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The social and genetic inheritance of educational attainment: Genes, parental education, and educational expansion

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ABSTRACT

Recently, several genome-wide association studies of educational attainment have found education-related genetic variants and enabled the integration of human inheritance into social research. This study incorporates the newest education polygenic score (Lee et al., 2018) into sociological research, and tests three gene-environment interaction hypotheses on status attainment. Using the Health and Retirement Study (N = 7599), I report three findings. First, a standard deviation increase in the education polygenic score is associated with a 58% increase in the likelihood of advancing to the next level of education, while a standard deviation increase in parental education results in a 53% increase. Second, supporting the Saunders hypothesis, the genetic effect becomes 11% smaller when parental education is one standard deviation higher, indicating that highly educated parents are more able to preserve their family's elite status in the next generation. Finally, the genetic effect is slightly greater for the younger cohort (1942–59) than the older cohort (1920–41). The findings strengthen the existing literature on the social influences in helping children achieve their innate talents.

1. Introduction

The idea of the American Dream has led many to believe meritocracy is the primary mechanism for success in the United States (U.S.) and that opportunities to attain higher education are equally distributed. However, as reported by the Organization for Economic Cooperation and Development (OECD, 2016: 74–89), in the U.S., children of college graduates are twice as likely to enroll in college as are those of high school graduates, and are six times more likely to go to college than are those of high school dropouts. The strong evidence of parents' education influencing children's education leads to questions about the Dream and suggests that it is necessary to understand the mechanisms underlying this intergenerational transmission to mitigate the achievement gaps.

Sociologists usually consider the intergenerational transmission of education a measure of social mobility. In the U.S., intergenerational mobility depends largely on both parent's and children's educational attainment (Blau and Duncan, 1967). However, genetic heritability also plays a role (Eckland, 1967; Duncan, 1968; Behrman and Taubman, 1989; Jencks and Tach, 2006; Nielsen, 2006; Nielsen and Roos, 2015). Failure to discern the contributions of both social and biological pathways leads to a weak conclusion that this transmission simply represents the effect of social inheritance. Past studies using twins and siblings (who frequently live in the same environment) were not able to account for specific genetic effects nor were they able to distinguish genetic effects from environmental effects. I seek to solve this by incorporating an education polygenic score, a measure of the effects of specific genetic variants on educational attainment.

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Education is regarded as an achievement in Sociology highly associated with ascribed status. The family a child is born into has a profound influence on that child's personal achievements. Wealthier families are frequently better able to provide offspring with superior resources, and these, in turn, assure their success. A recent study has shown that the U.S. recession in 2008 had a greater impact on the financial resources of poor families (Pfeffer et al., 2013). This study suggests that children from poor families are disadvantaged when competing for slots in the best colleges and universities when there are unpredictable shocks to the economy. Sociologists also have begun to consider the effect of genetic potential in several hypotheses about gene-environment interactions and their effect on status-related outcomes (Nielsen, 2016).

In the recent years, a few social scientists have incorporated a new genetic measure, the polygenic score, into their studies. A polygenic score is a measure summarizing the genetic findings from genome-wide association studies (GWASs) predicting traits of interest. It picks up the significant genes and their magnitude in predicting a phenotype, and thus is predictive of the trait. These GWAS findings are generally considered credible. For educational outcomes, the education polygenic scores of the earlier version GWASs' results were used in some studies recently. One of the earliest efforts was made by Conley et al. (2015). They used the genetic polygenic score constructed from Rietveld et al. (2014) to study the effects of genetic and social inheritance on children's education. Although the approach of the paper was a breakthrough at the time, the GWAS they used for the polygenic score only identified 3 genome-wide significant single nucleotide polymorphisms (SNPs) then. Therefore, both the main effects and the interaction results they reported were weak.

As sample sizes increased, recent GWASs identified more SNPs that are significantly related to educational attainment (Okbay et al., 2016; Lee et al., 2018). The latest study found 1271 SNPs from a sample of 1.1 million individuals (Lee et al., 2018). Few social scientists have taken an advantage of the new result to answer their questions of interest so far. One working paper written by economists (Papageorge and Thom, 2018) used the score to predict education and several labor market outcomes. They also explored the gene-environment interaction effects on the outcomes. However, while they reported many novel results, the explanations they offered were not thorough since their focus was to survey the effects of genes on a variety of status outcomes. Moreover, the paper fell short of providing sociological interpretation of the results, and thus limited its contribution to understanding society.

This study incorporates the newest genetic polygenic score constructed from Lee et al. (2018)'s GWAS results into the status attainment research. In addition, I examine the moderating effects of parental education and historical events on the relationship between genetic variants and educational attainment. My study population is over the age of 50, thus were educated during the 20th century's stable gradual expansion of higher education in the U.S. The genetic potential for education is likely realized more in the younger of them due to their greater opportunities for higher education in the period.

While preliminary efforts of adding polygenic scores to social science models have been made (Conley et al., 2015; Davies et al., 2015; Domingue et al., 2015; Krapohl and Plomin, 2015; Barth et al., 2018; Belsky et al., 2018; Liu, 2018; Papageorge and Thom, 2018; Trejo et al., 2018), this study endeavors to bring three contributions toward the field: (1) I examine the effects of social inheritance on an offspring's educational achievement while controlling genetic heritability using the most recent genome-wide association (GWAS) results (Lee et al., 2018) in the Health and Retirement Study (HRS). (2) This study tests the three gene-environment interaction hypotheses on status attainment and describes the influences of parental education on the realization of genetic potential. (3) Finally, I explore how the changes in the macro educational environment in the 20th century America influence individuals' achievement of their genetic potential.

2. Theoretical background

2.1. Traditional model of educational attainment

Individuals possess two statuses, ascribed status and achieved status. Ascribed status refers to the status individuals acquire at birth, such as gender, ethnicity, genetic predisposition, parental education, and family SES. In contrast, achieved status consists of the attributes an individual earns, such as educational and occupational success. Sociologists often consider these latter achievements as indicators of social mobility.

In their seminal status attainment model, Blau and Duncan (1967) examined the connection between ascribed and achieved status. They contended that a father's education and occupation are influential factors in a child's educational and occupational attainments, and thus the ascribed status has a significant impact on social mobility. In their analysis, the correlation between parental education and children's education is 0.310, indicating that a one-year increase in a father's educational attainment leads to a 0.310-year increase in a child's education. Their general results show that status attainment can be reproduced across generations to some extent. Although the relationship between the variables is not strong, the mechanisms underlying the intergenerational mobility have been explored since then.

Soon after Blau and Duncan's seminal status attainment model, the Wisconsin Model was developed to explain the intergenerational transmission of educational attainment by taking social psychological variables into account, such as the influence and aspirations of significant others (Sewell et al., 1969). Using a broader framework, Jonsson et al. (2011) analyzed intergenerational reproduction of occupation, which is tightly linked with education. They identified four kinds of resources: human capital, cultural capital, social network, and economic resources. Human capital includes the cognitive skills and abilities shared by parents and children. Cultural capital refers to shared aspirations, culture and personal tastes. Social network indicates a family's social ties within the community that might connect them to additional resources. Economic resources are the family's income and other financial endeavors. By transmitting all of these resources to children, parents reproduce their advantage or disadvantage in the next generation.

These studies have established the transmission of educational attainment from parent to child. Their explanations are often built

on the assumption that the effects of ascribed status are transmitted socially. However, biological heritability between parents and children also connects parental achievement with their children's. Thus, the genetic pathway may confound the social influence. Without considering an individual's inborn talents, sociologists can hardly know whether opportunities for higher education are distributed equally among all Americans at a certain point in time. Likewise, whether an individual's success is the result of personal effort rather than SES background cannot be fully understood without considering how biological and social factors influence each other.

2.2. The integration of biological accounts

2.2.1. Theoretical framework

Although environmental components are relatively obvious, genetic factors cannot be ignored when considering an individual's IQ (Eckland, 1967). The methods did not exist at the time to differentiate between hereditary and environmental components (Duncan, 1968). Scarr and Weinberg's (1978) study on the IQ of adoptees and biological children was an early attempt at disentangling the effects of biology and the environment effects on intelligence. They reported strong effects for biological parents' IQ rather than adoptive parents' IQ on children, suggesting that genes accounted for a large portion of the effects of family background.

Researchers began to collect data on twins and siblings to analyze the environmental and biological influences on social mobility. Using data for male twins born in the U.S. from 1917 through 1927, economists Behrman and Taubman (1989) found that genetics accounted for more than 80% of the observed variation in schooling. By implementing the behavioral genetic models (i.e., ACE models), which decompose the total variance in outcome variables into heritability (A), shared environment (i.e., shared by siblings; C), and non-shared environment (i.e., measurement errors and individual-specific differences; E), Nielsen (2006) showed that, for adolescents' verbal IQ, grade point average, and college plans, the genetic component explained about 50%–70% of the variance, the unshared environmental component accounted for 30%–40%, but shared environment explained only as much as 10%. To revisit this question using a new polygenic score, the first hypothesis I test is: both parental education and genetic predisposition have a positive impact on educational attainment.

