A Chip Off The Old Block? Genetics and The Intergenerational Transmission of Socioeconomic Status

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Abstract

We study how one generation's genetics affects the SES of the next. We link the Dutch Lifelines Biobank genetic data to annual tax records for 2006-2022 and exploit randomness in the genetic transmission across generations to identify causal genetic effects. Our genetic measure is the polygenic index for educational attainment. An individual's genetics influences their offspring's education, income, and wealth, with effect sizes comparable to leading estimates of family environment. Genetic transmission explains roughly half of these effects. We find no evidence of genotypic assortative mating, but an individual's genetics causes them to select reproductive partners from higher socioeconomic backgrounds.

1 Introduction

Rising economic inequality in many developed countries has made promoting economic mobility a key priority for policy makers. Designing effective policies to address this challenge requires a better understanding of how socioeconomic advantage is transmitted across generations (Conley et al. 2015; Black et al. 2020). Economists and other social scientists have studied extensively the advantages conferred by growing up in favorable environmental conditions, such as having more educated parents (Tsai, Liu, Chou, & Grossman, 2011; Chevalier, Harmon, O'Sullivan, & Walker, 2013; Dickson, Gregg, & Robinson, 2016), attending higher-quality schools, or living in lower-poverty neighborhoods (Chetty, Hendren, & Katz, 2016). However, we still know far less about the role of genetics in driving the persistence of socioeconomic status (SES) across generations.

How does the genetics of one generation affect the economic prospects of the next? Answering this question presents two key challenges: measurement and identification. The measurement challenge arises because most datasets containing molecular genetic data were designed to study health and contain little or no socioeconomic information. We address this limitation by linking genetic data from the Dutch Lifelines Biobank with administrative longitudinal tax records. These tax records provide detailed annual data on wealth, income, savings, and investments from 2006 to 2022. They cover the entire population of taxpayers in the Netherlands, enabling us to link an individual's genetic data not only to their own tax records but also to those of their family members. As an individual-level measure of one's genetics, we use the polygenic index (PGI) for educational attainment, the most widely studied PGI in social genomics. In our data, this linear index constructed from millions of genetic markers explains approximately 7% of the cross-sectional variation in years of schooling.

The identification challenge requires isolating exogenous variation in genetics, holding one's socioeconomic background constant. Human biology provides a source of exogenous variation. Each person has two copies of every genetic marker. During conception, only

one of a parent's two copies is transmitted to the offspring. It is random which copy is transmitted. The practical implication is that, once we condition on the PGIs of one's parents, the remaining variation in one's PGI is random. To demonstrate it, we show that, while one's PGI is associated with the SES of one's parents, this correlation disappears once we condition on the PGIs of the parents.

The starting point for our analysis is what we call "the reference"—the Lifelines participant whose genetic variation we study. We leverage the randomness in the genetic transmission from the reference's parents to the reference to identify the causal effects of the reference's genetics on their offspring's SES. In practice, we regress the offspring's SES on the reference's PGI while controlling for the sum of the PGIs of the reference's parents. We also estimate the causal effects of the reference's PGI on their own SES by regressing the reference's SES on their PGI while controlling for the sum of the PGIs of their parents. To implement this empirical strategy, we link the genetic data of the reference and their parents to the tax records of both the reference and their offspring. Our main sample consists of about 7,000 individuals in the "reference generation" and 14,000 in the "offspring generation", with the latter restricted to individuals aged 30 or older in 2022. These samples yield about 62,000 and 119,000 annual observations, respectively.

We find that the genetics of one generation causally impacts the education, income, and wealth of the next. For example, increasing the reference's PGI rank by 10 percentiles raises their offspring's education by 0.12 years of schooling–or 0.18 years when adjusting for measurement error in the PGI. This effect size is comparable to some of the most credible estimates of the causal impact of family environment, which leverage the quasi-random assignment of adopted children into adoptive families. For instance, Black et al. (2020) estimate that increasing an adoptive parent's net wealth rank by 10 percentiles raises the education of their adopted child's education by 0.1 years of schooling. Moreover, we find that the impact of the reference's PGI on their offspring's SES is not much smaller than its effects on their own SES. To illustrate, increasing the reference's PGI rank by 10 percentiles

raises their own education by 0.16 years of schooling. This suggests that the effects of a shock to one generation's genetics are strongly persistent across generations.

We next examine the channels through which the genetics of one generation may influence the economic prospects of the next, distinguishing between three key mechanisms. First, a person's genetics can indirectly influence their offspring's SES through purely environmental pathways, a process we refer to as *genetic nurture*. Even genetic variants that a person does not pass down to their offspring may still affect their offspring's SES by influencing the person's characteristics and behaviors, which in turn shape how their children are raised. Second, the genetic variants inherited by the offspring direct influence their SES through genetic effects, a process we refer to as the *genetic transmission*.Third, a person's genetics may affect their offspring's SES through assortative mating. If a person's genetics influences their choice of reproductive partner, this can impact the offspring's SES in two ways: (1) the offspring inherits half of their genetics from their other biological parent and (2) the characteristics and behaviors of this other biological parent also shape the offspring's upbringing.

