Socioeconomic status, historical context and genetic variants in shaping human fertility behaviour

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Abstract

This study demonstrates that human fertility behaviour is a consequence of the complex interplay among individuals' gene, socioeconomic status (SES) and the historical context in which they live. Drawing on approximately 9,869 genetic samples from the Health and Retirement Study, I first investigate how childhood and adulthood SES (measured by father's education and own education) moderate the impact of the genetic predictors of the number of children ever born and age at the first birth. I then consider differences across birth cohorts in the genetic influence on fertility, and cohort variations in the moderating effects of SES on the genetic influence. The analyses suggest that the genetic influence on NEB is greater for individuals who are from disadvantaged SES background. In other words, the SES disparity in NEB manifests only for individuals with a higher genetic propensity to NEB, but not for those with lower genetic propensities. Such a relationship is not observed in AFB. The preliminary results show non-significant PGS × Cohort interactions, suggesting no significant changes in genetic influence on fertility behaviours across cohorts.

Extended abstract

Introduction

Fertility has been a central interest of research within the discipline of demography and sociology during the past few decades. The United States has witnessed dramatic changes in fertility rates and childbearing since the 1930s (Bailey and Hershbein 2018). Human reproductive behaviours are complex traits resulting from multiple, not necessarily independent, factors. Genetic predisposition, socioeconomic status (SES), and historical context are all indicators of fertility. A recent genome-wide association study (GWAS) (Barban et al. 2016) identified 12 lead genetic variants associated with reproductive behaviours – i.e. number of children ever born (NEB) and age at first birth (AFB). These genetic variants, involved in various biological pathways such as follicle stimulation, sperm differentiation, and ovulation defects, play essential roles in fertility outcomes.

From a sociological point of view, socioeconomic status has long been recognised as a strong predictor of reproductive behaviour (Balbo et al. 2013; Mills et al. 2011). Higher educational achievement is associated with postponement of family formation and childbearing, and reduced family size. In developed countries such as the United States, education is negatively associated with the number of children ever born for both men and women (Hopcroft 2006; Huber et al. 2010; Weeden et al 2006). Moreover, since the 1930s the US has experienced economic crisis, wartime, sex revolution, and changes in family planning policies. These changes all contribute to the vary fertility behaviours in the United States.

This study seeks to bridge the three dimensions of inquiry – genetic predisposition, SES and historical/environmental context – to further understand human reproductive behaviours. As shown by gene-environment interaction (G×E) studies (Tropf and Mandemakers 2017; Liu and

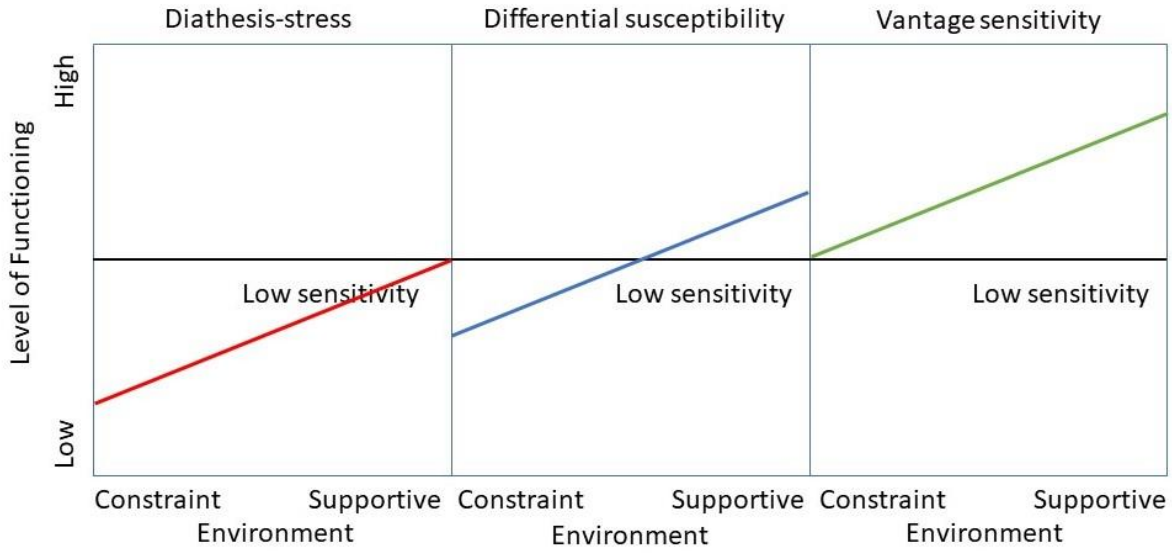
Guo 2016; Weddow et al. 2018), genetic, socioeconomic and contextual factors do not act independently, but interactively affect behaviours and traits. Past research casts doubt on the causal effect of education on reproductive outcomes, suggesting that family background characteristics may cause spurious associations between education and fertility behaviours (Tropf and Mandemakers 2017). Both SES at childhood and adulthood can be critical in forming the relationship between genetic predisposition and reproductive outcomes. This calls for an integration of genetic research and demography in the investigation of fertility.