The actualization of genes can be shaped by the environment, especially when barriers are small and resources are adequate (Bronfenbrenner and Ceci, 1994; Perry, 2002; Shanahan and Hofer, 2005). When opportunities for the realization of an individual's genetic potential increase, the influence of genetic components therefore also increases. Results from Behrman and Taubman (1989) and Nielsen (2006) mentioned above thus indicate that the educational opportunities for higher education in the 20th century America were relatively equal.

Specifically on status-related outcomes, there are three main gene-environment interaction hypotheses (Nielsen, 2016). First, the Scarr-Rowe Hypothesis (Tucker-Drob and Bates, 2016) contends that genetic potentials are realized more thoroughly when family SES is better, e.g., children from disadvantaged backgrounds, who are often constrained from fulfilling their potentials (Guo and Stearns, 2002). Another variant of the Scarr-Rowe Hypothesis is an initial increment of gene realization when socioeconomic status is low, which levels off after the environment reaches a threshold.

In the Pareto Hypothesis (Pareto, 1909), genetic potentials reach a peak when individuals are from middle class families, but are realized only weakly in both the poorest and the wealthiest families because the environments are too harsh and suppressed for the poorer children to move up and too protective and abundant for the rich children to move down. Hence, Pareto hypothesized a curvilinear relationship between genetic realization and SES.

The Saunders Hypothesis (Saunders, 2010) suggests the opposite of the Scarr-Rowe Hypothesis. In his analyses of the National Child Development Study in England, Saunders found that social mobility in England depended largely on meritocracy, since (assuming merit is genetic) the genetic-based predicted intergenerational social mobility pattern is almost the same as the actual pattern. However, Saunders also found that middle-class families could still prevent their offspring from falling into the working class. Therefore, according to Saunders, the realization of genetic potentials is constrained by high status families' preservation of higher education for their children irrespective of their innate abilities. However, unlike Pareto, Saunders did not hold that the low-status families restrict their children's opportunities to realize their genetic potentials.

If any of these hypotheses are supported, the paths between the polygenic score and family SES should be predictable. To support the Scarr-Rowe Hypothesis (Hypothesis 2a), the interaction term between the polygenic score and family SES should be positive, i.e., the genetic component effects are greater in higher status families. If Pareto's Hypothesis (Hypothesis 2b) is correct, the interaction term should be positive for middle class families, but negative or less positive for the lowest and the highest status families. If the Saunders Hypothesis (Hypothesis 2c) is supported, the interaction term should be negative, i.e., the genetic component effects are weaker for the those having better socioeconomic statuses.

Researchers have also tested the environmental influences on the realization of genetic potential and the findings of most of these studies support the Scarr-Rowe Hypothesis. For example, Guo and Stearns (2002) used a large sibling sample to study the heritability and social influences on intelligence. Their results indicated that, for children who live in disadvantaged environments, the realization of their genetic potential is limited. Other researchers have demonstrated that genes explain nearly zero variation in IQ for children under the age of 8 in low-SES families, while they account for more than half of the variation for children in affluent families (Turkheimer et al., 2003; Tucker-Drob et al., 2011).

2.2.2. Genetic data, GWAS, and polygenic score

Estimates of genetic effects and heritability (provided by twin studies) led to a greater understanding of the effects of genetics and the environment (Behrman and Taubman, 1989; Nielsen, 2006). However, the methodology could not then identify the specific and

overlapping genes within pairs and also could not distinguish genetic effects from environmental effects, which was problematic as the equal-environments assumption (EEA) holds that twins are equally exposed to environmental influences relevant to the trait under study (Freese, 2008). Thus, similarities among the pairs could not be readily attributed to genes.

As molecular genetic data became available, studies started to use this information to understand human behaviors. Among the 3 billion base pairs in the human genome, about 99% are the same across individuals. The base pairs that vary by individuals are called the single-nucleotide polymorphisms (SNPs). Locations of the base pairs (called *loci.*) have been mapped, so every SNP has its own location code. Researchers can use this information to locate which variants are associated with traits of interest.

The newly developed Genome-wide association study (GWAS) enables the identification of variants that are associated with the traits of interest. For humans, there are about 8–10 million common SNPs for which at least one percent of the population has the rarely observed allelic variant in the reference population. Mostly, laboratories genotype only about a million of these tagged SNPs and impute others (Belsky and Israel, 2014; Manuck and McCaffery, 2014). The ability of GWAS to include many genetic variants and its hypothesis-free nature provide researchers with an unbiased and comprehensive method. Only the common disease-common variant assumption, which holds that multiple SNPs contribute to a single disease or trait and thus each of them only has a small effect on the outcome, is required. So far, the assumption has mostly been supported (Visscher et al., 2012).

To illustrate the GWAS methodology, the form of the regressions used for outcome Y at a single SNP_j is:

$$\mathrm{Y} = X^{'}\beta + \beta_j SNP_j + \varepsilon$$

where SNP_j is the number of a reference allele an individual possesses. If the reference allele is CG at a particular SNP, an individual has CG on both chromatids will have a value of 2 at the SNP. The number of alleles therefore can be 0, 1, or 2 for each SNP. In addition to SNP_j, $X^{'}$ is a vector of other control variables including principal components for ancestral similarities. For millions of SNPs included in the analysis, the above regressions would be estimated millions of times, one for each SNP.

For social scientists, since the focus is not identifying new loci and the raw findings from GWAS are not really a concern, a summary score of individual genetic predisposition for traits can be constructed and incorporated into studies. By multiplying the whole-genome risk alleles of individuals by the resulting GWAS betas, Purcell et al. (2009) constructed a polygenic score (PGS) and predicted the incidence of schizophrenia successfully. Usually, the whole-genome polygenic score, which assumes that a large number of SNPs each contribute infinitesimal effects to the phenotype, is used in the studies. The score takes the form below:

$$\mathrm{PGS}_i = \sum_j \widehat{\beta}_j SNP_{ij}$$

where $\widehat{\beta}_j$ is the estimated beta for SNP_{ij} in GWAS, and SNP_{ij} is the number of risk alleles an individual i has for the particular SNP. The PGS was then standardized to enable the interpretation of the results.

2.2.3. GWAS and educational attainment

Recent efforts in exploring genetic variants related to education were fruitful. Using data from 126,559 individuals, Rietveld et al. (2013) identified three independent SNPs (rs9320913, rs11584700, and rs4851266) associated with years of education and college completion; although the linear PGS of these SNPs can only account for 2 percent of the variation in educational attainment, the results have been replicated (Rietveld et al., 2014). More recent GWAS reported 74 SNPs using 293,723 individuals (Okbay et al., 2016) and 1271 genome-wide significant SNPs from 1.1 million individuals (Lee et al., 2018). The newest PGS constructed by the whole-genome SNPs explained 10.6% of the variance in educational attainment in the Health and Retirement Study (HRS) incrementally. The highest PGS quintile is also associated with a 40% higher college completion rate in HRS. In this study, this PGS is used as the genetic source of educational attainment.

Studies have used the whole-genome PGS derived from Rietveld and colleagues' study (2013) to predict education in the Framingham Heart Study (FHS) and the Health and Retirement Study (HRS). For example, Conley et al. (2015) found that one-sixth of the correlation between parental and children's education can be explained by genetic inheritance, and the genetic effect does not vary by maternal education after controlling for children's genetic score. They concluded that the policies fostering equal educational opportunities might have a trivial impact on intergenerational mobility, since parental education did not moderate the genetic effects. Other studies have shown that the PGS of these three SNPs: 1) is positively associated with adolescents' educational achievement and has a negative interaction effect with parental education on years of education when using the sibling fixed effects model (Domingue et al., 2015); 2) accounts for at least 3 percent of the variance in children's educational achievement (Krapohl and Plomin, 2015); 3) has an interaction effect with fathers' social class when predicting education; and, 4) are strongly associated with income at age 46 (Davies et al., 2015).

The PGS generated using Okbay et al. (2016) has also been used in several studies. Liu (2018) used the three-generation PGS from the Framingham Heart Study (FHS) to address the social and genetic pathways. Liu showed that only one-fifth of the transmission of educational attainment can be explained by genetic inheritance and a half of this genetic transmission is mediated by parental education.

More recently, the PGS constructed from Lee et al. (2018) was also incorporated into several studies. The new PGS was again found to be related to years of schooling, and the effect was moderated by social environment. For example, school context was found to be interacting with the PGS (Trejo et al., 2018). Although the interaction between PGS and school-level socioeconomic status varies across dataset, the interaction effect was significant. Additionally, studies also explored the effect of the new PGS on status attainment and

other labor market outcomes. Results showed that higher polygenic scores are associated with higher education, better labor market outcomes, and upward social mobility (Belsky et al., 2018; Papageorge and Thom, 2018). A one standard deviation increase in the PGS results in an increase of 0.58–0.83 years in schooling in the analysis of the HRS data (Papageorge and Thom, 2018).

