The effect of education on spousal education: A genetic approach

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ABSTRACT

We investigate the causal effect of education on spousal education using a sample of couples from the Health and Retirement Study. We estimate reduced-form linear matching functions derived from a parsimonious matching model which links spouses' education. Using OLS we find that an additional year in husband's (resp. wife's) education is associated with an average increase in wife's (resp. husband's) education of 0.41 years —95% CI: 0.37, 0.45 (resp. 0.63 years —95% CI: 0.57, 0.68). To deal with endogeneity issues due to measurement error and omitted variables, we use a measure of genetic propensity (polygenic score) for educational attainment as an instrumental variable. Assuming that our instrument is valid, our 2SLS estimate suggests that an additional year in husband's (resp. wife's) education increases wife's (resp. husband's) education by about 0.49 years —95% CI: 0.35, 0.62 (resp. 0.76 —95% CI: 0.56, 0.96). Since greater genetic propensity for educational attainment has been linked to a range of personality and cognitive skills, we allow for the possibility that the exclusion restriction is violated using the plausible exogenous approach by Conley et al. (2012). A positive causal effect of education on spousal education cannot be ruled out, as long as one standard deviation increase in husband's (wife's) genetic propensity for education directly increases wife's (husband's) education by less than 0.2 (0.3) years.

1. Introduction

Assortative mating on education —that individuals with similar education match with one another more frequently than would be expected under a random mating pattern or that partners' educational attainments are positively correlated— has been studied in economics since the seminal work by Becker (1973). Many social scientists have documented a strong and increasing educational homogamy[1] (e.g., Bruze, 2011; Chiappori et al., 2009; Greenwood et al., 2014; Schwartz and Mare, 2005). More recently, Gihleb and Lang (2016) have warned about the use of inappropriate statistical techniques when studying the evolution of assortative mating over time, and concluded that there is little evidence supporting an increase in educational homogamy. Mogstad et al. (2019) have shown that assortative mating has been declining over time among college graduates, whereas the low-educated have been increasingly sorting into internally homogeneous marriages.

In this paper, we focus on the impact of education on spousal education. We estimate linear matching functions derived from a parsimonious matching model where individuals match on human capital. The model allows us to derive linear matching functions which link wife's (resp. husband's) human capital to husband's (resp. wife's) human capital. In practice, human capital is not observed by the econometrician, and instead we use years of education. Under classical measurement error, it is well-known that the OLS regression estimate of the slope of wife's (resp. husband's) education on husband's (resp. wife's) education will be biased towards zero. A valid instrumental variable can fix this, and help us in dealing with other endogeneity concerns such as omitted variables bias (e.g., if individuals match on other characteristics correlated with human capital). We use genetic data to obtain potentially valid instrumental variables and infer the causal effect of education on spousal education using a sample of couples from the Health and Retirement Study (HRS).

We use a polygenic score for educational attainment as an instrumental variable for educational attainment. The polygenic score —a single quantitative measure of genetic predisposition based on genetic variants present in the entire genome (see Plomin et al., 2009)— is constructed to predict educational attainment of married individuals using data from

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[1] Education homogamy refers to the tendency of spouses being similar to each other in terms of educational attainment, educational qualification or field of study.

the HRS, building upon the recent findings from a large scale GWAS of educational attainment (Lee et al., 2018), and following recent work in economics (Barth et al., 2020; Papageorge and Thom, 2020).[2]

We are the first to rely on molecular data to exploit potential exogenous variation in educational attainment, allowing for the possibility that our instrument violates the exogeneity condition using the approach by Conley et al. (2012), in a marriage market application. Our OLS estimates of the matching functions show that an additional year in husband's (resp. wife's) education is associated with an average increase of 0.41 years in wife's education (resp. 0.63 years in husband's education). We also find that a one standard deviation increase in husband's educational attainment polygenic score (EA PGS) increases husband's education by about 0.64 years and wife's education by about half of this magnitude, 0.31. Our IV (2SLS) estimates, using husband's EA PGS as the (excluded) instrument for wife's education, suggest that an additional year in husband's education increases wife's education by about 0.49 years. Using wife's EA PGS as the excluded instrument for husband's education, we find that an additional year in wife's education increases husband's education by about 0.76 years.

While the educational attainment polygenic score (EA PGS) is a relevant instrument for education and is considered to be randomly assigned at conception (Mendelian randomization), at least after accounting for population stratification, this is a necessary but *not* a sufficient condition to use the EA PGS as a valid instrumental variable.[3] Polygenic scores for spousal education may affect own education above and beyond their effects on spousal education, and hence violate the exclusion restriction.[4]

Greater polygenic propensity for educational attainment has been linked to higher cognitive aptitude, self-control, and interpersonal skills in childhood (Belsky et al., 2016; Rabinowitz et al., 2019), and more recently, to larger brains (Elliott et al., 2018),[5] but also to lower scores on the ADHD (attention deficit hyperactivity disorder) index (de Zeeuw et al., 2014). Since intelligence and personality, amongst other attributes, are relevant in the marriage market (Dupuy and Galichon, 2014; Lundberg, 2012), it is important to assess the consequences of departing from the exclusion restriction.

To allow for the possibility that the exogeneity condition is violated, we relax the exclusion restriction following the approach in Conley et al. (2012) whose implementation is carefully discussed by Clarke and Matta (2018).[6] In particular, we allow for the own EA PGS to have a direct effect on spousal education. We cannot rule out a positive causal effect of husband's education on wife's education so long as a one standard deviation increase in husband's EA PGS directly increases wife's education by less than 0.2 years. Similarly, we cannot rule out a positive causal effect of wife's education on husband's education so long as a one standard deviation increase in wife's EA PGS directly increases husband's education by less than 0.3 years.

There is an extensive literature on education in the marriage market, which studies empirical matching patterns or the effect of education, via adjusting for observable characteristics (Chiappori et al., 2016; Oreffice and Quintana-Domeque, 2010), using within-siblings or within-twins variation (Huang et al., 2009) or instrumental variables (Lefgren and McIntyre, 2006).

Larsen et al. (2015) claim that using the variation in male educational attainment induced by the WWII G.I. Bill may provide the most transparent identification strategy to date: their findings suggest that the additional education received by returning veterans caused them to "sort" into wives with significantly higher levels of education. While theirs is an interesting identification strategy, it only exploits *cohort* variation.

Earlier work had studied the impact of male scarcity on marital assortative mating using the large shock that WWI caused to the number of French men (Abramitzky et al., 2011), used quarter of birth as a (weak) instrument for female education (Lefgren and McIntyre, 2006), or data on twins to assess assortative mating and how education is productive in marriage (Huang et al., 2009).[7]

Our work is also related to studies on genetic assortativeness.[8] These articles use genetic information from large scale GWASs that are also the core of our analysis. While they are instrumental for our analysis, our work departs from them, if only because our focus is on the causal effect of education on spousal education, and not spousal resemblance at the genotypic level.[9] Moreover, we find that genetic assortativeness on education polygenic scores is much smaller than assortativeness on education, and that it essentially disappears after controlling for education and population stratification, consistent with recent work by Barth et al. (2020).

Our research also broadly speaks to the increasing "genoeconomics" literature that studies the genetic determinants of socioeconomic outcomes (Barban et al., 2021; Beauchamp et al., 2011; Benjamin et al., 2007; Conley et al., 2014). While a few studies in economics have used genome-wide polygenic score as an instrumental variable (see also Böckerman et al., 2019; von Hinke Kessler Scholder et al., 2016), we are the first to study the causal effect of education on spousal education using genetic data as a source of potential plausible exogenous variation using the approach by Conley et al. (2012).

As we shall see, our IV results are valid for a range of mild violations of the exclusion restriction, directly tackling the issue of *pleiotropy*, which in the context of genome-wide scores leads to concerns about the number of potential pathways through which the score could influence the outcome.[10] Hence, our work complements and expands the economic literature using genes (or genetic markers) as instrumental variables (e.g., Cawley et al., 2011; Fletcher and Lehrer, 2011; von Hinke Kessler Scholder et al., 2011; 2013; 2016; 2014; Norton and Han, 2008).

The rest of the paper is organized as follows. Section 2 derives reduced-form linear matching functions and discusses how to identify the causal effect of education on spousal education. Section 3 defines the polygenic score for education and how to handle potential deviations from IV assumptions. Section 4 describes the data sources, the construction of the polygenic score and presents some descriptive statistics. Section 5 contains the main estimates. Section 6 concludes the paper.