We find that generally genetic transmission accounts for about half of the effect of one generation's PGI on the SES of the next, implying that the remaining 50% must be driven by genetic nurture and assortative mating. However, there are two notable exceptions. First, genetic transmission explains only a third of the effect of one generation's genetics on the next generation's housing assets and on broader wealth measures that include housing assets, such as total assets and net wealth. This finding is consistent with housing policies in the Netherlands that previously enabled higher-SES parents to financially assist their children in purchasing their first homes. Second, genetic transmission appears to account for approximately 80% of the effects on the next generation's individual earnings and individual income. We speculate that this is because, while higher-SES parents can give their children a leg up in educational attainment and homeownership, they may have less direct influence over their offspring's success in the labor market. Overall, these findings suggest that by focusing

primarily on genetic transmission, the economics literature may have underestimated the broader role of genetics in the intergenerational transmission of SES.

Finally, we examine the role of assortative mating. We find that an exogenous increase in a person's PGI for educational attainment does not lead them to select a partner with a higher PGI. This suggests that, at least with respect to genetic predisposition for educational attainment, we find no evidence of assortative mating. Nevertheless, we find that a person's genetics influences their choice of a reproductive partner from a higher socioeconomic background. Specifically, an exogenous increase in a person's PGI leads them to select partners who are more educated and whose parents have higher income and wealth. These findings reinforce our earlier results, suggesting that a substantial portion of the effects of an individual's genetics on the next generation operate indirectly through the environment in which the offspring are raised—an environment shaped not only by the individual's genetically-influenced characteristics and behaviors but also molded by the reproductive partner they choose.

This paper contributes to the extensive economics literature on the intergenerational transmission of SES (Bowles & Gintis, 2002; Björklund, Lindahl, & Plug, 2006; Sacerdote, 2007; Black, Devereux, Lundborg, & Majlesi, 2020; Fagereng, Mogstad, & Rønning, 2021; Beauchamp, Schmitz, McGue, & Lee, 2023). To our knowledge, it is one of the first to use molecular genetic data to study how the genetics of one generation causally impacts the economic prospects of the next. Most prior studies lacked genetic data and instead inferred genetics' role indirectly by combining model assumptions with data on the similarity between the outcomes of different family members (e.g., adopted child vs. biological parent; dizygotic vs. monozygotic twins; or biological-biological sibling pairs vs. biological-adopted sibling pairs). Compared to these approaches, our estimates have greater generalizability than previous estimates—while previous methods relied on special samples (e.g., twins or adoptees), our approach applies universally. Beyond estimating these effects, this paper contributes by shedding light on the mechanisms through which the genetics of one generation shapes the

economic prospects of the next. The economics literature on genetics and intergenerational SES has focused almost exclusively on the genetic transmission channel. We quantify the relative importance of this channel and provide evidence that genetic nurture and assortative mating also play a significant role.

It also speaks to a growing literature in social genomics that exploitx the exogenous genetic variation to study the causal effects of one's genetics on otheir own SES (Belsky et al., 2018; Muslimova, Van Kippersluis, Rietveld, Von Hinke, & Meddens, 2020; Burik, Kweon, & Koellinger, 2021; Okbay et al., 2022; Barcellos, Carvalho, & Turley, 2021; Houmark, Ronda, & Rosholm, 2024; Carvalho, 2024; Buser, Ahlskog, Johannesson, Koellinger, & Oskarsson, 2024). This paper makes two contributions to this literature. First, it estimate these effects using rich longitudinal tax records with detailed administrative data on both income and wealth. Second, it extends the literature by examining how an individual's genetics affects their offspring's SES and comparing these intergenerational impacts to the effects of the individual's genetics on their own SES.

The rest of the paper is structured as follows. Section 2 describes our data. Section 3 introduces the genetic measure used in the empirical analysis. Section 4 outlines our empirical strategy. Section 5 shows our results. Section 6 explores the mechanisms that drive our results. Section 7 concludes.

2 Data

We merge genetic data from the Dutch Lifelines Biobank with administrative tax records made available by the Dutch tax authorities (Belastingdienst) and Statistics Netherlands (CBS). The tax records include detailed data on both income and wealth for the entire population of the Netherlands. We supplement these data with information on completed education from both surveys and administrative data.

2.1 Dutch tax administrative records

We use data on annual income (before taxes) and wealth for the period between 2006 and 2022.[1] [2] Data on income are available both at the individual and the household level.[3] We also use data on annual individual earnings.[4] There is information on various components of net wealth, all measured at the household level. These include total assets, total debt, financial wealth, housing wealth, business assets, other assets, mortgage debt, and other debt. Financial wealth is further divided into (i) checking and savings balances and (ii) bonds and shares.[5] In some of our analyses, we measure income and wealth in terms of percentile ranks, which are calculated within calendar year and year of birth. When estimating the effects on levels, we winsorize the top and bottom 1% of the data.[6]

2.2 The Dutch Lifelines Biobank

Lifelines is a prospective cohort of 167,729 individuals who, at the time of recruitment, lived in Northern Netherlands (i.e., in the provinces of Drenthe, Friesland and Groningen). The sample size corresponds to about 10% of this region's population. Recruitment occurred between 2006 and 2013 using a two-step procedure (Scholtens et al., 2015). First, general practitioners recruited individuals of ages 25 to 49.[7] These original participants were then encouraged to invite their family members to also enroll in the study (no age restrictions applied to these family members). Because of this recruitment design, Lifelines contains multiple members of the same family, which will be instrumental for our empirical strategy. To date, approximately 78,700 Lifelines participants have been genotyped. The genotypic

[1] Wealth is measured as of January 1st of each year.

[2] Income data are available also for 2003, 2004, and 2005.

[3] Household income is calculated by aggregating the individual incomes of everyone living in the household. CBS provides a household-specific identifier.