Besides, the gene-environment analysis also showed negative interactions between childhood SES and PGS when predicting earning at least a high school diploma, but positive interactions when predicting having at least a college degree (Papageorge and Thom, 2018). These results suggest that the Saunders hypothesis is supported for having a degree less than college and the Scarr-Rowe hypothesis is supported for earning a college degree and above. However, these papers might have ignored that college matriculation is often dependent on a high school diploma by treating educational attainment as either a continuous process or without conditions; by using the continuation ratio logistic model to test the gene-environment interaction hypotheses, I attempt to address this issue.

2.3. Historical changes and genetic effects on educational attainment

The actualization of genetic potentials can be influenced by either macro-historical changes or the proximal environmental influences on individuals via a gene-by-environment interaction (G × E). For example, Branigan et al. (2013), in their meta-analysis of educational attainment across countries, found that the genetic component explains more variance in education for men than for women and is also stronger for those born after 1950 than for those born before 1950. In the U.S., Nielsen and Roos (2015) used sibling data to estimate the variance in educational achievement explained by heritability, shared environment, and non-shared environmental, and found that the variance in education explained by the genetic component decreased and that explained by shared environment increased as of 2000. The declining effect of the genetic component indicates the opportunity to attain higher education in the U.S. has become less equal over the past six decades.

Heath et al. (1985) studied Norwegian twins and found that family background had a larger impact on the educational attainment of Norwegians born before 1940, although the patterns varied between genders across time periods (variance due to genetic predisposition increased for males after World War II, it remained relatively stable for females). The authors maintained that the increase in the fraction of heritability was mainly due to the Norwegian government's adoption of liberal social and educational policies following the war, and that more opportunities for education were available for males than females at that time.

According to the above studies, I therefore test two hypotheses. If opportunity became more unequal, genetic effects would decline over time (Hypothesis 3a: became unequal), while if opportunity became more equal, the effects of genes would increase (Hypothesis 3b: equalization).

I use cohorts consisting of middle-aged to older individuals (born between 1924 and 1959) who were exposed to federal policies implemented after WWII that expanded higher education and encouraged youths to pursue secondary educations (Trow, 1972, 2007; Mumper et al., 2011). The nationwide expansion in higher education also implies that the effects of parental education would probably be attenuated in the later cohorts. Thus, the final hypothesis to test is: if educational opportunities become equal, the moderating effects of parental education would decline in the latter cohorts (Hypothesis 4).

3. Data and methods

3.1. Data

I used data from the Health and Retirement Study (HRS) (http://hrsonline.isr.umich.edu/), a nationally representative longitudinal survey of U.S. adults over the age of 50 sponsored by the National Institute on Aging (NIA) which has been continuously administered since 1992 with data collected every two years; the Institute for Social Research (ISR) at the University of Michigan collects the data. Information on social, economic, and other factors related to the antecedents and consequences of retirement are included; HRS also obtained saliva specimens from respondents in 2006 and 2008. Of specimens provided, 13,129 samples were genotyped using the Illumina HumanOmni2.5-4v1 array at the Center for Inherited Disease Research and 12,507 passed the quality control process at the Genetics Coordinating Center of the University of Washington. However, because the GWAS results for educational attainment were based on Caucasians (Rietveld et al., 2013; Okbay et al., 2016; Lee et al., 2018), only 8353 individuals have the calculated polygenic score provided by HRS. To reduce the potential mortality selection bias among the older respondents, I then limited the samples to 7986 cases who were born between 1920 and 1959; of these, 35 individuals with unknown educational degree were deleted; 351 individuals who lacked data on parental education were dropped; 1 individual without the sampling weight was excluded, resulting in a final sample size of 7599 (see Supplementary Table 4–6 for the multiple imputation results).

3.2. Variable measurement

3.2.1. Educational attainment

My outcome variable is educational attainment. HRS's range of values for this variable was 0–17 years and above. I used the categories reconstructed from the HRS 2016 Tracker file to measure educational attainment; I recombined these categories into: No degree, GED/High school diploma, Two-year college degree/Some college, Four-year college degree, Master, and Professional degree (Ph.D., M.D., J.D.).

3.2.2. Parental education

I used the highest years of education attained by father or mother instead of including both as the parental educational attainment

measure for fewer missing values. HRS's values for these variables ranged from 0 to 17 years and above. I standardized parental education according to the respondent's cohort to adjust for the differential distribution of parental educational attainment across cohorts. To account for the possible estimation bias due to using the highest education of father or mother, a dummy variable was created to indicate which parent's education was included in the models. The dummy variable was coded as 0 for using father's education and 1 for using mother's education.

3.2.3. Polygenic score

The education polygenic score (PGS) for European ancestry was used in this study. Lee et al. (2018) constructed the score for the Health and Retirement Study in Plink using the LD-adjusted weights from their GWAS results. The final GWAS-based polygenic score contains 1,144,251 SNPs for European-ancestry individuals. It was standardized in this study for interpretation.

3.2.4. Cohorts

I used categorical cohort to examine the historical changes hypotheses. I defined categorical cohort using the six HRS cohorts, which are as follows: Aging & Health Dynamics cohort (AHEAD), born 1905–1924, which entered in 1993 and was surveyed in 1995, and 1998 through 2012; Children of the Depression cohort (CODA), born between 1924 and 1930, surveyed from 1998 through 2010; Health and Retirement cohort (HRS), born between 1931 and 1941, surveyed from 1992 through 2012; War Babies cohort (WB), born between 1942 and 1947, surveyed from 1998 through 2012; Early Boomers cohort (EBB), born between 1948 and 1953, surveyed from 2004 through 2012; and Mid Boomers cohort (MBB), born between 1954 and 1959, entered in 2010 and was also surveyed in 2012. I combined the War Babies cohort, Early Boomers cohort and the Mid Boomers cohort to increase the sample size of the youngest cohort in the cohort-separated analysis. Respondents were restricted to those who were born between 1920 and 1959 to reduce the influence of the morbidity or mortality selection issue.

3.2.5. Control variables

Control variables included gender, respondent's birth region, degree of urbanization of respondent's residential location at age 10, father's occupation at age 16, self-rated childhood SES, and the survey wave. The father's occupation and the childhood SES variables are loaded with missing values, especially in the younger cohort where the question was not asked in the same way. A missing category was generated for the missing values in each of the control variables.

Population stratification was also considered since the allele frequency differences due to systematic ancestry differences can result in spurious associations between SNPs and traits. For example, one of the famous examples is the chopsticks gene (Hamer and Sirota, 2000). The chopsticks gene is just an artifact resulting from the different frequency of genetic variants between Asian and Caucasians. To solve the issue, previous researchers have conducted principal components analyses to identify the potential ancestral differences in SNPs in genotype data (Price et al., 2006). Following the most-frequently used approach, I report the findings with, and without, controlling the largest 10 principal components.

3.3. Analytic strategy

I used the continuation ratio logistic model to better study the determinants of individuals' transition between stages when they fulfill the requirement or complete the previous stage. The general continuation ratio logit model is

$$\ln \frac{\Pr(y > j | y \geq j)}{1 - \Pr(y > j | y \geq j)} = \theta_j + x^{'}\beta$$

where $x^{'}$ is the transpose of the vector of covariates, θ_j is the cut point for the j th category compares to the lowest category, and β is the coefficients of the covariates which are assumed to be the same across contrasts.

The continuation ratio model is similar to the ordered logit model in the sense that they each have only one set of coefficients, i.e., both of them require the proportional odds assumption. However, the continuation ratio model allows interactions between the dummy stage variables and the other independent variables of interest, and therefore relaxes the assumption (Allison, 2012: 186). If the totally unconstrained model is requested, ordinary binary logistic regression models, which estimate several models by using the conditional samples can provide the different parameters (Agresti, 2006: 192).

To address my hypotheses, I first included the parental education in the model and added the PGS to determine the relative effects of social and genetic inheritance on educational attainment. The model (x_s indicates the main effects and control variables) is

$$\ln \frac{\Pr(y > j | y \geq j)}{1 - \Pr(y > j | y \geq j)} = \theta_j + \beta_1(\textit{Parental Education}) + \beta_2(\textit{Polygenic Score}) + \beta_s x_s$$

Next, to test the moderating hypothesis and determine whether the data supports the Scarr-Rowe Hypothesis or the Saunders Hypothesis, I added a single interaction term between parental education and PGS: $\beta_3(\textit{Parental Education} \times \textit{Polygenic Score})$. If β_3 is positive, the data supports the Scarr-Rowe Hypothesis; if β_3 is negative, the Saunders Hypothesis is supported. I included two more interaction terms in the model to assess whether the data supports the Pareto Hypothesis, as indicated by a U-shaped relationship between parental education and the realization of genetic potentials: $\beta_4(\textit{Parental Education}^2) + \beta_5(\textit{Parental Education}^2 \times \textit{Polygenic Score})$. Here, β_4 is the coefficient for parental education squared and β_5 indicates the moderating effect of parental education on genes when parental education is extremely high or low. According to the Pareto Hypothesis, β_3 should be positive since children

Table 1
Descriptive statistics for full sample and cohorts within the sample.