There are three aims of this study. First, I examine how SES at a younger age and in the adulthood moderates the genetic influence on fertility behaviours measured by NEB and AFB. Second, I consider differences across birth cohorts in the genetic influence based on the proposition that cohort differences reflect changes in the socio-historical context which individuals lives unfold. Third, I investigate cohort variations in the moderating effects of SES on the genetic influence. The dataset I use is the Health and Retirement Study (HRS) with both SES and genotypic information.

Theoretical Framework and Hypotheses

Genetic liability interacts with environmental factors such as SES and family background. The *diathesis-stress model* (also known as the social trigger/compensation model) suggests that some individuals with genetic "risks" of fertility problems could be disproportionately susceptible to environmental conditions or behaviours that are childbearing unfriendly (Ellis et al. 2011). The *vantage sensitivity* model, stipulating that some individuals disproportionately benefit from supportive conditions due to their genetic makeup (Pluess & Belsky, 2013). The *differential susceptibility model* indicates that individuals with specific genotypes are more sensitive to environmental conditions. Compared to other people, individuals with a higher genetic propensity for age at first childbearing may find it easier to fulfil their fertility intentions in favourable environments, whereas the same individuals are more likely to postpone childbearing if the opportunity cost is high. See *Figure 1* for a graphical representation of the three models.

Figure 1. A graphical representation of the three G×E hypotheses tested.



In this study, I use SES as an indicator of the social environment. I focus on socioeconomic factors both in childhood and in adulthood to capture the life course dynamics. Individuals from lower SES family background in childhood are associated with adolescent pregnancy and a higher number of children (Singh, Darroch and Frost 2001; van Roode et al. 2017). This relationship can be explained by pathways such as "intergenerational transmission" of social norms for earlier childbearing, or less parental control (Barber 2001). Economic theorists suggest that reproductive behaviours are influenced by costs and opportunity costs associated with having children (Becker 2009). The direct expenses or raising children, as long as lost career opportunities or income may be higher for those with higher SES, or those with higher levels of education. Associations between low childhood and adult SES and earlier parenthood appears persistent among countries (van Roode et al. 2017). Given the relationship between SES and reproductive outcomes, I expect that *genetic influences on fertility behaviours are socio-economically moderated* (Hypothesis 1).

I also use cohort effects in this study as an indicator of historical change to examine whether the genetic influence on reproductive behaviours and the moderating effects of SES on the genetic influence are contingent on historical background. A cohort is a group of individuals who share the experience in the same historical contexts and events in the same period (Ryder 1965). Different cohorts may have different exposures to historical and social events at different stages of their lives (Liu and Guo, 2015). Jalovaara and Andersson (2018) show that childlessness was highest among the highly educated in older female cohorts in the Nordic countries, but the patterns have changed over the cohorts as childlessness has increased among the low educated and remained relatively stable among higher educated women. Tropf et al. (2015) demonstrate that economic uncertainty appears to override freedom from normative constraints to encourage the activation of genetic effects on decisions about childbearing. For women who entered adulthood during the Second World War, when significant environmental constraints forced postponement of childbearing, there is no significant genetic influence on the age at first birth. In contrast, for individuals born in the 1930s who experienced liberalisation of cultural values and changing sexual norms at reproductive age, the heritability of fertility traits showed a sharp peak. In this study, I use a nationally representative, longitudinal sample to examine the cohort effect in fertility. I expect that the *genetic influence on fertility behaviours changes across cohorts, and the cohort differences in the genetic influence on fertility are greater for individuals with a higher genetic predisposition.* (Hypothesis 2).

Methods

Data

Data for this study is from the Health and Retirement Study (HRS), which is an ongoing nationally representative, biennial longitudinal panel study of over 26,000 American individuals aged 50 and above (Ofstedal et al. 2011). The survey was designed to study the metrics of family, employment, wealth and health. In 2006, HRS initiated an enhanced face-to-face (EFTF) interview. In addition to the core interview, the EFTF interview includes a set of physical performance tests, anthropometric measurements, blood and saliva samples and a self-administered questionnaire on psychosocial topics. This study links HRS survey data compiled by Rand Corporation (Bugliari et al. 2018) with the most updated publicly available

HRS polygenic scores (Ware et al. 2018). The HRS survey data consists of 34,679 persons over age 50, among which 10,961 were genotyped between 2006-2012. I limited the sample to individuals of European genetic ancestry based on the GWAS meta-analysis on reproductive behaviours.

Dependent variable

I study two dependent variables in this study – the number of children ever born (NEB) and age at first birth (AFB). The number of children born to the respondent was reported by each respondent individually. Information on respondents' childbearing histories is derived from a roster of living biological children, including each child's age. In combination with the respondent's birth year, these data allowed calculation of the timing of first birth. The data exclude children who have died as well as children who may have been given up for adoption at birth.

