# Fertility, Genes, and the Sexual Revolution*

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**Abstract**

Fertility has a genetic component typically neglected by economists. Using data from the UK Biobank, we analyze the extent to which genes (measured by polygenic indexes) and the environment (measured by birth cohort) affect age at first birth, completed fertility, and the probability of ever having had a child. There is evidence of gene-by-environment interactions, with more recent cohorts displaying greater genetic heterogeneity across all outcomes. A decomposition analysis shows that greater exposure to contraceptive pill usage plays a key role in mediating these cohort effects. Leveraging exogenous variation in genetic endowment across sisters and mother-daughter pairs in family fixed effects models, we find that the pill diffusion increases the age at first birth, particularly for women with greater genetic predisposition to delay motherhood. The results suggest that genes are more effective in socially more progressive environments.

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# 1 Introduction

Understanding fertility decisions has been at the center of economic research since the 1960s, paving the way to the analysis of some of the most profound transformations of the last 100 years which link fertility to female education, life cycle labor supply and wages, improvements in home production technology, child health and well-being, economic growth, and female empowerment.[1] Although many studies have hinted at the relevance of genetic endowments to reproductive fitness, none of them explicitly examined the interplay between genetic markers and socioeconomic factors in shaping female fertility behavior. Ours is the first paper that integrates sociogenomics into the economics of fertility.

Most of what we know that relates human genetics (nature) and environment (nurture) to fertility behavior comes from heritability studies (e.g., Mills et al., 2021).[2] These models, however, do not tell us much about how genes are associated with fertility behavior, other than providing an estimate of the proportion of the variation in the outcome of a decision that is attributable to genetic differences. Moreover, by suppressing the role played by genes, heritability studies cannot go beyond the nature versus nurture distinction, which is known to oversimplify, and mischaracterize our understanding of, behavior (e.g., Houmark et al., 2024). Although genes are fixed at conception, the environment where individuals live, which may influence their decisions, is likely to change, giving rise to gene-by-environment interaction ($G \times E$) effects on fertility outcomes. Exploring whether genetic predispositions interact differently with different environmental factors can reveal new heterogeneities and offer insights into the evolution of inequality in reproductive behavior. This is one of main contributions of our study.

Specifically, we analyze fertility behavior of British women born from the late 1930s to the late 1960s. Leveraging detailed molecular genetic data from the UK Biobank (UKB), one of the world's largest publicly available genomic resources, we measure genetic endowments using outcome-specific polygenic indexes (PGIs). Variations in each of these indexes have been shown to predict a wide range of fertility traits and choices (Barban et al., 2016). We focus on three outcomes (i.e., age at first birth, completed fertility, and the probability of having ever had a child), which offer a comprehensive picture of women's decisions over their reproductive life cycle. Our proxy of environment is year of birth. This represents a broad measure of the changes faced by women in the UKB, as they went through very different socioeconomic circumstances at the same point in their life (and fertile) cycle.[3]

[1] Some of the pioneering contributions are Becker (1960, 1981); Becker et al. (1990); Galor and Weil (2000); Goldin and Katz (2002); de la Croix and Doepke (2003); Greenwood et al. (2005). For a recent extensive survey, see Doepke et al. (2023).

[2] Until recently, such studies relied on twins or related individuals (e.g. Sacerdote, 2007; Fagereng et al., 2021).

[3] Earlier contributions that use birth cohort as a measure of the changes in the environment which can

Genetic differences across women have a large positive impact on all outcomes, and so do birth cohort differences. A 10-year increase in year of birth implies a rise in the age at motherhood of 1.2 years, a reduction in the number of children ever born of 0.2, and a decline in the probability of having ever had a child of 6 percentage points.

For the first time, we show that the $G \times E$ interactions amplify the observed cohort effects, and that genetic influences vary depending on the position women have in the PGI distribution. For example, across the 30 cohorts of the UKB, women with a low genetic predisposition to delay motherhood (i.e., in the bottom decile of the PGI distribution) see a small increase in the age at first birth from about 23.5 to just over 24 years. Women in the top decile of the PGI distribution face a postponement nearly five times larger, from 25 to almost 30 years of age. Similar significant widening gaps by PGI intensity emerge for the other two outcomes. In the case of the probability of having ever had a child, for instance, a 10 year increase in the year of birth is associated with a 3 percentage point reduction if a woman has a high genetic predisposition to have children and a 9 percentage point reduction if she has a low predisposition. This evidence suggests that genetic influences on fertility have likely grown in importance for more recent cohorts, whose social norms and economic opportunities might have been more conducive to female emancipation (e.g., Doepke et al., 2023). These results do not emerge among men, whose genetic propensities might have surfaced when environmental risks were less advantageous to female (but not male) susceptibilities.

To gain a deeper understanding of the gene-by-environment interactions, we perform a decomposition analysis where we identify three factors that could mediate the cohort effects. The factors, all defined at the district-year level, are exposure to environments with different contraceptive pill usage, exposure to milieux characterized by different shares of women with university (or higher) degrees, and exposure to contexts with different shares of women employed in highly skilled jobs.[4] Many studies have documented the impact of these factors on fertility and female economic progress (e.g., McCrary and Royer, 2011; Buera and Kaboski, 2012; Eckstein et al., 2019).[5] We find that the reinforcements of motherhood delay and of the increase in childlessness driven by the cohort effects are powerfully mediated by pill exposure. The other two mediators play a more limited role.

modify the influence of genetic risk factors include, among others, Rosenquist et al. (2015), Domingue et al. (2016), and Herd et al. (2019).

[4]More precisely, pill exposure is defined by the share of pill users among all childless women aged 18 or more in the same local authority district (LAD) of birth as the focal woman and matched to the calendar year in which she was 18 years old. College exposure is the proportion of women with a university (or higher) qualification by LAD and year of birth. High-occupation exposure is the share of women aged 18–30 and in the same LAD as the focal woman whose first job required a high-skill content and linked to the year when she was 18 years old. We shall come back to these measures with greater detail in Section 3.

[5]Female emancipation may have been facilitated by other factors, such as the legalization of abortion (Myers, 2017) and the rise of feminism (Goldin and Katz, 2002). Demand-side factors, such as equal pay and sex discrimination legislations, could have played a crucial role too (e.g., Bailey et al., 2024). Analyzing the influence of these additional channels is interesting but left for future research.

Changes in the diffusion of contraceptive pill across cohorts account for about half of the observed delay of age at first birth, and for the entire change in the probability of remaining childless. These factors, however, do not contribute in explaining the observed decrease in completed fertility. In addition, we find that pill diffusion mediates the effect of birth cohort across the entire genetic distribution, suggesting that the diffusion of the contraceptive pill did not overshadow the role of genetic endowments.

In further analysis, we take advantage of random genetic variation between first-degree relatives, either sisters or mother-daughter pairs. In both cases, the degree of genetic variation is reduced in comparison to what we observe over the whole population, while the differences in the external environment are again relatively small among sisters but larger between mothers and daughters. Our fixed effects results confirm that growing up in a more progressive environment magnifies the genetic predisposition to postpone motherhood, intensifying preexistent differentials across women. Overall, our findings are consistent with the notion that the profound shifts in attitudes towards egalitarian gender roles and personal freedom, something we refer to as the the sexual revolution of the 1960s and 1970s, interacted with individual genetic predisposition, amplifying diversity in reproductive behavior. To support this interpretation, we provide fresh descriptive evidence of the relationship between gender role norms and pill exposure.

The interpretation of the gene-by-environment interplay we give in this context is in the spirit of a social control mechanism, according to which genetic influences are mediated by structural constraints, social norms, and — more broadly — culture (e.g., Guiso et al., 2006; Bisin and Verdier, 2011). Social norms, such as those shaped by the sexual revolution, are expected to channel genetic influences into fertility behavior, either magnifying or mitigating their impacts on outcomes. Evolving more rapidly than genes, culture can create different environments that expose genes to new selective pressures and adaptations (Richerson et al., 2010), which in turn may enhance or inhibit female reproductive fitness. The association of genetic influences on fertility outcomes can then be viewed as a proxy of success of genetic predispositions, and, to the extent that social norms enable women to postpone births or not to have children at all, the influence exerted by genes is likely to be higher in areas and times that allow for a variety of alternative behaviors (see, among others, Engzell and Tropf, 2019; Herd et al., 2019).

Our paper adds to the extensive literature on the economics of fertility mentioned at the very start, integrating the new sociogenomic approach into the main analysis for the first time. This is important in and of itself, given the recognized, but largely untested, significance granted to the biological architecture of human reproduction. An additional, distinct contribution is that we identify meaningful $G{\times}E$ effects and provide an innovative interpretation based on culture.

This, in turn, gives us an opportunity to speak to the burgeoning economic literature that uses polygenic indexes to identify gene-environment interactions. A recent overview

can be found in Biroli et al. (2022). Most of this research focuses on education, wealth, and health, and uses PGIs for educational attainment, smoking, or body mass index. For instance, Barcellos et al. (2018) show that genes moderate the effect of education on health, whereby education-driven improvements in weight are larger for individuals with high genetic predisposition to obesity. Ronda et al. (2022) find that the educational returns to genetic endowments are attenuated by childhood disadvantage, meaning that children who experience childhood disadvantage are unable to realize their full educational potential (see also Papageorge and Thom, 2020). Parents are found to invest in their children differently, depending on a child's genetic endowment (e.g., Sanz-de Galdeano and Terskaya, 2023; Houmark et al., 2024), and there is evidence of complementarities in skill formation (Muslimova et al., 2020). Barth et al. (2020) document that genes are more strongly related to wealth among individuals who have greater autonomy over their financial decisions, while those with lower educational genetic endowments are likely to benefit from outsourcing their investment decisions.[6] Our study opens up this research strand to fertility choices.

The remainder of the paper is organised as follows. Section 2 introduces basic concepts of molecular genetics and the main genetic quantities used in our study. Section 3 presents the data and descriptive analysis, while Section 4 shows the basic evidence on the gene-by-cohort interactions. Section 5 goes deeper into understanding the interplay between genes and environment presenting a mediation analysis, family fixed effects models, and results on male fertility. It also provides evidence of the association of our environmental mediators with gender role norms. Section 6 concludes.

# 2 Measuring Genes

Recent advances in molecular genetics and massive improvements in technology have made it cost effective to measure millions of genetic variants in the human genome for large samples of individuals (Conley and Fletcher, 2017). To analyze the impact of genes directly, a standard approach in the recent sociogenomic literature is to summarize all of an individual's genetic endowment in a so-called polygenic index (e.g., Papageorge and Thom, 2020; Houmark et al., 2024).

Polygenic indexes (PGIs), also known as polygenic scores, are numerical values that summarize the estimated genetic contribution to a particular fertility outcome (often referred to as phenotype or trait), based on an individual's genetic makeup. Formally, for each individual $i$, a PGI is the weighted average of a distinct genetic variant, known

[6] Black et al. (2020) find instead little evidence of gene-environment interactions to explain the inter-generational transmission of wealth. Their work uses a sample of adoptees, but no genomic data. Using a subsample of adoptees in the UKB and polygenic indexes to proxy genetic endowments, Cheesman et al. (2020) instead find strong gene-environment influences.

as single nucleotide polymorphism (SNP), weighted by the empirical association between the phenotype itself and the same SNP at each genetic site, i.e.,

$$\text{PGI}_i = \sum_{s=1}^{S} w_s a_{is}, \tag{1}$$

where $a_{is} \in \{0, 1, 2\}$ is the allele count for each SNP $s$, $S$ is the total number of observable SNPs in the data, and $w_s$ is a weight given by the genome-wide association study (GWAS) regression coefficient.[7] For each individual $i$, therefore, the PGI is a scalar, which can be interpreted as a measure of an individual's genetic association with the phenotype relative to the population. The range of possible values of a PGI depends on $S$ and converges to a normal distribution if the number of independent SNPs is sufficiently large. We follow common practise and standardize each PGI by subtracting its mean and dividing it by its standard deviation.

A few remarks are in order. First, genetic variants are not randomly observed across genetic addresses. Their occurrence varies according to a block structure, known as linkage disequilibrium (Nei and Li, 1973). Several methodologies have been proposed to account for this feature. We follow the approach introduced by Privé et al. (2020), which re-weights GWAS summary statistics using linkage disequilibrium between SNPs from a reference data, in order to avoid the potential bias due to the correlation structure of genetic variants.

Second, PGIs are ancestry specific and not portable across populations because the genetic architecture underlying each trait can vary significantly between different ancestral groups. This variation is due to differences in linkage disequilibrium patterns, which affect how genetic variants are associated with each other, and ancestry-specific allele frequencies, where certain genetic variants may be common in one population but rare or absent in another (Duncan et al., 2019). For this reason, we restrict our focus on individuals of European ancestry, and cannot generalize our findings to individuals of non-European ancestry (see also Campbell et al., 2005; Martin et al., 2017). To adjust for population stratification directly, our analysis will also include a series of principal components of the genetic data, which explicitly account for ancestry differences, minimize the influence of spurious associations, and maximise statistical power.

[7] A GWAS scans the entire genome for SNPs associated with a particular outcome. This is done through linear regression models where the outcome of interest is regressed on SNP counts and it is iterated for all the (millions of) SNPs available in the data. Results are corrected for multiple testing, where the convention is of one million independent tests, leading to a standard GWAS statistical significance threshold of $10 \times 10^{-8}$. Our work is based on the results from a large GWAS meta-analysis on reproductive behavior by Barban et al. (2016) and its follow-ups (Mills et al., 2021; Mathieson et al., 2023). Evidence presented in these studies implicates mechanisms related to reproductive health, puberty timing, and evolutionary fitness in the biological pathways that link the indexes to fertility phenotypes. Pioneering contributions in social science research include Beauchamp et al. (2011), Benjamin et al. (2012), and Okbay et al. (2016).

Third, if the weights $w_s$ are derived from the same data used to perform the GWAS, overfitting will likely lead to biased estimates (Wray et al., 2013). To avoid this problem, we use PGIs from the Polygenic Index Repository which divides the sample into three equally sized subsamples and, for each partition, the summary statistics of the other two partitions are used as independent weights in the calculation of expression (1) (for further details, see Becker et al., 2021).[8] We thus end up with an estimating sample that is one-third of the original UK Biobank population.

Fourth, we use the PGIs based on the GWAS for age at first birth and number of children ever born (Barban et al., 2016). These indexes are based on sex-specific meta-analyses of genome-wide associations computed on individuals with European ancestry. To control for secular changes in fertility behavior, the GWAS analysis protocol includes quadratic polynomials in year of birth as controls. As a sensitivity exercise, we replace the two fertility-specific PGIs with the PGI for educational attainment (EA) based on the most recent GWAS by Lee et al. (2018). All three indexes are included in the Becker et al. (2021)'s repository.

There are important advantages regarding the use of polygenic indexes as direct measures of genetic endowment in our context. We emphasize two. The first recognizes that fertility outcomes have extremely high degrees of polygenicity, consistent with a genetic architecture that may be influenced by negative selection (O'Connor et al., 2019). PGIs therefore acknowledge that each individual falls on a continuum of genetic predispositions which result from small contributions of multiple genetic variants (Mills et al., 2021). The second advantage is that PGIs allow us to be agnostic about the precise biological processes underlying its corresponding phenotype, especially because this is likely to be determined by 'distal' genetic influences scattered across the entire genome (Belsky and Israel, 2014).

# 3 Data

## 3.1 The UK Biobank and Sample Selection

The UK Biobank (UKB) is a population-based prospective study established by the UK National Health Service (NHS).[9] Between 2006 and 2010, invitations were mailed to 9.2 million NHS registered individuals aged 40–69 (born between 1934 and 1971), who lived up to 25 miles from one of 22 study assessment centers throughout the UK. A sample of half a million individuals agreed to participate (implying about 5.5% response rate) of which around 270,000 were women. As part of the survey, study participants

[8] In the main analysis, we use the partition UKB3 based on a random sample of individuals with no third degree or closer relatives.

[9] For a detailed description of the data as well as access and governance issues, see Allen et al. (2012).

went through an assessment that included: a self-completed touch-screen questionnaire, a computer-assisted interview, the collection of physical and functional measures, and the collection of blood, urine, and saliva samples. All physical and medical measures (e.g., anthropometrics and blood pressure) were gathered by trained nurses or healthcare practitioners.

Every participant in the study was genotyped. This makes the UK Biobank one of the largest publicly available genetic resources in the world. Although the survey has limited information on respondents' socioeconomic background and most of the information on family environment is available retrospectively, it provides geo-coordinates for both place of birth and current residence at interview, with one kilometer grid resolution.[10] We assign respondents with available geographical coordinates to their region and local authority district (LAD) both at birth and at the time of interview.[11] As shown in Appendix Figure A1, the geographic distribution of the respondents' area of birth is widespread across the country.

To address the selection bias induced by volunteers' participation in the study, we adopt the weighting procedure proposed by van Alten et al. (2022), which uses inverse probability weights based on UK Census microdata.[12] This approach corrects the UKB sampling based on the proximity to recruitment centers, and reweighs the sample to be nationally representative. Because participants to all biobanks (not just the UKB) are usually healthier than the general population, these studies may suffer from a healthy-volunteer bias. van Alten et al. (2022) show that their weighting strategy reduces this source of bias by up to 80%.

In the analysis, we exclude respondents from Northern Ireland (who represent 6.2% of the whole sample), those born outside the British Isles (7.7%), and those for whom the area of birth is missing (0.16%). We also exclude individuals born in 1934–37 and 1970–71, since only few respondents were born in those years (a total of 1.3% of the original sample), making cross-cohort comparisons problematic. As already mentioned, we restrict our attention to individuals with European ancestry to adjust for population stratification, and we consider only one-third of the original UKB sample to avoid overfitting issues, focusing on women with no third-degree or closer relatives. These restrictions lead to a final sample of approximately 68,500 unrelated women.