	Total		AHEAD, CODA, & HRS 1920–41		WB, EBB, & MBB 1942–59	
Variables	Mean	S.E.	Mean	S.E.	Mean	S.E.
Degree						
No Degree	.112	.004	.145	.005	.058	.004
GED/High School	.575	.006	.585	.007	.558	.009
2-yr/some college	.054	.003	.041	.003	.075	.005
4-yr college	.153	.004	.138	.005	.178	.007
MA	.083	.003	.069	.004	.106	.006
PhD	.024	.002	.023	.002	.025	.003
Year of Education	13.428	.050	13.003	.037	14.135	.116
Standardized Polygenic Score	-.001	.011	.019	.015	-.034	.019
Parental Education in Years	11.090	.036	10.531	.046	12.020	.053
Standardized Parental Education	.001	.012	.002	.015	-.001	.019
Parental Education Indicator (Father = 0)	.364	.006	.352	.007	.385	.009
Female	.579	.006	.557	.007	.616	.009
Birth Year	1938.697	.104	1932.893	.082	1948.371	.078
Cohort						
AHEAD (1920–1924)	.046	.002	.074	.004	–	–
CODA (1924–1930)	.163	.004	.261	.006	–	–
HRS (1931–1941)	.416	.006	.665	.007	–	–
WB (1942–1947)	.164	.004	–	–	.438	.009
EBB & MBB (1948–1959)	.211	.005	–	–	.562	.009
Region						
Northeast	.189	.004	.184	.006	.197	.007
Midwest	.364	.006	.365	.007	.363	.009
South	.275	.005	.294	.007	.244	.008
West	.108	.004	.107	.004	.111	.006
Other	.020	.002	.028	.002	.007	.002
Missing	.043	.002	.022	.002	.078	.005
Urbanicity						
Urban	.511	.006	.493	.007	.540	.009
Rural	.466	.006	.475	.007	.450	.009
Missing	.024	.002	.032	.003	.011	.002
Foreign Born or Missing	.031	.002	.036	.003	.023	.003
Father's Occupation at 16						
Labor	.346	.005	.402	.007	.253	.008
Farmer	.152	.004	.205	.006	.064	.005
Blue Collar	.124	.004	.142	.005	.095	.005
White Collar	.127	.004	.141	.005	.104	.006
Missing or Others	.250	.005	.109	.005	.485	.009
Self-Rated Childhood SES						
Poor	.222	.005	.279	.007	.127	.006
Average	.510	.006	.603	.007	.355	.009
Well off	.052	.003	.055	.003	.046	.004
Missing	.217	.005	.063	.004	.472	.009
2008 Survey	.468	.006	.472	.007	.462	.009
N	7599		4749		2850	

from middle class families are more likely to realize their genetic potential and β_5 should be negative due to the crystallizing of the class structure for the highest and lowest social classes.

I then tested the interaction terms between the PGS, and cohort (β_7) to assess changes in genetic effects over time to understand whether educational opportunity became equal or unequal in the mid-20th century. The model now is:

$$\ln\frac{\Pr(y>j|y\geq j)}{1-\Pr(y>j|y\geq j)}=\theta_j+\beta_1(Parental\ Education)+\beta_2(Polygenic\ Score)+\beta_3(Parental\ Education\times Polygenic\ Score)+\beta_6(Cohort)$$
$$+\beta_7(Polygenic\ Score\times Cohort)+\beta_s x_s$$

If the hypothesis that educational opportunity became less equal is supported, β_7 should be negative, since in this scenario the younger cohorts are less likely to actualize their genetic potential; in contrast, if the equalization hypothesis is supported, β_7 should have a positive trend (smaller genetic effects for older cohorts but greater for younger cohorts).

Finally, to test Hypothesis 4, I separated my study population into two aggregated cohorts (1920–41, and 1942–59) to illustrate the decline of the moderating effects of parental education.

The Health and Retirement Study sample is a stratified multi-stage sample of United States households. The genetic data were collected in the 2006 and 2008 biomarker sub-sample survey, and the sample weights for the sub-sample were provided by the HRS in the two waves. The genetic sample collected in 2006 was a randomly selected half of the 2006 sample, and the other half was selected for the collection of the biomarkers in 2008. To accommodate the survey design, I applied the sample weights and controlled for the

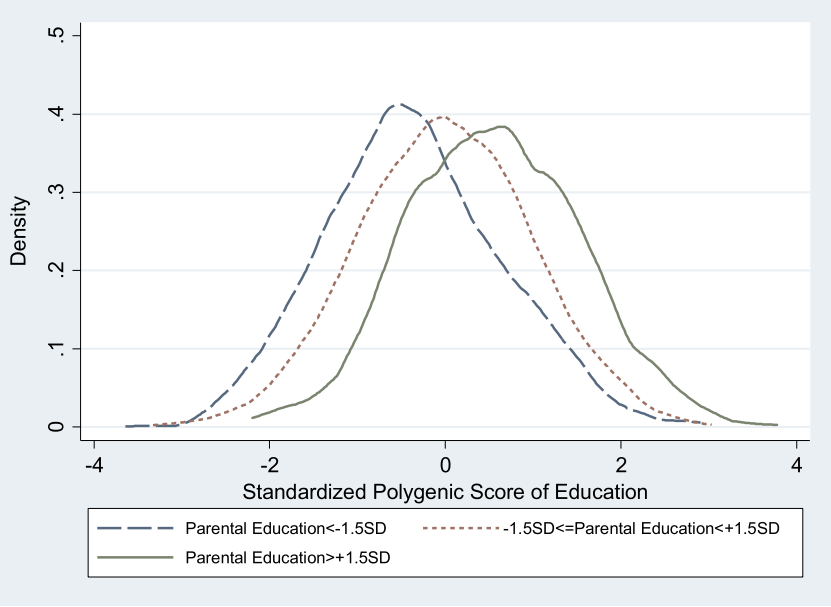

Fig. 1. Correlation between the standardized parental education and Offspring's standardized polygenic score of education.

survey year in all of the models described above.

4. Results

4.1. Descriptive statistics

Table 1 shows the descriptive statistics of the characteristics of the HRS dataset. First, 58% of subjects reported their highest degree as a GED or high school graduation. In the older cohorts, more individuals have no degree, compared with the younger cohorts; a greater proportion of the younger cohort earns secondary and postsecondary degrees. Second, the PGS of education is around 0 after standardization within the analytic sample. The score is slightly lower in the younger cohorts than in the older cohorts. Third, on average, parents had approximately 11 years of education, with the parents of younger cohort members achieving about 2 more years than the parents of members of older cohorts. After standardizing parental education by cohorts, the means are around 0 for each aggregated cohort and overall. Finally, there are more females (58%) than males, the respondents were about 70 years old (mean birth year of 1938) when the genetic data were collected, more individuals were born in the Midwest, and about half lived in urban areas when they were young.

Fig. 1 illustrates the correlation between parental education and the individual's PGS. The Pearson correlation coefficient is 0.23 and is significant at the $p < .000$ level, thus the higher the parental education, the greater the PGS for education. The figure suggests that the effects of parental education can be genetically transmitted, which is often ignored in sociological studies.

4.2. Continuation ratio models predicting educational attainment

Table 2 shows the results from the continuation ratio models. Models 1 and 2 are the traditional educational attainment models, which only include the demographic and SES variables. The PGS was added to models 3 through 8. In models 9 through 14, I controlled for population stratification by entering 10 principal components to the models.

The results from models 1 and 2 support the traditional status attainment model. Model 1 considers the effects of parental education on offspring's educational attainment. Individuals whose parents have a standard deviation more education are 75% [$e^{0.562} = 1.754$] more likely to advance to the next stage and, after other control variables are accounted for in Model 2, the positive effect of parental education still holds.

In Model 3, the PGS effect size of 0.520 indicates that a standard deviation increase in the PGS is associated with a 68% increase in the likelihood of advancing to the next educational level. The inclusion of parental education in Model 4 reduces the genetic effect to 0.439, and the effect of parental education also decreases from 0.562 in Model 1 to 0.492 in Model 4. After controlling for other variables except for population stratification, the effects of both the PGS and parental education only fluctuate slightly.

In Model 7, I tested the Scarr-Rowe Hypothesis against the Saunders Hypothesis and in Model 8 I tested the Pareto Hypothesis. The significant negative interaction effect in Model 7 indicates that parental education negatively moderates the influence of genes. In Model 8, the two interaction terms show no signs of supporting the hypothesized inverted U-shaped effect of parental education on the realization of genes. The Saunders Hypothesis appears to be supported by the evidence.

Table 2
Continuation ratio model predicting educational attainment (Weighted).