Childhood SES is measured by father's years of education. Adulthood SES is based on the respondent's years of education.

Cohort is based on a respondent's birth year. The HRS includes seven birth cohorts at the time of this analysis: The Study of Assets and Health Dynamics Among the Oldest Old (AHEAD) cohort (born before 1924); the Children of Depression (CODA) cohort (born 1924 to 1930); the HRS cohort (born 1931 to 1941) the War Baby (WB) cohort (born 1942 to 1947); the Early Baby Boomers (EBB) cohort (born 1948 to 1953); the Mid Baby Boomers (MBB) cohort (born 1954 to 60), and the Late Baby Boomer (LBB) cohort (1960 to 65). Because for LBB cohort, there are only 61 observations with genotyped data, this study only includes the first six cohorts.

We use polygenic scores (PGS) to measure genetic predictors for NEB and AFB. The PGS is extracted from the public available HRS polygenic score – release 3 dataset. The score was created using results from a 2016 GWAS (Barban et al. 2016) for reproductive behaviours. Both PGS for AFB and NEB are standardised.

In this analysis, we controlled for age, sex (except for sex stratified models), the most significant ten principal components to account for population stratification (Price et al. 2006).

Analytic Strategy

Multivariate analysis is used in the examination of the interaction effects of fertility genes, SES and cohort on fertility outcomes. Specifically:

$$
\begin{aligned}
Fertility_i &= \beta_0 + \beta_1 PGS_i + \beta_2 SES_i + \beta_3 Cohort_i \\
&+ \beta_4 (PGS_i \times SES_i) + \beta_5 (PGS_i \times Cohort_i) + \beta_6 (SES_i \times Cohort_i) \\
&+ \beta_7 (PGS_i \times SES_i \times Cohort_i) \\
&+ \sum_p \beta_p X_{pi} + \varepsilon_i
\end{aligned}
$$

where Fertility outcome for individual i is predicted by PGS, SES, cohort and their interaction terms. X_{pi} represents covariates for p = 1, …, p.

Preliminary results

The summary statistics by cohort and gender are reported in Table 1. I examined bivariate associations of PGS, SES and cohort with NEB and AFB. PGS for both NEB and AFB are standardised. Higher scores of NEB indicate greater genetic propensities to have more children. Higher scores of AFB suggests greater genetic propensities to have children at an older age. PGS is positively associated with their related phenotypes (See Figure 2). Table 2 and Figure 3 show that the PGS-NEB correlation is stronger from earlier cohorts than for more recent cohorts, where the 1924-1930 CODA cohort has the strongest correlations. The CODA cohort was sexually mature right after the Second World War and experienced less social constraints on their fertility behaviours. Higher childhood SES for women does not predict less NEB in the earliest cohort. The association between women's education and NEB becomes stronger from the HRS cohort to the EBB cohort, for the most recent MBB cohort, the relationship between education NEB disappears. For men, childhood SES is not associated with NEB. Genetic predictors are associated with men's NEB across all the cohorts and the pattern is similar with women.

The association between PGS and AFB is more complex to summarise (Table 3). There is a declining in the strength of association from the earliest cohort to WB for women, but spikes at EBB cohort and then disappears for the MBB cohort. For male, the PGS does not predict AFB significantly after accounting for SES. Adulthood SES is important in predicting AFB for men and women. Education has become more and more important over the years in predicting AFB. The higher the PGS, the older age people will have their first child.

The result from Table 4 displays results testing for interactions of SES and PGS on NEB and AFB. Results from NEB models are consistent with the *diathesis-stress model* that the genetic influence on NEB is greater for individuals who are from disadvantaged SES background. In other words, the SES disparity in NEB manifests only for individuals with a genetic propensity to NEB, but not for those with lower genetic propensities. The interaction relationship is plotted in Figure 4. Such a relationship is not observed in AFB. The preliminary results show non-significant PGS × Cohort interactions, suggesting no significant changes in genetic influence on fertility behaviours across cohorts.

In Table 5, we found cohort differences in the moderating effects of childhood SES on the gentic influence. The three-way PGS × Father's education × Cohort is negative and the effect size is rather small (coefficient = -0.006 se = 0.002). Figure 5 plots the three-way interactions. The cohort differences in the association between PGS and childhood SES disappears in most recent cohorts.