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[2] Rather than focusing on a limited number of genetic variants, the polygenic scores (PGSs) use the entire information in the DNA (or a large proportion of it) to construct a measure of genetic predisposition to higher educational attainment (Conley et al., 2015; Domingue et al., 2014; Plomin et al., 2009; Ward et al., 2014).

[3] As discussed by Beauchamp et al. (2011), the use of genetic markers as instrumental variables was anticipated by Davey Smith and Ebrahim (2003) and pioneered in economic research by Ding et al. (2009), who used genes as instrumental variables for health in studying the impact of health on academic outcomes.

[4] Many researchers argue that it is very unlikely (if not impossible) that any markers satisfying the exclusion restriction will ever be found in many economic applications (Cawley et al., 2011; Conley, 2009).

[5] Brain size is positively related with cognitive scores (Elliott et al., 2018).

[6] `plausexog` in Stata: https://ideas.repec.org/c/boc/bocode/s457832.html

[7] More generally an IV approach to instrument for market conditions, such as sex ratios, had been used by Angrist (2002) and Charles and Luoh (2010), for instance.

[8] Using data from the HRS, Domingue et al. (2014) find that spouses are more genetically similar than two people chosen at random. Guo et al. (2014) also find a positive similarity in genomic assortment in married couples by using the HRS and the Framingham Heart study. Conley et al. (2016), however, show that the increased level of assortative mating in education observed across birth cohorts from 1920 to 1955 does not correspond to an increase in similarity at the genotypic level.

[9] On the genetic similarity of spouses see also Zou et al. (2015).

[10] Recent work by van Kippersluis and Rietveld (2018a) and van Kippersluis and Rietveld (2018b) expands the plausible exogenous approach in Conley et al. (2012) in a world with *heterogeneous* first-stage effects but with *constant* reduced-form effects.

2. Reduced-Form linear matching functions

While several studies have used (and estimated) linear matching functions linking spouses incomes, occupations and/or human capital measures (Chiappori et al., 2016; Ermisch et al., 2006; Lam and Schoeni, 1993; 1994; Oreffice and Quintana-Domeque, 2010), these were based on implicit or explicit statistical (linear) decomposition exercises rather than derived from equilibrium matching models. In this section, we derive reduced-form linear matching functions from a parsimonious model of the marriage market. The main purpose of this section is to show that a linear matching function —to estimate the causal effect of education on spousal education— can be obtained from a very simple equilibrium model of the marriage market.

Our model is based on Browning et al. (2014)[11] and we follow their assumptions quite closely. First, human capital is the only marital trait —x is the human capital of women, y is the human capital of men— and the marital surplus —the difference in the utility generated by a couple formed by a woman with human capital x and a man with human capital y and their utility levels when single— is produced according to the function $h(x, y)$. Second, h is twice continuously differentiable, strictly increasing in both x and y (i.e., $h_x > 0$, $h_y > 0$) and strictly super-modular (i.e., $h_{xy} > 0$). Third, there is a continuum of men and women with a total mass each normalized to 1. Fourth, there are no search frictions: each woman (resp. man) has free access to the pool of all potential male (resp. female) spouses, with perfect knowledge of the marital traits of each other. Finally, x and y are independently and normally distributed before the matching takes place: $x \sim N(\mu_x, \sigma_x^2)$ and $y \sim N(\mu_y, \sigma_y^2)$.[12]

Deterministic matching function. Given that there are no search frictions (fourth assumption), matching arises due to preferences (i.e., marital surplus). Moreover, the property of the marital surplus function (by the second assumption) guarantees positive assortative mating on human capital.[13] Therefore, if a woman with human capital x is married to a man with human capital y, then the set of women with human capital levels above x must be the same measure as the set of men with human capital levels above y. Thus, given the third and fifth assumptions, for all x and y in the set of married couples,

$$1 - \Phi\left(\frac{x - \mu_x}{\sigma_x}\right) = 1 - \Phi\left(\frac{y - \mu_y}{\sigma_y}\right),$$

where Φ is the standard normal cumulative distribution function. Thus, the matching function that determines the human capital of the wife for each man with human capital y is given by

$$x = \left(\mu_x - \frac{\sigma_x}{\sigma_y}\mu_y\right) + \frac{\sigma_x}{\sigma_y}y,$$

or in compact notation,

$$x = \alpha + \beta y, \tag{1}$$

where $\alpha \equiv \left(\mu_x - \frac{\sigma_x}{\sigma_y}\mu_y\right)$ and $\beta \equiv \frac{\sigma_x}{\sigma_y}$. Similarly, the matching function that determines the human capital of the husband for each woman with human capital x is given by

$$y = \left(\mu_y - \frac{\sigma_y}{\sigma_x}\mu_x\right) + \frac{\sigma_y}{\sigma_x}x.$$

This simple model predicts perfect positive assortative mating in human capital:

$$Corr(x, y) = \frac{Cov(x, y)}{\sigma_x \sigma_y} = \frac{Cov(\alpha + \beta y, y)}{SD(\alpha + \beta y)SD(y)} = \beta\frac{Cov(y, y)}{\beta\sigma_y\sigma_y} = \frac{\sigma_y^2}{\sigma_y^2} = 1.$$

In addition, and as highlighted by Gihleb and Lang (2016), a regression of x on y will identify β, i.e. $\frac{\sigma_x}{\sigma_y}$:

$$\frac{Cov(x, y)}{V(y)} = \frac{Cov(\alpha + \beta y, y)}{V(y)} = \beta\frac{Cov(y, y)}{V(y)} = \beta\frac{\sigma_y^2}{\sigma_y^2} = \beta = \frac{\sigma_x}{\sigma_y},$$

while a regression of y on x will identify $\frac{1}{\beta}$, i.e. $\frac{\sigma_y}{\sigma_x}$:

$$\frac{Cov(x, y)}{V(x)} = \frac{\sigma_y}{\sigma_x}.$$

Stochastic matching function. In practice, we do not observe human capital, but a proxy of it, say years of education, or we are interested in years of education but our measure of education contains error. Hence, our proxies for human capital or education for wives and husbands are given by:

$$\widetilde{x} = x + \epsilon_x,$$

$$\widetilde{y} = y + \epsilon_y,$$

where measurement error for each measure is white noise: $\epsilon_x \sim N(0, \sigma_{\epsilon_x}^2)$ and $\epsilon_y \sim N(0, \sigma_{\epsilon_y}^2)$. Thus, equation (1) with measurement error becomes:

$$\widetilde{x} = \alpha + \beta\widetilde{y} + u, \tag{2}$$

where $u = \epsilon_x - \beta\epsilon_y$, and hence $Cov(\widetilde{y}, u) \neq 0$.

Now, the correlation between the human capital proxies among spouses is given by:

$$Corr(\widetilde{x}, \widetilde{y}) = \frac{Cov(\widetilde{x}, \widetilde{y})}{SD(\widetilde{x})SD(\widetilde{y})} = \frac{Cov(x, y)}{\sigma_x \sigma_y}\frac{\sigma_x \sigma_y}{SD(x + \epsilon_x)SD(y + \epsilon_y)}$$
$$= \frac{\sigma_x \sigma_y}{SD(x + \epsilon_x)SD(y + \epsilon_y)} < 1.$$

In addition, the regression of $\widetilde{x}$ on $\widetilde{y}$ will *not* identify β, i.e. $\frac{\sigma_x}{\sigma_y}$, but:

$$\frac{Cov(\widetilde{x}, \widetilde{y})}{V(\widetilde{y})} = \beta\frac{\sigma_y^2}{\sigma_{\epsilon_y}^2 + \sigma_y^2} = \frac{\sigma_x}{\sigma_y}\frac{\sigma_y^2}{\sigma_{\epsilon_y}^2 + \sigma_y^2}.$$

Similarly, the regression of $\widetilde{y}$ on $\widetilde{x}$ will *not* identify $\frac{1}{\beta}$, i.e. $\frac{\sigma_y}{\sigma_x}$, but:

$$\frac{Cov(\widetilde{x}, \widetilde{y})}{V(\widetilde{x})} = \frac{1}{\beta}\frac{\sigma_x^2}{\sigma_{\epsilon_x}^2 + \sigma_x^2} = \frac{\sigma_y}{\sigma_x}\frac{\sigma_x^2}{\sigma_{\epsilon_x}^2 + \sigma_x^2}.$$

A well-known method to deal with attenuation bias due to classical measurement error is instrumental variables. In general, instrumental variables can also help us to deal with other sources of endogeneity (Angrist and Krueger, 1999).