Variables	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12	Model 13	Model 14
Standardized Whole-genome Polygenic Score (PGS)			.520*** (.026)	.439*** (.026)	.501*** (.027)	.452*** (.026)	.459*** (.026)	.454*** (.028)	.524*** (.026)	.444*** (.026)	.506*** (.027)	.458*** (.027)	.465*** (.026)	.460*** (.029)
Standardized Parental Education	.562*** (.024)	.475*** (.025)		.492*** (.025)		.413*** (.025)	.421*** (.025)	.418*** (.027)		.504*** (.025)		.422*** (.025)	.429*** (.026)	.427*** (.027)
Parental Education*PGS							-.044** (.014)	-.041** (.015)					-.041** (.014)	-.038* (.015)
Parental Education^2								-.007 (.014)						-.007 (.014)
Parental Education^2*PGS								.005 (.013)						.004 (.013)
Parental Education Indicator (Father = 0)	-.174*** (.046)	-.107* (.050)		-.126* (.047)		-.069 (.050)	-.068 (.050)	-.067 (.050)		-.119* (.049)		-.064 (.051)	-.062 (.051)	-.062 (.051)
Father's Occupation at 16 (Labor = 0)														
Farmer		.123 (.086)			-.020 (.084)	-.007 (.086)	-.008 (.087)	-.007 (.087)			-.020 (.079)	-.000 (.081)	-.001 (.082)	.000 (.082)
Blue Collar		.426*** (.070)			.475*** (.070)	.358*** (.075)	.356*** (.075)	.357*** (.076)			.486*** (.071)	.377*** (.076)	.375*** (.076)	.376*** (.076)
White Collar		.614*** (.090)			.858*** (.088)	.549*** (.093)	.555*** (.093)	.560*** (.097)			.877*** (.089)	.571*** (.093)	.576*** (.093)	.582*** (.097)
Missing or Others		.137* (.068)			.219** (.071)	.123 (.073)	.128 (.073)	.130 (.074)			.232** (.069)	.142* (.071)	.146* (.071)	.149* (.071)
Self-Rated Childhood SES (Poor = 0)														
Average		.106 (.071)			.252*** (.072)	.131 (.074)	.130 (.073)	.129 (.073)			.267*** (.072)	.143 (.074)	.142 (.073)	.141 (.073)
Well off		.185 (.110)			.420*** (.116)	.151 (.116)	.155 (.117)	.157 (.116)			.434*** (.117)	.157 (.118)	.161 (.118)	.163 (.117)
Missing		.381*** (.099)			.535*** (.098)	.391*** (.097)	.384*** (.096)	.384*** (.095)			.539*** (.101)	.395*** (.100)	.388*** (.099)	.388*** (.098)
Foreign Born or Missing		.708*** (.184)			.533** (.170)	.567** (.172)	.559** (.173)	.561** (.173)			.526** (.169)	.545** (.173)	.538** (.174)	.540** (.175)
Female		-.407*** (.048)			-.442*** (.048)	-.423*** (.051)	-.421*** (.051)	-.421*** (.051)			-.451*** (.050)	-.434*** (.053)	-.432*** (.053)	-.432*** (.053)
Cohort (AHEAD 1920–1924 = 0)														
CODA (1924–1930)		.079 (.141)			.105 (.133)	.144 (.144)	.153 (.143)	.152 (.141)			.106 (.134)	.149 (.145)	.157 (.145)	.156 (.143)
HRS (1931–1941)		.214 (.112)			.291* (.110)	.309* (.117)	.318** (.117)	.317** (.114)			.294** (.109)	.314** (.116)	.323** (.115)	.322** (.113)
WB (1942–1947)		.586*** (.129)			.623*** (.130)	.682*** (.131)	.692*** (.131)	.690*** (.129)			.627*** (.129)	.686*** (.129)	.695*** (.129)	.693*** (.128)
EBB & MBB (1948–1959)		.550*** (.130)			.548*** (.121)	.659*** (.133)	.675*** (.133)	.673*** (.131)			.549*** (.123)	.654*** (.134)	.669*** (.134)	.667*** (.133)
Region (Northeast = 0)														
Midwest		-.099 (.068)			-.101 (.067)	-.103 (.070)	-.105 (.070)	-.106 (.070)			-.129 (.065)	-.130 (.068)	-.131 (.068)	-.131 (.068)
South		-.122 (.066)			-.147 (.074)	-.068 (.071)	-.066 (.070)	-.064 (.070)			-.132 (.081)	-.031 (.079)	-.029 (.078)	-.027 (.078)
West		-.000 (.078)			.046 (.083)	-.025 (.083)	-.024 (.083)	-.023 (.083)			.047 (.085)	-.010 (.084)	-.008 (.084)	-.007 (.084)
Other														

(*continued on next page*)

Table 2 (*continued*)

Variables	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8	Model 9	Model 10	Model 11	Model 12	Model 13	Model 14
		-.615*			-.574*	-.581*	-.566*	-.568*			-.583*	-.582*	-.568*	-.569*
		(.240)			(.225)	(.223)	(.224)	(.226)			(.224)	(.227)	(.227)	(.229)
Missing		-.338**			-.325*	-.271*	-.276*	-.275*			-.342*	-.280*	-.283*	-.282*
		(.115)			(.132)	(.121)	(.121)	(.120)			(.131)	(.121)	(.121)	(.121)
Urbanicity (Urban = 0)														
Rural		-.307***			-.331***	-.275***	-.273***	-.273***			-.338***	-.277***	-.275***	-.275***
		(.049)			(.046)	(.046)	(.046)	(.046)			(.047)	(.048)	(.048)	(.048)
Missing		−1.453***			−1.429***	−1.395***	−1.391***	−1.392***			−1.421***	−1.382***	−1.378***	−1.380***
		(.291)			(.275)	(.285)	(.287)	(.287)			(.276)	(.286)	(.288)	(.288)
Stage (GED/HS vs. No Degree = 0)														
2-yr/Some College	−2.929***	−3.098***	−2.889***	−3.047***	−3.129***	−3.226***	−3.243***	−3.245***	−2.897***	−3.064***	−3.141***	−3.243***	−3.258***	−3.260***
	(.061)	(.064)	(.063)	(.066)	(.066)	(.069)	(.068)	(.068)	(.063)	(.065)	(.067)	(.069)	(.068)	(.068)
4-yr College	−1.016***	−1.243***	-.972***	−1.185***	−1.294***	−1.426***	−1.443***	−1.445***	-.980***	−1.204***	−1.309***	−1.445***	−1.461***	−1.463***
	(.097)	(.098)	(.101)	(.100)	(.101)	(.102)	(.102)	(.101)	(.101)	(.101)	(.102)	(.103)	(.103)	(.102)
MA	−3.001***	−3.317***	−2.945***	−3.271***	−3.402***	−3.604***	−3.615***	−3.615***	−2.957***	−3.296***	−3.423***	−3.632***	−3.642***	−3.643***
	(.080)	(.080)	(.081)	(.087)	(.080)	(.086)	(.087)	(.087)	(.081)	(.086)	(.082)	(.088)	(.088)	(.088)
PhD	−3.903***	−4.288***	−3.839***	−4.246***	−4.395***	−4.647***	−4.646***	−4.646***	−3.859***	−4.281***	−4.425***	−4.685***	−4.684***	−4.683***
	(.145)	(.144)	(.161)	(.163)	(.156)	(.159)	(.158)	(.158)	(.160)	(.162)	(.158)	(.160)	(.159)	(.159)
2008 Survey	-.010	-.018	-.039	-.021	-.045	-.028	-.028	-.029	-.040	-.023	-.045	-.029	-.029	-.030
	(.047)	(.047)	(.043)	(.049)	(.046)	(.050)	(.050)	(.050)	(.043)	(.048)	(.046)	(.049)	(.049)	(.049)
Population Stratification														
PC1									5.514*	9.636***	4.816*	8.344***	8.294***	8.293***
									(2.090)	(2.055)	(2.318)	(2.289)	(2.298)	(2.309)
PC2									−2.042	−2.018	−5.113*	−4.346*	−4.292*	−4.307*
									(2.240)	(2.365)	(2.095)	(2.142)	(2.131)	(2.150)
PC3									1.535	1.050	−2.020	−2.136	−2.045	−2.014
									(2.308)	(2.516)	(1.998)	(2.170)	(2.155)	(2.138)
PC4									−1.089	−2.104	−1.592	−2.047	−2.109	−2.119
									(2.445)	(2.762)	(2.393)	(2.629)	(2.601)	(2.599)
PC5									2.032	2.337	2.954	2.973	3.003	3.003
									(1.871)	(2.162)	(1.876)	(2.014)	(2.006)	(2.002)
PC6									.520	.898	1.599	1.722	1.660	1.666
									(1.777)	(1.956)	(1.805)	(1.901)	(1.888)	(1.894)
PC7									-.477	−1.247	−1.925	−2.382	−2.343	−2.354
									(2.144)	(2.224)	(2.253)	(2.319)	(2.316)	(2.315)
PC8									3.685	3.518	4.117	3.878	3.777	3.749
									(2.161)	(2.199)	(2.222)	(2.283)	(2.264)	(2.258)
PC9									−1.998	−1.601	−2.372	−2.124	−2.175	−2.159
									(2.109)	(2.048)	(2.059)	(2.049)	(2.044)	(2.050)
PC10									−4.181*	−4.303*	−5.833**	−5.532**	−5.525**	−5.540**
									(1.830)	(1.794)	(2.064)	(2.025)	(2.021)	(2.018)
Constant	2.464***	2.275***	2.396***	2.529***	2.109***	2.267***	2.274***	2.281***	2.399***	2.535***	2.113***	2.260***	2.266***	2.274***
	(.051)	(.147)	(.054)	(.053)	(.136)	(.140)	(.138)	(.136)	(.054)	(.054)	(.136)	(.140)	(.139)	(.136)
Observations	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603	17,603
N	7600	7600	7600	7600	7600	7600	7600	7600	7600	7600	7600	7600	7600	7600

Standard errors in parentheses.
***p < 0.001, **p < 0.01, *p < 0.05.