[4] Earnings include both labor earnings and self-employed income.

[5] Our measures of wealth exclude pension funds. In the Netherlands, pension funds are not freely transferable and are therefore not considered assets.

[6] Data were deflated using the consumer price index made available by CBS. The base year is 2015.

[7] GPs were asked to exclude the following people from participation: those with terminal illness or severe mental illness, those unable to visit the GP, those unable to fill in questionnaires, or those unable to understand Dutch.

data of 78,038 of them passed the quality control process we implemented to ensure that the genetic data include only well-measured genetic variants.

2.3 Merging Tax Records and Genetic Data

The genetic data of Lifelines participants are merged with administrative data on their income and wealth, as well as the income and wealth of their family members - namely, their parents, adult children, spouse, and parents-in-law. Family members are identified using a parent-child linkage file ("KINDOUDERTAB"), which records the legal parents of each person living in the Netherlands. The term "spouse" is used to refer to a person with whom the Lifelines participant shares legal parenthood (i.e., both are legal parents of the same child) and "parents-in-law" to refer to that person's parents. In the Netherlands, legal parenthood is a reliable predictor for biological parenthood, as adoption is rare (affecting <0.5% of the Dutch population (CBS, 2008, 2010)). Any misclassification would bias our estimates towards zero, as adoptive parents and their adopted children do not share their genetics.

2.4 Educational attainment

Lifelines collected data on participants' completed education. In some of our analyses, we will also need data on the completed education of family members of Lifelines participants. For this reason, we complement the Lifelines self-reported data on education with administrative individual-level data on highest degree completed made available by CBS.[8]

3 Genetic Measure

Humans share over 99% of their genomes. However, they differ at specific loci called *Single nucleotide polymorphisms* (SNPs). While SNPs are not the only type of genetic variation

[8]The CBS information is sourced from various administrative sources and population surveys.

in humans, they are the most common and widely studied. The human genome contains approximately 20 million SNPs. At most SNPs, individuals can have one of two possible genetic variants. In genetic data, one of these variants is arbitrarily designated as the "reference allele". Since each person has two copies of every chromosome, they may carry 0, 1, or 2 copies of the reference allele.

Our genetic measure is a polygenic index ("PGI"). To maximize predictive power, a PGI aggregates information from millions of genetic markers, each with a small influence on the outcome of interest. PGIs are widely used in the social sciences genetics literature (Becker et al., 2021). In practice, a PGI is a weighted sum of the number of reference alleles an individual carries across various SNPs:

$$G_i = \sum_{s}^{S} x_i^s w_s \tag{1}$$

where i indexes an individual and s indexes a SNP. G_i is individual i's PGI, x_i^s is the number of reference alleles (0, 1 or 2) the individual carries at SNP h, and w_s is the weight assigned to SNP s. Intuitively, greater weights are assigned to SNPs that are more strongly associated with the given outcome of interest.

In this paper, we use a PGI constructed to predict educational attainment ("EA"). To construct it, we used the publicly-available weights of Okbay et al. (2022).[9] In practice, this "Genome-Wide Association Study" (GWAS) consisted of a series of regressions – one for each SNP – of years of schooling on the individual's number of reference alleles at that SNP.[10] Roughly speaking, the GWAS weights are the coefficients from these regressions.

[9]Because effect sizes of each individual SNP are typically very small, a large sample is critical for constructing a PGI with high predictive power. Okbay et al.'s GWAS (2022) was originally conducted in a sample of about 3 million individuals. However, their publicly-available GWAS results are based on a smaller subsample of 765,283 individuals that excluded over 2 million participants of 23andMe. This is nevertheless the largest publicly-available GWAS for EA available to date. We standardize the PGI using its cross-sectional standard deviation.

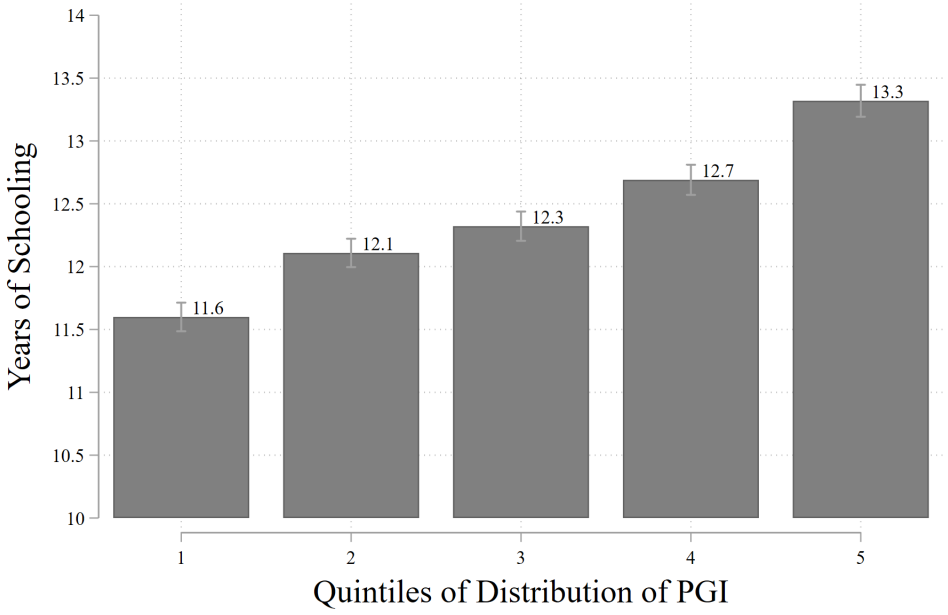
[10]The regressions controlled for sex, age, and the first principal components of the genetic data. The principal components are used to control for population stratification: subgroups in a European ancestry population will have different values of $E[x_{ik}]$ due to different ancestral backgrounds. This could lead to spurious associations between x_{ik} and EA if these different subgroups differ in their EA for environmental reasons (Price et al., 2006).