IV to the rescue. Assume that we have a potential valid instrument z_y for $\widetilde{y}$. In particular, suppose that z_y is a measure of genetic predisposition to higher educational attainment (Lee et al., 2018). The two well-known conditions for instrument validity are the following:

IV1: Relevance. The instrument z_y must be correlated with the endogenous variable $\widetilde{y}$:

$$Cov(\widetilde{y}, z_y) \neq 0.$$

IV2: Exogeneity. The instrument z_y must be uncorrelated with the error term u:

$$Cov(u, z_y) = 0.$$

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[11] See section 7.2.3.

[12] Browning et al. (2014) refer to x and y as income, focus on the transferable utility case, allow for different masses of men and women, and assume that x and y are uniformly distributed to solve for the closed-form solution of the matching function.

[13] The assumption that marital surplus is strictly increasing in both arguments guarantees positive assortative mating in a non-transferable utility context. The assumption that the cross-derivative is strictly positive guarantees positive assortative mating in a transferable utility context.

As long as these two conditions hold,

$$\frac{Cov(\widetilde{x}, z_y)}{Cov(\widetilde{y}, z_y)} = \beta.$$

In this paper, we will use z_y to estimate the causal effect of y on x, and z_x to estimate the causal effect of x on y.

Of course, the model used to derive the linear reduced-form matching functions has many limitations. In practice, people match on multiple characteristics (Chiappori et al., 2012; 2018) not included in the model. Moreover, search frictions may play a role. Thus, our model cannot help us in providing a structural interpretation of our causal estimates (e.g., search vs. preferences). Indeed, in our parsimonious model the causal effect of husband's education on wife's education β is $\frac{\sigma_x}{\sigma_y}$, while the causal effect of wife's education on husband's education is $\frac{\sigma_y}{\sigma_x}$, and this prediction is totally counterfactual. However, the model makes three valid points: first, it shows that a linear matching function can be obtained from a simple parsimonious equilibrium model of the marriage market; second, it shows that assortative mating cannot be measured in terms of regression coefficients (Gihleb and Lang, 2016), because changes in σ_x (or σ_y) over time will lead to increases (decreases) in regression coefficients, which cannot be interpreted as increases (decreases) in assortative mating; finally, under this simple model, IV estimates will be larger than the OLS estimates, a prediction which is borne out by the data, at least, qualitatively.

In the next section, we discuss the construction of a genetic IV and the potential violation of the exclusion restriction, for instance, by the omission of other characteristics which are relevant in the marriage market but are not included in our one-dimensional parsimonious model. To tackle potential violations of the exclusion restriction, we will follow the plausible exogenous approach by Conley et al. (2012).

3. Building a potentially valid genetic IV

3.1. Polygenic scores

Recent advances in molecular genetics have made it possible and relatively inexpensive to measure millions of genetic variants in a single study. The most common type of genetic variation among people is called single nucleotide polymorphism (SNP). SNPs are genetic markers that have two variants called alleles. Since individuals inherit two copies for each SNP, one from each parent, there are three possible outcomes: 0, 1 or 2 copies of a specific allele. SNPs occur normally throughout a person's DNA. Each SNP represents a difference in a single DNA building block, called a nucleotide. For example, a SNP may indicate that, in a certain stretch of DNA, a nucleotide cytosine is replaced with the nucleotide thymine among some individuals.

SNPs are usually indicated by their position in the DNA, their possible nucleotides and by an identification number. They occur once in every 300 nucleotides on average, which means there are roughly 10 million SNPs in the human genome. A large part of current genetic research aims to identify the function of these genetic variants and their relationship to different diseases. Genome-wide association studies (GWASs) have been used to identify SNPs associated to particular diseases or traits. A drawback of GWAS is that, given the polygenic nature of human diseases and traits, most identified variants confer relatively small increments in risk, and explain only a small proportion of heritability. A common solution is to use the results from a GWAS and compile a polygenic score (PGS) for a phenotype aggregating thousands of SNPs across the genome and weighting them by the strength of their association.

There are two main reasons to use a PGS to describe the genetic susceptibility to a trait in social sciences (Belsky and Israel, 2014; Schmitz and Conley, 2017). First, complex health outcomes or behaviors are usually highly polygenic, i.e., reflect the influence or aggregate effect of many different genes (Visscher et al., 2008). PGSs assume that individuals fall somewhere on a continuum of genetic predisposition resulting from small contributions from many genetic variants. Second, a single genetic variant has too small of an effect in explaining complex phenotypes, i.e., no single gene produces a symptom or trait at a detectable level, unless the sample size is extremely large.

A PGS for individual i can be calculated as the sum of the allele counts a_{ij} (0, 1 or 2) for each SNP $j = 1, \ldots, M$, multiplied by a weight q_j, that is:

$$PGS_i = \sum_{j=1}^{M} q_j a_{ij},$$

where weights q_j are transformations of GWAS coefficients. A polygenic score is therefore a linear combination of the effects of multiple SNPs on the trait of interest. SNPs are not independent in the genome but their occurrence varies according to a block structure called *linkage disequilibrium* (LD). Using unadjusted GWAS coefficients as weights could reduce the accuracy of PGSs and yield to imprecise estimates (Barth et al., 2020). Different methods have been proposed to account for linkage disequilibrium in the construction of polygenic scores.

In this paper, we use a Bayesian method called LDpred (Vilhjálmsson et al., 2015) to derive the weights q_j. Weights are based on the association results from the GWAS on educational attainment by Lee et al. (2018), where HRS has been removed from the analysis, to avoid overfitting.[14] LDpred assumes a point-normal mixture prior for the distribution of effect sizes and takes into account the correlation structure of SNPs by estimating the LD patterns from a reference sample of unrelated individuals. The weight for each variant is set to be equal to the mean of the posterior distribution (approximated via MCMC simulation) after accounting for LD. LDpred requires an assumption about the fraction of SNPs which are truly associated with the outcome. A common choice for polygenic traits, followed in this study, is to assume that all the SNPs are associated with the outcome of interest (Barcellos et al., 2018; Barth et al., 2020). The scale of PGSs depends on the number of SNPs included in the score. For comparability purposes, we standardize a score by subtracting its mean and dividing it by its standard deviation.[15]

Using PGSs rather than single genetic markers has several advantages. First, they are "hypothesis-free" measures that do *not* require knowledge about the biological processes involved. This is particularly important when the phenotype of interest is complex, i.e., influenced by a large number of genes, or when its biological mechanisms are not yet fully understood (Belsky and Israel, 2014). Second, using a score, rather than single genes, is a possible *solution* to overcome the low predictive power of single genes, especially for behavioral traits. For example, in Lee et al. (2018) —the most recent GWAS on educational attainment— the top genome-wide significant SNP explains around 0.01% of the variation in years of schooling, while a linear polygenic score from all measured SNPs explains 10.6% of the same variable in the HRS sample.[16]

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[14] An alternative method to estimate weights for a polygenic score consists of selecting independent SNPs with a statistical procedure called *pruning*. The selected independent SNPs are then used to calculate the score, avoiding possible bias due to oversampling DNA regions that are highly genotyped. The range of possible values that a PGS can take depends on the number of SNPs included, tending to a normal distribution if the number of independent SNPs included in the score is sufficiently high. Simulation studies have shown that LDpred leads to more precise estimates for polygenic scores in case of highly polygenic traits (Vilhjálmsson et al., 2015)

[15] PGSs were standardized at the population level to be as close as possible to an indicator of genetic predisposition of the HRS population with European ancestry. Our regression sample is slightly positive selected in the PGSs: the average husband's (wife's) EA PGS is 0.19 (0.15). We then restandardized the scores based on the regression sample. While this is obviously immaterial for our IV estimates, it facilitates the numerical interpretation of the coefficients on the PGSs in terms of standard deviations.