Table 3
Cohort differences in continuation ratio model predicting educational attainment (Weighted).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Variables				1920–41 AHEAD, CODA, & HRS		1942–59 WB, EBB, & MBB	
Standardized Whole-genome Polygenic Score (PGS)	.465***	.310*	.318*	.442***	.449***	.476***	.484***
	(.026)	(.143)	(.144)	(.035)	(.035)	(.037)	(.038)
Standardized Parental Education	.429***	.433***	.329**	.431***	.442***	.430***	.436***
	(.026)	(.025)	(.106)	(.034)	(.036)	(.041)	(.042)
Parental Education*PGS	-.041**	-.042**	-.040**		-.060*		-.041
	(.014)	(.014)	(.014)		(.028)		(.024)
Female	-.432***	-.432***	-.433***	-.483***	-.485***	-.389***	-.384***
	(.053)	(.053)	(.052)	(.057)	(.057)	(.076)	(.075)
Cohort (AHEAD 1920–1924 = 0)							
CODA (1924–1930)	.157	.102	.093	.120	.131	–	–
	(.145)	(.144)	(.142)	(.136)	(.135)		
HRS (1931–1941)	.323**	.265*	.241*	.252*	.263*	–	–
	(.115)	(.119)	(.116)	(.108)	(.107)		
WB (1942–1947)	.695***	.640***	.649***	–	–	Ref.	Ref.
	(.129)	(.129)	(.126)				
EBB & MBB (1948–1959)	.669***	.628***	.623***	–	–	-.034	-.029
	(.134)	(.133)	(.128)			(.100)	(.099)
PGS*Cohort (AHEAD 1920–1924 = 0)							
PGS*CODA (1924–1930)		.192	.176				
		(.163)	(.163)				
PGS*HRS (1931–1941)		.224	.192				
		(.149)	(.153)				
PGS*WB (1942–1947)		.213	.222				
		(.149)	(.149)				
PGS*EBB & MBB (1948–1959)		.069	.073				
		(.141)	(.143)				
Parental Education*Cohort (AHEAD 1920–1924 = 0)							
Parental Education*CODA (1924–1930)			.152				
			(.125)				
Parental Education*HRS (1931–1941)			.230*				
			(.110)				
Parental Education*WB (1942–1947)			.034				
			(.111)				
Parental Education*EBB & MBB (1948–1959)			.060				
			(.117)				
Parental Education Indicator (Father = 0)	-.062	-.072	-.078	-.052	-.049	-.117	-.116
	(.051)	(.051)	(.051)	(.056)	(.055)	(.074)	(.073)
Father's Occupation at 16 (Labor = 0)							
Farmer	-.001	-.009	.005	-.098	-.096	.349*	.347*
	(.082)	(.081)	(.084)	(.084)	(.085)	(.171)	(.173)
Blue Collar	.375***	.369***	.359***	.344***	.344***	.352**	.348**
	(.076)	(.077)	(.077)	(.097)	(.097)	(.125)	(.126)
White Collar	.576***	.565***	.547***	.560***	.568***	.520**	.525**
	(.093)	(.094)	(.092)	(.085)	(.086)	(.169)	(.169)
Missing or Others	.146*	.144*	.143*	.040	.048	.300**	.304**
	(.071)	(.071)	(.071)	(.087)	(.088)	(.112)	(.112)
Self-Rated Childhood SES (Poor = 0)							
Average	.142	.141	.136	.178**	.177**	.071	.072
	(.073)	(.073)	(.072)	(.061)	(.061)	(.157)	(.156)
Well off	.161	.148	.151	.329**	.341**	-.064	-.063
	(.118)	(.116)	(.115)	(.121)	(.120)	(.211)	(.212)
Missing	.388***	.388***	.395***	.567**	.562**	.223	.216
	(.099)	(.100)	(.100)	(.174)	(.173)	(.203)	(.201)
Foreign Born or Missing	.538**	.527**	.528**	-.298	-.300	.997***	.988***
	(.174)	(.174)	(.174)	(.196)	(.194)	(.232)	(.233)
Region (Northeast = 0)							
Midwest	-.131	-.132	-.133	-.095	-.100	-.168	-.168
	(.068)	(.068)	(.068)	(.078)	(.077)	(.113)	(.113)
South	-.029	-.021	-.017	.052	.054	-.105	-.103
	(.078)	(.078)	(.079)	(.087)	(.087)	(.139)	(.138)
West	-.008	-.001	-.002	.118	.111	-.146	-.139
	(.084)	(.083)	(.084)	(.121)	(.120)	(.172)	(.172)
Other	-.568*	-.577*	-.580*	.113	.125	-.418	-.413
	(.227)	(.226)	(.228)	(.196)	(.196)	(.443)	(.441)
Missing	-.283*	-.287*	-.297*	-.285	-.292	-.460*	-.463*
	(.121)	(.123)	(.121)	(.235)	(.234)	(.178)	(.178)

(*continued on next page*)

Table 3 (*continued*)

Variables	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
				1920–41 AHEAD, CODA, & HRS		1942–59 WB, EBB, & MBB	
Urbanicity (Urban = 0)							
Rural	-.275***	-.273***	-.280***	-.317***	-.314***	-.274***	-.272***
	(.048)	(.048)	(.048)	(.045)	(.045)	(.075)	(.076)
Missing	−1.378***	−1.395***	−1.414***	−1.488***	−1.486***	-.747	-.739
	(.288)	(.291)	(.289)	(.272)	(.274)	(.538)	(.537)
Stage (GED/HS vs. No Degree = 0)							
2-yr/Some College	−3.258***	−3.272***	−3.286***	−3.055***	−3.072***	−3.621***	−3.643***
	(.068)	(.069)	(.070)	(.080)	(.081)	(.121)	(.122)
4-yr College	−1.461***	−1.474***	−1.491***	-.767***	-.786***	−2.098***	−2.120***
	(.103)	(.102)	(.105)	(.123)	(.124)	(.153)	(.153)
MA	−3.642***	−3.652***	−3.667***	−3.198***	−3.205***	−4.159***	−4.177***
	(.088)	(.087)	(.087)	(.111)	(.111)	(.151)	(.155)
PhD	−4.684***	−4.695***	−4.706***	−4.187***	−4.181***	−5.238***	−5.243***
	(.159)	(.158)	(.159)	(.182)	(.180)	(.242)	(.242)
2008 Survey	-.029	-.033	-.036	.016	.017	-.051	-.053
	(.049)	(.049)	(.049)	(.062)	(.062)	(.076)	(.076)
Population Stratification							
PC1	8.294***	8.544***	8.605***	9.806**	9.861**	8.083*	7.999*
	(2.298)	(2.300)	(2.282)	(2.995)	(3.035)	(3.666)	(3.675)
PC2	−4.292*	−4.203	−4.239	−4.462	−4.569	−3.990	−3.861
	(2.131)	(2.116)	(2.121)	(2.595)	(2.624)	(3.180)	(3.168)
PC3	−2.045	−2.069	−2.060	.871	.963	−4.065	−3.945
	(2.155)	(2.147)	(2.149)	(2.470)	(2.469)	(3.215)	(3.160)
PC4	−2.109	−2.008	−2.016	−1.523	−1.617	−2.162	−2.205
	(2.601)	(2.594)	(2.589)	(2.350)	(2.324)	(4.313)	(4.278)
PC5	3.003	2.845	2.784	4.251	4.370	2.002	1.993
	(2.006)	(2.054)	(2.062)	(2.779)	(2.752)	(2.853)	(2.845)
PC6	1.660	1.794	1.693	3.717	3.649	-.594	-.670
	(1.888)	(1.876)	(1.880)	(2.126)	(2.120)	(2.930)	(2.910)
PC7	−2.343	−2.188	−2.113	.095	-.025	−4.524	−4.398
	(2.316)	(2.318)	(2.303)	(2.906)	(2.877)	(3.698)	(3.695)
PC8	3.777	3.792	3.848	1.452	1.325	5.634	5.526
	(2.264)	(2.243)	(2.255)	(2.387)	(2.361)	(3.493)	(3.484)
PC9	−2.175	−2.279	−2.234	−2.673	−2.702	−1.048	−1.107
	(2.044)	(2.072)	(2.067)	(2.709)	(2.694)	(3.201)	(3.170)
PC10	−5.525**	−5.670**	−5.809**	−1.642	−1.632	−8.722**	−8.716**
	(2.021)	(2.022)	(2.016)	(2.711)	(2.706)	(2.937)	(2.945)
Constant	2.266***	2.334***	2.359***	2.057***	2.065***	3.427***	3.447***
	(.139)	(.129)	(.126)	(.140)	(.139)	(.210)	(.213)
Observations	17,603	17,603	17,603	10,827	10,827	6776	6776
N	7599	7599	7599	4749	4749	2850	2850

Standard errors in parentheses.
***p < .001, **p < .01, *p < .05.