Table 1. Analytic sample summary statistics by cohort and gender

	AHEAD Mean (SE)	CODA Mean (SE)	HRS Mean (SE)	WB Mean (SE)	EBB Mean (SE)	MBB Mean (SE)	Total Mean (SE)
Female							
NEB	2.757	3.063	2.988	2.437	2.179	2.126	2.701
	(1.337)	(1.302)	(1.320)	(1.149)	(1.085)	(1.026)	(1.288)
AFS	25.30	23.74	22.67	22.49	24.78	25.47	23.60
	(4.798)	(4.357)	(4.153)	(4.451)	(5.583)	(5.941)	(4.828)
Father's Education	8.159	8.843	9.620	10.41	11.29	12.01	9.948
	(1.031)	(3.401)	(3.578)	(3.437)	(3.399)	(3.249)	(3.483)
Education	12.43	12.65	12.72	13.17	13.80	13.83	13.00
	(2.272)	(2.410)	(2.270)	(2.295)	(2.217)	(2.117)	(2.324)
Birth Year	1919.3	1927.3	1936.3	1944.2	1950.5	1956.6	1938.4
	(3.249)	(1.952)	(3.099)	(1.713)	(1.697)	(1.797)	(10.92)
Observations	536	831	2025	890	658	580	5520
Male							
NEB	2.609	2.959	2.762	2.329	2.165	2.172	(1.275)
	(1.329)	(1.338)	(1.296)	(1.170)	(1.139)	(1.061)	26.40
AFS	28.54	26.67	25.66	25.47	26.68	27.72	(5.526)
	(5.361)	(4.773)	(5.149)	(5.593)	(5.986)	(6.300)	10.27
Father's Education	8.182	9.292	9.847	10.69	11.48	12.18	(3.545)
	(1.712)	(3.626)	(3.565)	(3.563)	(3.242)	(3.147)	13.42
Education	12.96	13.09	13.13	13.75	14.14	13.78	(2.742)
	(3.001)	(3.028)	(2.845)	(2.535)	(2.302)	(2.292)	1939.3
Birth Year	1919.6	1927.2	1936.3	1944.5	1950.5	1956.5	(10.86)
	(3.207)	(1.937)	(3.188)	(1.760)	(1.706)	(1.662)	(1.275)
Observations	335	683	1687	607	655	534	4501

Note: NEB = number of children ever born; AFB = age at first birth
Darker shading means higher score.

Figure 2. Genetic Predisposition and fertility behaviours in the Health and Retirement Study.