[16] This refers to the incremental (additional) R-squared in a regression of years of education on sex, birth year, the interaction between sex and birth year, and the first ten principal components of the genetic relatedness matrix. A one stan-

Third, complete genome-wide association results are *publicly* available. PGSs can be calculated from consortia data for a range of phenotypes. The results published by these consortia are based on a meta-analysis of a large number of cohort studies. The predictive power of a polygenic score is inflated if the samples are not independent, i.e., the same sample was used in the original calculation of association results. For this reason, it is common to use genetic association results from independent studies or to rerun the association results excluding the cohort to which the score is applied, which is exactly how we proceed.

3.2. Genetic IV

There is a vast literature in statistics and epidemiology that focuses on methodological aspects related to genetic IV (e.g., Burgess et al., 2015; Davey Smith and Ebrahim, 2003; Davies et al., 2015; Didelez and Sheehan, 2007; Glymour et al., 2012; Kang et al., 2016; Lawlor et al., 2008; Sheehan et al., 2008; Windmeijer et al., 2018). von Hinke Kessler Scholder et al. (2016) and van Kippersluis and Rietveld (2018b) carefully examine the conditions needed for genetic variants to be used as valid instrumental variables.[17]

The reduced-form linear matching function (2) clarifies the necessary requirements, relevance and exogeneity, for a valid instrument.[18] The relevance assumption (**IV1**) requires that the husband's polygenic score for education, z_y, is linearly related with husband's education, $\tilde{y}$. While the use of one or few genetic variants can be weakly associated with education (weak instrument problem), our polygenic score is relevant and has been shown to robustly affect education (Lee et al., 2018; Okbay et al., 2016; Rietveld et al., 2013). Moreover, the score predicts education differences between siblings (Rietveld et al., 2014). The exogeneity assumption (**IV2**) requires that the husband's polygenic score, z_y, is uncorrelated with the error term u.[19]

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dard deviation increase in the polygenic score is associated with an increase in educational level of 0.84 years (SE=0.026). See Supplementary Table 38, panel B, in Lee et al. (2018).

[17] von Hinke Kessler Scholder et al. (2016) and Böckerman et al. (2019) use polygenic scores for body mass index as IV. Previous studies based on candidate genes have investigated: the effect of obesity or body fat mass on labor market outcomes (Norton and Han, 2008), on medical costs (Cawley and Meyerhoefer, 2012), or on educational attainment (von Hinke Kessler Scholder et al., 2012); the impact of poor health on academic performance (Ding et al., 2009; Fletcher and Lehrer, 2011); the effect of cigarette smoking on BMI (Wehby et al., 2012); the effect of alcohol exposure in utero on child academic achievement (von Hinke Kessler Scholder et al., 2014); the effects of cigarette quitting during pregnancy on different health behaviors (Wehby et al., 2013); the effect of child/adolescent height on different health and human capital outcomes (von Hinke Kessler Scholder et al., 2013). More recently, Cawley et al. (2019) investigate whether an individual's BMI is affected by the polygenic risk score for BMI of their full sibling when controlling for the individual's own polygenic risk score for BMI. They do not find evidence for such an effect.

[18] The usual motivation for using a genetic instrumental variable (IV) is the fact that individuals' genotypes are randomly allocated at conception: such a quasi-experimental design is called *Mendelian randomization* (Davey Smith and Ebrahim, 2003). However, randomization is *not* a sufficient condition to use genetic data as valid instrumental variables, as recently emphasized by Davies et al. (2018) and van Kippersluis and Rietveld (2018b).

[19] In common genetic IV studies that investigate the effect of one individual's treatment on the *same* individual's outcome, by using a genetic variant of his as instrument, the exclusion restriction can be violated mainly in four situations (von Hinke Kessler Scholder et al., 2016): (i) when parents' behavior or preferences are affected by the genotype; (ii) when the mechanisms, through which genetic variants affect the exposure variable, imply changes in behaviors or preferences that affect directly the outcome; (iii) when the genetic instrument is correlated with other genetic variants that affect the outcome (*Linkage Disequilibrium*); (iv) when disruptive influences of the risk factor on the outcome are limited by foetal or post-natal development processes (*Canalization*).

3.3. IV in practice: Statistical inference and deviating from exogeneity

While instrument relevance (**IV1**) is usually assessed by means of well-known rules of thumb on the value of the first-stage F statistic (Staiger and Stock, 1997; Stock and Yogo, 2005), recent work by Lee et al. (2020) shows that a true 5 percent test requires an F greater than 104.7. Following Lee et al. (2020), we construct IV confidence intervals which are dependent on the value of the first-stage F statistic: If $F > 104.7$, the 95% CI is $\hat{\beta}_{IV} \pm 1.96 \times \widehat{SE}(\hat{\beta}_{IV})$.[20] Otherwise, the 95% CI is $\hat{\beta}_{IV} \pm t_{x,y} \times \widehat{SE}(\hat{\beta}_{IV})$. The critical values $t_{x,y}$ are taken from Table 3 in Lee et al. (2020), where x corresponds to the integer part of $\sqrt{F}$, and y corresponds to the decimal part of $\sqrt{F}$. For example, if F is 69, then $\sqrt{F}$ is 8.3, and the critical value $t_{8,3}$ at the 5% significance level is 2.06 instead of 1.96.

The exogeneity assumption (**IV2**) has two components: independence and exclusion (Angrist and Pischke, 2014). As recently emphasized by van Kippersluis and Rietveld (2018a) and van Kippersluis and Rietveld (2018b) in the context of using polygenic scores as instrumental variables, independence is naturally satisfied when genetic variants are used as IV due to Mendelian randomization (Davey Smith and Ebrahim, 2003). However, the exclusion restriction is more difficult to assess. Consider the following equation:

$$\tilde{x} = \alpha + \beta \tilde{y} + \gamma z_y + u. \tag{3}$$

The exclusion restriction is satisfied when $\gamma = 0$, however, this cannot be directly assessed.[21]

To allow for the possibility that the exogeneity condition is violated, $\gamma \neq 0$, and that the husband's polygenic score for education affects wife's education above and beyond husband's education, we follow Conley et al. (2012) and implement "plausibly exogenous" estimation as carefully explained by Clarke and Matta (2018). The standard exogeneity assumption (**IV2**) requires γ to be zero in equation (3). However, when invoking "plausible exogeneity" we replace this assumption with the assumption that γ is close to, but not necessarily equal to, 0.[22] We can then compute $\beta(\gamma)$ as follows:

$$\beta(\gamma) = \frac{Cov(\tilde{x}, z_y)}{Cov(\tilde{y}, z_y)} - \frac{\gamma}{\frac{Cov(\tilde{y}, z_y)}{V(z_y)}},$$

so that $\beta(\gamma)$ equals the IV estimand minus γ divided by the first-stage coefficient. We follow the "union of confidence interval" (UCI) approach, which consists in finding bounds for the IV when the exclusion restriction is violated for a range of values of γ. We apply the same method to find bounds for the causal effect of wife's education on husband's education.

4. Data description

The data used in this paper come from the Health and Retirement Study (HRS), a national panel survey representative of Americans over

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[20] $\widehat{SE}(\hat{\beta}_{IV})$ is the robust standard error of $\hat{\beta}_{IV}$.

[21] Note that running a regression of $\tilde{x}$ on a constant, $\tilde{y}$ and z_y will not help us in testing whether $\gamma = 0$, since these regression estimates will suffer from collider bias. Rohrer (2018) and Cunningham (2021) offer excellent discussions and examples of collider bias.

[22] van Kippersluis and Rietveld (2018a) and van Kippersluis and Rietveld (2018b) suggests finding an estimate of the direct effect γ based on the reduced-form effect of the instrument for a sample with a zero first-stage. Their approach allows to exploit Mendelian randomization which is pleiotropy-robust. The approach consists in using the estimate of γ (if we reject that $\gamma = 0$) as an input for the plausibly exogenous approach. Their 'beyond plausible exogenous' approach relies on two assumptions: (1) the coexistence of heterogeneous first-stage effects with homogeneous direct effects across the zero-first-stage group and the full sample, and (2) the selection into the zero-first-stage subgroup should not be driven by the husband's (resp. wife's) EA PGS and wife's (resp. husband's) education.

the age of 50 and their spouses, interviewed every two years since 1992.[23] The survey contains detailed socio-demographic information. It consists of six cohorts: initial HRS cohort, born between 1931 and 1941 (first interviewed in 1992); the Study of Assets and Health Dynamics Among the Oldest Old (AHEAD) cohort, born before 1924 (first interviewed in 1993); Children of Depression (CODA) cohort, born between 1924 and 1930 (first interviewed in 1998); War Baby (WB) cohort, born between 1942 and 1947 (first interviewed in 1998); Early Baby Boomer (EBB) cohort, born between 1948 and 1953 (first interviewed in 2004); and Mid Baby Boomer (MBB) cohort, born between 1954 and 1959 (first interviewed in 2010).