Following the standard practice in social genomics, the sample is restricted to participants with European genetic ancestries.[11]

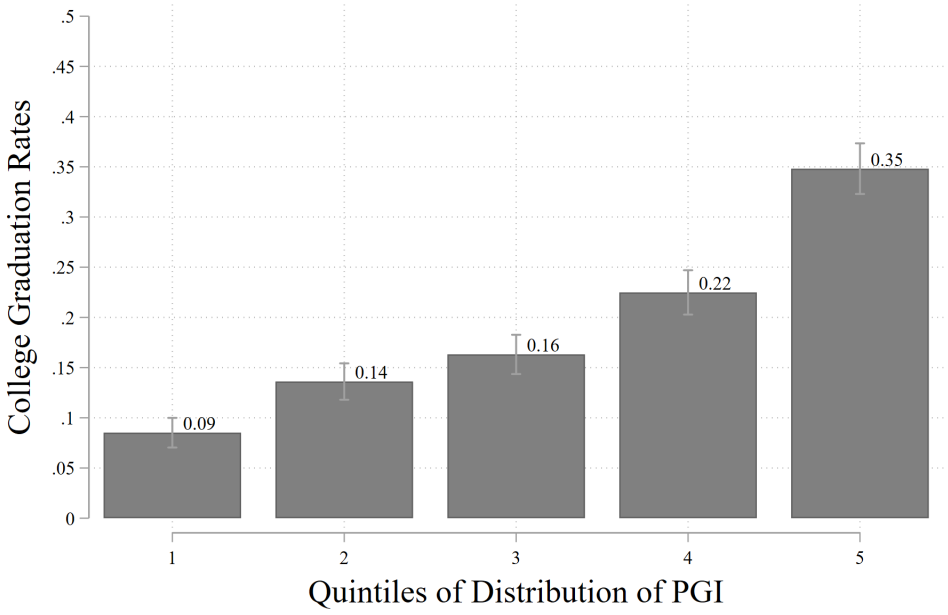
Figure 1 illustrates how the constructed PGI predicts completed education. We divide the sample into quintiles based on the PGI distribution and compute average education separately for each quintile. Figure 1A presents the average years of schooling for each quintile, while Figure 1B displays the fraction of individuals in each quintile who graduated from college. On average, individuals in the top quintile have 1.7 more years of schooling than those in the bottom quintile. They are also 26 percentage points more likely to graduate from college. The PGI explains approximately 7% of the cross-sectional variation in years of schooling and 5% of the variation in college graduation. Figures 1C shows that individuals with higher PGIs have on average more wealth.

However, Figures 1A, 1B, and 1C cannot be interpreted causally, as they confound genetic effects with environmental influences. In particular, the quintiles differ not only in their genetic makeup but also in their socioeconomic backgrounds. Figure 1D shows, for each quintile, the average net wealth (in percentile ranks) *of the previous generation*, rather than the current one. Individuals with higher PGIs tend to come from higher-SES backgrounds. This figure illustrates the longstanding challenge of disentangling genetic effects from environmental influences. In the next section, we discuss how to isolate the former.

[11]We exclude individuals with different genetic ancestries to avoid the *problem of portability:* due to differing *linkage disequilibrium* patterns among groups with different ancestries, constructing a PGI based on a GWAS conducted on a different ancestry group results in large measurement error of this PGI and lower predictiveness (Bitarello & Mathieson, 2020; Privé et al., 2022). The data providers flagged 249 genotyped respondents as being of non-European ancestry, as based on a combination of variables reported by the respondents and the genetic data, assessing ancestry using a principal components analysis.

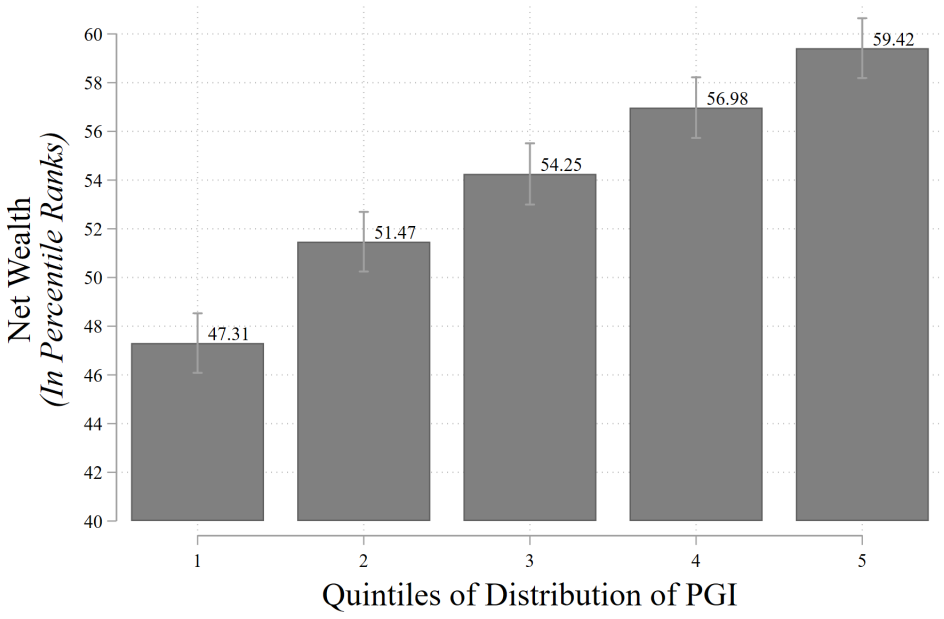


(a)