[10]The grid coordinate data are provided in the British National Grid (i.e., OSBS 1936) projection. OSGB1936 is the Ordnance Survey National Grid geographic reference system used in Great Britain.

[11]Our definition of local authority district is based on the 2018 Census boundaries, which leads to 380 LADs. The data are available from the Office for National Statistics (England and Wales), the National Records of Scotland, and the Northern Ireland Statistics and Research Agency.

[12]Selectivity bias can arise because of the possible correlation of genetics (and thus PGIs) to participation bias Schoeler et al. (e.g., 2023) and to mortality bias as shown in the Health Retirement Study by Domingue et al. (2017).

## 3.2 Fertility Outcomes and Descriptive Statistics

The UKB collects fertility histories, in which each woman reports her age at first live birth and the total number of children she ever had. Based on this information, we construct the following three outcomes: age at first birth (A1B); number of children ever born or completed fertility (NEB); and ever had children (EVER), which is the complement of childlessness. To minimize right censoring issues for NEB and EVER, the sample is restricted to women who are aged 45 or more at interview.

As Britain went through profound socioeconomic transformations since World War II, so did the reproductive behavior of the women in the UK Biobank sample (e.g., Guinnane, 2011). Appendix Figure A2 displays the time trends in our outcomes by birth cohort. First births have been postponed by more than three years (from nearly 24.5 up to 27.5 years) over the sample period. As women in the early birth cohorts had on average 2 or more children, those in more recent cohorts had fewer than 1.6 children.[13] Finally, the fraction of women who ever had a child decreased from about 88% among women born at the start of the UKB to almost 70% among those born in the late 1960s.[14]

Table 1 shows summary statistics for the three outcomes and standardized PGIs. It also reports descriptive statistics on three environmental moderators, which we describe in the next section, and respondents' own early life conditions, including their self-reported birth weight (in kilograms), smoking status of their mothers during pregnancy, and whether they were breastfed. These measures have been used to proxy individual socioeconomic background and shown to be correlated to later outcomes (e.g., Almond et al., 2018). We shall use them in a series of robustness checks.

Since one of our main objectives is to investigate the extent of gene-environment effects on fertility taking a broad stand on what constitutes $E$, that is, defining it as calendar time, we check whether our PGIs are stable across cohorts. We explore this issue by separately regressing each PGI on birth cohorts (grouped in five-year bands), controlling also for the first 10 principal components of the full matrix of SNP data to account for population stratification. The results are in Appendix Table A1. As mentioned in Section 2, the GWAS regressions underpinning the polygenic indexes include quadratic polynomials in years of birth as a way to account for secular changes in fertility behavior and possible selection bias in the discovery sample. Despite this, the first three columns of the table show that the PGIs are significantly associated with some of the most recent cohorts. For A1B, six (out of the 15) pairwise cohort group comparisons show statistically significant associations with the polygenic index. This result, which emerges also for

[13] Some women could have had more children after the last available interview. This, however, will not change the general picture given in Figure A2.

[14] Although the first in vitro fertilization (IVF) baby was born in 1978, it took a long time before the treatment became widely available (Zhao et al., 2011) and its success rate was initially low (Lundborg et al., 2017). In the UKB, only 31 women gave birth through IVF, that is, less than 0.02% of the births in the sample.

five of the comparisons for NEB and EA, is consistent with the findings reported in Beauchamp (2016) and Kong et al. (2018), suggesting a small cross-cohorts variation in genetic endowments.[15]

To overcome this issue and ground our analysis on time-invariant genetic predispositions, we adjust each PGI by birth cohort and replicate the previous exercise. The new estimates in the last three columns of Appendix Table A1 show that the adjusted indexes are no longer associated with birth cohorts. In the rest of the analysis, therefore, we use this adjusted standardized version of each of the PGIs as our measure of genetic endowment. The kernel-smoothed densities of the indexes are plotted in Appendix Figure A3. We cannot reject the null hypothesis that they are normally distributed.

Finally, polygenic indexes for different outcomes are likely to pick up similar genetic material, given non-negligible phenotypic overlaps. This is reflected by the genetic correlations reported in Appendix Table A2, which shows also the cross-correlations of all outcomes. As expected, NEB is negatively correlated with A1B, and its PGI is also inversely correlated to the polygenic index of A1B. The genetic correlation of education and age at first birth is the highest (0.90), followed by a correlation of –0.62 between PGI EA and PGI A1B.

# 4 Gene-by-Cohort Interplay on Fertility

We now examine the influence of genes on fertility and how this varies across women's birth cohorts, using year of birth as the broad measure of the environment in which women make their decisions. Figure 1 displays the trends in age at motherhood (A1B), number of children (NEB), and ever had children (EVER) across 5-year cohorts by two groups of women, those in the top decile and those in the bottom decile of their PGI distributions.[16] Specifically, women in the top decile of PGI A1B are referred to as late-fertility-$G$ individuals and those in the bottom decile as early-fertility individuals. Women in the bottom decile of PGI NEB are instead labelled low-fertility-$G$ women, while those in the top decile as high-fertility-$G$ individuals.

Figure 1 confirms the aggregate trends reported in Appendix Figure A2. It also illustrates important differences by genetic endowment. Women with early fertility $G$ experience a postponement of motherhood of about seven months, with their age at first birth going from 23.5 years among those in older cohorts to 24.2 years among those in recent cohorts. Late-fertility-$G$ women, instead, have their first birth at age 25.5 if they come from early UKB cohorts and at age 29.5 if they are from later cohorts. Women with late fertility $G$ show not only a higher age at motherhood but also an increasing trend over

[15] The variation is quantitatively modest and accounts for only up to 7% of a standard deviation of each polygenic index.

[16] More precisely, these are means conditional on birth cohorts and PGI deciles.

birth years. Mirroring patterns emerge for NEB and EVER. In particular, the difference in NEB between women with low- and high-fertility-G is 0.5 and insignificant among older cohorts and 0.7 and significant among younger cohorts. Similarly, the corresponding gaps in EVER are less than 10 and more than 20 percentage points, indicating a change in the share of women who ever had a child in excess of 100%. This evidence indicates a clear divergence in fertility choices among women with different genetic endowments, a difference which amplifies over time.

To test these differences by cohort more formally, we estimate the following model:

$$\begin{aligned} Y_i = \theta_0 + \theta_1 Cohort_i + \theta_2 G_i^Y + \theta_3 (G_i^Y \times Cohort_i) + \mathcal{X}_i' \boldsymbol{\delta} \\ + \sum_{p=1}^{10} \gamma_{0p} \mathrm{PC}_i^p + \sum_{p=1}^{10} \gamma_{1p} (\mathrm{PC}_i^p \times Cohort_i) + \varepsilon_i, \end{aligned} \tag{2}$$

where $Y_i$ is the fertility outcome for woman $i$, $G_i^Y$ is the outcome-specific polygenic index for individual $i$, and $Cohort_i$ is a linear trend in $i$'s year of birth.[17] We are interested in $\theta_3$, which reflects $G \times E$ interactions on fertility, where $E$ is captured by $Cohort$. $\mathcal{X}$ is a vector of controls, comprising a WWII indicator that equals 1 if a woman is born between 1939 and 1945, and 0 otherwise, and district of birth fixed effects. To account for population stratification, we use the first 10 principal components of the full matrix of SNP data, $\mathrm{PC}_i^p$, and their interactions with $Cohort$. These allow us to control also for the possible interplay between ancestral commonality and birth cohort, absorbing any potential stratification bias from the $G \times Cohort$ term.[18] Finally, $\varepsilon_i$ is an individual specific idiosyncratic error term.

Table 2 summarizes the results. Each column reports the estimates from equation (2) for a different fertility outcome, and inference is based on standard errors robust to heteroskedasticity. All the estimates in the table but one, i.e., $\theta_2$ in column (c), are statistically significant at the tighter $p$-value threshold of 0.005, which Benjamin et al. (2018) argue should be the standard of evidence for claims of new discoveries.

Genetic penetrance plays a key role in explaining the observed variation in all outcomes. A one-standard-deviation increase in $G$ is significantly associated with: (i) a postponement of age at first birth by 0.87 years; (ii) an increase in the total number of children born to a woman by 0.11; and (iii) a higher chance of ever having a child by one percentage point. The environment, proxied by $Cohort$, plays an equally important role. Being born 10 years later leads to: (i) a postponement of A1B by 1.2 years; (ii) a reduction in NEB by 0.2 children; and (iii) a 6 percentage point decrease in the probability of EVER.

A direct way of assessing the interplay between genes and environment is to focus on

[17] Given the linear patterns observed in Figure 1 and Appendix Figure A2, we opted for a simple linear cohort trend instead of higher-order polynomial specifications.

[18] The 10 PCs are demeaned before interacting them with $Cohort$ as suggested by Keller (2014).

the $G{\times}Cohort$ estimates. We find evidence of statistically significant gene-by-environment interactions for all outcomes. In particular, the postponement of age at first birth led by younger cohorts is reinforced by $G \times Cohort$. Conversely, the reductions in family size and in the probability of having ever had a child associated with more recent cohorts are weakened by the gene-by-environment interaction.[19]

This evidence lends support to the notion of environmentally mediated genetic effects on fertility-related behaviors, which have long been posited by standard economic theories of fertility (e.g., Becker, 1981), but never properly documented. It also suggests that genetic influences on fertility have likely grown in importance for more recent cohorts, whose social norms and economic opportunities might have allowed a broad range of life-course alternatives (see, among others, Kohler et al., 1999; Doepke et al., 2023). Put differently, older cohorts, whose early lives were affected by traditional gender norms and relatively limited economic prospects, seem to exhibit low levels of genetic influence. Later cohorts, which went through major changes in the environment they lived in (e.g., educational expansions and the sexual revolution), reflect stronger genetic effects. This is a new, intriguing result and it is the focus of the rest of the paper.

# 5 Understanding the Gene-by-Cohort Interplay

Using birth cohort as our measure of environmental risk allows us to encompass a host of socioeconomic factors, which might have affected fertility across the several cohorts represented in the UK Biobank. In what follows, we focus on three factors that a large economic literature has shown to be crucial determinants of fertility choices and that capture potentially different aspects of female empowerment and changing social norms. These are the availability of oral contraception, female educational attainment, and female employment in highly skilled jobs.

We perform three exercises. First, to quantify the contribution of each factor in mediating the cohort effect on fertility, we use a decomposition approach and test whether the mediating effects differ by genetic endowment. Second, to account for shared environmental risks, we estimate family-based models. Although the influence of genes is more limited in these models, they can help us to uncover the presence of $G \times E$ effects even when the variation in $G$ or the differences in $G$ and $E$ are more restricted than in the general population. Finally, we explore male fertility and assess if this is also characterised by a gene-by-cohort interplay and whether or not it is mediated by the same moderators mentioned above.

[19] All results are robust to the inclusion of $Cohort{\times}\mathcal{X}$, $G{\times}\mathcal{X}$, and $G{\times}$PC interactions. These additional estimates are in Appendix Table A3.

## 5.1 Decomposition Analysis

We start by decomposing the total effect of *Cohort* on $Y$ into a direct effect and an indirect mediator effect, and then we test if the indirect effect is different at different points of the PGI distribution. To do this, we use a moderated mediation approach, adapted from Hayes and Preacher (2013).[20] The term 'moderated mediation' underlines that $G$ moderates the pathway between the mediator and $Y$.

*Setup* — Let $\mathbf{M_i}$ be a vector of moderators that account for the birth cohort effect on fertility for each woman $i$. To establish that *Cohort* is related to a specific mediator, we estimate the following relationship, one for each moderator $j$:

$$M_{ij} = \kappa_j + \alpha_j Cohort_i + \mathbf{X_i}'\boldsymbol{\lambda} + \zeta_{ij}, \tag{3}$$

where $M_{ij}$ is the mediator $j$ ($j = \{1, 2, 3\}$) for woman $i$, $Cohort_i$ is the linear trend in year of birth for $i$, $\mathbf{X_i}$ is a vector of individual-specific controls, and $\zeta_i$ is an idiosyncratic error term.[21] The next equation shows the association of $Cohort_i$ with the outcome $Y_i$ when controlling for $G$, all the mediators $\mathbf{M}$, and the interactions between $G$ and $\mathbf{M}$, i.e.:

$$Y_i = \varsigma + \tau Cohort_i + \varphi G_i^Y + \sum_{j=1}^{J} \beta_j M_{ij} + \sum_{j=1}^{J} \mu_j G_i^Y M_{ij} + \mathbf{X_i}'\boldsymbol{\rho} + \xi_i. \tag{4}$$

A simplified graphical representation of the model is shown in Appendix Figure A4. This setup allows us to decompose the total effect of $Cohort_i$ on $Y_i$ into: (i) the direct effect, holding both genetic endowment and mediators constant, which is given by $\tau$ in model (4); and (ii) the indirect effects through each mediator $j$ at different values of $G$, that is, $\alpha_j(\beta_j + \mu_j G_i^Y)$ from both equations (3) and (4). We estimate the full model using a structural equation method and compute bootstrap standard errors with 1,000 iterations for the direct and indirect effects.

*Mediators* — As mentioned, we consider three mediators. The first, labelled $M_1$, is the change in availability of the modern oral contraception, the "contraceptive pill". Many identify the diffusion of the pill as an indicator of gender-equal norms and one of the main channels leading to female empowerment (Goldin and Katz, 2002; Bailey, 2006). A related literature documents the key role played by gender identity norms in fostering women's emancipation (e.g., Alesina et al., 2013; Bau and Fernández, 2023; Doepke et al., 2023). We thus take the diffusion of the pill and its associated gender norms as an environmental

[20] A recent mediation analysis in the field of genoeconomics is used by Abdellaoui et al. (2022) who study the association of birth order with a spouse's PGI and whether this association is mediated by socioeconomic status. See Huber (2021) for a review.

[21] In the analysis, $\mathbf{X}$ includes a WWII dummy, district fixed effects, the first ten demeaned principal components of the full matrix of SNP data and their interactions with the cohort linear trend.

measure relevant to female fertility decisions.[22]

The contraceptive pill was first introduced in the UK in 1961. Initially accessible only to married women, its availability was extended to everyone in England and Wales in 1967, in 1968 in Scotland, and in 1972 in Northern Ireland. Women in the UK Biobank are asked retrospectively whether they have ever used the pill and at which age they began taking it. For each local authority district (LAD) in the data, we compute the proportion of childless women aged 18 or above who ever took the pill. We then link this variable to each woman when she was 18 years old.

Panel A of Figure 2 shows the pill diffusion by birth cohort and region broadly defined. Contraceptive pill usage varies substantially across cohorts. Women born in the earlier years of the UK Biobank did not have access to the pill until they were in their mid-to-late twenties, while those from more recent cohorts faced an environment in which approximately 80% of the 18 years old in their area used the pill. Women born between 1950 and 1965, who represent a large fraction of those observed in the UKB, experienced the sharpest variation as pill usage rose from less than 10% to more than 70%. The data also reveal some (albeit smaller) regional variation, with London having pioneered pill usage, especially during the 1970s, and Scotland lagging 5–8 percentage points behind.[23]

The second measure of environmental influence is female college education, $M_2$. The growth in female schooling has been found to be a salient contributor to the reduced fertility observed over a considerable part of the twentieth century (e.g. McCrary and Royer, 2011; Eckstein et al., 2019). Women in the UK Biobank are asked to report their highest qualification. We use this information to construct the share of women with a college degree by LAD and year of birth, which is then matched to each woman in the sample.[24] Panel B of Figure 2 displays the evolution of $M_2$ across birth cohorts and broad geographic regions. We observe an increasing trend for the whole country, with large differences by region. About 10% of women born in the late 1930s have a college degree if they are from the North East, a relatively poor area of the UK. The share grows to 30% among women from the same region but born in the late 1960s. The corresponding figures for women from the wealthier East of England counties are 30% and 50%, respectively.

Our last mediator is the proportion of women working in highly skilled occupations, $M_3$. The UKB has a subsample of approximately 58,000 women with detailed employment

[22]Many commentators argue that the diffusion of the contraceptive pill is the single most important determinants of fertility change in Britain during the 1960s and 1970s (e.g., Murphy, 1993). As mentioned in the Introduction, other supply-side factors (such as the legalization of abortion) or demand-side factors (such as the introduction of sex discrimination legislation) might have played a role. They are not analyzed here. Notice also that the dates on respondents' abortions are not available in the UK Biobank.

[23]More spatial variation emerges by LAD, which is the geographic unit of analysis used in estimation.

[24]The UKB also asks individuals to report the age at which they completed their full time education. When the direct information on qualifications is missing, we define a woman as having a college degree if her age at completed education is 22 or above.

history information covering all the occupations they held. Each occupation is mapped to a 4-digit SOC2000 code which we group in nine main categories: managers and senior officials, professional occupations, associate professional and technical occupations, administrative and secretarial occupations, skilled trades occupations, personal service occupations, sales and customer service occupations, process, plant and machine operatives, and elementary occupations. Using the O*NET-SOC taxonomy, we then combine jobs that require low skills (elementary occupations), medium skills (administrative and secretarial occupations, skilled trades occupations, personal service occupations, sales and customer service occupations, process, plant and machine operatives) or high skills (managers and senior officials, professional occupations, associate professional and technical occupations). From this, we compute the proportion of women aged 18–30 whose first job required a high-skill content by LAD. Finally we match this new variable to each woman when she was 18 years old. Panel C of Figure 2 shows a smooth increase in $M_3$ from about 17% among women born before WWII to roughly 40% among those born in the mid-1950s. For all subsequent birth cohorts, this share remains quite stable. The differences in $M_3$ by regions are small, and similar to those found for $M_1$.