Between 2006 and 2012, the HRS genotyped about 20,000 respondents who provided DNA samples and signed consent. DNA samples were genotyped using the Illumina Human Omni-2.5 Quad BeadChip, with coverage of approximately 2.5 million single nucleotide polymorphisms (SNPs). Current genetic data available for research also include imputation of approximately 21 million DNA variants from the 1000Genomes Project.[24] Following recommendations of the genotyping center, we removed individuals with a genotyping rate <95% and SNPs with minor allele frequency (MAF) less than 1%, with p-value < 0.0001 on the test for Hardy-Weinberg equilibrium, and with missing call rate greater than 5%. The resulting genetic sample includes 15,445 individuals and information for 8,391,857 genetic variants.

The survey interviews the respondents of eligible birth years repeatedly, as well as their married spouses or partners regardless of age. Since we are interested in couples rather than in the longitudinal structure of the data, we build a cross-section. We include any individual interviewed at least once. The original sample (RAND HRS Data) contains 37,319 individuals: We focus on individuals for which the genetic data are available after the quality control described above, 15,445 in total, excluding 21,874 respondents from the original survey. We restrict the sample to only White respondents, also excluding Hispanics. We consider only heterosexual couples at their first marriage. In particular, we exclude never married partners, people who are divorced or widowed at the time of the first interview, and people who have been already married or widowed more than once when entering the survey. We also drop respondents whose spouse has never been interviewed, couples where the spousal age gap is ten years or more, couples in which at least one of the two spouses has zero years of education, and those in which at least one of the two spouses was born outside the US or born in the US but with missing census division of origin.[25] We also exclude individuals born before 1920 who might have been exposed to the Spanish Flu and born after 1959 which is the end of the last HRS cohort (Mid Baby Boomer). This yields a working sample of 1,562 couples (3,124 individuals).

The main variables used in our empirical analysis are education (measured as the number of completed years of schooling) and the polygenic scores for education (EA PGSs). As previously discussed, we generated a polygenic score based on the most recent GWAS results on educational attainment available (Lee et al., 2018). Since the HRS was part of the educational attainment consortium, we obtained the list of association results calculated excluding the HRS from the meta-analysis from the Social Science Genetic Association Consortium.[26] Using these

summary statistics, we constructed linear polygenic scores weighted for their effect sizes in the meta-analysis. We constructed the scores using the software LDpred and PLINK (Vilhjálmsson et al., 2015).[27]

Table 1 provides the basic descriptive statistics for our sample of husbands and wives. These individuals were born between 1920 and 1959. On average, husbands –with 13.6 years of education– are more educated than their wives –with 13.3 years of education. If we compare our sample of husbands and wives with its non-genotyped counterpart (N=2,468), i.e., where at least one of the spouses has not been genotyped, we find that our sample is on average about three years younger, and more educated (0.51 years more for wives, 0.79 years more for husbands). This is consistent with Barth et al. (2020). See Tables A1 and A2 in the online appendix.

Fig. 1 presents a scatter plot of years of education for both spouses, with both the conditional mean function of wife's education given husband's education and the linear prediction of wife's education given husband's education. Except for the 7 couples (0.45% of the sample) where the husband has 5 years of education or fewer, the linear regression closely matches the conditional mean function.

In Table 2, panel A, we present some correlates of years of education and educational attainment polygenic scores (EA PGSs). There is a strong positive linear relationship between spousal years of education (0.5647, p-value<0.0001), consistent with positive assortative mating in education, and confirming the relationship displayed in Fig. 1. We also

Table 1
Descriptive statistics.

	Mean	SD	Min	Max
Husband's Year of Birth	1937.892	9.043	1920	1959
Husband's Years of Education	13.618	2.686	2	17
Husband's EA PGS	0.000	1.000	−3.172	3.418
Wife's Year of Birth	1940.12	8.92	1920	1959
Wife's Years of Education	13.321	2.195	3	17
Wife's EA PGS	−0.000	1.000	−3.181	2.875

Note: The number of observations is 1,562. The descriptive statistics are based on white non-Hispanic couples in their first marriage, with at most 10 years of age difference and born in the US. Individuals born between 1920 and 1959. Both spouses have been interviewed at least once and provided DNA sample.
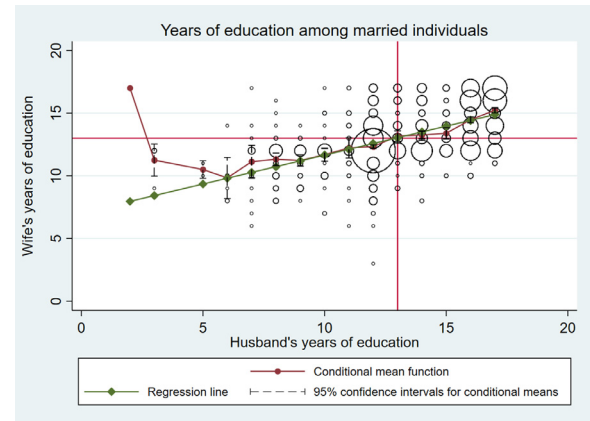

Fig. 1. Scatterplot of years of education among married individuals. Note: The size of each circle is proportional to the number of married couples by years of education of each spouse. The horizontal line denotes the median education for wives. The vertical line denotes the median education for husbands.

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[23] For the non-genetic data, we used the RAND HRS Data files, Version N.

[24] For details on quality control of the HRS genetic data, please see here. Data are available for research via the database of Genotypes and Phenotypes.

[25] Census Divisions are groupings of states and the District of Columbia that are subdivisions of the four census regions (Northeast, Midwest, South, and West). There are nine Census divisions: New England, Mid Atlantic, East North Central, West North Central, South Atlantic, East South Central, West South Central, Mountain, Pacific.

[26] Because of data sharing agreements, results are calculated from association results that exclude also 23andMe from the meta-analysis. Complete genetic association results on educational attainment are available here, see acknowledgments for data conditions.

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[27] Genetic data are based on best call genotypes imputed to 1000 Genome. LD structure is estimated from the HRS genotypic data (only individuals with European ancestry) using a LD window of $M/3000$, where M is the number of included SNPs. The prior used to construct the score assumes that there is a probability $p = 1$ that a SNP has a non-zero association.

Table 2
Correlations and contingency tables for education and educational attainment polygenic scores.

Panel A. Correlations	Husband's Education	Wife's Education	Husband's EA PGS	Wife's EA PGS	
Husband's Education	1				
Wife's Education	0.5647	1			
	[<0.0001]				
Husband's EA PGS	0.2648	0.1867	1		
	[<0.0001]	[<0.0001]			
Wife's EA PGS	0.1986	0.2881	0.1319	1	
	[<0.0001]	[<0.0001]	[<0.0001]		
Correlation between Adjusted Wife's EA PGS and Adjusted Husband's EA PGS					0.0496
					[0.0498]

Panel B. Contingency table for education, conditional probability of husband's education

	Wife's Low Education	Wife's High Education
Husband's Low Education	82.17	17.83
Husband's High Education	36.96	63.04
Pearson test	$\chi^2(1) = 332.1$	
	[<0.001]	

Panel C. Contingency table for EA PGSs, conditional probability of husband's EA PGSs

	Unadjusted		*Adjusted*	
	Wife's Low EA PGS	Wife's High EA PGS	Wife's Low EA PGS	Wife's High EA PGS
Husband's Low EA PGS	54.03	45.97	50.83	49.17
Husband's High EA PGS	45.97	54.03	49.17	50.83
Pearson test	$\chi^2(1) = 10.2$		$\chi^2(1) = 0.4328$	
	[0.001]		[0.511]	

Note: In Panel A adjusted husband's (wife's) EA PGS is the residual from a regression of the husband's (wife's) EA PGS on husband's (wife's) years of education and 10 principal components of the husband's (wife's) genetic data. In Panels B and C: low is defined as below the median and high is defined as above the median; Each cell reports the conditional probability of husband's education (EA PGS) given his wife's education (EA PGS). The row probabilities sum to 100. Adjusted conditional probabilities are based on the residual EA PGSs. p-values are reported in brackets.

find some evidence, albeit weaker, of assortative mating on the EA PGS (0.1319, p-value<0.0001). The correlation in EA PGSs is less than one quarter of the correlation in years of education. Moreover, the correlation between adjusted EA PGSs, obtained after regressing the spousal EA PGS on spousal years of education, and the 10 principal components of the spousal genetic data to account for population stratification (Beauchamp et al., 2011)[28], is much smaller (0.0496, p-value=0.0498). The motivation for this statistical decomposition is the following: If the correlation between polygenic scores of spouses reflects the correlation between educational attainment of spouses rather than the correlation between other spousal characteristics uncorrelated with education, we should expect this correlation to be zero except for sampling variation. While the correlation is not zero, it is substantially smaller than 0.1319.