In Models 9 through 14, where the population stratification is controlled for, the main effects of PGS, parental education, and the interaction terms still hold. The effect size of the PGS increases by a small 0.04 in Model 9 compared to Model 3. In models 10 and 12, the parental education coefficient decreases only slightly after controlling for population stratification. In Model 13, the significant negative effect of the interaction between parental education and PGS stays the same. The coefficient of −0.041 means that a standard deviation increase in parental education reduces 9% of the genetic effect [0.041/0.465 = 0.088]. Combined with the strong and positive effect of parental education, this significant negative interaction indicates that parents in better positions are capable in preserving the elite status in the next generation. The pattern shown in Model 8 holds in Model 14 as well.

These results support hypotheses 1 and 2c. The PGSs and parental education have significant positive effects on the transition to higher levels of education. Moreover, the main effects of parental education and PGSs are independent from each other to some extent. Only slight drops or increases are found in the analyses when including other variables in the models. An auxiliary analysis (not shown) using ordinary least square models also indicates that including the polygenic score alone explains 10.7% of the variation in years of schooling with population stratification considered, while parental education and other control variables account for 25.1%. When I only added the population stratification to the model (which already controls for SES and control variables) R^2 increased to 25.4%, and when PGS was also added, the R^2 increased to 30.5%. These results show that the PGS explains about 5.1% [30.5%–25.4%] of the variation in years of education, and support the independent main effects of both genes and social factors on educational attainment.

For the second hypothesis, the significant parental education × PGS interaction terms suggest that parental education moderates the genetic influences on education. The negative results further support the Saunders' argument (hypothesis 2c). This suggests that highly educated parents can protect their children from downward mobility.

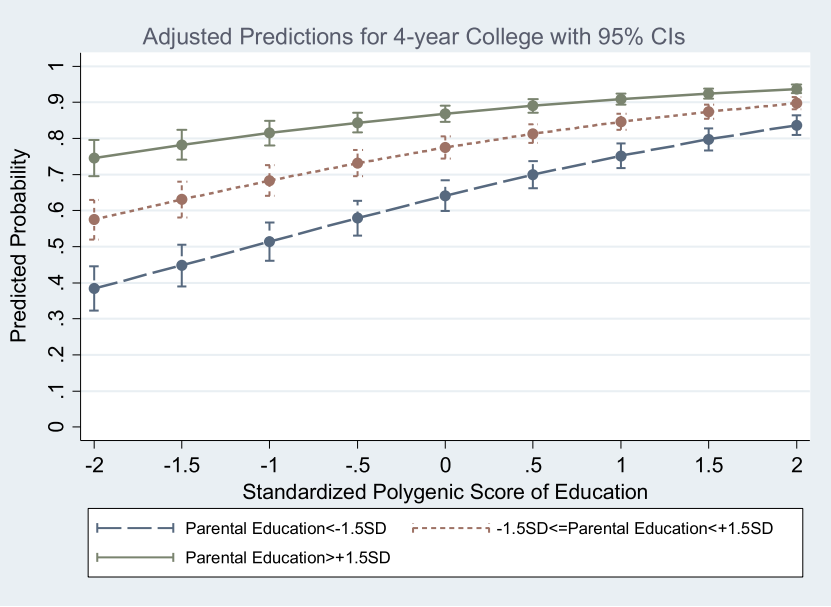

Fig. 2. Predicted probabilities for advancing into a four-year college by holding covariates at means.

4.3. Cohort differences

To examine whether the opportunity became more unequal or equal in the 20th century, interaction terms between PGS and cohorts were added to the preferred model (Model 13 in Table 2). The results for the cohort differences are shown in Table 3. The PGS is slightly stronger in the younger cohorts, as can be seen from the interaction terms in Model 2 and the main effect of the PGS in Model 4 and Model 6 (0.442 vs. 0.476). However, the PGS × Cohort interaction terms in Model 2 are positive but insignificant. Moreover, while there is a trend of increasing genetic effect over time and thus supporting the equalization hypothesis, the interaction term for the youngest cohort is weak. To strengthen the equalization hypothesis, Model 3 further includes the Parental education × Cohort interaction terms and shows a declining effect of parental education in the younger cohorts.

Hypothesis 4 argues that the moderating effect of parental education would be weaker in the later cohorts if educational opportunities became equal. Therefore, the cohort-separated analyses were added to the table. Models 4 through 7 demonstrate that the Parental education × PGS interaction term is significant for the older cohort in Model 5, and insignificant for the younger cohort in Model 7. This suggests that the moderating impact of parental education on the PGS is greater for the older cohort than the younger cohort. The result thus shows some supports for the hypothesis of equalization in the U.S. education system from the 1920s through the 1970s.

4.4. Predicted probability

Fig. 2 shows the predicted probability from Model 1 (Table 3) of advancing to a four-year college for different levels of parental education and the PGS with other variables held at means (see Supplementary section 1.1 for discussion on interaction terms in logistic models). The standardized parental education is divided into three groups based on a 1.5 standard deviation cutoff, hence, there are three lines plotted, as follows: 1) parental education less than −1.5 standard deviations; 2) parental education between −1.5 and 1.5 standard deviations; and, 3) parental education greater than 1.5 standard deviations. In the graph, parental education affects the probability of going to a four-year college the most when an individual's genetic potential for education is lower. However, as the PGS increases, the distances between the three lines shrink and the lines converge in the end. The marginal effect of the interaction term shown in Fig. 3 describes the negative moderating pattern. When holding the covariates at means, the slope of the interaction term is negative, indicating again as the PGS increases, the influence of parental education on entering a college decreases.

The cohort differences can be seen in Fig. 4. The two plots in Fig. 4 shows the predicted probability for a cohort of attending a four-year college for the three levels of parental education with covariates held at means. For the younger cohort (age 55), the level of parental education affects the predicted probability less significantly, while the differences between the three levels of parental education at the lower end of the PGS are wider for the older cohort. Fig. 4 also shows a trend that the younger the cohort, the higher the probability of going to a four-year college for individuals at every level of parental education. This supports the equalization hypothesis.

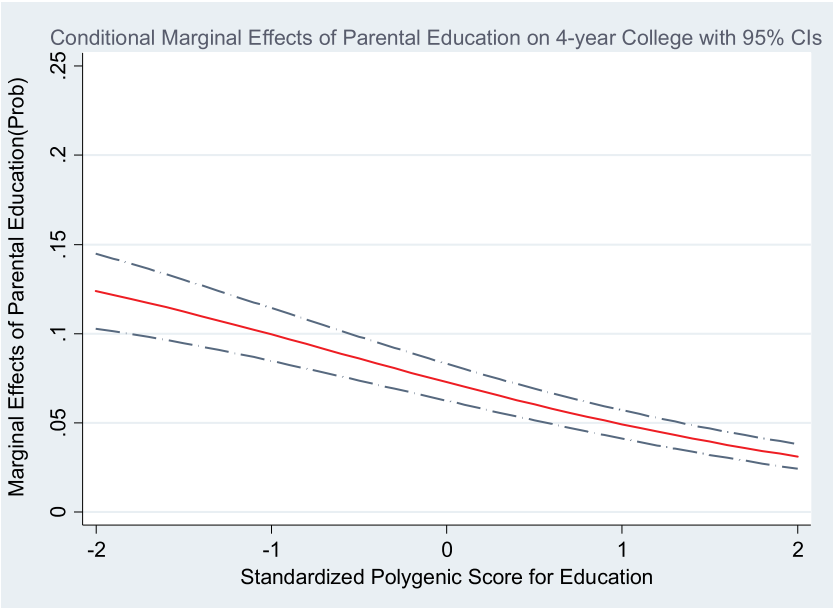

Fig. 3. Marginal effects of standardized parental education on advancing into a four-year college for different values of standardized polygenic score of education by holding covariates at means.

5. Conclusion and discussion

This study has used the latest available genetic measurement to address longstanding issues regarding educational attainment in sociology. I have demonstrated that both genetic components and traditional parental education are positively related to educational transitions and have independent effects on educational attainment. The inclusion of the genetic component does not challenge the effect of parental education, affirming the sociological status attainment model. A one standard deviation increase in the parental education leads to a 53% increase in the likelihood of advancing to the next level of education while a one standard deviation increase in the polygenic score results in a 58% increase in the likelihood of advancement. The significant gene-environment interaction effects of the genetic component and parental education further enrich our understanding of the educational transition. My results, which indicate negative interactions between the PGS and parental education, suggest that resourceful parents are able to assure advantages in schooling for their offspring, even if their child is less endowed. Individuals from disadvantaged backgrounds are also likely to obtain higher education, but only if they are talented. This pattern supports the Saunders Hypothesis but does not support either the Scarr-Rowe or the Pareto hypotheses.