*Results* — The structural estimates of model (3)–(4) for A1B are reported in Table 3, while those for NEB and EVER are in Appendix Tables A4 and A5, respectively. In all tables, columns (a)–(c) show a strong positive association of each mediator with *Cohort* (i.e., $\alpha_j > 0$ in equation (3)), indicating that women from later cohorts in the sample are exposed to greater pill utilization, higher female college education, and more elevated female shares in highly skilled jobs. This is consistent with the notion that younger women are exposed to a more progressive socioeconomic environment.

Column (d) reports the *Cohort*-outcome associations, conditional on $\mathbf{M}$ and $G \times \mathbf{M}$ interactions. Across all three outcomes, the impact of $G$ on $Y$, $\varphi$, is always positive and significant. Being born 10 years later leads to a significant postponement of motherhood by 0.47 years on average (Table 3) and to a significant reduction in the number of children ever born by 0.24 (Appendix Table A4), but to no change in the probability of ever having had a child (Appendix Table A5).

Exposure to an environment with a 10-percentage-point greater pill usage increases A1B by 0.18 years (see $\beta_1$ in Table 3), and this grows to 0.23 years, if we also consider the significant $G \times M_1$ effect of 0.005. Similarly, a 10-percentage-point increase in the share of women with a college degree implies nearly a quarter of motherhood postponement, which rises to 0.29 years if compounded with $\mu_2$, i.e., the impact of $G \times M_2$. The influence of greater exposure to high-skill female employment, instead, is smaller and statistically indistinguishable from zero.

With the exception of the $\beta_2$ estimate, according to which a 10-percentage point increase in the share of women with a college qualification implies a small reduction of

0.03 children in completed fertility, the impacts of $\mathbf{M}$ and $G \times \mathbf{M}$ on NEB are otherwise generally weak. This suggests that the *Cohort-Y* relationship in this case is not heavily affected by our three mediators (see the results in Appendix Table A4). In the case of EVER, instead, given that $\tau$ is statistically insignificant, the *Cohort-Y* effect turns out to be entirely mediated by pill usage and female college education (Appendix Table A5).

Table 4 presents the decomposition results. The table shows the direct effect of an increase in *Cohort* by one year and the effect of each mediator at the bottom, middle (i.e., the fifth), and top decile of the genetic distribution, $G1$, $G5$, and $G10$, respectively. At the bottom of each column, we report the total effect.

Consider women in the top decile of the PGI A1B distribution, which corresponds to having the highest genetic predisposition to postpone motherhood (column (c)). The direct cohort effect for these women is estimated to be 0.047 (reflecting the $\tau$ estimate in the first row, column (d) of Table 3) and the mediator effect is 0.110 (=0.084+0.021+0.005), leading to the estimated total effect of 0.157 (=0.047+0.110). This means that for an average woman being born 10 years later amounts to an increase in the A1B of nearly 19 months, which can be decomposed into a direct effect of almost six months, a pill exposure effect of 10 months, a college exposure effect of another 3 months, and a negligible contribution of the high-skill occupation effect.

Figure 3 graphically displays the total and indirect effects for each outcome, while the corresponding percentages accounted for by each mediator are in Table 5, by PGI decile and outcome. Four results are worth stressing. First, there is a strong gradient in the total cohort effect by genetic predisposition across all outcomes. Being 10 years younger implies a postponement of motherhood by 0.8 years among women in the bottom decile of the PGI distribution, and by 1.6 years among women in the top decile. Second, pill exposure, $M_1$, claims the lion's share of the total cohort effects on A1B, accounting for more than 50% of the association in the top half of the PGI distribution, at least three times more than what the college exposure mediator explains. In the case of EVER, the cohort effect is more than fully mediated by $M_1$ across the entire PGI distribution, explaining almost 150% at top decile and just over 100% in the bottom decile.

Third, although female college education plays a more modest role than pill exposure (accounting for about 8–21% of the total cohort effect across the three outcomes), it is the only statistically significant mediator for NEB. Fourth, local exposure to greater female employment in highly skilled jobs does not mediate the cohort effect for any outcome, except for A1B for which it reduces age at motherhood among women with a more pronounced genetic predisposition of having children early.

In sum, these results confirm, and qualify, our earlier evidence. The interplay between each of the PGIs (our measures of genetic susceptibility) and the mediators of environmental risk (pill exposure, in particular) affects women's fertility outcomes in line with the changing social times across cohorts. Put differently, gene-environment interactions

reinforce the postponement of motherhood and the reduction in the probability of having ever had a child (or the increase in childlessness), with both processes being more strongly mediated by pill usage. In spite of the fact that the $G{\times}Pill$ gradient across women at the top and at the bottom of the PGI distributions is not strongly statistically significant, this new evidence suggests that pill exposure favors women with greater genetic propensity to delay motherhood and women with a lower biological susceptibility to remain childless. While this latter effect tends to level the playing field across women, the former effect exacerbates existing inequalities. As the environment becomes more conducive to female emancipation, so are genetic predispositions to fertility capable to exert their influences more fully, although possibly to different degrees for different women and in directions that could be socially undesirable.

*Robustness Checks* — We perform a number of sensitivity exercises, whose results are displayed in Appendix Figures A5–A7, where panel A in each figure reports the baseline findings shown in Figure 3. In the first exercise, we re-estimate equations (3) and (4) after including proxies of early life conditions in $\mathbf{X}$. These are the respondent's birth weight, and indicators of smoking status of the respondent's mother during pregnancy, and whether the respondent was breastfed. Such measures have been shown to be correlated to pre- and post-natal parental investments and predictive of both early and later child achievement (e.g., Almond et al., 2018). Information on these controls is available for at most 55% of the samples used to estimate our benchmark specification. Despite the loss in sample size, the new estimates reported in panels B are by and large similar to those shown in panels A, except that pill exposure mediates the cohort effect slightly less than before (Appendix Figure A5). This may partly reflect a positive correlation between favorable early life conditions and pill usage at the local area level.

Replacing the outcome-specific PGIs used in the baseline specification with the PGI EA or adding PGI EA as an additional control does not affect our results (see panels C and D in Appendix Figures A5–A7). This confirms the well known genetic overlap picked up by our PGIs and PGI EA (e.g., Mills et al., 2021), and it also emphasizes nuanced differences among them.

To deal with possible selective internal migration, we re-fit the model of equations (3) and (4) on the subsample of 'stayers', i.e., women whose area of residence at the time of interview is the same as that observed at birth. This leaves about half of the original sample.[25] Most of the baseline results are upheld (see panel E of Appendix Figures A5–A7), with the exception that the mediating effect of pill exposure is even greater for A1B (especially at the bottom of the PGI distribution, i.e., among early-fertility-$G$ women)

[25]For this exercise, we use 40 NUTS2 statistical areas, which are larger than LADs. This is because we aim to identify meaningful moves rather than short-distance relocations, while keeping a fine level of granularity. If we had LADs as our geographical unit in this analysis, we would have retained only 22% of the original sample.

and EVER (across the entire PGI distribution). The importance of exposure to the pill usage, therefore, seems to be even more crucial for less mobile women.

In our last check, we cluster standard errors at the LAD level. While this level of clustering conforms to the measurement of the mediators, it may not be appropriate for *Cohort* and PGIs, which vary at the individual level. Polygenic indexes, however, are known to have a complex geographic structure which reflects population stratification and its possible interaction with all mediators. In the main analysis, we account for these specific aspects by including the first 10 principal components of the genetic data and their interactions with *Cohort*, but other clustering dimensions might be missed. The results in panel F of Appendix Figures A5–A7 show standard errors that are essentially identical to those found with the baseline specification.

Taken all together, these findings uphold our previous estimates. They reiterate the importance that the diffusion of the oral contraception technology had in shaping women's fertility decisions. Importantly, and with the usual caveat on statistical significance mentioned above for the main decomposition results, they suggest that a higher exposure to pill usage magnifies motherhood postponement among women with greater genetic susceptibility to delay age at first birth and attenuates the probability of having ever had children among women with lower biological propensity to remain childless.

## 5.2 Family-Based Models

The measures of $E$ used so far have focused on aggregate *external* forces, which women were exposed to at birth or while growing up, and which have been shown to be relevant to female fertility. Fertility decisions, however, could also be shaped by the environment women face within their own family of origin. This *internal* environment may be affected by physical risk factors (e.g., parental resources, parenting styles, family norms, housing, and neighborhoods) as well as parental genotypes that are not genetically transmitted from parents to offspring, but might yet affect child phenotypes, a phenomenon known as "genetic nurture" (Kong et al., 2018; Houmark et al., 2024).

Family-based models can be used to account for shared internal environmental risks. The richness of the UK Biobank data enables us to perform two different exercises that exploit random genetic variation across blood relatives. In the first, we estimate sister fixed effects (SFE) models, which have been extensively used in economic research on fertility (e.g. Rosenzweig and Wolpin, 1995). By comparing siblings who grew up in similar (internal and external) environments, these models allow us to assess the importance of $G \times E$ effects, even when the influence of both genetic and social transmissions is heavily restrained (Belsky et al., 2018) or when parents shape their offspring's outcomes by responding to their genetic endowment differentials (e.g., Muslimova et al., 2020; Fletcher et al., 2023). In the second exercise, we consider mother-daughter pairs and perform a

family fixed effect analysis. In this case, genetic endowments continue to be similar as in the previous exercise (although in a way that differs from the SFE model), but the differences in environmental risks are substantially greater.

To fit family-based models we use a specific sample of Becker et al. (2021)'s PGI repository, which includes first grade relatives. Given the importance of pill exposure in mediating the cohort effects, we now consider oral contraception as our only measure of $E$, although controlling for the *Cohort* trend.[26] For each woman $i$ in family $f$, we then estimate:

$$\begin{aligned} Y_{if} = \phi_0 + \phi_1 Pill_{if} + \phi_2 G^Y_{if} + \phi_3 Pill_{if} \times G^Y_{ij} + \mathbf{X_{if}}'\boldsymbol{\pi} \\ + \sum_{p=1}^{10} \eta_{0p} \mathrm{PC}^p_{if} + \sum_{p=1}^{10} \eta_{1p} \mathrm{PC}^p_{i} \times Pill_{if} + \vartheta_f + \upsilon_{if}, \end{aligned} \tag{5}$$

where $Pill$ is the pill exposure measure described earlier, the vector $\mathbf{X_{if}}$ includes the usual control variables plus a linear trend in the year of birth, $\vartheta_f$ measures the time-invariant family unobserved components shared among sisters or mother-daughter pairs, $\upsilon$ is an individual idiosyncratic shock, and all the other terms have the same definition as before.

*Sister Comparisons* — This analysis is performed on a sample of approximately 12,000 biological sisters. Appendix Table A6 reports summary statistics on this sample. The SFE estimates are summarized in Table 6. Columns (a), (c), and (e) provide a benchmark, estimating equation (5) without family fixed effects, while the remaining columns also account for $\vartheta_f$.

Columns (a), (c), and (e) reveal that the random genetic variation across sisters exerts an influence on all three outcomes that is always statistically significant and quantitatively similar to what we observe in the original sample of women reported in Table 2. Controlling for sister fixed effects in columns (b), (d), and (f) reduces genetic penetrance by about 55% in the case of A1B and 20% in the case of NEB and EVER, but $\phi_2$ remains always positive and highly significant. This attenuation is not surprising, since on average one-half of the SNPs inherited from parents are shared among sisters.[27]

A 10 percentage point increase in pill exposure delays age at first birth by 0.32 years, a large and statistically significant effect that grows slightly when we account for sister fixed effects.[28] This is striking, given the high correlation in $Pill$ observed among sisters in the

[26] It is worth noting that the pill exposure correlation between mothers and daughters is 0.05, whereas it is 0.84 among sisters.

[27] The raw correlation between sisters' PGI A1B is around 0.57. The correlation between siblings is usually slightly higher than between parental and child genotypes because of assortative mating (Torvik et al., 2022).

[28] To benchmark these estimates, we re-estimated the baseline specification (5), where $Pill$ is the measure of environmental risk, while also controlling for *Cohort*. Besides a strong positive impact of *Cohort* on A1B, these additional results reported in Appendix Table A9 show a statistically significantly positive $\phi_1$ estimate which is 15% lower than the corresponding estimate in column (a) of Table 6.

UKB. Therefore, however similar the internal environmental risks faced by sisters might be, small differences in external risks are associated with meaningful differences in A1B: women who grew up in more progressive milieux with greater pill exposure postpone their first birth further. The other two outcomes, instead, are unaffected by pill exposure. In part, this could be a result of the low statistical power of the (smaller) sample of sisters, but it may also reflect the possibility that a more intense exposure to pill utilization does not affect as much the quantum of fertility as it does its timing. Bailey (2006) finds similar evidence.

The lack of impact on NEB and EVER emerges also for gene-by-environment interactions. This is likely the cumulative result of muted direct effects of both $G$ and $Pill$ on both outcomes. However, the $G \times Pill$ interplay on A1B, $\phi_3$, is positive and significant, albeit about one-third of its comparator in the full sample, $\theta_3$, in Table 2. As in that case, the $\phi_3$ effect amplifies the increase in the age at first birth associated with a greater pill usage exposure, supporting the notion that genetic susceptibilities to fertility tend to surface more distinctly when environmental risks (or societal cultural norms) are more favorable to female emancipation.

*Mother-Daughter Comparisons* — This analysis uses about 2,000 mother-daughter dyads, in which daughters were born on average around 1965 and mothers around 1942 (see Appendix Table A6). Mirroring the previous exercise, variation in genetic influences is limited, but now we leverage greater variation in the external environment. Using the same two specifications as before, equation (5) is estimated on pairs of one mother and one daughter.[29] Since, obviously, childlessness cannot be measured on the subsample of mothers, we perform the analysis only on A1B and NEB. The results are in Table 7.[30]

Unlike the case of the SFE model, the impact of genes, $\phi_2$, on A1B becomes statistically insignificant when family fixed effects are accounted for in column (b). It is instead large and significant on NEB (see column (d) of Table 7). Pill exposure is strongly associated with both outcomes and in the expected direction. In the case of A1B, $\phi_1$ is substantial, with a 10 percentage points increase delaying motherhood by 1.4 years on average (column (b)), and this is reinforced by the gene-environment interaction, $\phi_3$. In the case of NEB, however, the $G \times Pill$ impact is indistinguishable from zero. The large role played by pill usage exposure on the timing of motherhood reveals that striking differences in social norms faced by mothers and daughters in the UK Biobank may trump genetic influences even after controlling for shared unobserved family characteristics, including intra-family cultural values that are stable across generations.[31]

[29] We restrict the analysis to the oldest daughter when two or more daughters are in the sample. The raw correlation between mothers and daughters' PGI A1B is about 0.55.

[30] The raw intergenerational correlation is sizeable and statistically significant for both outcomes, going from 0.18 for NEB to 0.28 for A1B, respectively.

[31] Replacing $Pill$ with $Cohort$ leads to qualitatively similar estimates. These are reported in Appendix Table A8.

We close this subsection emphasizing that, irrespective of how we control for unobserved intra-household environmental factors which are fixed either across sisters (e.g., internal socialization and family norms) or within mother-daughter pairs (e.g., parenting habits and family role models), the gene-by-environment estimates invariably suggest that the genetic effects on the timing of motherhood are strongest when social norms are more progressive.

## 5.3 Male Fertility

Although not core to our study, we briefly explore male fertility. This is important for at least two reasons. One is that we know little about male fertility and its determinants,[32] and essentially nothing about the gene-environment interactions that could affect it. The other reason is that it allows us to qualify our understanding of the gene-by-cohort effect on female fertility we have elaborated so far. If there is a $G \times Cohort$ effect on male fertility that is mediated by the same factors as its female counterpart, then the same changes in social norms must affect male and female fertility decisions alike. If instead this specific gene-by-environment effect for men does not exist or it is not mediated by the same processes, then male genetic predispositions might have exerted their influence differently, possibly even before the sexual revolution.

For this analysis, we focus only on NEB and EVER, since information on men's A1B is not collected in the UK Biobank. We apply the same sample selections and use the same variable definitions as those employed for the sample of women described in Section 3 and summarized in Appendix Table A10. Appendix Figure A2 reports the time trends in the two outcomes by birth cohort, along with the female trends discussed earlier. The reduction in the number of children ever born and the decline in the proportion of men who ever had a child are steeper than what we found for women. This uphold the male retreat-from-fertility phenomenon illustrated by Bratsberg et al. (2022).

Appendix Figure A8, which displays the same trends separating out men in the top decile from men in the bottom decile of the PGI NEB distribution, shows that the gap between the two groups of men is relatively stable over time. This suggests that the gene-by-cohort interactions are likely to be unimportant for male fertility. The estimates in Appendix Table A11, which are obtained from fitting model (2) on men, confirm this conjecture. Although the $\theta_1$ and $\theta_2$ estimates for both outcomes are in line with those found for women in Table 2, just smaller in magnitude, the impact of $G \times Cohort$ is trivial. The changing times faced by men in the UKB seem to have left their fertility behavior unchanged.

To check this possibility more directly, we repeat the mediation analysis of subsection

[32] Some of the recent contributions in this literature include Kearney and Wilson (2018) and Giuntella et al. (2022). They do not use genetic data.