In panel B, we present some tabulations to explore assortative mating in both years of education and EA PGSs, both above and below the median. The tabulations for education in the first row indicate that the probability of a husband being low educated if the wife is low educated is 82.2%, while the probability of a husband being low educated if the wife is high educated is 17.8%. The second row reveals that the probability of a husband being highly educated if the wife is highly educated is 63% while the probability of him being high educated if the wife is

low educated is 37%. This again reveals the presence of positive assortative mating on education ($\chi^2(1) = 332.1$, p-value<0.0001). Indeed, the main diagonal (low-low, high-high) contains 73% of couples, while under random educational matching we would expect 50% of couples in the main diagonal.

Finally, in Panel C, the tabulations for (unadjusted) EA PGSs reveal some degree of assortative mating ($\chi^2(1) = 10.2$, p-value=0.001), but much lower than that for education. Indeed, the probability of a husband having a low (high) EA PGS if the wife has a low (high) EA PGS is 54%, while the probability of a husband having a high (low) EA PGS if the wife has a low (high) EA PGS is 46%. The main diagonal (low-low, high-high) contains 54% of couples, while under random score matching we would expect 50% of couples in the main diagonal. Focusing on the adjusted EA PGSs, the tabulation reveals that the main diagonal contains 51% of couples, and that we cannot reject that spousal EA PGSs are independent ($\chi^2(1) = 0.4328$, p-value=0.511). Our findings are consistent with Barth et al. (2020), who fail to reject the null hypothesis of random sorting in EA PGSs. Once again, this finding is consistent with the idea that the spousal genetic relationship should disappear once we focus on the part of the polygenic scores uncorrelated with educational attainment, if the relationship between spousal polygenic scores reflects the relationship between spousal educational attainments.

Interestingly, we find similar correlations and contingency tables for education among the non-genotyped respondents. The correlation is 0.6140 (p-value<0.0001) and the entries in the contingency table are also very similar or essentially the same for the conditional probability of husband's low education. See Table A3 in the online appendix.

5. OLS And IV estimates

5.1. OLS Estimates of the matching equation

We first present our OLS estimates of equation (2) in Table 3, in column 1, and additional versions of it with control variables, in columns 2–5. Column 1 shows that an additional year in husband's education is positively associated with an average increase of 0.461 (95% CI: 0.422, 0.501) in the number of years of wife's education, and that 32% of the

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[28] Population stratification refers to the situation in which there is a systematic relationship between the allele frequency and the outcome of interest in different subgroups of the population. Genetic similarity is often correlated with geographical proximity. It is possible to control for the non-random distribution of genes across populations and account for differences in genetic structures within populations in three ways. First, genome-wide analysis should be based on ethnic *homogeneous* populations, for example restricting the analysis to individuals of European ancestry or controlling for geographical origin. Second, only *unrelated* individuals should be included in the analysis to avoid family structure or cryptic relatedness. Last, population structure can be approximated by running a *principal components analysis* (PCA) on the entire genotype and using the principal components as control variables in the analysis (Beauchamp et al., 2011; Price et al., 2006). PCA is the most common method used to control for population stratification in a GWAS. In our application, the first ten genetic principal components for each spouse using genome-wide principal components function as ancestry markers.

Table 3
OLS estimates of the matching function.

	Dependent variable: Wife's Education				
	(1)	(2)	(3)	(4)	(5)
Husband's Education	0.461	0.448	0.432	0.422	0.407
	(0.422, 0.501)	(0.407, 0.489)	(0.391, 0.472)	(0.382, 0.463)	(0.366, 0.449)
Wife's EA PGS			0.402	0.367	0.383
			(0.313, 0.491)	(0.277, 0.458)	(0.292, 0.475)
Observations	1,562	1,562	1,562	1,562	1,562
R-squared	0.319	0.337	0.351	0.368	0.387
PCAs	No	No	No	Yes	Yes
Demographics	No	Yes	No	No	Yes

Note: 95% confidence intervals based on robust standard errors in parentheses. PCAs: 10 first principal components of the husband's and the wife's genetic data. Demographics: year of birth of the wife, year of birth of the husband, 8 indicators of the wife's region (Census division) of birth and 8 indicators of the husband's region (Census division) of birth.

variation in wife's education is explained by husband's education. This positive correlation is consistent with previous research (e.g., see Table 7 in Chiappori et al., 2018). In column 2 we add demographic controls (i.e., year of birth of each spouse, and 8 indicators for each spouse region (Census division) of birth). The estimated coefficient remains very similar, 0.448 (95% CI: 0.407, 0.489), and the explanatory power increases from 32% to 34%.

From column 3 to column 5 we use genetic information. In column 3 we add the wife's EA PGS for education, without any other control variables, and obtain a similar coefficient for husband's education, 0.432 (95% CI: 0.391, 0.472), and a higher explanatory power, 35%. A one standard deviation increase in the wife's EA PGS is associated with an increase in the wife's educational attainment of 0.402 years (95% CI: 0.313, 0.491). In column 4 we account for population stratification adding the wife's and husband's ten first principal components of the principal component analysis to genotypic data: the coefficient on husband's education is estimated at 0.422 (95% CI: 0.382, 0.463), and the one on the wife's EA PGS at 0.367 (95% CI: 0.277, 0.458). Finally, in column 5, we look at the relationship between wife's and husband's education netting out the influence of the wife's EA PGS, population stratification and demographic controls. Our results indicate that an additional year in husband's education is associated with an average increase of 0.407 (95% CI: 0.366, 0.449) in the number of years of wife's education.

Table A4 in the online appendix contains the same OLS analysis for the regressions of husband's education on wife's education: the OLS point estimates range from 0.691 (95% CI: 0.640, 0.742), in column 1, to 0.626 (95% CI: 0.573, 0.679), in column 5.

5.2. OLS Estimates of first-stage and reduced-form equations

Table 4 contains the estimates of the first-stage regression equation, where husband's education is regressed against husband's EA PGS (our plausible exogenous instrument). In the first three columns, we do not adjust for the wife's EA PGS. Column 1, which does not include any controls, shows that a one standard deviation increase in the husband's EA PGS is associated with an average increase in husband's education of 0.711 years (95% CI: 0.585, 0.837). When accounting for population stratification (column 2), the magnitude decreases to 0.661 (95% CI: 0.532, 0.790), and after adding demographic characteristics to the population stratification controls (column 3), our point estimate is 0.678 (95% CI: 0.551, 0.805).

When focusing on columns 4–6, which adjust for the wife's EA PGS, we find that a one standard deviation increase in the husband's EA PGS is associated with an average increase in husband's education of 0.652 years (95% CI: 0.527, 0.777), column 4. Assuming that polygenic scores are as good as randomly assigned, at least after accounting for population stratification, column 5 shows a substantial causal effect of

polygenic scores on educational attainment: a one standard deviation increase in husband's EA PGS (resp. wife's EA PGS) increases average husband's education by 0.622 years — 95% CI: 0.494, 0.750 (resp. 0.407 — 95% CI: 0.280, 0.534). Finally, column 6 shows similar magnitudes, 0.642 — 95% CI: 0.516, 0.768 (resp. 0.418 — 95% CI: 0.293, 0.543) netting out the influence of demographic characteristics.