My findings also suggest that educational opportunity in the 20th century U.S. equalized, and they provide evidence for the effects of historical changes on realizing genetic potential. Compared to Conley et al. (2015) and Conley and Domingue (2016), who used the less powerful whole-genome results from Rietveld et al. (2013), my results not only confirms the effect of genes on educational attainment but also provides evidence for the gene-environment interactions on both the micro and macro levels.

On the micro level, unlike the weak and slightly positive interaction between the PGS and maternal education reported by Conley

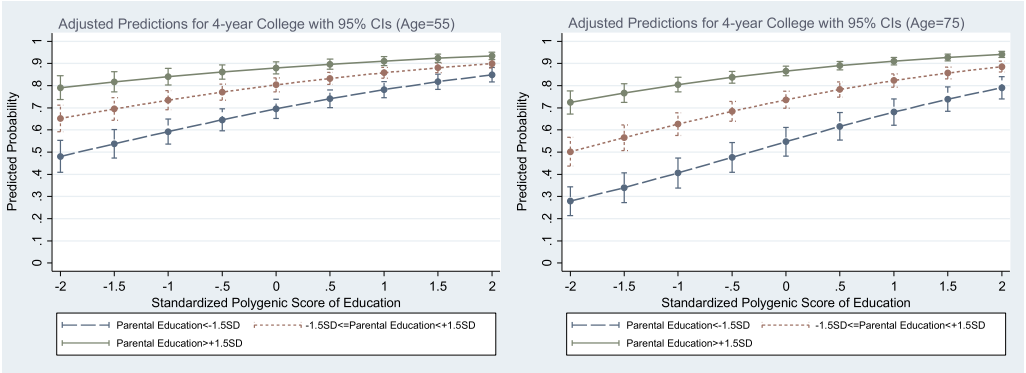

Fig. 4. Cohort differences in the predicted probabilities for advancing into a four-year college by holding covariates at means.

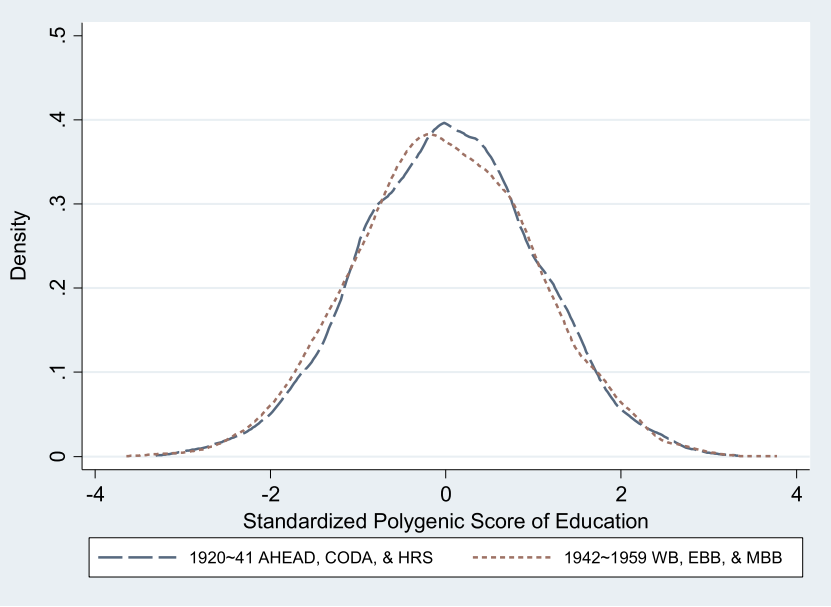

Fig. 5. Correlation between cohorts and standardized polygenic score of education.

et al. (2015), but similar to Papageorge and Thom (2018), I found stronger but negative interaction effects of PGS and parental education, which suggests that the Saunders Hypothesis might be supported. On the macro level, similar but different from the significant interaction between birth year and PGS in Conley and Domingue's (2016) work, I found a weak positive relationship between genetic effects and educational transition over time, which might support the equalization theory.

Some of my results are inconsistent with those of previous studies (Conley et al., 2015; Conley and Domingue, 2016; Papageorge and Thom, 2018). This might be due to the new GWAS results I used to construct my PGS or to the different models I used in my analysis. The continuation ratio logit model approximates the reality more closely because it estimates the odds of entering the next stage conditioned on completing the previous stage.

My results indicate that the effect of the PGS generally increased as higher education opportunities have expanded in the 20th century, but the effect dropped in the youngest cohort. An explanation for the declining effect would be the saturation argument suggested by Raftery and Hout (1993) in their maximally maintained inequality (MMI) hypothesis. The MMI hypothesis states that the expansion of higher education aims to equalize the impact of family SES on educational attainment by increasing opportunities for all. However, as the supply of this level of education surpasses the demand, familial influences decrease at the particular level but manifest more strongly at the next higher level. Thus, inequality persists at the maximum level of education whenever there is at least a higher level that is not saturated. Saturation refers to the likelihood that all offspring from advantaged families attain that level of education. For example, when all the children from wealthy families obtain a high school diploma, the relative advantage of attaining a high school degree for them decreases from then on. The high school level of education is thus saturated, meaning that the inequality of attending high school diminishes after the high school level expands (i.e., an increase in the numbers of students allowed to enroll). The same concept can apply to other levels of education with equal force.

If the effect of genes is regarded as meritocratic ability, when levels of education are saturated, genetic influence might also decline. Roksa et al. (2007) demonstrated that the effect of parental education is slightly weaker for the post-WWII cohort born before the 1970s. Their results also suggest that father's occupation had a smaller impact on attending college for this cohort in the 1980s (a cohort members of which are participants in the Health and Retirement Study, my data source).

A study using the Swedish Twins Registry data from 1929 to 1958 (Okbay et al., 2016) might support the argument. The authors reported the decreasing effect of the all-SNP score throughout the birth cohorts and suggested this was a consequence of the liberal reform of the educational system in the 1950s and 1960s, which extended compulsory education and postponed school tracking. Thus, as higher education became nearly universal, both the selectivity of higher education and the variation of educational attainment dropped and genetic effects declined.

The educational expansion in higher education thus offers an explanation to the fluctuated PGS effects over time as the genetic effect increased during the initial expansion and declined when the higher levels of education are accessible by most. The new results should therefore provide new insights on the impact of micro and macro environment on the realization of individual potential.

With respect to policy-related implications, since both genetics and parental education are important for educational attainment, resource redistribution policies might improve the achievement of the children from deprived families. The significant moderating effect of parental education also implies that welfare policies could help individuals from poor families to perform better academically.

The empirical support I found in my results for the equalization hypothesis shows the impact of liberal policies on educational opportunities in the mid-20th century. Future studies can investigate the changes in genetic effects after the 1980s' conservative

Reagan revolution; the work of Roksa et al. (2007) examined the changing effects of social inheritance and demonstrated that the influence of having a parent with an undergraduate degree or higher on entering four-year college increased significantly again in the 1990s. Researchers might consider genetic effect changes during the same period in the future.

This paper also has several limitations. First, the HRS dataset does not include parental genetic information; parents' genes may influence their own educational attainment and SES, thereby affecting children's genetic realization. Second, utilizing the sibling fixed-effect model might allow for better control of other unobserved variables; however, sibling information is not available in the HRS data.

Third, HRS retroactively obtained the parental education, thus increasing the chances of respondents forgetting or subjectively modifying their parents' educational attainment, potentially resulting in recall bias. Another age-related issue is the mortality or morbidity selection bias of the HRS sample (Domingue et al., 2017). Individuals who have a better polygenic score for education might live longer. Fig. 5 shows that although there is a weak correlation between age and polygenic score (Pearson's $r = 0.05$, $p < .000$), the distribution of the polygenic score for education is similar across cohorts. Supplementary analysis applying the inverse probability weights (IPW) as Domingue et al. (2017) suggested shows the similar results (Supplementary Tables 1–3).

Fourth, the subjects in this study are non-Hispanic whites due to the availability of GWAS results, and the PGS used in my analyses was constructed by using the meta-analysis of several cross-national cohorts using the genetics data at a single time from each participant. However, it is possible that the effects of each SNP have changed over time, and the SNPs that matter significantly at one point in time might not be significant at other times (Kulminski et al., 2015). Therefore, caution is needed when generalizing the cohort-specific results.

In spite of these limitations, by including genetic PGS in the analysis, my results strengthen the sociological explanation of educational attainment. As genetic data become widely available and molecular genetics advances, future studies can take advantage of the burgeoning opportunities to not only explore the genetic effects on sociological outcomes, but further solidify the sociological accounts of social behaviors and status-related outcomes.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssresearch.2019.102387.

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