5.1 on the sample of men. We keep the same definition of pill exposure, $M_1$ as before, but the other two mediators — the fraction of individuals with university qualifications or more by LAD and cohort, $M_2$, and the fraction of 18–30 year old individuals working in high-skill jobs by LAD and cohort, $M_3$ — are now computed on male data and linked to each man in the sample when he was 18 years old. The estimates in Appendix Table A12 are summarized in Figure 4. The small negative direct effects of *Cohort* on both outcomes are not explained by our three mediators, with the possible exception of $M_3$ which accounts for about 11% of the cohort effect on EVER among low-fertility-$G$ men, i.e., those with greater genetic propensity to remain childless.

In sum, male genetic susceptibilities to fertility have likely surfaced before the sexual revolution, when environmental risks were less favorable to female predispositions. Other factors may have become more conducive to mediating the secular retreat from fertility among men in the UKB sample, such as deindustrialization, globalization, declining health, and changes in the wage structure (e.g., Huttunen and Kellokumpu, 2016; Kearney and Wilson, 2018; Giuntella et al., 2022). Pinning down these mechanisms goes beyond the scope of the paper and is left for future research.

## 5.4 Gender Role Norms and the Pill Revolution

We conclude with suggestive evidence on the association of our environmental mediators with gender norms. Ideally, we would like to have a measure of norms collected around the time when women in the UKB were making their fertility decisions. Unfortunately, such data do not exist. We thus use data from the British Household Panel Survey and its successor, the UK Household Longitudinal Survey, covering the 30-year period from 1992 to 2021.

To match the UKB cohort coverage with the BHPS-UKHLS data, we restrict the analysis to women born between 1935 and 1970. For each individual in this sample, we have direct information on beliefs about gender roles. In particular, respondents are asked if they agree with the following five claims: (i) "Pre-school child suffers if mother works"; (ii) "Family suffers if mother works full-time"; (iii) "Husband and wife should contribute to household income"; (iv) "Husband should earn, wife should stay at home"; (v) "Employers should help mothers combine jobs and childcare". Respondents' agreement with each of these statements is rated according to a 5-point scale, where 1=strongly agree, 2=agree, 3=neither agree nor disagree, 4=disagree, and 5=strongly disagree. We analyze each index separately and also define a comprehensive index by summing responses across the five questions after inverting the scale for questions (iii) and (v). The summary index therefore varies between 5 and 25, with higher values indicating more egalitarian norms between the sexes and lower values capturing more traditional gender role attitudes.

We next average each gender role norm measure across individuals from the same

cohort, $c$, in the same local authority district, $j$, and label this variable $GenderNorms_{jc}$. For each of the five belief variables and the summary measure, we then estimate the following model:

$$GenderNorms_{jc} = \psi_0 + \psi_1 M_{1,jc} + \psi_2 M_{2,jc} + \psi_3 M_{3,jc} + \eta_j + \nu_{jc} \tag{6}$$

where $M_1, M_2, M_3$ are defined in subsection 5.1 and refer to contraceptive pill usage, share of women with college education, and share of women in highly skilled occupations in local authority district $j$ and cohort $c$, respectively, and $\eta_j$ refers to LAD fixed effects. We do not include cohort fixed effects, because these are absorbed into the three mediators. Our coefficients of interests are the three $\psi_j$'s. A positive estimate would indicate that areas where women were more exposed to pill usage ($\psi_1$), or to a greater share of female college graduates ($\psi_2$), or a greater share of women in high-skill occupations ($\psi_3$) are also the areas with more equal beliefs about gender roles.

The OLS results in Table 8 show that exposure to greater pill diffusion is almost invariably associated with more egalitarian gender norms. This emerges strongly for four of the five attitude measures (the exception being in column (c) for the response that both partners should contribute to household income) and for the summary index in column (f) of panel A. The correlations of $M_2$ and $M_3$ with $GenderNorms_{jc}$ are instead much weaker (at least one order of magnitude smaller than in the case of $M_1$), they are sometimes wrong signed (as in the case of $M_2$ in columns (a) and (b)), and they are usually statistically indistinguishable from zero.

To strengthen the credibility of these results, we repeat the exercise after replacing gender role norms with other attitudinal variables, which may be only weakly related to female empowerment and are therefore expected to be broadly uncorrelated with our fertility mediators. We label them 'placebo norms'. These cover a wide range of values and norm-revealing habits, from wearing a poppy on Remembrance Day (a very popular display of support in the UK for the service of war veterans and their families) to environmental attitudes (e.g., wearing extra clothes rather than turning up heating, or not making a purchase because of excess packaging), and from social views (such as state ownership of public services and industries) to attitudes toward car characteristics (such as style, comfort, safety, and reliability).

The results from this second exercise are in panel B of Table 8. For the items in columns (a)–(d), responses are dichotomized into a binary variable, taking value 1 if women are in agreement with each statement, and 0 otherwise. Columns (e) and (f) show estimates for composite items, ranging from 0 to 5 and from 0 to 2, respectively. In all cases, we find almost no correlation between these placebo norms and our mediators. Only two out of the 18 coefficients are statistically significant at the 10% level.

Albeit not causal, the estimates in panel A illustrate that areas that were historically

exposed to greater pill utilization (from the 1960s through to the 1980s) are also the areas with more egalitarian gender norms in recent years (from the mid 1990s to the early 2020s). Although on a much shorter time scale, this result echoes Alesina et al. (2013)'s findings on the origins of gender roles. It also offers suggestive evidence that differences in gender attitudes across cohorts are associated with the technological fertility discoveries which allowed women to benefit from a broader range of life opportunities, in part facilitated by favorable gene-by-environment interactions.

# 6 Conclusion

Although fertility is believed to have a strong biological component, economic research on this relationship is scant. In this paper, for the first time, we systematically investigate genetic influences on female fertility and document the existence of gene-by-environment interactions. Using data from the UK Biobank, one of the largest genomic resources in the world, and leveraging the latest developments in molecular genetics, we summarize individual genetic endowments in polygenic indexes and capture the broader social environment with the year of birth among women born between the late 1930s and the late 1960s.

We find a strong positive impact of genetic predispositions on women's age at first birth, the total number of children they have, and the probability they ever had a child; and we document a strong impact of birth cohort, with later-born women postponing motherhood more, having fewer children, and facing a higher likelihood of remaining childless. We also show that cohort of birth modifies the influence of genetic endowments on all outcomes: the more recent a cohort, the higher the genetic influence. This suggests the presence of a gene-by-environment interplay, which amplifies the direct effect of genetic endowments and becomes more apparent when social norms and economic conditions enable women to expand the set of their life-course opportunities. This interaction does not emerge among men, whose genetic predispositions might have emerged when environmental risks were less advantageous to female (but not male) susceptibilities.

To understand the gene-cohort interactions on female fertility, we decompose the cohort effect into a direct impact and an indirect impact, the latter going through three separate channels (i.e., local exposure to the oral contraceptive diffusion, local exposure to female college attainment, and local exposure to female employment in highly skilled occupations) and being allowed to differ by intensity of the polygenic indexes. Pill exposure turns out to be the primary mediator, as it accounts for half of the positive cohort effect on age at first birth, especially among women with a high genetic propensity to delay motherhood, and it entirely explains the negative effect on the probability of ever having had a child, especially among women with a high propensity to have children. The diffusion of the oral contraceptive might have led the emergence of gender-egalitarian

norms and epitomized one aspect of the sexual revolution that has accompanied women's empowerment (Goldin and Katz, 2002).

The gene-by-pill-exposure effect on age at first birth persists even when we analyze a subsample of sisters and control for sister fixed effects or a subsample of matched mother-daughter pairs and account for family fixed effects. Taking advantage of the random variation in gene allocation among first-degree relatives, the former approach confirms a significant postponement of motherhood even when it is driven by (possibly small) between-sister differences in exposure to pill diffusion. The latter approach, in which the differences in environmental risks are much larger by definition, identifies a substantial impact on age at first birth due to considerable variation in pill exposure between mothers and daughters.

The existence of gene-environment effects on female fertility, which reinforce the influence of genetic predispositions and surface more strongly if progressive social norms expand women's economic possibilities, may have material implications. With many advanced countries facing sustained fertility decline, greater labor market insecurity, and a variety of family policies that shape fertility decisions (e.g., Doepke et al., 2023), new social environments and new gene-by-environment interactions are likely to arise. These, in turn, can affect existing, or engender new, inequalities if the impact of the environment differs by genetic endowment. The evidence that women with greater genetic propensity to delaying motherhood benefit more from the pill revolution offers a compelling example.

These considerations lead to exciting new areas for future research, with special attention again given to the role played by genetic endowments. One is to identify the channels through which women with greater genetic propensity to anticipate birth did not benefit from the pill revolution: Is it a question of worse information? Or lower economic incentives? Knowing this is policy relevant. Access to oral contraception, which is recognized as a key step towards equal opportunity (Bailey, 2020), may have to be accompanied by other interventions if women do not equally take advantage of this technology.[33] This could also be the case for future innovations. Another area is to examine a wider range of recent external environmental risks, such as the availability of in vitro fertilization treatments (Lundborg et al., 2017), the changing role of gender stereotypes (Bertrand, 2020), and the emergence of globalization and new forms of work (Autor et al., 2024). Another yet is to gain a deeper understanding of the male retreat from fertility (Bratsberg et al., 2022) and childlessness more generally (Baudin et al., 2015).

[33]For a critical appraisal of the relationship between access to contraception and fertility among rural households in a developing country, see Dupas et al. (2024).

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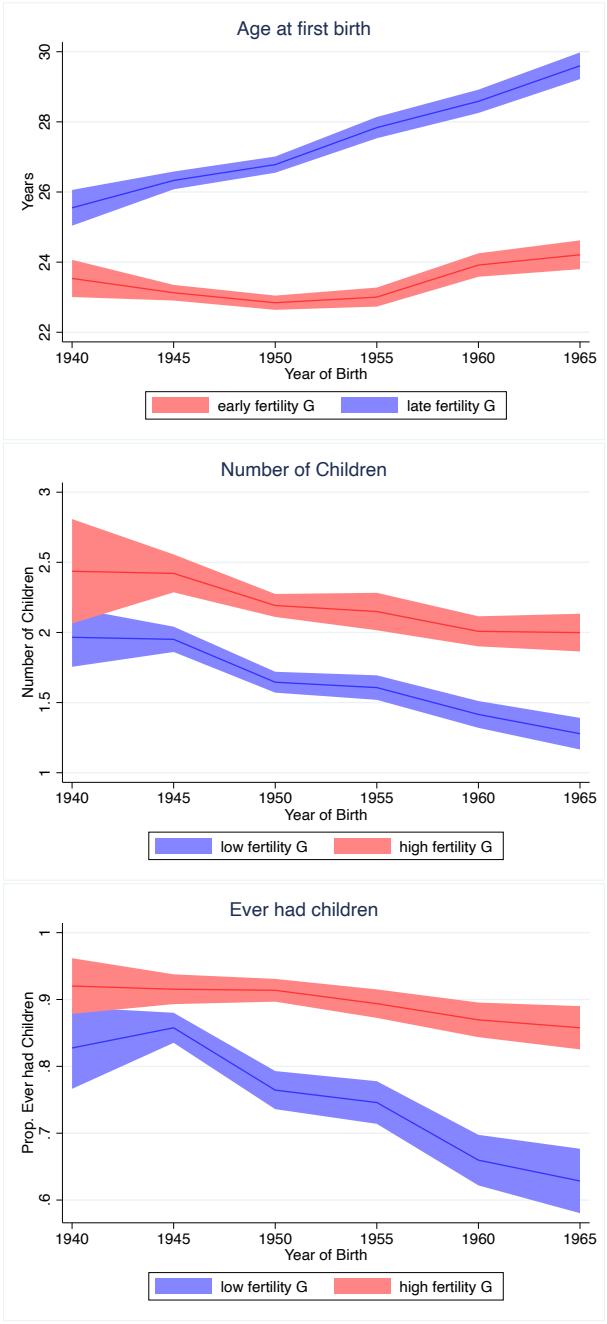
# Figures and Tables

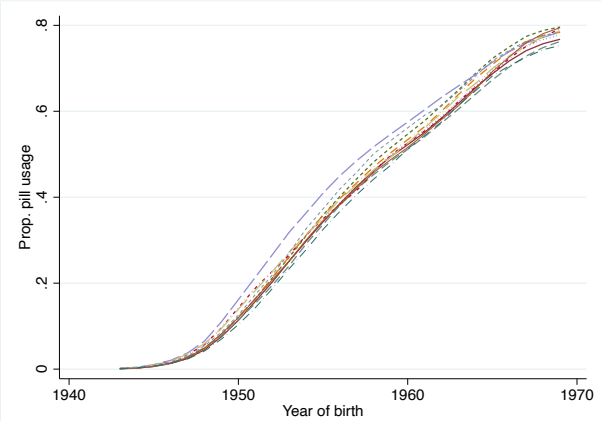
Figure 1: Fertility Outcomes by Birth Cohort and Genetic Endowment in the UK Biobank



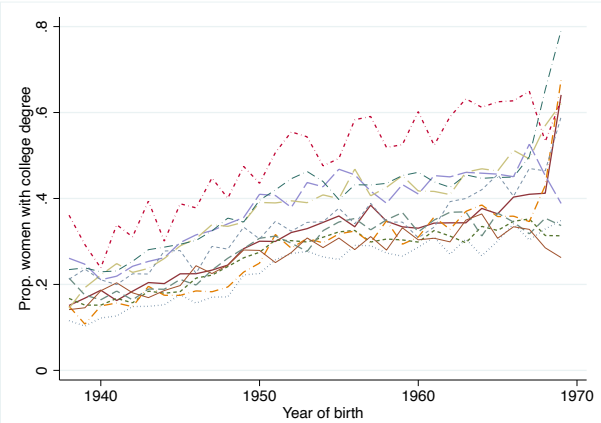

*Notes*: Each panel displays the estimated 5-year cohort associations with the outcome for the bottom and top decile of the outcome-specific PGI. The shaded areas are the corresponding 95% confidence intervals. 'Early' ('late') fertility $G$ corresponds to the bottom (top) decile of the PGI A1B. 'Low' ('high') fertility $G$ corresponds to the bottom (top) decile of the PGI NEB. In blue (red), we indicate women who are genetically more (less) predisposed to delay motherhood, to have fewer children, and to remain childless. For NEB and EVER, we restrict the sample to women aged 45 years old or more at the time of interview.

Figure 2: Mediators

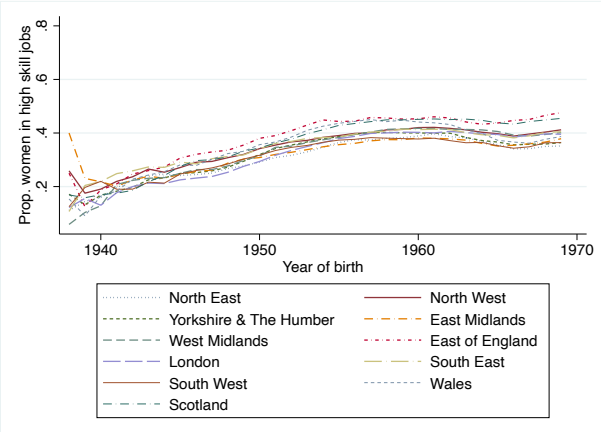
Panel A: Pill Exposure




Panel B: Female College

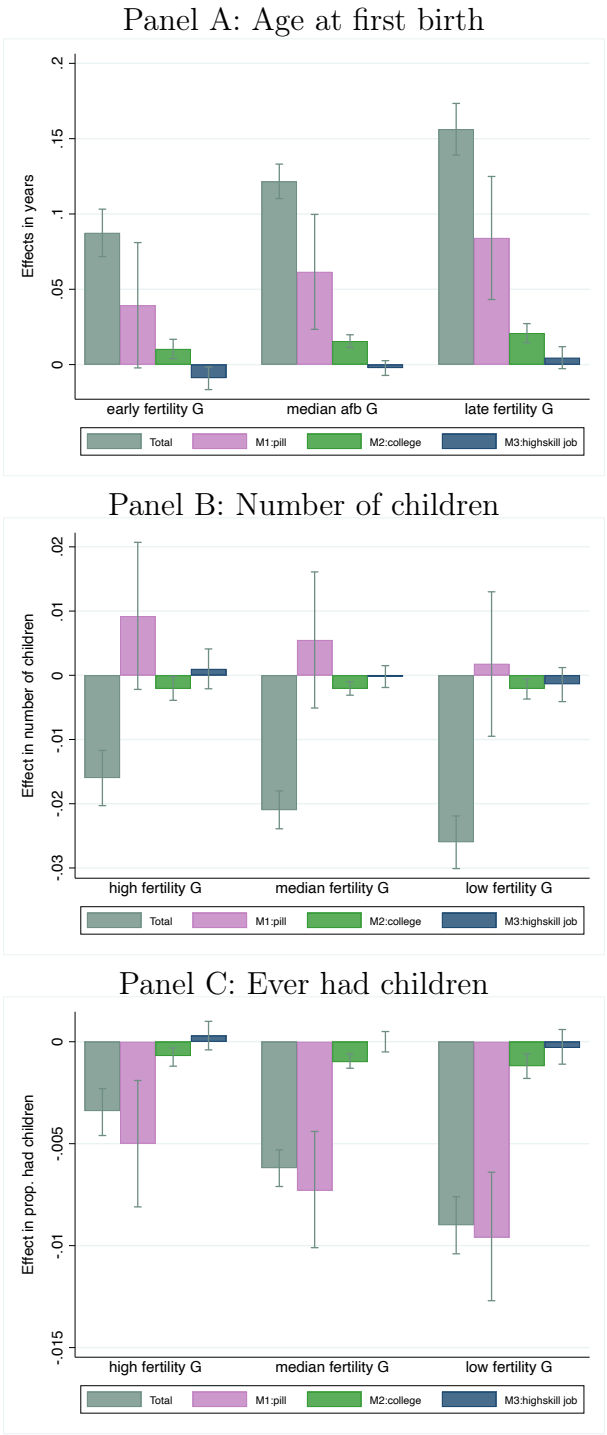



Panel C: Female High Skill Jobs




*Notes*: The figure in Panel A shows the proportion of childless women who ever took the birth control pill by broad geographic region of birth and year of birth. The proportion of pill usage corresponds to when each woman was 18 years old. The figure in Panel B shows the proportion of women with a college degree by year and region of birth. The figure in Panel C shows the proportion of women whose first occupation was in a high skill job. As for the pill, the share of women in high skill jobs is calculated by year and district and linked to each woman when she was 18 years old. The data come from the UK Biobank, please see Section 4.1 for more details.