The last row in Table 4 reports the first-stage F statistic on the instrument. They are well above and beyond the critical values in Stock and Yogo (2005). Moreover, following Lee et al. (2020), IV inference will be dependent on the first-stage F statistic. In particular, while the critical value for a 95% IV confidence interval is 1.96 for columns (1), (3) and (4), since each has an F greater than 104.7, the critical values are 1.98 for columns (2) and (6), and 1.99 for column (5), based on Table 3 in Lee et al. (2020).[29]

Table A5 in the online appendix contains the OLS first-stage regressions of wife's education on wife's EA PGS. If anything, the instrument appears to be stronger. Indeed, based on Lee et al. (2020), all the critical values for a 95% IV confidence interval are 1.96, except for column (5), which is 1.98.

In Table 5 we turn to the reduced-form estimates, where wife's education is regressed against husband's EA PGS (our plausible exogenous instrument). As in Table 4, we present two set of estimates, without adjusting (columns 1–3) and adjusting for the wife's EA PGS (columns 4–6). Column 1, which does not include any controls, shows that a one standard deviation increase in the husband's EA PGS is associated with an average increase in wife's education of 0.410 years (95% CI: 0.306, 0.513). After adding population stratification controls (column 2), our point estimate decreases to 0.345 (95% CI: 0.240, 0.450), and after including demographic characteristics in addition to the controls for population stratification (column 3), our point estimate becomes 0.360 (95% CI: 0.257, 0.464).

We then shift our attention to reduced-form estimates adjusted for the wife's EA PGS (columns 4–6). Column 4 shows that a one standard deviation increase in husband's EA PGS (resp. wife's EA PGS) is associated with an average increase in wife's education of 0.332 years — 95% CI: 0.232, 0.432— (resp. 0.588 — 95% CI: 0.486, 0.691). In column 5 we assume conditional random assignment of polygenic scores, after accounting for population stratification, and find that a one standard deviation increase in husband's (resp. wife's) EA PGS increases wife's education by 0.294 years — 95% CI: 0.191, 0.397 (resp. 0.536 — 95% CI: 0.432, 0.640). Similar effects are found after netting out the influence of demographic characteristics, 0.313 — 95% CI: 0.213, 0.414 (resp. 0.549 — 95% CI: 0.446, 0.652).

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[29] Note that Table 3 in Lee et al. (2020) reports critical values up to an F of 99.99, with a corresponding critical value of 1.98. Thus, we use 1.98 as the critical value for any F between 100 and 104.7.

Table 4
OLS estimates of the first stage.

Dependent variable: Husband's Education						
	(1)	(2)	(3)	(4)	(5)	(6)
Husband's EA PGS	0.711	0.661	0.678	0.652	0.622	0.642
	(0.585, 0.837)	(0.532, 0.790)	(0.551, 0.805)	(0.527, 0.777)	(0.494, 0.750)	(0.516, 0.768)
Wife's EA PGS				0.447	0.407	0.418
				(0.324, 0.571)	(0.280, 0.534)	(0.293, 0.543)
Observations	1,562	1,562	1,562	1,562	1,562	1,562
R-squared	0.070	0.106	0.144	0.097	0.128	0.166
PCAs	No	Yes	Yes	No	Yes	Yes
Demographics	No	No	Yes	No	No	Yes
First-stage F statistic	122.816	100.857	109.241	105.226	91.265	99.920

Note: 95% confidence intervals based on robust standard errors in parentheses. PCAs: 10 first principal components of the husband's and the wife's genetic data. Demographics: year of birth of the wife, year of birth of the husband, 8 indicators of the wife's region (Census division) of birth and 8 indicators of the husband's region (Census division) of birth. First-stage F statistic is the statistic of the test of the coefficient on Husband's EA PGS being zero.

Table 5
OLS estimates of the reduced form.

Dependent variable: Wife's Education						
	(1)	(2)	(3)	(4)	(5)	(6)
Husband's EA PGS	0.410	0.345	0.360	0.332	0.294	0.313
	(0.306, 0.513)	(0.240, 0.450)	(0.257, 0.464)	(0.232, 0.432)	(0.191, 0.397)	(0.213, 0.414)
Wife's EA PGS				0.588	0.536	0.549
				(0.486, 0.691)	(0.432, 0.640)	(0.446, 0.652)
Observations	1,562	1,562	1,562	1,562	1,562	1,562
R-squared	0.035	0.083	0.127	0.106	0.139	0.185
PCAs	No	Yes	Yes	No	Yes	Yes
Demographics	No	No	Yes	No	No	Yes

Note: 95% confidence intervals based on robust standard errors in parentheses. PCAs: 10 first principal components of the husband's and the wife's genetic data. Demographics: year of birth of the wife, year of birth of the husband, 8 indicators of the wife's region (Census division) of birth and 8 indicators of the husband's region (Census division) of birth.

Table 6
IV (2SLS) estimates of the matching function.

Dependent variable: Wife's Education						
	(1)	(2)	(3)	(4)	(5)	(6)
Husband's Education	0.576	0.522	0.531	0.509	0.473	0.488
	(0.452, 0.700)	(0.388, 0.656)	(0.402, 0.661)	(0.378, 0.640)	(0.332, 0.613)	(0.352, 0.624)
		[1.98]			[1.99]	[1.98]
Wife's EA PGS				0.361	0.344	0.345
				(0.249, 0.472)	(0.234, 0.454)	(0.236, 0.455)
Observations	1,562	1,562	1,562	1,562	1,562	1,562
PCAs	No	Yes	Yes	No	Yes	Yes
Demographics	No	No	Yes	No	No	Yes

Note: 95% confidence intervals based on robust standard errors in parentheses. The confidence intervals in *italics* are constructed using either 1.96 or the critical values in Table 3 of Lee et al. (2020). When using the critical values in Lee et al. (2020), these are reported in brackets. PCAs: 10 first principal components of the husband's and the wife's genetic data. Demographics: year of birth of the wife, year of birth of the husband, 8 indicators of the wife's region (Census division) of birth and 8 indicators of the husband's region (Census division) of birth.

Table A6 in the online appendix contains the reduced-form estimates of the effects of the polygenic scores on husband's years of education.

5.3. IV Estimates of the matching equation

If our instruments are *valid* (i.e., relevant and exogenous), the causal effect of husband's education on wife's education is given by the reduced-form coefficient on the husband's EA PGS inflated (divided) by the first-stage coefficient on the husband's EA PGS. Looking at the regression conditional on the wife's EA PGS estimates in Tables 4 and 5, columns 4–6, these ratios are 0.509 (column 4 divided by column 1), 0.473 (column 5 divided by column 2) and 0.488 (column 6 divided by column 3), which are well-known to be numerically equivalent to the IV point estimates using 2SLS displayed in Table 6. An additional year in

husband's education increases average wife's educational attainment by about 0.5 years: in words, and in our matching context, this means that if we take two men, who are observationally equivalent in the marriage market, and we increase the educational attainment of one of them by one more year of education, the one with higher education will be expected to have a wife with about half a year more of education. Note that the 95% CIs are based on robust standard errors and the critical value 1.96 when $F > 104.7$, or the corresponding critical value in Table 3 of Lee et al. (2020) when $F < 104.7$.

The IV estimates of the causal effect of wife's education on husbands' education are reported in Table A7 in the online appendix. Again, assuming that our instrument is valid, an additional year in wife's education increases average husband's educational attainment by about 0.76

Table 7

Comparison of estimates of education on spousal education.

	(1)	(2)	(3)
Panel A. Causal effect on Wife's Education			
OLS 95% CI	(0.391, 0.472)	(0.382, 0.463)	(0.366, 0.449)
IV (2SLS) 95% CI	(0.378, 0.640)	(0.332, 0.613)	(0.352, 0.624)
IV (UCI) 95% bounds with $\gamma \in [0, 0.2]$	(0.065, 0.640)	(-0.000, 0.613)	(0.035, 0.624)
Panel B. Causal effect on Husband's Education			
OLS 95% CI	(0.601, 0.706)	(0.590, 0.697)	(0.573, 0.679)
IV (2SLS) 95% CI	(0.579, 0.942)	(0.553, 0.966)	(0.562, 0.961)
IV (UCI) 95% bounds with $\gamma \in [0, 0.2]$	(0.237, 0.942)	(0.177, 0.966)	(0.197, 0.961)
PCAs	No	Yes	Yes
Demographics	No	No	Yes

Note: In panel A, all regressions adjust for the wife's EA PGS in panel A. In panel B, all regressions adjust for the husband's EA PGS. IV bounds are based on the approach developed by Conley et al. (2012) and estimated using the plausexog Stata command as described by Clarke and Matta (2018): IV (UCI: Union of Confidence Interval) approach consists in finding bounds for the IV when the exclusion restriction is violated ($\gamma \neq 0$) by choosing a range of values for γ, in our case, with a minimum of 0 and a maximum of 0.2. 5% significance level is obtained using either 1.96 or the critical values in Table 3 of Lee et al. (2020).

years: this means that if we take two women, who are observationally equivalent in the marriage market, and we increase the educational attainment of one of them by one more year of education, the one with higher education will be expected to have a husband with about three quarters of a year more of education.

