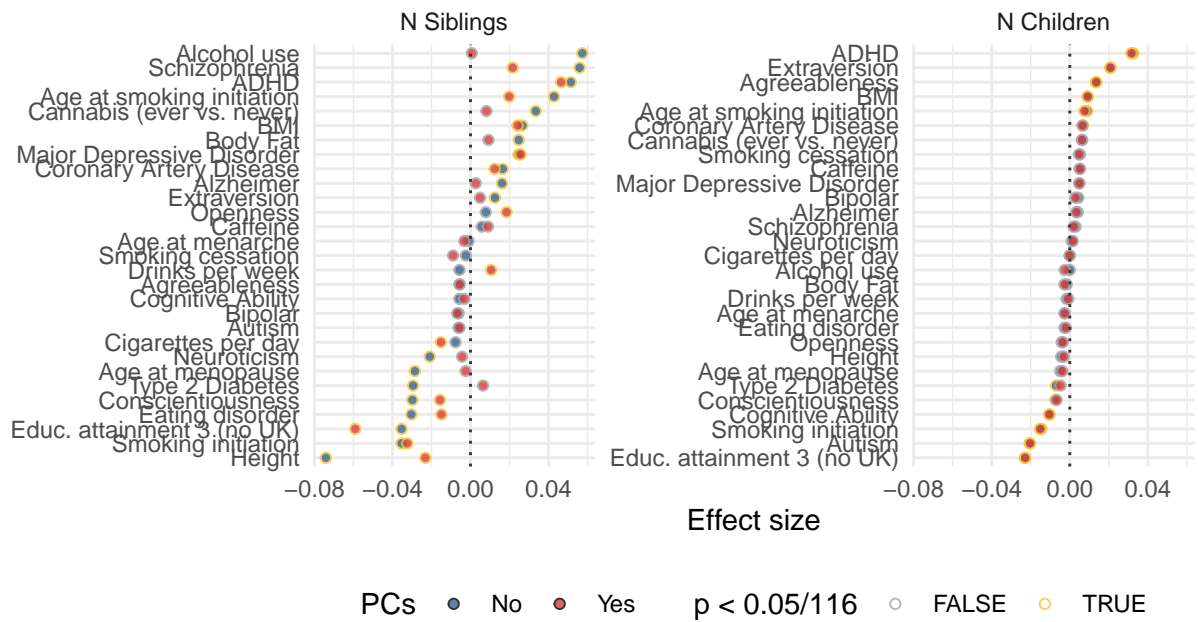


Figure 18: Partial correlations with number of children

Table 4: Top 10 correlations between polygenic scores

PGS	PGS	Correlation
Educ. attainment 2 (no UK)	Educ. attainment 3 (no UK)	0.89
Hip circumference	Waist circumference	0.807
BMI	Waist circumference	0.753
Waist circumference	Waist-hip ratio	0.711
BMI	Hip circumference	0.697
Body Fat	Waist circumference	0.435
BMI	Body Fat	0.425
BMI	Waist-hip ratio	0.425
Body Fat	Hip circumference	0.385
ADHD	Autism	0.328

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