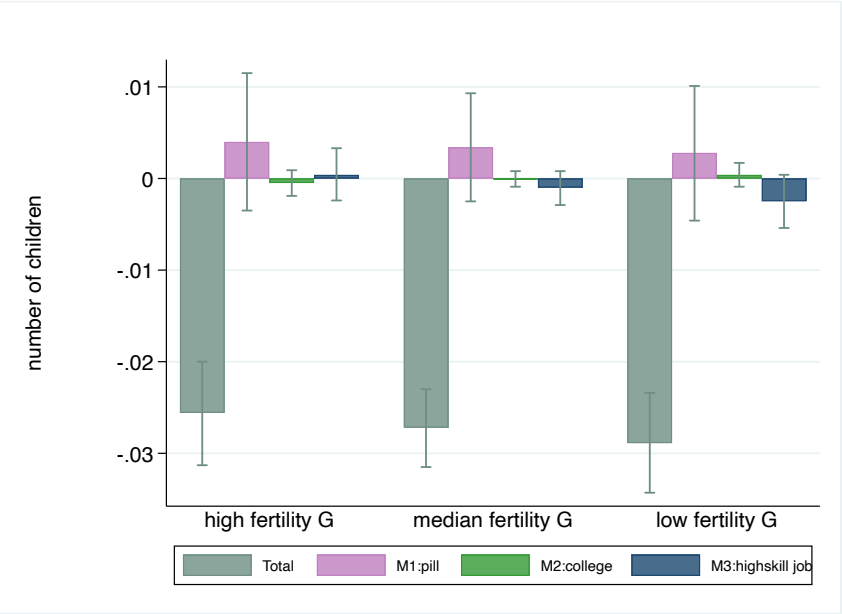
Figure 3: Decomposing the effect of the birth cohort on female fertility outcomes



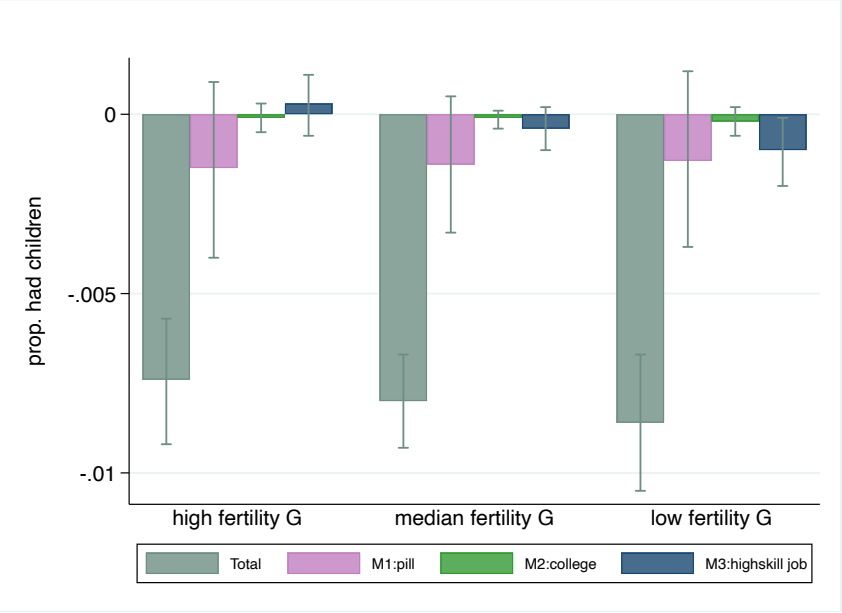

*Notes*: Each panel shows the total effect and the indirect effect for each mediation calculated at the 1st, 5th and 10th decile of the outcome-specific PGI distribution. The mediators are pill exposure (pink), women with a college degree (green) and women working in high skill jobs (blue). Early (late) fertility G corresponds to the 1st (10th) decile of the PGI for A1B. High (low) fertility G corresponds to the 10th (1st) decile of the PGI for NEB. The estimates come from Table 4 and the S.E. are bootstrapped. See the text for more details.

Figure 4: Decomposing the effect of the birth cohort on male fertility outcomes

Panel A: Number of children




Panel B: Ever had children




*Notes*: Each panel shows the total effect and the indirect effect for each mediation calculated at the 1st, 5th and 10th decile of the outcome-specific PGI distribution. The mediators are pill exposure (pink), men with a college degree (green) and men working in high skill jobs (blue). High (low) fertility G corresponds to the 10th (1st) decile of the PGI for NEB. The estimates come from Table 4 and the S.E. are bootstrapped. See the text for more details.

Table 1: Summary Statistics

| Variable | Mean | SD | $N$ |
|---|---|---|---|
| Outcomes | | | |
| Age at first birth (A1B) | 24.87 | 4.79 | 46,759 |
| Number of children (NEB) | 1.88 | 1.23 | 62,680 |
| Ever had children (EVER) | 0.83 | 0.38 | 62,680 |
| Measures of genetic assessment ($G$) | | | |
| PGI A1B | 0 | 1 | 68,449 |
| PGI NEB | 0 | 1 | 68,449 |
| PGI EA | 0 | 1 | 68,449 |
| Mediators (%) | | | |
| Pill exposure | 32.66 | 27.59 | 68,449 |
| Female college | 32.32 | 16.72 | 68,449 |
| Female high skill job | 33.56 | 11.41 | 68,449 |
| Woman's early life controls | | | |
| Own mother smoking during pregnancy (=1) | 0.32 | 0.47 | 59,113 |
| Own birth weight (kg) | 3.22 | 0.65 | 44,613 |
| Was breastfed (=1) | 0.62 | 0.49 | 55,233 |

*Source:* UK Biobank.
*Notes*: SD stands for standard deviation, while $N$ for the number of women in the estimation sample. EA stands for educational attainment. All mediators are computed on the UK Biobank. 'Pill exposure' is measured by the proportion of childless women who used the pill for the first time by age 18 in the local authority district (LAD) of birth of each woman in the sample. 'Female college' corresponds to the proportion of women with a college degree by LAD and year of birth. 'Female high skill job' is the proportion of women aged 18–30 whose first occupation was a high skill job in a given LAD and linked to each woman in the UK Biobank when she was 18 years old. Statistics are weighted using sampling weights.

Table 2: Baseline Estimates

| | | Age at first birth (A1B) | Number of children (NEB) | Ever had children (EVER) |
|---|---|---|---|---|
| | | (a) | (b) | (c) |
| $Cohort$ | $(\theta_1)$ | 0.120*** | -0.021*** | -0.006*** |
| | | (0.006) | (0.002) | (0.0004) |
| $G$ | $(\theta_2)$ | 0.867*** | 0.107*** | 0.010** |
| | | (0.056) | (0.017) | (0.004) |
| $G \times Cohort$ | $(\theta_3)$ | 0.033*** | 0.004*** | 0.002*** |
| | | (0.004) | (0.001) | (0.0003) |
| Observations | | 46,759 | 62,680 | 62,680 |
| $R^2$ | | 0.1584 | 0.0502 | 0.0445 |
| District FEs | | ✓ | ✓ | ✓ |
| War FE | | ✓ | ✓ | ✓ |
| PCs | $(\boldsymbol{\gamma}_0)$ | ✓ | ✓ | ✓ |
| PCs$\times Cohort$ | $(\boldsymbol{\gamma}_1)$ | ✓ | ✓ | ✓ |
| Mean of Dep. Var. | | 25.47 | 1.835 | 0.825 |
| SD of Dep. Var. | | 4.550 | 1.153 | 0.380 |

*Notes*: Obtained from equation (2). Each column corresponds to a specific outcome. $G$ refers to PGI A1B in column (a) and to PGI NEB in columns (b) and (c). All regressions include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise; district (LAD) fixed effects; the first 10 (demeaned) principal components (PCs) of the full matrix of SNP data; and the interactions between each of the 10 PCs and $Cohort$. All estimates are weighted using the sampling weights constructed by van Alten et al. (2022). 'Observations' is the number of women in the analysis. Standard errors are robust to heteroskedasticity.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 3: Moderated Mediation Estimates for Age at First Birth

| | | Pill usage ($M_1$) | Female with college degree ($M_2$) | Female in high-skill occupations ($M_3$) | Age at first birth (A1B) |
|---|---|---|---|---|---|
| | | (a) | (b) | (c) | (d) |
| *Cohort* | | 3.509*** | 0.668*** | 0.654*** | 0.047** |
| | | (0.008) | (0.017) | (0.011) | (0.021) |
| $M_1$ | ($\beta_1$) | | | | 0.018*** |
| | | | | | (0.006) |
| $M_2$ | ($\beta_2$) | | | | 0.023*** |
| | | | | | (0.003) |
| $M_3$ | ($\beta_3$) | | | | -0.004 |
| | | | | | (0.004) |
| $G$ | ($\varphi$) | | | | 1.279*** |
| | | | | | (0.029) |
| $G \times M_1$ | ($\mu_1$) | | | | 0.005*** |
| | | | | | (0.002) |
| $G \times M_2$ | ($\mu_2$) | | | | 0.006** |
| | | | | | (0.003) |
| $G \times M_3$ | ($\mu_3$) | | | | 0.009** |
| | | | | | (0.004) |

*Notes*: Obtained from equations (3) and (4). The parameters associated to *Cohort* in the first row are $\alpha_1$, $\alpha_2$, and $\alpha_3$ in columns (a), (b), and (c), respectively, and $\tau$ in column (d). 'Pill usage ($M_1$)' is the proportion of childless women aged 18 or more who ever took the pill in the local authority district (LAD) of birth of each woman in the sample. 'Female with college degree ($M_2$)' is the proportion of women with a college degree or more by LAD and year of birth. 'Female in high skill occupations ($M_3$)' is the proportion of women aged 18–30 whose first job was in a high skill occupation by LAD and linked to each woman in the UK Biobank when she was 18 years old. $M_1$, $M_2$, and $M_3$ are expressed in percentage points, and $\beta_1, \beta_2$ and $\beta_3$ are the corresponding parameters from equation (4). $G$ refers to the PGI A1B. The number of women in the analysis is 46,759. All regressions include the same variables reported in the notes to Table 2. The same notes also provide details on sampling weights and standard errors.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 4: Decomposition Estimates

| | Age at first birth (A1B) | | | Number of children (NEB) | | | Ever had children (EVER) | | |
|---|---|---|---|---|---|---|---|---|---|
| | $G1$ | $G5$ | $G10$ | $G1$ | $G5$ | $G10$ | $G1$ | $G5$ | $G10$ |
| | (a) | (b) | (c) | (d) | (e) | (f) | (g) | (h) | (i) |
| Direct effect | 0.047** | 0.047** | 0.047** | -0.024*** | -0.024*** | -0.024*** | 0.002 | 0.002 | 0.002 |
| | [0.027] | [0.027] | [0.027] | [0.000] | [0.000] | [0.000] | [0.202] | [0.202] | [0.202] |
| $M_1$ | 0.039* | 0.062** | 0.084*** | 0.002 | 0.006 | 0.009 | -0.010*** | -0.007*** | -0.005*** |
| | [0.062] | [0.002] | [0.0001] | [0.763] | [0.317] | [0.123] | [0.000] | [0.000] | [0.002] |
| $M_2$ | 0.010** | 0.016*** | 0.021*** | -0.002** | -0.002*** | -0.002** | -0.001*** | -0.001*** | -0.0007*** |
| | [0.001] | [0.000] | [0.000] | [0.0133] | [0.000] | [0.031] | [0.000] | [0.000] | [0.002] |
| $M_3$ | -0.009** | -0.002 | 0.005 | -0.001 | -0.0002 | 0.001 | -0.0003 | 0.000 | 0.0003 |
| | [0.022] | [0.378] | [0.235] | [0.289] | [0.808] | [0.534] | [0.541] | [0.911] | [0.380] |
| Total effect | 0.088*** | 0.122*** | 0.157*** | -0.026*** | -0.021*** | -0.016*** | -0.009*** | -0.006*** | -0.003*** |
| | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] |
| Observations | 46,759 | 46,759 | 46,759 | 62,680 | 62,680 | 62,680 | 62,680 | 62,680 | 62,680 |

*Notes*: Obtained from equations (3) and (4) and from the results reported in Table 3 for A1B and Appendix Tables A4 and A5 for NEB and EVER, respectively. Indirect effects refer to $M_1$ (exposure to pill utilization), $M_2$ (exposure to female college education), and $M_3$ (exposure to female high-skill occupations). Total effect is the sum of the direct effects and the three indirect effects. The effects are moderated by $G$, for which we report the estimates based on three values of the $G$ distribution, i.e., $G1$, $G5$, and $G10$, which correspond respectively to the first (bottom), fifth, and tenth (top) decile of PGI A1B for A1B (also defined early fertility $G$, medium fertility $G$, and late fertility $G$), and PGI NEB for NEB and EVER (also defined low, medium, high fertility $G$). 'Observations' is the number of women used in the analysis. Bootstrapped $p$-values are reported in square brackets. For other details on the estimated specifications an sampling weights, see the notes to Table 2.

* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 5: Share of the *Cohort* Effect Accounted for by the Three Mediators

| | | | |
|---|---|---|---|
| A: Age at first birth (A1B) | | | |
| | Early fertility $G$ | Medium fertility $G$ | Late fertility $G$ |
| | $G1$ | $G5$ | $G10$ |
| $M_1$ | 0.450* | 0.506*** | 0.538*** |
| | (0.242) | (0.164) | (0.124) |
| $M_2$ | 0.119** | 0.128*** | 0.134*** |
| | (0.037) | (0.018) | (0.023) |
| $M_3$ | -0.103** | -0.019 | 0.029 |
| | (0.044) | (0.021) | (0.025) |
| B: Number of children (NEB) | | | |
| | Low fertility $G$ | Medium fertility $G$ | High fertility $G$ |
| | $G1$ | $G5$ | $G10$ |
| $M_1$ | -0.068 | -0.263 | -0.577 |
| | (0.224) | (0.258) | (0.410) |
| $M_2$ | 0.082** | 0.100*** | 0.128* |
| | (0.034) | (0.027) | (0.069) |
| $M_3$ | 0.055 | 0.010 | -0.063 |
| | (0.056) | (0.042) | (0.102) |
| C: Ever had children (EVER) | | | |
| | Low fertility $G$ | Medium fertility $G$ | High fertility $G$ |
| | $G1$ | $G5$ | $G10$ |
| $M_1$ | 1.060*** | 1.171*** | 1.461*** |
| | (0.172) | (0.248) | (0.504) |
| $M_2$ | 0.132*** | 0.154*** | 0.211** |
| | (0.035) | (0.029) | (0.088) |
| $M_3$ | 0.028 | -0.004 | -0.090 |
| | (0.049) | (0.041) | (0.105) |

*Notes*: Obtained from Table 4 and Figure 3. Bootstrapped standard errors are in parentheses.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 6: Estimates from the Sisters' Subsample

| | | Age at first birth (A1B) | | Number of children (NEB) | | Ever had children (EVER) | |
|---|---|---|---|---|---|---|---|
| | | (a) | (b) | (c) | (d) | (e) | (f) |
| *Pill* | ($\phi_1$) | 0.032** | 0.042*** | -0.003 | -0.005 | -0.001 | -0.001 |
| | | (0.013) | (0.016) | (0.003) | (0.005) | (0.001) | (0.001) |
| *G* | ($\phi_2$) | 0.942*** | 0.419*** | 0.108*** | 0.087** | 0.031*** | 0.025** |
| | | (0.084) | (0.143) | (0.021) | (0.037) | (0.006) | (0.010) |
| $G \times Pill$ | ($\phi_3$) | 0.012*** | 0.009* | 0.002*** | 0.001 | 0.0004* | 0.0004 |
| | | (0.003) | (0.005) | (0.0006) | (0.001) | (0.0002) | (0.0004) |
| *Cohort* | | -0.025 | -0.020 | -0.014 | -0.012 | -0.001 | -0.001 |
| | | (0.0464) | (0.057) | (0.012) | (0.019) | (0.004) | (0.005) |
| Observations | | 6,689 | 6,413 | 11,990 | 11,476 | 11,990 | 11,476 |
| $R^2$ | | 0.2256 | 0.7589 | 0.0900 | 0.6822 | 0.0703 | 0.6610 |
| District FEs | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| War FE | | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs | ($\boldsymbol{\eta}_0$) | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs $\times Pill$ | ($\boldsymbol{\eta}_1$) | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Family FEs | ($\vartheta_f$) | | ✓ | | ✓ | | ✓ |
| Mean of Dep. Var. | | 24.99 | 25.02 | 1.849 | 1.847 | 0.822 | 0.821 |
| SD of Dep. Var. | | 4.405 | 4.392 | 1.157 | 1.158 | 0.382 | 0.383 |