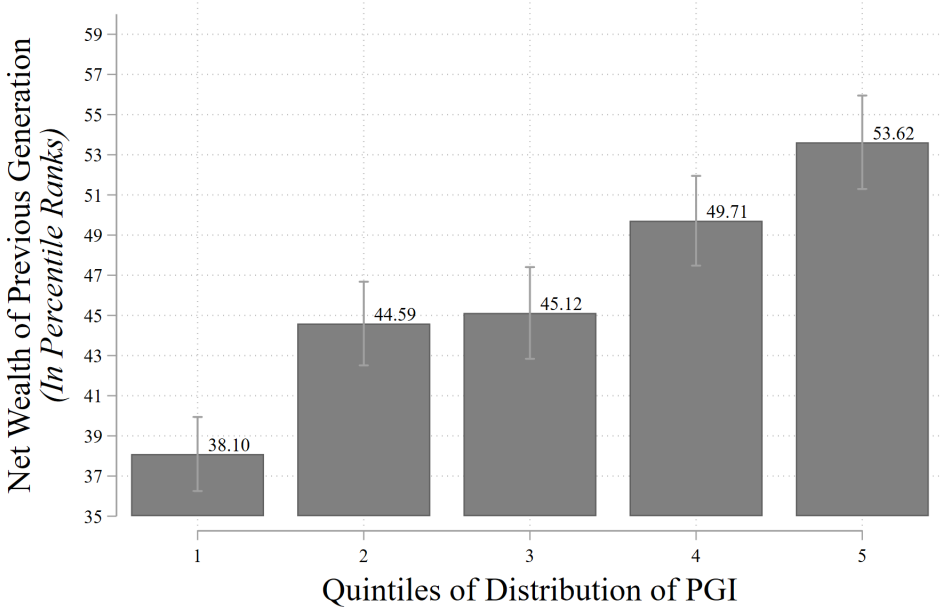


(b)

Figure 1: Panel a presents mean years of schooling across EA PGI quintiles. Panel b presents the college graduation rate across EA PGI quintiles. Error bars show 95% confidence intervals.



(c)



(d)

Figure 1: Panel c presents mean net wealth across EA PGI quintiles. Panel d presents mean net wealth of one's parents across EA PGI quintiles. Mean net wealth is measured as one's percentile rank position in the Dutch net wealth distribution, given one's year of birth. Error bars show 95% confidence intervals.

4 Empirical Strategy

4.1 The Stochastic Nature of Genetic Inheritance

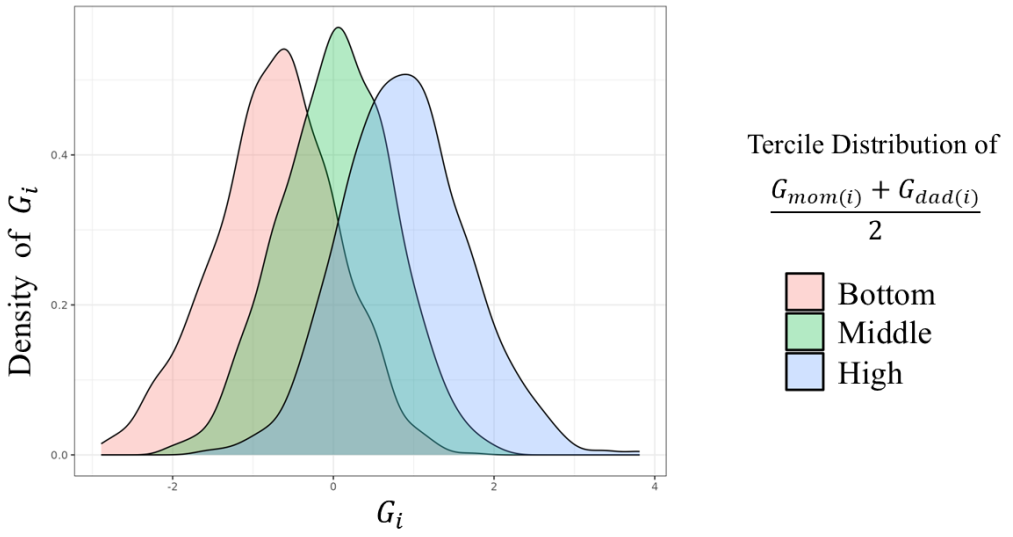
Humans have two copies of each genetic marker. During conception, each parent transmits only one of their two copies to their offspring, and the selection of which copy is transmitted occurs at random. A practical implication of this randomness is that, once we condition on the sum of the PGIs of an individual's parents, the remaining variation in the individual's PGI is effectively random. In particular, an individual's PGI can be expressed as the average PGI of their biological parents plus a random component:

$$G_i = \frac{G_i^{mom} + G_i^{dad}}{2} + \delta_i \tag{2}$$

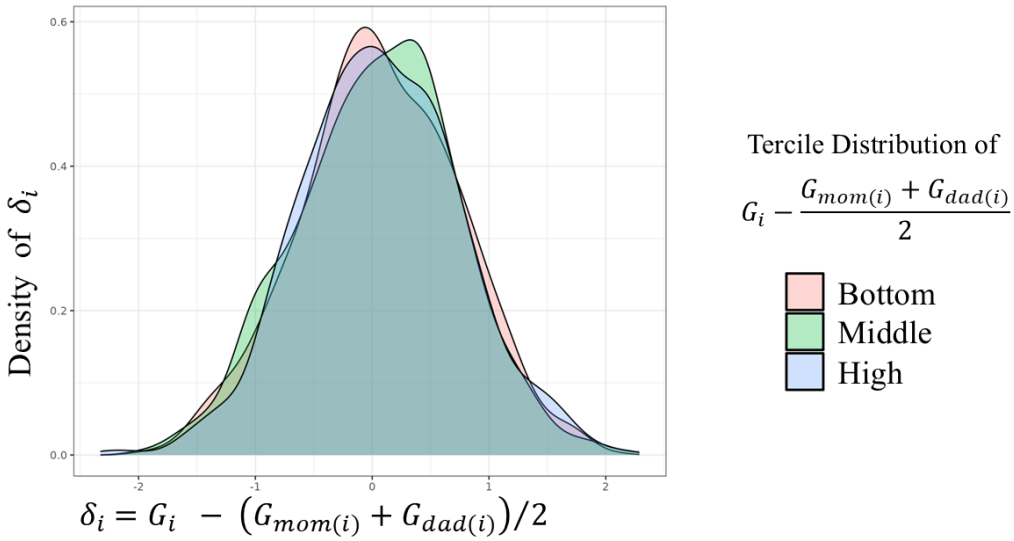
where G_i^{mom} and G_i^{dad} denote the PGIs of individual i's biological mother and father, respectively, and δ_i represents the random component. This random component can be thought of as the deviation from an individual's expected PGI, which is equal to the average PGI of their biological parents. Its variation is substantial: its cross-sectional variance is half as large as the cross-sectional variance of the PGI.

We now illustrate that the two terms on the right-hand side of equation (2) are orthogonal to each other. The top panel of Figure 2 plots the density of individuals' PGIs, G_i, separately for those whose parents have low, intermediate, and high PGIs. Unsurprisingly, individuals with higher-PGI parents tend to have higher PGIs themselves, a direct consequence of genetic inheritance. In contrast, the bottom panel of Figure 2 plots the density of the random component, δ_i, for the same three groups. They completely overlap, appearing virtually indistinguishable. Figure 2b demonstrates that individuals with different genetic ancestries have equal chances in this "genetic lottery".

Next, we show that the random component is orthogonal to the environmental circumstances into which an individual is born. We begin by documenting that an individual's PGI is associated with their parents' SES, reinforcing the pattern observed in Figure 1D.



(a)



(b)

Figure 2: Panel (a) shows the density of the EA PGI, stratified by parental PGI terciles, where parental PGI is defined as the average of one's mother's and father's PGI. Panel (b) presents the density of the residual PGI (one's PGI minus parental PGI), plotted separately for each parental PGI tercile.

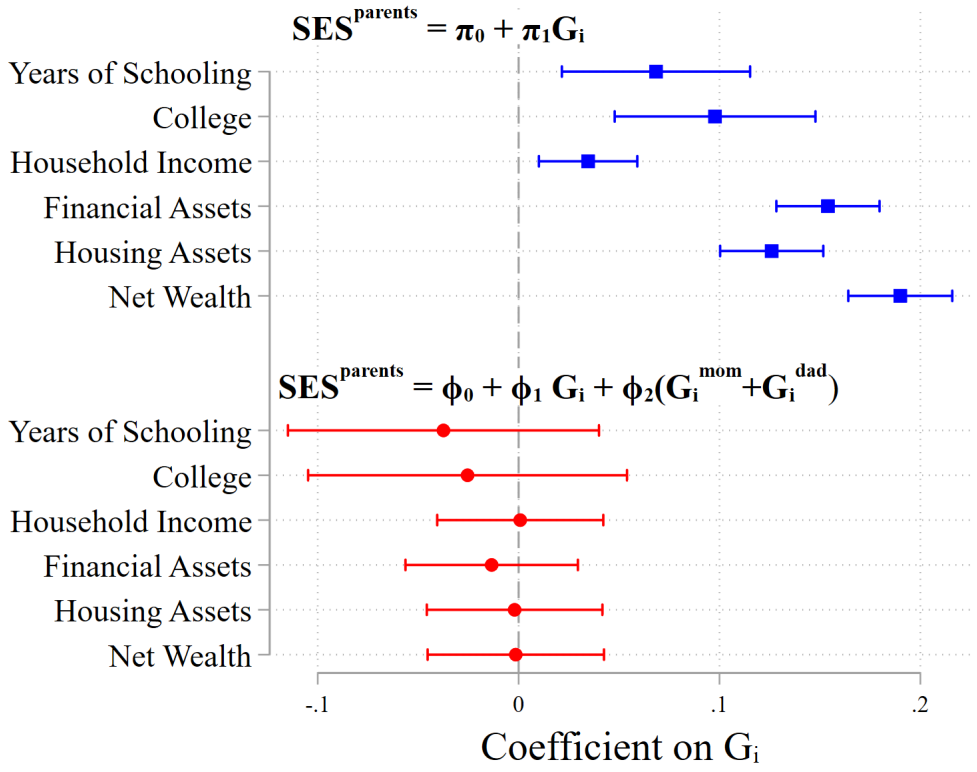


Figure 3: The effect of the EA PGI of the reference person on measures of socioeconomic status (SES) of their parents. The upper panel of the figure (blue dots and bars) shows point estimates and 95% confidence intervals of π_1, the coefficient of a regression of the SES indicator of the parents on the reference person's PGI. The bottom panel of the figure (red dots and bars) shows point estimates and 95% confidence intervals of ϕ_1, the coefficient of a regression of the SES indicator of the parents on the reference person's PGI, conditional on the sum parents' PGIs. Standard errors are clustered to account for serial correlation in annually measured outcomes. All regressions were estimated within the sample for which we had (1) PGIs of the reference person, (2) the sum of imputed PGIs of reference person's parents and (3) the relevant measure of SES for at least one of these parents of the reference person.