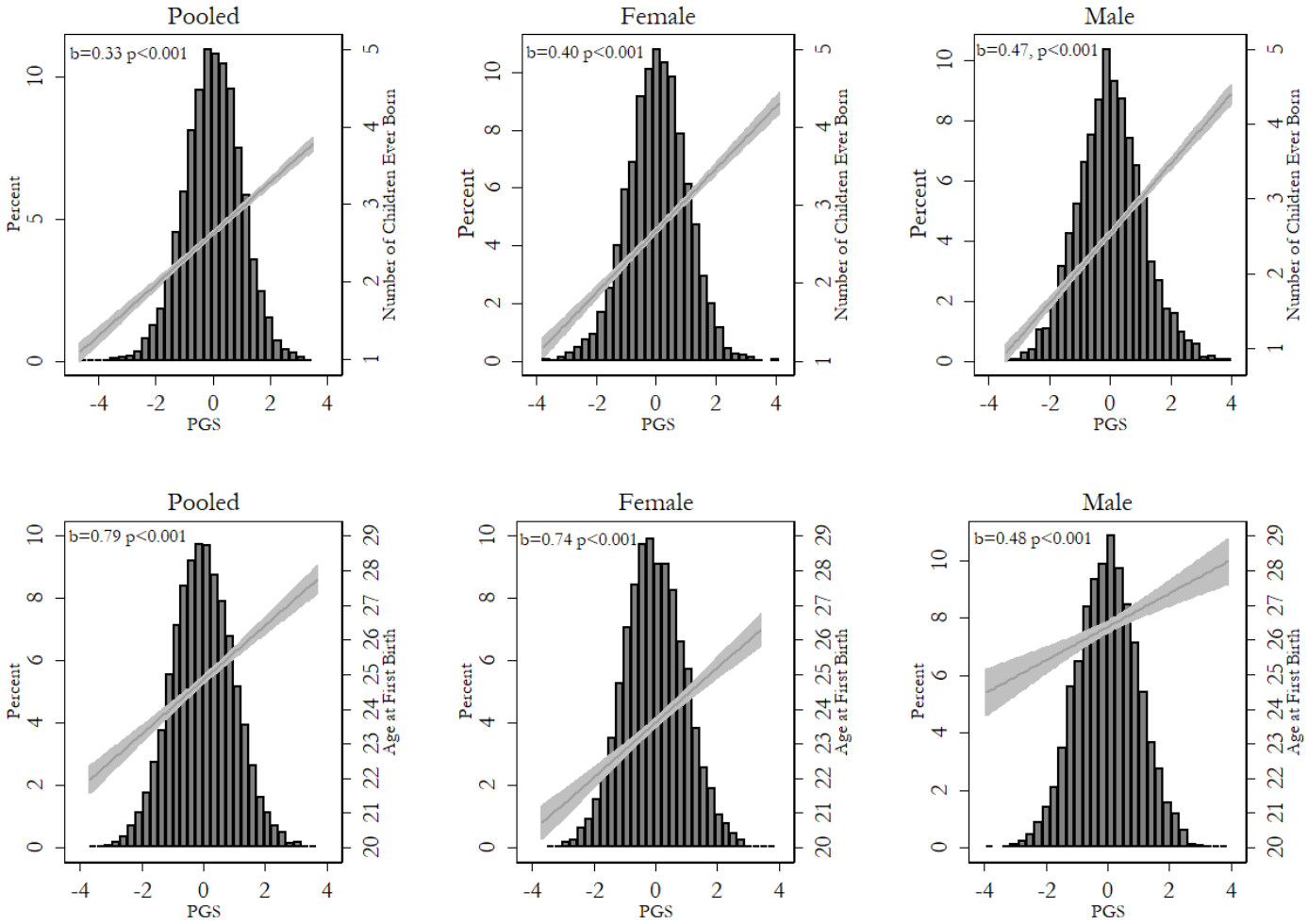


Table 2 Genetic and socioeconomic associations with NEB by cohort and gender

	AHEAD Beta (std.err.)	CODA Beta (std.err.)	HRS Beta (std.err.)	WB Beta (std.err.)	EBB Beta (std.err.)	MBB Beta (std.err.)	Total Beta (std.err.)
Female NEB							
PGS_NEBf	0.374***	0.420***	0.383***	0.364***	0.287***	0.188***	0.349***
	(0.052)	(0.039)	(0.025)	(0.036)	(0.049)	(0.049)	(0.015)
Father's Education	-0.0793	0.0203	0.00458	-0.0157	-0.000476	-0.0218	-0.00385
	(0.042)	(0.013)	(0.007)	(0.011)	(0.013)	(0.014)	(0.005)
Education	-0.0198	-0.00228	-0.0739***	-0.0974***	-0.0504*	-0.0219	-0.0571***
	(0.023)	(0.021)	(0.012)	(0.017)	(0.022)	(0.023)	(0.008)
Age	0.0585***	0.0120	-0.0242**	-0.0599**	-0.0280	0.00184	-0.0183***
	(0.015)	(0.020)	(0.008)	(0.020)	(0.023)	(0.024)	(0.001)
Constant	7.970***	3.581**	2.239***	0.685	1.568	2.745**	2.299***
	(1.282)	(1.375)	(0.489)	(1.048)	(1.074)	(1.006)	(0.139)
Observations	488	674	1838	796	577	521	4894
Male NEB							
PGS_NEBm	0.430***	0.459***	0.445***	0.345***	0.295***	0.121*	0.385***
	(0.050)	(0.036)	(0.021)	(0.042)	(0.041)	(0.056)	(0.014)
Father's Education	0.000328	-0.00932	-0.00653	0.0161	-0.00275	-0.0133	-0.00384
	(0.043)	(0.011)	(0.007)	(0.014)	(0.013)	(0.015)	(0.005)
Education	0.0261	-0.00360	-0.0236*	-0.0310	0.0438*	0.0127	-0.00958
	(0.021)	(0.014)	(0.010)	(0.023)	(0.020)	(0.022)	(0.007)
Age	-0.0543**	0.0131	0.0438***	0.0240	0.0104	0.0312	0.0213***
	(0.020)	(0.021)	(0.008)	(0.025)	(0.026)	(0.027)	(0.002)
Constant	6.369***	2.092	0.380	1.279	0.991	0.927	1.428***
	(1.626)	(1.480)	(0.470)	(1.345)	(1.239)	(1.115)	(0.137)
Observations	308	584	1545	544	587	487	4055

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

PGS = polygenic score; NEB=number of children ever born; f = female; m=male.

Table 3 Genetic and socioeconomic associations with AFB by cohort and gender

	AHEAD Beta (std.err.)	CODA Beta (std.err.)	HRS Beta (std.err.)	WB Beta (std.err.)	EBB Beta (std.err.)	MBB Beta (std.err.)	Total Beta (std.err.)
Female AFB							
PGS_AFBf	0.485*	0.522**	0.211*	0.304	0.741**	-0.0486	0.348***
	(0.226)	(0.176)	(0.100)	(0.172)	(0.250)	(0.290)	(0.074)
Father's Education	0.180	0.0166	0.0191	0.155**	0.177*	0.181*	0.102***
	(0.189)	(0.053)	(0.030)	(0.049)	(0.076)	(0.087)	(0.022)
Education	0.533***	0.441***	0.549***	0.570***	0.753***	1.061***	0.613***
	(0.106)	(0.091)	(0.054)	(0.075)	(0.139)	(0.142)	(0.037)
Birth Year	0.266***	0.212**	0.106***	-0.0163	-0.291*	0.0593	0.0232***
	(0.072)	(0.080)	(0.031)	(0.084)	(0.128)	(0.132)	(0.007)
Constant	-3.184	3.413	9.317***	14.26**	25.66***	6.346	13.37***
	(6.016)	(5.642)	(1.944)	(4.481)	(5.881)	(5.648)	(0.634)
Observations	488	669	1808	772	562	507	4806
Male AFB							
PGS_AFBm	0.0815	-0.0149	0.131	0.249	0.568*	0.413	0.234**
	(0.349)	(0.202)	(0.131)	(0.231)	(0.244)	(0.307)	(0.088)
Father's Education	0.147	0.0643	0.0288	-0.0283	0.259**	0.161	0.0933***
	(0.225)	(0.053)	(0.040)	(0.073)	(0.080)	(0.102)	(0.028)
Education	0.160	0.231***	0.386***	0.716***	0.414**	0.702***	0.389***
	(0.116)	(0.067)	(0.050)	(0.102)	(0.134)	(0.138)	(0.035)
Birth Year	0.406***	0.265**	0.0395	-0.00357	-0.172	0.0217	0.0173*
	(0.103)	(0.096)	(0.039)	(0.135)	(0.150)	(0.164)	(0.009)
Constant	-6.024	4.668	17.89***	16.04*	25.84***	15.31*	19.20***
	(8.443)	(6.718)	(2.444)	(7.071)	(6.983)	(6.651)	(0.759)
Observations	308	578	1529	534	580	486	4015