*Notes*: Obtained from model (5). Each column refers to a different regression. *G* corresponds to PGI A1B in columns (a) and (b) and to PGI NEB in colums (c)–(f). All specifications include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions between the first 10 demeaned PCs and pill exposure (PCs×*Pill*). Columns (b), (d), and (f) include family FEs. All regressions are weighted using the sampling weights constructed by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust to heteroskedasticity and clustered at the family level.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 7: Estimates for the Mother-Daughter Subsample

| | | Age at first birth (A1B) | | Number of children (NEB) | |
|---|---|---|---|---|---|
| | | (a) | (b) | (c) | (d) |
| *Pill* | ($\phi_1$) | 0.198*** | 0.140*** | -0.034*** | -0.023** |
| | | (0.017) | (0.028) | (0.010) | (0.011) |
| *G* | ($\phi_2$) | 0.366*** | 0.283 | 0.106 | 0.240*** |
| | | (0.120) | (0.248) | (0.066) | (0.092) |
| $G \times Pill$ | ($\phi_3$) | 0.012*** | 0.0150*** | 0.001 | 0.001 |
| | | (0.003) | (0.004) | (0.001) | (0.002) |
| *Cohort* | | -0.329*** | -0.169* | 0.042 | 0.022 |
| | | (0.053) | (0.089) | (0.029) | (0.033) |
| Observations | | 2,222 | 2,108 | 1,452 | 1,352 |
| $R^2$ | | 0.440 | 0.881 | 0.442 | 0.838 |
| District FEs | | ✓ | ✓ | ✓ | ✓ |
| War FE | | ✓ | ✓ | ✓ | ✓ |
| PCs | ($\boldsymbol{\eta}_0$) | ✓ | ✓ | ✓ | ✓ |
| PCs $\times Pill$ | ($\boldsymbol{\eta}_1$) | ✓ | ✓ | ✓ | ✓ |
| Family FEs | ($\vartheta_f$) | | ✓ | | ✓ |
| Mean of Dep. Var. | | 24.33 | 24.30 | 2.161 | 2.178 |
| SD of Dep. Var. | | 4.742 | 4.725 | 1.234 | 1.229 |

*Notes*: See the notes to Table 6 for details.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table 8: The Relationship of the Three Environmental Mediators with Gender Role Norms

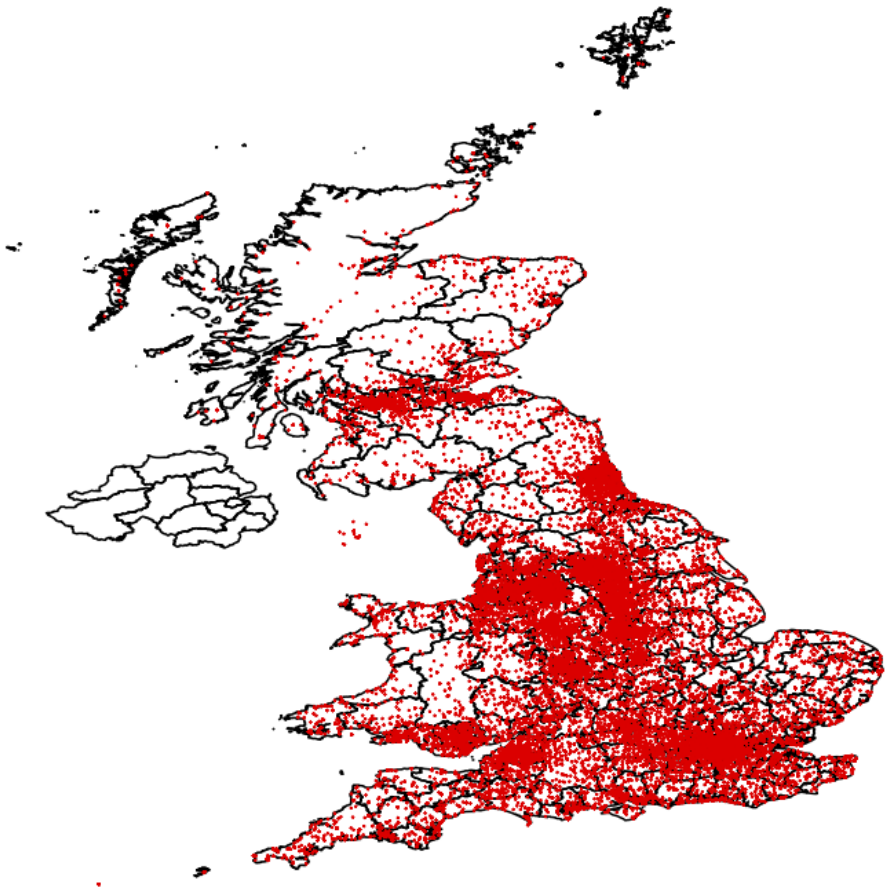
| | | (a) | (b) | (c) | (d) | (e) | (f) |
|---|---|---|---|---|---|---|---|
| A: Gender norms | | Pre-school child does not suffers if mother works | Family does not suffers if mother works FT | Husband and wife should both contribute to HH income | Husband should earn, wife stay at home (reverse scale) | Employer should help mothers combine jobs and childcare | Summary index |
| $M_1$ (*Pill*) | ($\psi_1$) | 0.005*** | 0.004*** | –0.001*** | 0.006*** | 0.002*** | 0.016*** |
| | | (0.0003) | (0.0003) | (0.0002) | (0.0004) | (0.0002) | (0.0008) |
| $M_2$ (*College*) | ($\psi_2$) | –0.0005** | –0.008*** | –0.0001 | 0.0003 | 0.0001 | –0.0009 |
| | | (0.0002) | (0.0003) | (0.0002) | (0.0003) | (0.0003) | (0.0008) |
| $M_3$ (*Skilled jobs*) | ($\psi_3$) | 0.0002 | 0.0001 | –0.0008 | 0.001** | 0.0005 | 0.0012 |
| | | (0.0006) | (0.0006) | (0.0005) | (0.005) | (0.0003) | (0.0016) |
| Observations | | 7,341 | 7,341 | 7,341 | 7,341 | 7,341 | 7,341 |
| $R^2$ | | 0.2575 | 0.2502 | 0.2704 | 0.3274 | 0.2927 | 0.2895 |
| Mean of Dep. Var. | | 3.05 | 3.02 | 1.62 | 3.62 | 1.85 | 13.16 |
| B: Placebo norms | | Doesn't buy because of excess packaging | Wears a poppy on Remembrance Day | Public services ought to be state owned | Wears extra, doesn't turn up heating | Important features for a car | Lights off, and recycled paper |
| $M_1$ (*Pill*) | ($\psi_1$) | 0.0001 | –0.0006 | -0.0003 | –0.0000 | 0.0008 | 0.0001 |
| | | (0.0002) | (0.0005) | (0.0002) | (0.0003) | (0.0005) | (0.0001) |
| $M_2$ (*College*) | ($\psi_2$) | –0.0004 | –0.0002 | –0.0001 | –0.0000 | 0.0009* | –0.0000 |
| | | (0.0003) | (0.0004) | (0.0002) | (0.0002) | (0.0005) | (0.0001) |
| $M_3$ (*Skilled jobs*) | ($\psi_3$) | –0.0002 | –0.0009 | 0.0007* | 0.0001 | 0.0013 | 0.0002 |
| | | (0.0005) | (0.0009) | (0.0004) | (0.0006) | (0.0009) | (0.0003) |
| Observations | | 4,081 | 1,957 | 5,570 | 4,081 | 6,880 | 7,347 |
| $R^2$ | | 0.3372 | 0.5373 | 0.3116 | 0.3227 | 0.2369 | 0.2156 |
| Mean of Dep. Var. | | 0.24 | 0.70 | 0.38 | 0.77 | 3.08 | 1.14 |

*Notes*: Obtained from OLS estimation of equation (6). Each column refers to a different regression. Column (e) of panel B refers to the sum of five binary features when buying a car: comfort, safety, reliability, interior space/boot size, style/design. Column (f) of panel B refers to the sum of two binary measures of environmental habits: switches lights off in empty rooms and buys recycled products. All regressions include district FEs. 'Observations' refers to the number of district-cohort observations in both panels.

* Significant at 10%; ** significant at 5%; *** significant at 1%.

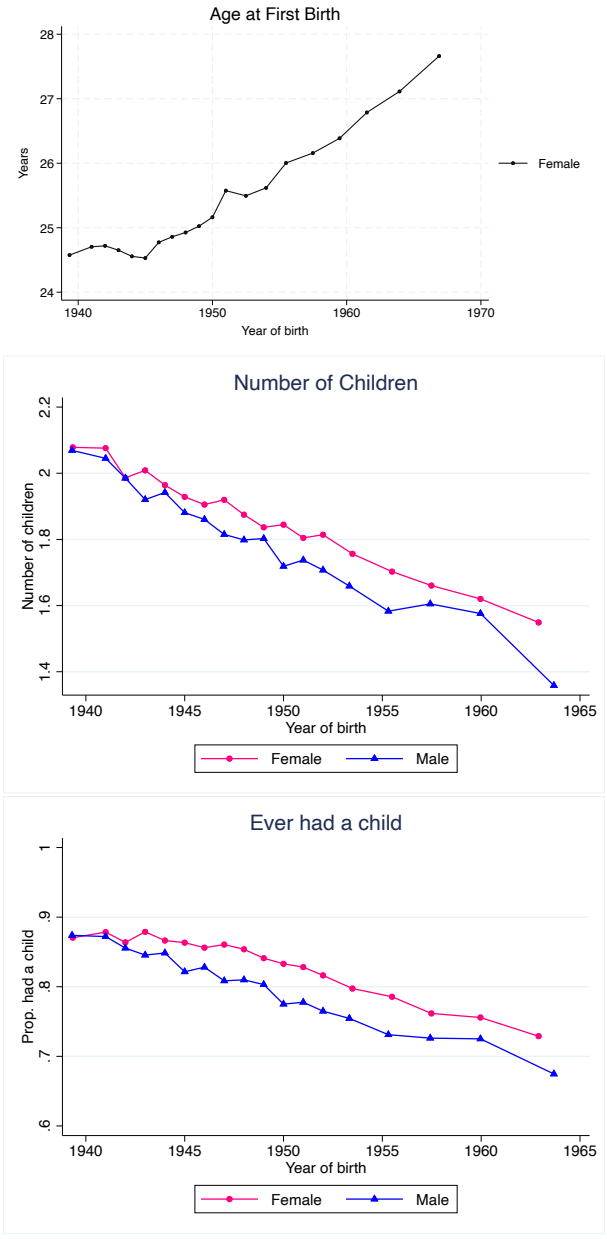
# For Online Publication

Figure A1: Birth Location for Each UK Biobank Respondent



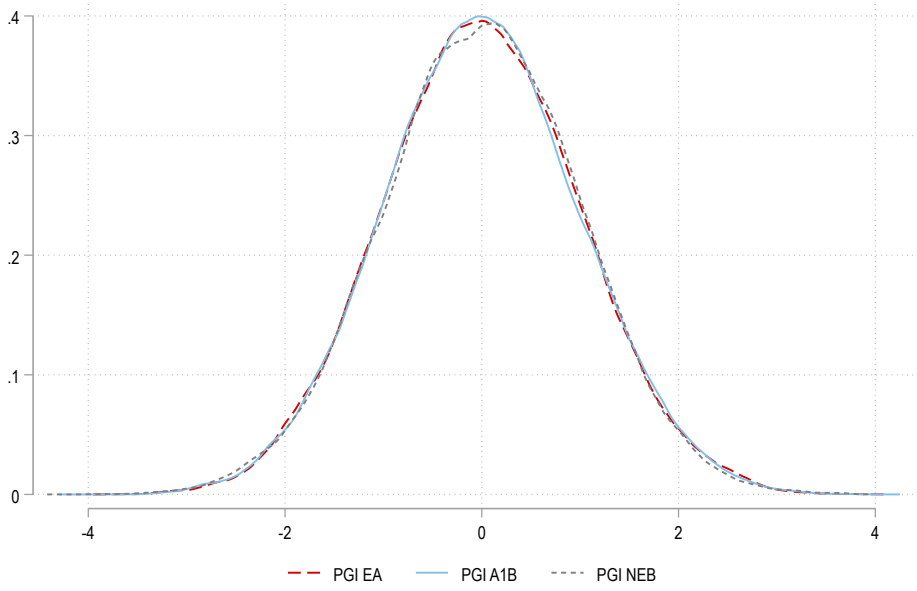
*Note*: Each dot in the map corresponds to the birth location of a UK Biobank respondent.

Figure A2: Fertility Outcomes by Sex and Birth Cohort in the UK Biobank



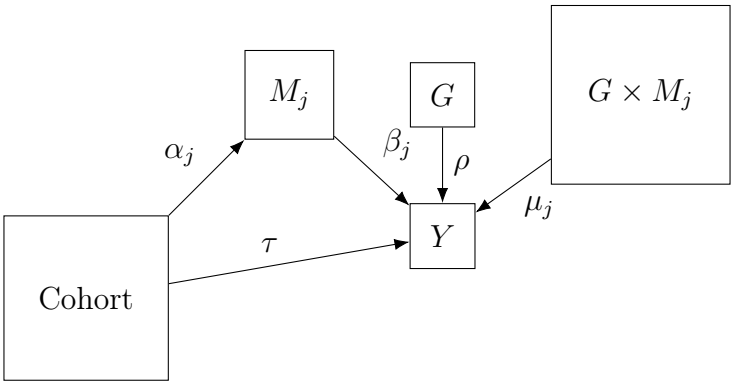

*Notes*: Each panel displays time trends in the outcomes: age at first birth (A1B - available only for women), number of children (NEB), and ever had a child (EVER). Each figure is constructed with the working samples used in estimation. For NEB and EVER, we restrict the sample to individuals aged 45 years old or more at the time of the interview.

Figure A3: Distribution of the polygenic indexes



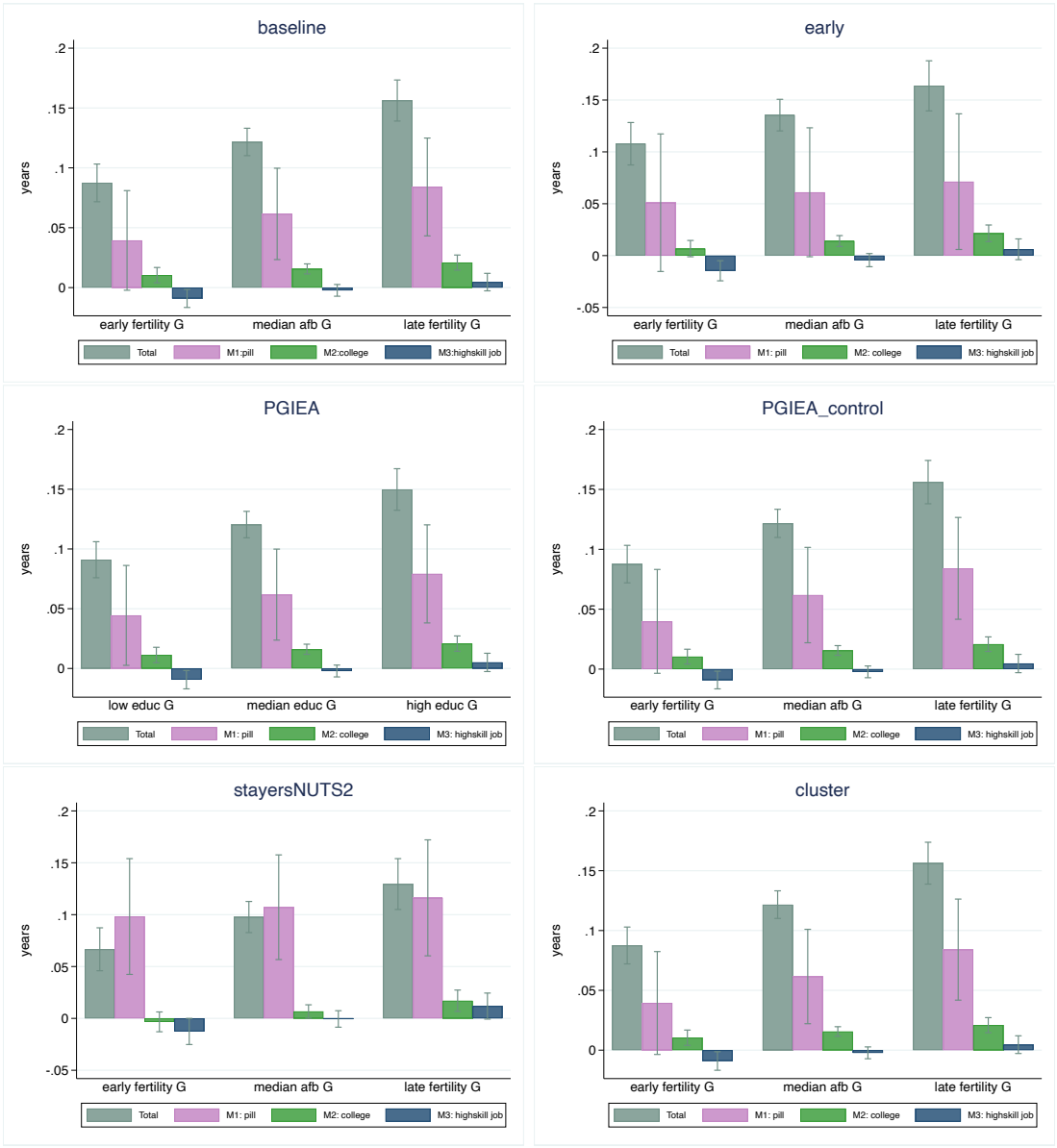

*Notes*: The figure plots the standardized kernel-smoothed density of three polygenic indexes for educational attainment, age at first birth and number of children (PGI EA, PGI A1B, and PGI NEB, respectively).