Specifically, we estimate:

$$Y_i^{parents}(t) = \pi_0 + \pi_1 G_i + u_i^{parents}(t) \tag{3}$$

where $Y_i^{parents}(t)$ represents the SES of individual i's parents in year t. The top panel of Figure 3 displays the coefficient on individual i's PGI, π_1. Each row corresponds to a different regression using a different proxy for the parents' SES. Across all specifications, the coefficients are positive, indicating that individuals with higher PGIs tend to come from higher socioeconomic backgrounds—their parents are more educated, earn higher incomes, and hold greater wealth.

Next, we examine how this relationship changes once we control for the sum of the parents' PGIs. Specifically, we estimate:

$$Y_i^{parents}(t) = \phi_0 + \phi_1 G_i + \phi_2 (G_i^{mom} + G_i^{dad}) + u_i^{parents}(t) \tag{4}$$

The bottom panel of Figure 3 reports the coefficient on individual i's PGI, ϕ_1. Once we control for the parents' PGIs, the relationship between an individual's PGI and their parents' SES vanishes, conforming that the remaining variation in the individual's PGI is orthogonal to the environmental circumstances into which they are born. This independence allow us to isolate genetic effects from environmental influences.

4.2 Regression analyses

We exploit the randomness in genetic transmission across generations to study how an individual's genetics affects both their own SES and the SES of the next generation. Central to our analysis is what we call "the reference"—the individual whose genetic variation we study. We exploit the randomness in the genetic transmission from the reference's parents to the reference to identify the causal effects of the reference's genetics. Specifically, we leverage that, once we control for the genetics of the reference's parents, the remaining variation in

the reference's genetics is random. Using this setup, we estimate two types of effects. First, we examine how the reference's genetics influence their own SES, which we refer to as the *same-generation genetic effect*. Second, we assess how the reference's genetics affect their offspring's SES, which we refer to as the *next-generation genetic effect*.

To estimate the same-generation genetic effect, we regress the reference's SES on their PGI while controlling for the sum of the PGIs of the reference's parents:

$$Y_i(t) = \alpha_0 + \alpha_1 G_i + \alpha_2 (G_i^{mom} + G_i^{dad}) + \mathbf{X}_i(t)\boldsymbol{\alpha}_3 + u_i(t) \tag{5}$$

where $Y_i(t)$ is the reference's SES measured in year t, G_i is the reference's PGI, G_i^{mom} is the PGI of the reference's mother, G_i^{dad} is the PGI of the reference's father, and $\mathbf{X}_i(t)$ is a vector of controls.

Similarly, we estimate the next-generation genetic effect by regressing the SES of the reference's offspring on the reference's PGI while controlling for the sum of the PGIs of the reference's parents:

$$Y_{i,k}^{child}(t) = \beta_0 + \beta_1 G_i + \beta_2 (G_i^{mom} + G_i^{dad}) + \mathbf{X}_i(t)\boldsymbol{\beta}_3 + u_{i,k}^{child}(t) \tag{6}$$

where $Y_{i,k}^{child}(t)$ is the SES of the reference's k-th child. Notice that equations (5) and (6) share the same right-hand side. The only distinction between the two equations is that equation (6) replaces the reference's with the SES of the reference's offspring.

The object of interest in equation (6) is the coefficient on the reference's PGI, β_1. It estimates the causal impacts on the reference's offspring of the reference inheriting specific genetic variants, holding constant any environmental factors determined before the reference's conception–including the environmental circumstances into which the reference is born. In contrast, the coefficient on the sum of the PGIs of the reference's parents, β_2, cannot be given a causal interpretation. Nevertheless, β_1 should be thought of as a lower-bound estimate of the effect of interest because the weights used to compute the PGI are estimated with error

(Becker et al., 2021; Sanz-de Galdeano & Terskaya, 2023; van Kippersluis et al., 2023). In Table 4, we show how our estimates change when we adjust for measurement error.

4.3 Sample and Summary statistics

To implement our empirical strategy, we require genetic data from the reference, their mother, and their father. However, Lifelines includes only a few cases where a participant and both biological parents were genotyped. Nevertheless, if we have genetic data from one of the participant's parents or from a participant's full sibling in addition to the participant's own genetic data, we can impute the genotypes of the participant's parents using *Mendelian imputation*, a methodology recently developed by Young et al. (2022). Crucially, this method differs from imputation techniques typically used by economists, as it relies on the laws of genetic inheritance–recombination and Mendelian segregation–rather than statistical correlations. Intuitively, if a full sibling of the participant carriers a reference allele at a given SNP that the participant does not have, this allele must have been transmitted from their parents to the sibling but not to the participant. Any remaining information about the participant's parental genotypes is imputed using population averages. Importantly, while the imputed genotypes of the participant's parents are measured with error, the estimates of the causal effects of the participant's will remain unbiased. This is because bias arises from genetic variants shared between the participant and their parents–precisely the portion that can be imputed. In contrast, the portion that cannot be imputed consists of the parents' genetic variants that were not inherited by the participant, which are unrelated to the participant's genetic variants. See Young et al. (2022) for more details.