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

PGS = polygenic score; AFB=age at first birth; f = female; m=male.

Figure 3. Visualization of PGS, childhood SES and adulthood SES association with reproductive measures.

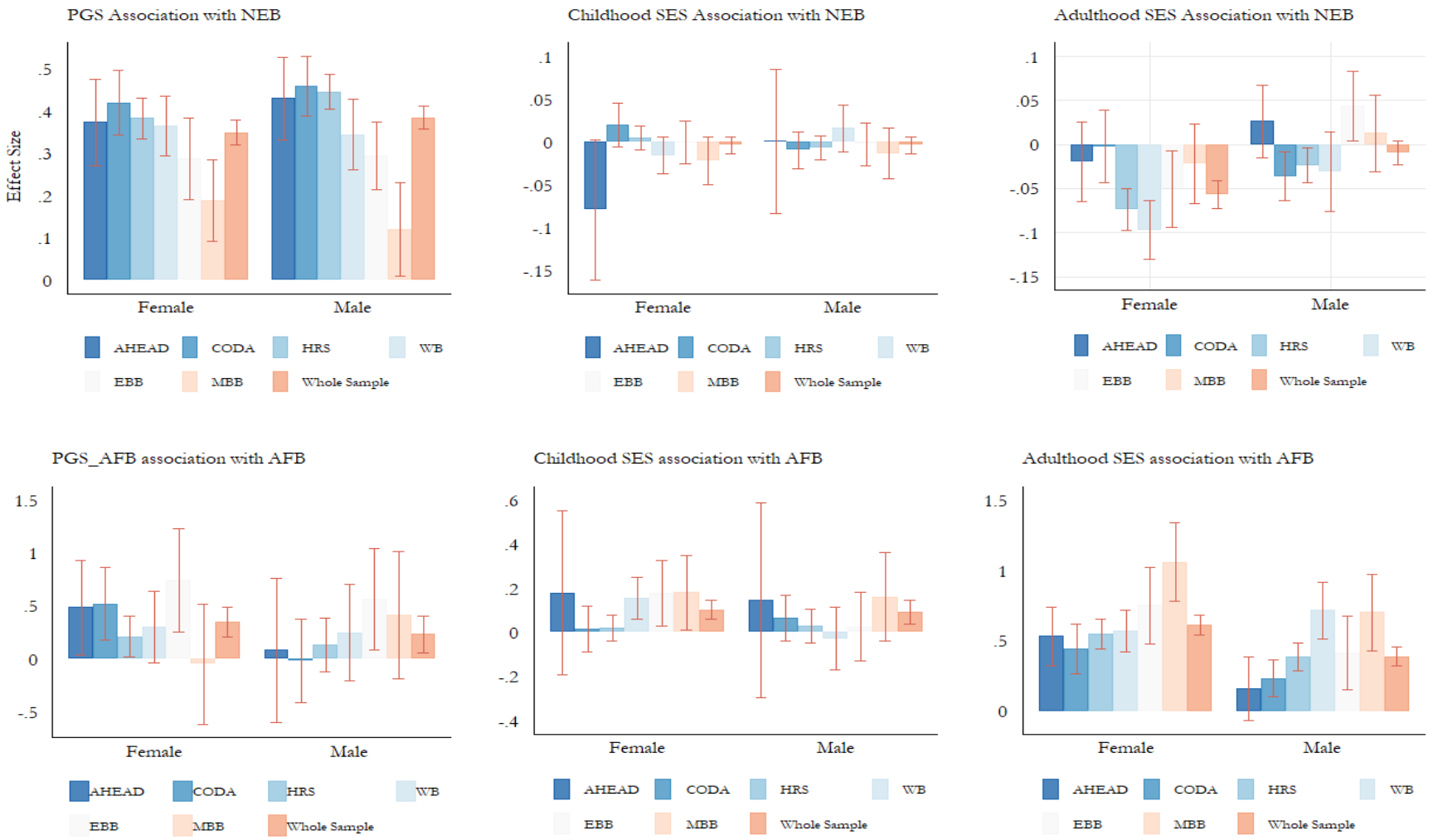


Table 4. Coefficients (standard errors) of models assessing moderating effects of SES on the genetic association with fertility (Hypothesis 1).