Figure A4: A Graphical Representation of the Moderated Mediation Model



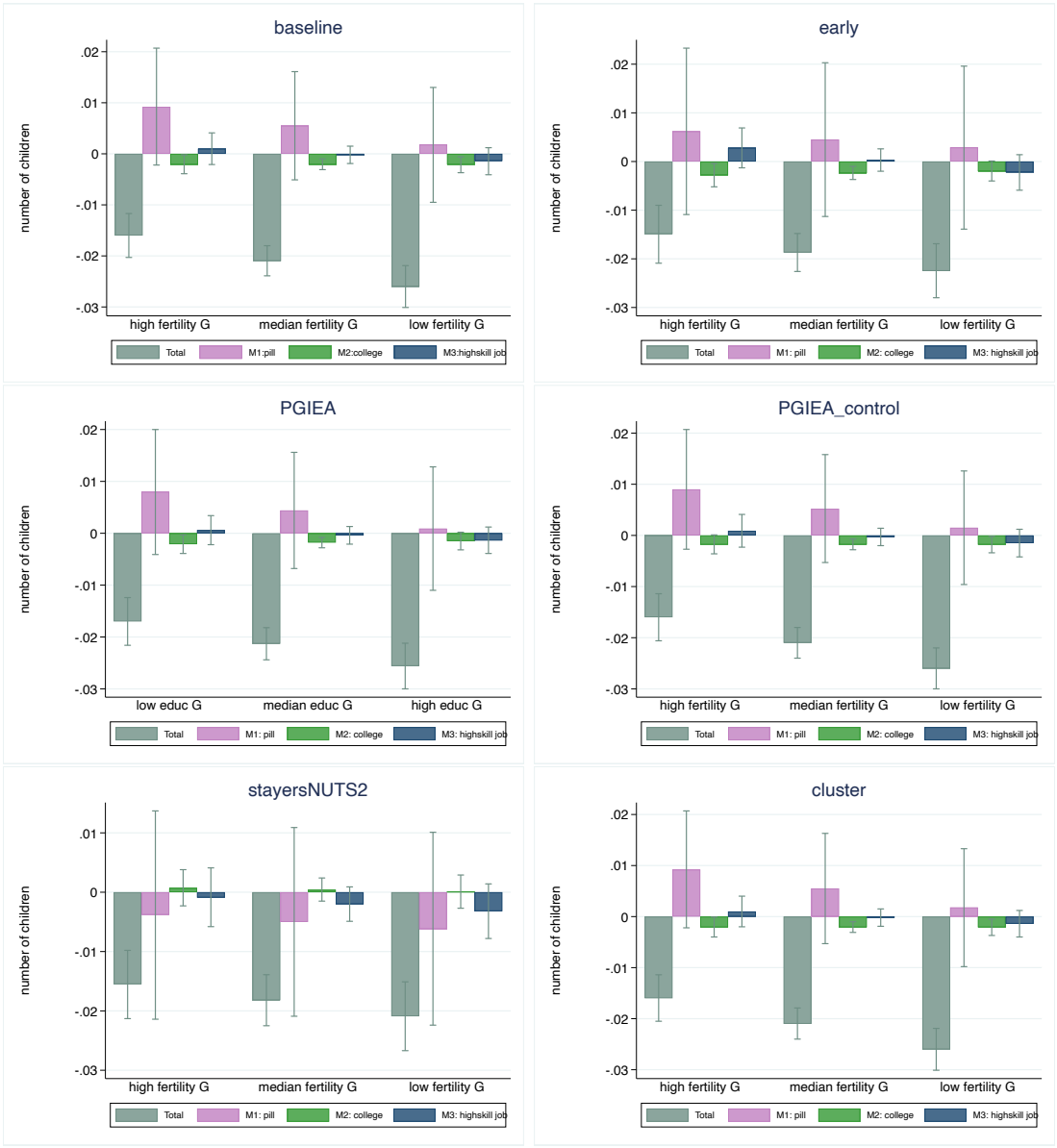

*Notes*: For simplicity, the figure show just one moderator $M_j$, instead of visualizing all three mediators used in the analysis.

Figure A5: Robustness checks on the mediation results for age at first birth



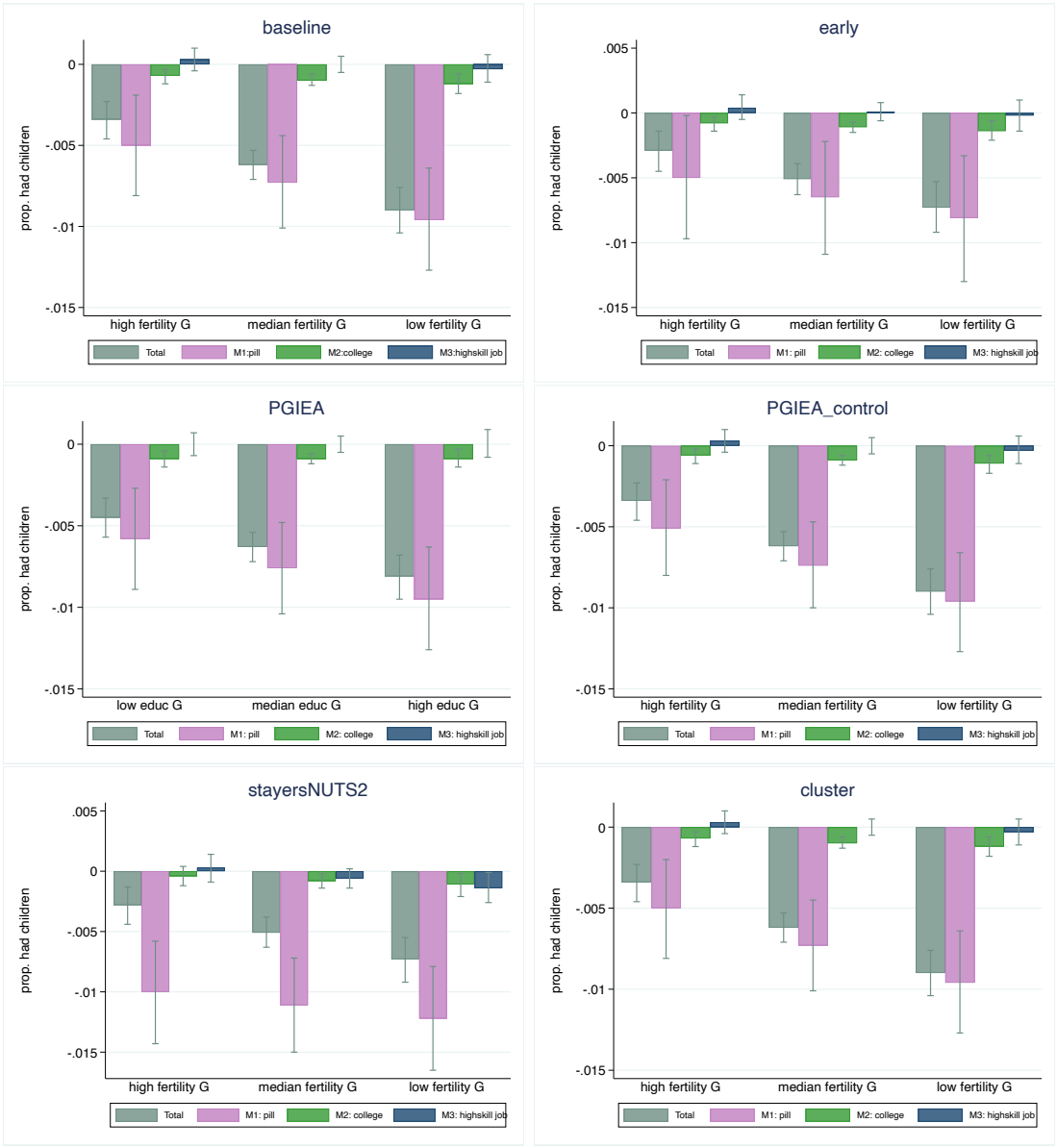

*Notes*: Each panel shows the total effect and the indirect effect for each mediation calculated at the 1st, 5th and 10th decile of the PGI for A1B distribution. The mediators are pill exposure (pink), women with a college degree (green) and women working in high skill jobs (blue). Early (late) fertility G corresponds to the 1st (10th) decile of the PGI for A1B. The plot baseline corresponds to the baseline mediation model (as in Figure 3), early includes early life controls, PGI EA is a model where PGI A1B is replaced by PGI EA, PGIEA_control includes the PGI EA as control, stayers NUTS are individuals which have not moved from the NUTS2 of birth, cluster is a model where S.E. are clustered at the district level. See the text for more details. All C.I. are bootstrapped.

Figure A6: Robustness checks on the mediation results for number of children



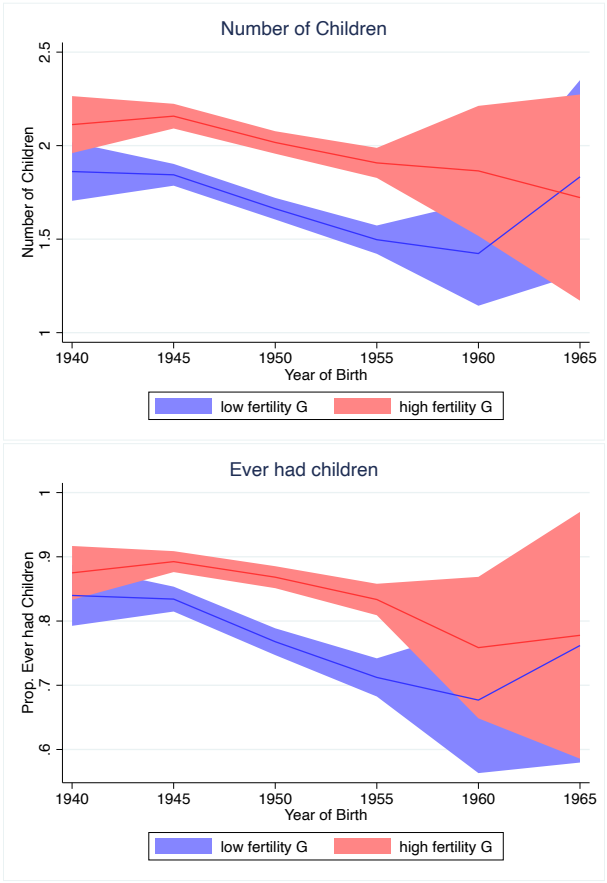

*Notes*: Each panel shows the total effect and the indirect effect for each mediation calculated at the 1st, 5th and 10th decile of the PGI for NEB distribution. The mediators are pill exposure (pink), women with a college degree (green) and women working in high skill jobs (blue). High (low) fertility G corresponds to the 10th (1st) decile of the PGI for NEB. The plot baseline corresponds to the baseline mediation model, early includes early life controls, PGI EA is a model where PGI NEB is replaced by PGI EA, PGIEA_control includes the PGI EA as control, stayers NUTS are individuals which have not moved from the NUTS2 of birth, cluster is a model where S.E. are clustered at the district level. See the text for more details. All C.I. are bootstrapped.

Figure A7: Robustness checks on the mediation results for ever had children




*Notes*: Each panel shows the total effect and the indirect effect for each mediation calculated at the 1st, 5th and 10th decile of the PGI for NEB distribution. The mediators are pill exposure (pink), women with a college degree (green) and women working in high skill jobs (blue). High (low) fertility G corresponds to the 10th (1st) decile of the PGI for NEB. The plot baseline corresponds to the baseline mediation model, early includes early life controls, PGI EA is a model where PGI NEB is replaced by PGI EA, PGIEA_control includes the PGI EA as control, stayers NUTS are individuals which have not moved from the NUTS2 of birth, cluster is a model where S.E. are clustered at the district level. See the text for more details. All C.I. are bootstrapped.

Figure A8: Male Fertility Outcomes by Birth Cohort in the UK Biobank




*Notes*: Each panel displays time trends in the outcomes for the 1st and the 10th decile of the PGI NEB. The outcomes are number of children (NEB) and ever had a child (EVER). We restrict the sample to men aged 45 years old or more at the time of the interview. High (low) fertility G corresponds to the 10th (1st) decile of the PGI for NEB.

Table A1: Stability of the Polygenic Indexes Across Birth Cohorts

| | PGI A1B Not Std (a) | PGI NEB Not Std (b) | PGI EA Not Std (c) | PGI A1B (d) | PGI NEB (e) | PGI EA (f) |
|---|---|---|---|---|---|---|
| 1940–1944 (cohort 2) | -0.006 | -0.001 | -0.005 | 0.001 | 0.004 | 0.009 |
| | (0.030) | (0.034) | (0.030) | (0.032) | (0.035) | (0.032) |
| 1945–1949 (cohort 3) | -0.021 | 0.025 | -0.015 | 0.002 | 0.005 | 0.012 |
| | (0.030) | (0.033) | (0.030) | (0.032) | (0.034) | (0.031) |
| 1950–1954 (cohort 4) | -0.048 | 0.023 | -0.047 | -0.001 | 0.008 | 0.009 |
| | (0.030) | (0.034) | (0.030) | (0.032) | (0.035) | (0.032) |
| 1955–1959 (cohort 5) | -0.062** | 0.049 | -0.052* | -0.005 | 0.008 | 0.003 |
| | (0.031) | (0.034) | (0.031) | (0.033) | (0.035) | (0.032) |
| 1960–1964 (cohort 6) | -0.077** | 0.058* | -0.069** | -0.004 | 0.008 | 0.003 |
| | (0.031) | (0.035) | (0.031) | (0.033) | (0.035) | (0.033) |
| 1965–1969 (cohort 7) | -0.056 | 0.075** | -0.039 | -0.005 | 0.008 | 0.002 |
| | (0.034) | (0.037) | (0.034) | (0.035) | (0.038) | (0.035) |
| Observations | 68,449 | 68,449 | 68,449 | 68,449 | 68,449 | 68,449 |
| pvalue test cohort2-cohort3 | 0.382 | 0.125 | 0.552 | 0.955 | 0.932 | 0.869 |
| pvalue test cohort2-cohort4 | 0.021 | 0.182 | 0.022 | 0.952 | 0.846 | 0.984 |
| pvalue test cohort2-cohort5 | 0.003 | 0.008 | 0.015 | 0.776 | 0.812 | 0.725 |
| pvalue test cohort2-cohort6 | 0.000 | 0.003 | 0.001 | 0.813 | 0.828 | 0.757 |
| pvalue test cohort2-cohort7 | 0.038 | 0.002 | 0.154 | 0.822 | 0.876 | 0.751 |
| pvalue test cohort3-cohort4 | 0.114 | 0.908 | 0.061 | 0.902 | 0.903 | 0.848 |
| pvalue test cohort3-cohort5 | 0.021 | 0.176 | 0.040 | 0.718 | 0.864 | 0.587 |
| pvalue test cohort3-cohort6 | 0.003 | 0.076 | 0.004 | 0.761 | 0.879 | 0.627 |
| pvalue test cohort3-cohort7 | 0.133 | 0.033 | 0.299 | 0.780 | 0.922 | 0.644 |
| pvalue test cohort4-cohort5 | 0.453 | 0.168 | 0.814 | 0.816 | 0.960 | 0.733 |
| pvalue test cohort4-cohort6 | 0.139 | 0.075 | 0.271 | 0.853 | 0.970 | 0.766 |
| pvalue test cohort4-cohort7 | 0.743 | 0.032 | 0.730 | 0.856 | 0.994 | 0.759 |
| pvalue test cohort5-cohort6 | 0.458 | 0.657 | 0.394 | 0.973 | 0.993 | 0.980 |
| pvalue test cohort5-cohort7 | 0.798 | 0.290 | 0.602 | 0.997 | 0.975 | 0.974 |
| pvalue test cohort6-cohort7 | 0.396 | 0.497 | 0.230 | 0.980 | 0.982 | 0.958 |

*Notes*: The table presents estimates from regressions of each polygenic index on 5-years cohort indicators. The reference cohort corresponds to women born in 1938-39. All regressions include the first 10 principal components of the full matrix of the SNP data, as well as an indicator variable for cohorts born during WWII and LAD fixed effects. In columns (a)–(c), 'PGI Not Std' corresponds to the unstandardized PGI by year of birth for each of the three outcomes. In columns (d)–(f), the PGI is standardized and used throughout the paper. PGI A1B, PGI NEB and PGI EA correspond to the PGI for age at first birth, number of children, and educational attainment, respectively. The table also reports the $p$-value of the test for the differences between cohort coefficients. In all regressions, the mean of the dependent variable is zero. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women in the analysis. Standard errors are robust to heteroskedasticity. * Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A2: Cross-Correlations of Outcomes and PGIs

| Variables | A1B | NEB | PGI A1B | PGI NEB | PGI EA |
|---|---|---|---|---|---|
| A1B | 1.00 | | | | |
| NEB | -0.26 | 1.00 | | | |
| PGI A1B | 0.26 | -0.09 | 1.00 | | |
| PGI NEB | -0.16 | 0.11 | -0.62 | 1.00 | |
| PGI EA | 0.25 | -0.06 | 0.90 | -0.36 | 1.00 |

*Notes*: The table reports the cross-correlations of the fertility outcomes under analysis and our three polygenic indexes (PGIs).