5.4. Plausible exogenous IV estimates

As discussed in the introduction, polygenic scores for spousal education may affect own education above and beyond their effects on spousal education, and hence violate the exclusion restriction. In this subsection, we relax the exclusion restriction following the "union of confidence interval" (UCI) approach developed by Conley et al. (2012). The UCI approach consists in finding bounds for the IV for a range of values of γ.

Table 7 compares the 95% CI intervals/bounds for OLS, IV and UCI estimates for the causal effects of education on wife's education (panel A) and husband's education (panel B). Both OLS and IV estimates generate a similar 95% lower bound in both panels. Since the minimum value for γ is set at 0, the 95% upper bounds in the IV and the UCI cases are exactly the same by construction. The bite of the UCI approach comes from the upper bound, $\gamma = 0.2$. In that case, we can see that the 95% lower bound ranges from - 0.000 (column 2) to 0.065 (column 1), in panel A, and from 0.177 (column 2) to 0.237 (column 1), in panel B.

Our interpretation of Table 7 is that we need to have a sufficiently large and positive γ, $\gamma \geq 0.2$, to rule out a positive causal effect of husband's education on wife's education. In words, a one standard deviation in husband's EA PGS must directly increase wife's education in 0.2 or more years to nullify the causal effect of husband's education on wife's education.[30] A similar, even stronger, argument can be made for the causal effect of wife's education on husband's education.[31]

Figs. 2 and 3 summarize the findings of Table 7 and the key findings of our paper.

Figures A1 (resp. Figure A2) in the online appendix displays the UCI 95% range of lower bounds for column 3 and panel A (resp. panel B) in Table 7. For completeness, we also present Figure A3 (resp. Figure A4) in the online appendix, which displays the UCI 95% bounds analysis for column 3 in Table 7 and panel A (resp. panel B) when $\gamma \in [-0.2, 0.2]$.
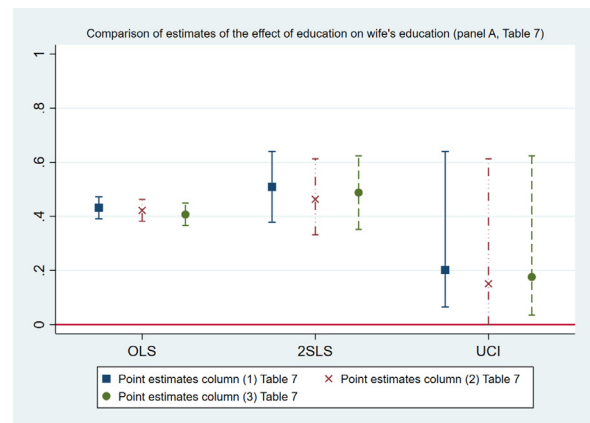

Fig. 2. Comparison of estimates of the effect of education on wife's education. Note: Point estimates and 95% bounds/confidence intervals. UCI with $\gamma \in [0, 0.2]$ See footer of Table 7. The horizontal line denotes a zero causal effect.
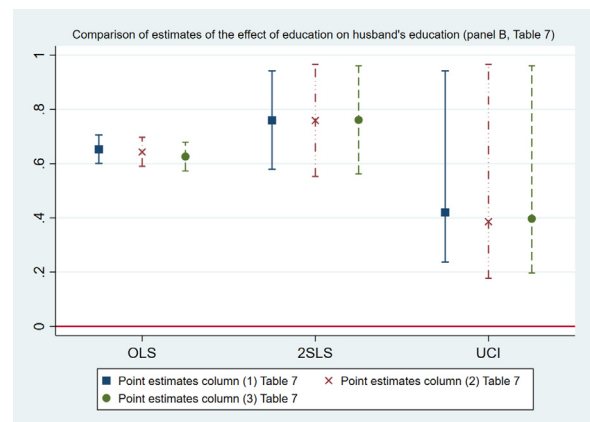

Fig. 3. Comparison of estimates of the effect of education on husband's education. Note: See footer of Fig. 2.

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[30] Note that Table 5 shows that a one standard deviation increase in husband's PGS is associated with an increase of 0.313 (95% CI: 0.213, 0.414) in the years of schooling of the wife, column 6. Thus, 0.2 seems a relevant magnitude at first glance. Moreover, the estimate of β when $\gamma = 0.2$ is 0.176 ($= \frac{0.313}{0.642} - \frac{0.2}{0.642}$). This means that when $\gamma = 0.2$, 43% of the OLS estimate (0.407) or 36% of the IV estimate (0.488) reflects a causal effect.

[31] Note that the lower bounds in panel B are much larger than in panel A.

Our results are very similar using OLS and IV, and the bounds analysis suggests that, for mild violations of the exclusion restriction, $\gamma < 0.2$, our IV findings are able to reveal a positive causal effect of education on spousal education.

5.5. Robustness check: Conditional Mendelian randomization

One concern of our previous IV analysis is that the independence assumption is satisfied only within families. For this reason, and inspired by Ronda et al. (2020), we also run our IV analysis conditioning on family characteristics. In particular, we use education of the mother and education of the father for both spouses. Reassuringly, our IV findings are robust to controlling for parental education.

Intuitively, given the addition of parental education of both spouses, the violation of the exclusion restriction needs to be a bit milder, $\gamma < 0.1$. However, the addition of these controls in our matching context can open endogeneity channels. Moreover, adding these controls makes our sample size decrease from 1,562 to 1,286 observations, a reduction in sample size of almost 20%. For these reasons, these findings are presented as a robustness check in Table A8 in the online appendix and must be interpreted with caution.

6. Conclusions

This is the first paper to present a genetic-IV strategy to estimate the causal effect of education on spousal education. Our IV (2SLS) estimates suggest that an additional year in husband's (resp. wife's) education increases wife's (resp. husband's) education by about 0.5 (resp. 0.75) years. Even if the husband's (resp. wife's) educational attainment polygenic score (EA PGS) has a direct effect on wife's (resp. husband's) education over and above husband's (resp. wife's) education, we cannot rule out a positive effect of husband's (resp. wife's) on wife's (resp. husband's) education, so long as one standard deviation increase in husband's (resp. wife's) EA PGS directly increases wife's (resp. husband's) education by less than 0.2 (resp. 0.3) years.

Of course, our study has limitations, and it is important to acknowledge them and highlight avenues for future research. We have identified three main potential drawbacks. The first two relate to the internal validity of our estimates and the external validity of our findings, respectively. The third one is about the exact mechanism behind the documented positive causal effect of education on spousal education.

First, while we use a sample from a nationally representative survey (HRS), our results refer to an HRS sample of individuals who are on average 70 years old, still alive, who got married on average 40 years ago and have been married to each other ever since. Second, while we allow for the possibility that the exclusion restriction is violated, we assume that the other requirement buried in the exogeneity assumption —the independence assumption— holds due to Mendelian randomization. However, one may argue that Mendelian randomization holds conditional on family fixed effects, but not across families. While our results are robust to controlling for parental education, future work can extend our analysis and address these two limitations by using new datasets where within-sibling variation in polygenic scores can be exploited and investigate whether our results are replicated.

Finally, future research should try to pin down the exact mechanism behind our findings: Do more educated husbands become more likely to encounter potential wives that are more educated? And/or do more educated husbands become more attractive to more educated wives, holding constant the likelihood of meeting an educated spouse? In other words, is the causal effect of education on spousal education documented in this study mainly due to search or preferences? (Bruze, 2011).

Acknowledgements

Supplementary material

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