	NEB		AFB	
	Childhood SES	Adulthood SES	Childhood SES	Adulthood SES
PGS	0.391***	0.416***	0.487**	0.210
	(0.032)	(0.056)	(0.165)	(0.282)
Father's Education	-0.0131***		0.211***	
	(0.004)		(0.017)	
PGS × Father's Education	-0.00873**		0.00464	
	(0.003)		0.487**	
Education		-0.0339***		0.502***
		(0.005)		(0.023)
PGS × Education		-0.00907*		0.0143
		(0.004)		(0.021)
Age	0.381***	0.375***	(0.016)	-0.692*
	(0.054)	(0.051)	-0.667*	(0.269)
Age^2	-0.00261***	-0.00258***	(0.289)	0.00645***
	(0.000)	(0.000)	0.00631**	(0.002)
Cohort	0.873***	0.827***	(0.002)	0.322
	(0.169)	(0.160)	0.462	(0.847)
Age × Cohort	-0.0134***	-0.0127***	(0.914)	-0.00207
	(0.003)	(0.003)	-0.00336	(0.014)
Female	0.0672***	0.0759***	(0.015)	-2.538***
	(0.019)	(0.018)	-2.611***	(0.084)
Constant	-10.89***	-10.27***	40.04***	36.57***
	(1.876)	(1.773)	(10.144)	(9.388)
N	8878	9929	8878	9929

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

NEB = Number of children ever born

AFB = Age at first birth

PGS = Polygenic score

Figure 4. Genetic Association with NEB for individuals with different genetic propensity.

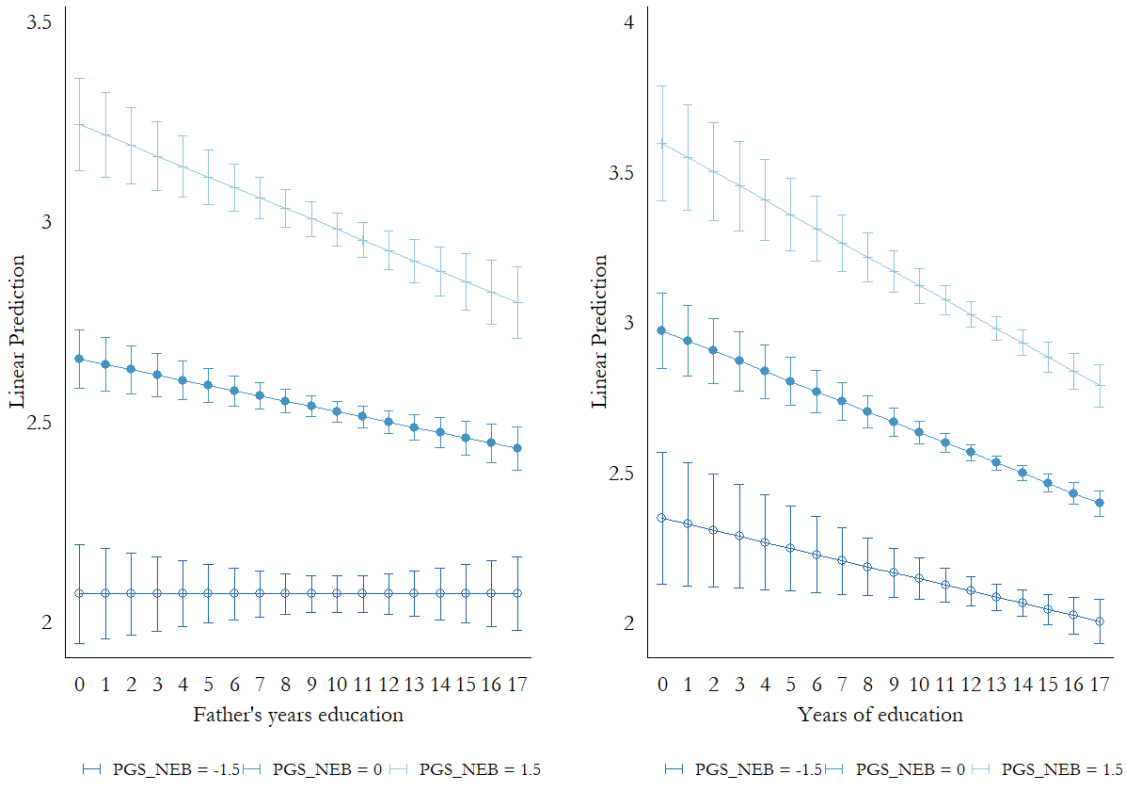


Table 5. Coefficients (standard errors) of models assessing cohort differences in the genetic association with NEB and AFB (Hypothesis 2).