Table A3: Robustness: Estimates based on Different Sets of Controls

| | Age at first birth (A1B) | | | Number of children (NEB) | | | Ever had children (EVER) | | |
|---|---|---|---|---|---|---|---|---|---|
| | (a) | (b) | (c) | (d) | (e) | (f) | (g) | (h) | (i) |
| *Cohort* | 0.128*** | 0.096** | 0.095** | -0.022*** | -0.023** | -0.023** | -0.007*** | -0.006* | -0.006* |
| | (0.004) | (0.039) | (0.039) | (0.001) | (0.010) | (0.010) | (0.0003) | (0.003) | (0.003) |
| $G$ | 0.771*** | 1.275*** | 1.436*** | 0.091*** | 0.183** | 0.187** | 0.018*** | 0.037 | 0.044* |
| | (0.035) | (0.249) | (0.254) | (0.009) | (0.073) | (0.074) | (0.003) | (0.023) | (0.024) |
| $G \times Cohort$ | 0.031*** | 0.028*** | 0.020*** | 0.003*** | 0.002*** | 0.002** | 0.002*** | 0.002*** | 0.001*** |
| | (0.003) | (0.003) | (0.004) | (0.001) | (0.001) | (0.001) | (0.0002) | (0.0002) | (0.0003) |
| Observations | 48,861 | 48,861 | 48,861 | 65,656 | 65,656 | 65,656 | 65,656 | 65,656 | 65,656 |
| $R^2$ | 0.134 | 0.150 | 0.151 | 0.038 | 0.050 | 0.050 | 0.036 | 0.050 | 0.050 |
| District FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs×*Cohort* | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Controls×*Cohort*, Controls×$G$ | | ✓ | ✓ | | ✓ | ✓ | | ✓ | ✓ |
| PCs×$G$ | | | ✓ | | | ✓ | | | ✓ |
| Mean of Dep. Var. | 25.42 | 25.42 | 25.42 | 1.834 | 1.834 | 1.834 | 0.824 | 0.824 | 0.824 |
| SD of Dep. Var. | 4.557 | 4.557 | 4.557 | 1.156 | 1.156 | 1.156 | 0.381 | 0.381 | 0.381 |

*Notes*: Obtained from model (2). Each column corresponds to a different regression. $G$ refers respectively to the PGI A1B for A1B and to PGI NEB for NEB and EVER. All regressions include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 demeaned principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions PCs and year of birth (PCs×*Cohort*). Columns (a), (d) and (g) reports the baseline estimates. In columns (b), (e), and (h), we also control for the interactions between the controls (war and district fixed effects) and *Cohort*, and between the controls and $G$. Columns (c), (f) and (i) add interactions between PCs and $G$. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust to heteroskedasticity. * Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A4: Moderated mediation estimates for number of children

| | Pill exposure | Fem. college | Fem. high-skill jobs | Number of children |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Cohort trend | 3.5491*** | 0.7161*** | 0.8825*** | -0.0243*** |
| | (0.0088) | (0.0148) | (0.0115) | (0.0059) |
| Pill exposure | | | | 0.0016 |
| | | | | (0.0015) |
| Fem. college | | | | -0.0029*** |
| | | | | (0.0007) |
| Fem. high-skill jobs | | | | -0.0002 |
| | | | | (0.0010) |
| G | | | | 0.1530*** |
| | | | | (0.0076) |
| G×Pill exposure | | | | 0.0008* |
| | | | | (0.0005) |
| G×Fem. college | | | | 0.0001 |
| | | | | (0.0008) |
| G×Fem. high-skill jobs | | | | 0.0010 |
| | | | | (0.0011) |
| Observations | 62,680 | 62,680 | 62,680 | 62,680 |
| District FE | ✓ | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ | ✓ |
| Interactions PCs-trend | ✓ | ✓ | ✓ | ✓ |

*Notes*: Structural equation estimates of the moderated mediation equations (3-4). 'Pill exposure' is measured by the proportion of childless women who used the pill for the first time by age 18 in the local authority district (LAD) of birth of each woman in the sample. 'Female college' corresponds to the proportion of women with a college degree by LAD and year of birth. 'Female high skill job' is the proportion of women aged 18-30 whose first occupation was a high skill job in every LAD and linked to each woman in the UK Biobank when she was 18 years old. Pill exposure, female college and female high skill job are scaled to be in 100 percentage points. $G$ corresponds to the PGI NEB. All regressions include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise; the first 10 principal components of the full matrix of SNP data, district fixed effects, and the interactions between the first 10 demeaned PCs and the year of birth trend. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A5: Moderated mediation estimates for ever had children

| | Pill exposure | Fem. college | Fem. high-skill jobs | Ever had children |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Cohort trend | 3.5491*** | 0.7161*** | 0.8825*** | 0.0019 |
| | (0.0088) | (0.0148) | (0.0115) | (0.0015) |
| Pill exposure | | | | -0.0020*** |
| | | | | (0.0004) |
| Fem. college | | | | -0.0013*** |
| | | | | (0.0002) |
| Fem. high-skill jobs | | | | 0.0000 |
| | | | | (0.0003) |
| G | | | | 0.0375*** |
| | | | | (0.0020) |
| G×Pill exposure | | | | 0.0005*** |
| | | | | (0.0002) |
| G×Fem. college | | | | 0.0003 |
| | | | | (0.0002) |
| G×Fem. high-skill jobs | | | | 0.0003 |
| | | | | (0.0003) |
| Observations | 62,680 | 62,680 | 62,680 | 62,680 |
| District FE | ✓ | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ | ✓ |
| Interactions PCs-trend | ✓ | ✓ | ✓ | ✓ |

*Notes*: Structural equation estimates of the moderated mediation equations (3-4). 'Pill exposure' is measured by the proportion of childless women who used the pill for the first time by age 18 in the local authority district (LAD) of birth of each woman in the sample. 'Female college' corresponds to the proportion of women with a college degree by LAD and year of birth. 'Female high skill job' is the proportion of women aged 18-30 whose first occupation was a high skill job in every LAD and linked to each woman in the UK Biobank when she was 18 years old. Pill exposure, female college and female high skill job are scaled to be in 100 percentage points. $G$ corresponds to the PGI NEB. All regressions include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise; the first 10 principal components of the full matrix of SNP data, district fixed effects, and the interactions between the first 10 demeaned PCs and the year of birth trend. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A6: Summary Statistics for the Relative Samples

| | Sisters sample | | | Mother-daughter pairs sample | | |
|---|---|---|---|---|---|---|
| Variable | Mean | SD | *N* | Mean | SD | *N* |
| *Outcomes* | | | | | | |
| Age at first birth (A1B) | 24.68 | 4.68 | 9,062 | 24.78 | 4.99 | 2222 |
| Number of children (NEB) | 1.85 | 1.2 | 12,432 | 2.14 | 1.33 | 1452 |
| Ever had children (EVER) | 0.82 | 0.38 | 12,432 | 0.87 | 0.33 | 1452 |
| *Measures of genetic assessment* ($G$) | | | | | | |
| PGI A1B | -0.11 | 1.01 | 13,120 | -0.11 | 1.02 | 2251 |
| PGI NEB | 0.05 | 1 | 13,120 | 0.04 | 0.99 | 1480 |
| *Measure of environment (%)* | | | | | | |
| Pill exposure | 30.01 | 25.55 | 13,120 | | | |
| Pill exposure (A1B subsample) | | | | 47.82 | 32.37 | 2251 |
| Pill exposure (NEB subsample) | | | | 40.98 | 30.99 | 1480 |

*Notes*: SD refers to standard deviation, while $N$ refers to the number of women in the estimation sample. 'Pill exposure' is measured by the proportion of childless women who used the pill for the first time by age 18 in the local authority district (LAD) of birth of each woman in the sample. Statistics are weighted using the sampling weights.

Table A7: FORSE TOGLIAMO - NON VIENE PIU' GxCOHORT!!! Sisters G× Cohort Trend Estimates

| | Age at first birth | | Number of children | | Ever had children | |
|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Cohort trend | 0.0868*** | 0.1226*** | -0.0241*** | -0.0280*** | -0.0059*** | -0.0052*** |
| | (0.0149) | (0.0243) | (0.0032) | (0.0053) | (0.0011) | (0.0017) |
| G | 0.7432*** | 0.2834 | 0.0615* | 0.0694 | 0.0212** | 0.0115 |
| | (0.1292) | (0.2061) | (0.0342) | (0.0598) | (0.0101) | (0.0159) |
| G×Cohort trend | 0.0356*** | 0.0242 | 0.0071*** | 0.0036 | 0.0014* | 0.0017 |
| | (0.0095) | (0.0152) | (0.0025) | (0.0041) | (0.0008) | (0.0012) |
| Observations | 6,689 | 6,413 | 11,990 | 11,476 | 11,990 | 11,476 |
| R-squared | 0.2243 | 0.7581 | 0.0900 | 0.6822 | 0.0701 | 0.6609 |
| District FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| PCs-Cohort trend | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| Family FE | | ✓ | | ✓ | | ✓ |
| Mean of Dep. Var. | 24.99 | 25.02 | 1.849 | 1.847 | 0.822 | 0.821 |
| SD of De. Var. | 4.405 | 4.392 | 1.157 | 1.158 | 0.382 | 0.383 |

*Notes*: Obtained from the model (5) where $E$ corresponds to the linear cohort of birth. Each column corresponds to a specific outcome. $G$ corresponds respectively to the PGI A1B for the outcomes A1B, and the PGI NEB for the outcomes NEB and EVER. All columns include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions between the first 10 demeaned PCs and the year of birth trend (PCs-trend). Columns 2, 4 and 6 include family fixed effects. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust and clustered at the family level.

* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A8: FORSE TOGLIAMO!!! Mother-Daughter G× Cohort Trend Estimates

| | Age at first birth | | Number of children | |
|---|---|---|---|---|
| | (1) | (2) | (3) | (4) |
| Cohort trend | 0.2802*** | 0.2735*** | -0.0493*** | -0.0411*** |
| | (0.0197) | (0.0248) | (0.0084) | (0.0092) |
| G | 0.2772* | 0.0914 | 0.1014 | 0.2319** |
| | (0.1570) | (0.2635) | (0.0781) | (0.0988) |
| G×Cohort trend | 0.0345*** | 0.0445*** | 0.0021 | 0.0023 |
| | (0.0099) | (0.0121) | (0.0040) | (0.0044) |
| Observations | 2,222 | 2,108 | 1,452 | 1,352 |
| R-squared | 0.4164 | 0.8760 | 0.4337 | 0.8357 |
| District FE | ✓ | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ | ✓ |
| PCs-Cohort trend | ✓ | ✓ | ✓ | ✓ |
| Family FE | | ✓ | | ✓ |
| Mean of Dep. Var. | 24.33 | 24.30 | 2.161 | 2.178 |
| SD of De. Var. | 4.742 | 4.725 | 1.234 | 1.229 |

*Notes*: Obtained from the model (5) where $E$ corresponds to the linear cohort of birth. Each column corresponds to a specific outcome. $G$ corresponds respectively to the PGI A1B for the outcomes A1B, and the PGI NEB for the outcome NEB. All columns include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions between the first 10 demeaned PCs and the year of birth trend (PCs-trend). Columns 2 and 4 include family fixed effects. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust and clustered at the family level.

* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A9: Pill Estimates

| | Age at first birth (1) | Number of children (2) | Ever had children (3) |
|---|---|---|---|
| Pill exposure | 0.0267*** | 0.0004 | -0.0017*** |
| | (0.0033) | (0.0010) | (0.0003) |
| G | 0.9696*** | 0.1086*** | 0.0283*** |
| | (0.0276) | (0.0061) | (0.0019) |
| G×Pill exposure | 0.0089*** | 0.0008*** | 0.0005*** |
| | (0.0008) | (0.0002) | (0.0001) |
| Cohort trend | 0.0346*** | -0.0235*** | -0.0011 |
| | (0.0117) | (0.0036) | (0.0012) |
| Observations | 48,861 | 65,656 | 65,656 |
| R-squared | 0.1348 | 0.0381 | 0.0368 |
| District FE | ✓ | ✓ | ✓ |
| War FE | ✓ | ✓ | ✓ |
| PCs | ✓ | ✓ | ✓ |
| PCs-Pill exposure | ✓ | ✓ | ✓ |
| Mean of Dep. Var. | 25.42 | 1.834 | 0.824 |
| SD of Dep. Var. | 4.557 | 1.156 | 0.381 |

*Notes*: Obtained from the pill model (2) where instead of *Cohort* as a measure of the environment we use pill usage. Each column corresponds to a specific outcome. *G* corresponds respectively to the PGI A1B for the outcomes A1B, and the PGI NEB for the outcomes NEB and EVER. All regressions include: an indicator variable taking value 1 if a woman was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions between the first 10 demeaned PCs and the pill exposure (PCs-Pill exposure). All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of women used in the analysis. Standard errors are robust.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A10: Summary Statistics for Men

| Variable | Mean | SD | $N$ |
|---|---|---|---|
| *Outcomes* | | | |
| Number of children (NEB) | 1.84 | 1.29 | 39,225 |
| Ever had children (EVER) | 0.8 | 0.4 | 39,225 |
| *Measures of genetic assessment* ($G$) | | | |
| PGI NEB | 0 | 1 | 58,330 |
| *Mediators (%)* | | | |
| Pill exposure | 32.07 | 27.85 | 58,330 |
| Male college | 34.48 | 17.33 | 58,330 |
| Male high skill job | 43.17 | 11.92 | 58,330 |

*Notes*: SD refers to standard deviation, while $N$ refers to the number of men in the estimation sample. All mediators are derived from the UK Biobank. 'Pill exposure' is measured by the proportion of childless women who used the pill for the first time by age 18 in the local authority district (LAD) of birth of each man in the sample. 'Male college' corresponds to the proportion of men with a college degree by LAD and year of birth. 'Male high skill job' is the proportion of men aged 18–30 whose first occupation was a high skill job in every LAD and linked to each man in the UK Biobank when he was 18 years old. Statistics are weighted using the sampling weights.

Table A11: Baseline Estimates for Men

| | Number of children (1) | Ever had children (2) |
|---|---|---|
| Cohort trend | -0.0317*** | -0.0078*** |
| | (0.0022) | (0.0007) |
| G | 0.1050*** | 0.0227*** |
| | (0.0210) | (0.0058) |
| G×Cohort trend | 0.0024 | 0.0009 |
| | (0.0022) | (0.0006) |
| Observations | 39,225 | 39,225 |
| R-squared | 0.0237 | 0.0146 |
| District FE | ✓ | ✓ |
| War FE | ✓ | ✓ |
| PCs | ✓ | ✓ |
| PCs-trend | ✓ | ✓ |
| Mean of Dep. Var. | 1.844 | 0.798 |
| SD of Dep. Var. | 1.294 | 0.401 |

*Notes*: Obtained from the baseline model (2) estimated on the sample of men. Each column corresponds to a specific outcome. *G* corresponds to the PGI NEB for the outcomes NEB and EVER. All regressions include: an indicator variable taking value 1 if a man was born between 1939 and 1945, and 0 otherwise (War FE); the first 10 principal components of the full matrix of SNP data (PCs), district fixed effects, and the interactions between the first 10 demeaned PCs and the year of birth trend (PCs-trend). All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of men used in the analysis. Standard errors are robust.
* Significant at 10%; ** significant at 5%; *** significant at 1%.

Table A12: Decomposition Estimates for Men

| | Completed fertility | | | Ever had children | | |
|---|---|---|---|---|---|---|
| | G1 | G5 | G10 | G1 | G5 | G10 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Direct | -0.0387*** | -0.0387*** | -0.0387*** | -0.0063*** | -0.0063*** | -0.0063*** |
| | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] |
| M1: Pill exposure | 0.0079 | 0.0081 | 0.0084 | -0.0005 | -0.0010 | -0.0015 |
| | [0.195] | [0.129] | [0.205] | [0.793] | [0.512] | [0.421] |
| M2: Male college | -0.0005 | -0.0000 | 0.0004 | -0.0003 | -0.0001 | 0.0001 |
| | [0.594] | [0.964] | [0.661] | [0.282] | [0.581] | [0.700] |
| M3: Male high skill job | -0.0023 | -0.0005 | 0.0013 | -0.0009 | -0.0002 | 0.0006 |
| | [0.334] | [0.727] | [0.502] | [0.202] | [0.664] | [0.325] |
| Total | -0.0336*** | -0.0311*** | -0.0285*** | -0.0081*** | -0.0076*** | -0.0071*** |
| | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] | [0.000] |
| Observations | 39,225 | 39,225 | 39,225 | 39,225 | 39,225 | 39,225 |

*Notes*: Obtained from the moderated mediation model (3-4). The table reports the direct effect of the cohort trend on each outcome, the indirect effects coming from the three mediators (M1: Pill exposure, M2: Male college and M3: Male high skill job), and the Total effect which is the sum of the direct and indirect effects. Indirect effects are moderated by G we report their estimate based on three values of the G distribution. $G1$, $G5$ and $G10$ correspond respectively to the first, fifth, and tenth decile of PGI NEB (also defined low, medium, high fertility G). All models include: an indicator variable taking value 1 if a man was born between 1939 and 1945, and 0 otherwise; district fixed effects; the first 10 principal components of the full matrix of SNP data; and the interactions between the first 10 demeaned PCs and the year of birth trend. All regressions are weighted using the sampling weights created by van Alten et al. (2022). 'Observations' is the number of men used in the analysis. Bootstrapped p-values are reported in square brackets.
* Significant at 10%; ** significant at 5%; *** significant at 1%.