	NEB		AFB	
	Childhood SES	Adulthood SES	Childhood SES	Adulthood SES
PGS	0.269**	0.307*	-0.266	0.238
	(0.090)	(0.135)	(0.465)	(0.691)
Father's Education	-0.00741		-0.0506	
	(0.010)		(0.052)	
PGS × Father's Education	0.0187*		0.0503	
	0.269**		(0.047)	
Education		-0.00949		0.0472
		(0.012)		(0.058)
PGS × Education		0.0105		0.00211
		(0.010)		(0.052)
Cohort	0.919***	0.945***	-0.915	-1.729
	(0.177)	(0.168)	(0.954)	(0.883)
PGS × Cohort	0.0184	0.0184	0.248	-0.0374
	(0.026)	(0.040)	(0.136)	(0.220)
Father's Education × Cohort	-0.00172		0.0731***	
	(0.003)		(0.014)	
Education × Cohort		-0.00739*		0.140***
		(0.003)		(0.017)
PGS ×Father's Education × Cohort	-0.00604*		-0.0158	
	(0.002)		(0.013)	
PGS × Education × Cohort		-0.00463		0.00522
		(0.003)		(0.016)
Birth Year	0.386***	0.375***	-0.773**	-0.688**
	(0.054)	(0.051)	(0.290)	(0.267)
Birth Year^2	-0.00264***	-0.00257***	0.00689***	0.00632***
	(0.000)	(0.000)	(0.002)	(0.002)
Birth Year × Cohort	-0.0139***	-0.0130***	0.00764	0.000786
	(0.003)	(0.003)	(0.016)	(0.014)
Female	0.0641***	0.0728***	-2.618***	-2.556***
	(0.019)	(0.018)	(0.090)	(0.084)
Constant	-11.13***	-10.64***	46.85***	42.95***
	(1.895)	(1.783)	(10.212)	(9.353)
N	8878	9929	8878	9869

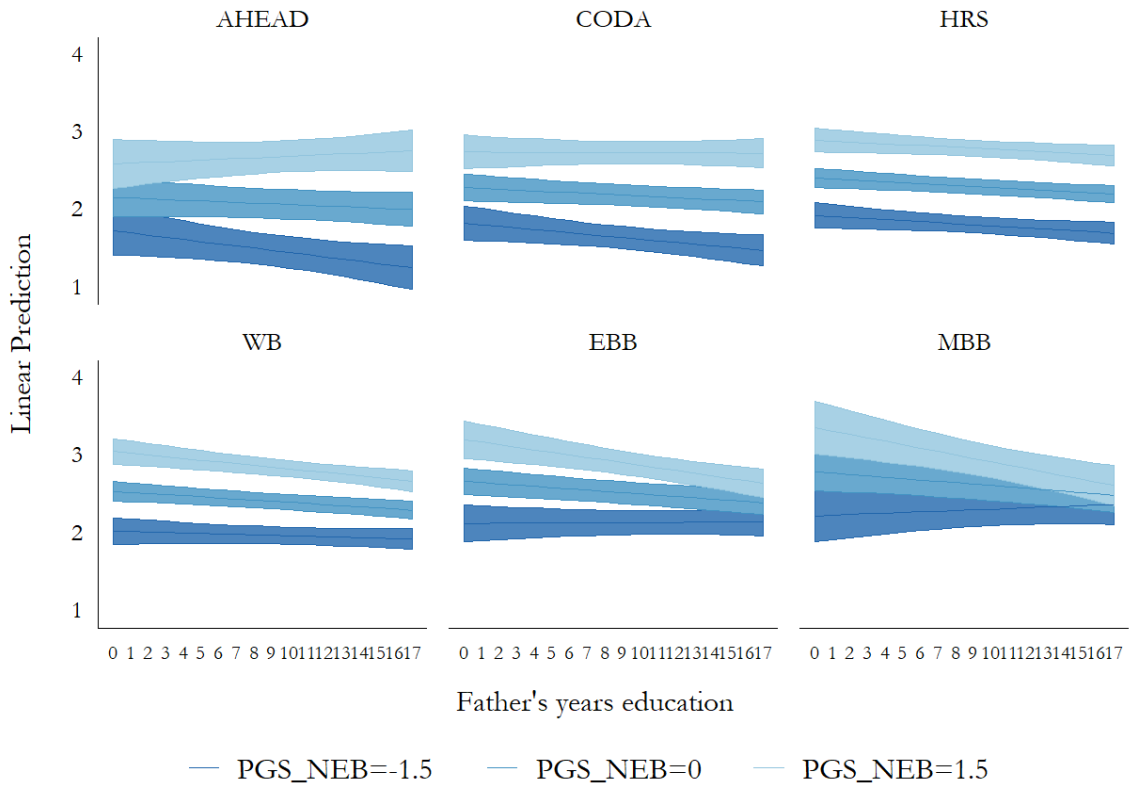
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

NEB = Number of children ever born

AFB = Age at first birth

PGS = Polygenic score

Figure 5. Association between PGS_NEB and childhood SES by cohort



Note: PGS_NEB = polygenic score for number of children ever born

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