The reference–the individual whose genetic variation we study–is always a Lifelines participant, as their genetic data are required for our analysis. Additionally, the imputation method described above requires that at least one parent or full sibling of the reference also participated in Lifelines, as their genetic data are needed for the imputation procedure. In contrast, to estimate next-generation genetic effects, we do not require genetic data from

the reference's offspring—only the offspring's socioeconomic outcomes from the CBS data. Because most offspring did not participate in Lifelines, survey data are not available for them. To ensure that the reference's offspring have completed their education and that the offspring's income and wealth reflect their socioeconomic status, we restrict the analysis to offspring who were at least 30 years old as of 2022, the most recent year in our data.

Our main sample includes 7,012 individuals from the "reference generation" and 14,109 individuals from the "offspring generation"–that is, the reference's offspring. The number of annual observations for these two samples is 62,259 and 119,326, respectively, meaning that, on average, each individual is observed for more than eight years.

Table 1 presents summary statistics on demographics, education, income, and wealth for these two samples. The first set of columns reports statistics for the reference generation while the second set reports statistics for the reference generation. Individuals in the reference generation were born between 1940 and 1969 (average birth year of 1957) and were, on average, 62.5 years old when observed. Individuals in the offspring generation were born between 1964 and 1991 (average birth year: of 1983.5) and were, on average, 37 years old at the time of observation. Naturally we observe the offspring generation at a younger age than the reference generation. In our sample, women are overrepresented in the reference generation, comprising approximately two-thirds of the reference individuals are women. In contrast, the gender distribution in the offspring generation is more balanced, with 49% women. The younger generation is more educated than the older generation. The offspring generation has, on average, 14.5 years of schooling, and more than half of them have graduated from college, whereas the reference generation has an average of 12.4 years of schooling, with less than a fifth of them holding a college degree. Because survey data from Lifelines are not available for most individuals in the offspring generation, their education information comes from CBS, while the education data for the reference generation come from Lifelines surveys. CBS education data are missing for a portion of the Dutch population, with missingness occurring more frequently in older cohorts. In our sample, this information is available for

approximately 90% of the offspring generation and for only 48% of the reference generation.

The younger generation earns more than the older generation. The average individual income is €32,188 for the reference generation and €48,187 for the offspring generation. However, due to differences in age at observation, the older generation has accumulated significantly more wealth. The average net wealth is €278,747 for the reference generation, compared to €110,182 for the offspring generation. Despite this difference, both generations have similar portfolio allocations: approximately two-thirds of total assets come from housing, and roughly one-fifth from financial assets. More than 90% of the financial assets are held in checking and savings accounts. Only one quarter of the reference generation and one fifth of the offspring generation hold bonds or stocks.

	Reference		**Reference's Offspring**	
	Mean	SD	Mean	SD
Demographics				
Male	0.32	0.47	0.51	0.50
Birth Year	1957.0	6.7	1983.5	6.9
Age	62.5	6.2	37.0	5.8
Education				
Years of Schooling	12.4	2.3	14.5	2.2
College Graduate	0.19	0.39	0.53	0.50
Income				
Individual Earnings	€20,906	€28,321	€46,225	€29,586
Individual Income	€32,188	€26,436	€48,187	€28,407
Household Income	€71,941	€43,636	€91,434	€47,710
Wealth				
Net Wealth	€278,747	€329,260	€110,182	€238,259
Total Assets	€383,428	€368,990	€291,613	€295,220
Total Debt	€100,198	€119,803	€180,044	€144,666
Housing Assets / Total Assets	0.65	0.33	0.67	0.37
Financial Assets / Total Assets	0.26	0.30	0.26	0.35
Checking & Savings / Fin. Assets	0.91	0.22	0.94	0.17
Bonds & Stocks / Fin. Assets	0.08	0.20	0.05	0.16
1 if Has Bonds or Stocks	0.25	0.44	0.20	0.40
Number of Individuals	7,012		14,109	
Number of Annual Observations	62,259		119,326	

Table 1: This table shows summary statistics, separately for the reference generation and the offspring generation. The education variables are available for 6,868 individuals (98% out of the total of 7,012 individuals) in the reference generation and for 12,681 individuals (90% out of the total of 14,109 individuals) in the offspring generation.

5 Results

5.1 Same-Generation Genetic Effects

We begin by examining how the reference's genetics influences their own SES. Estimates from equation (5) are presented in Table 2. The left panel reports results with outcomes measured in levels, while the right panel presents results with outcomes measured in percentile ranks. Odd-numbered columns show estimates of α_1, the coefficient on the reference's PGI, while even-numbered columns report robust standard errors–standard errors are clustered at the level of the reference. All regressions control for the sum of the PGIs of the reference's parents, as well as the reference's year of birth and gender, and year-of-measurement fixed effects for the time-variant variables. Only the coefficient on the reference's PGI is reported in the table.

	Levels		**Percentile Rank**	
	α_1	S.E.	α_1	S.E.
Education				
Years of Schooling	0.46	0.07		
College Graduate	0.07	0.01		
Income				
Individual Earnings	€1,751	€415	1.47	0.55
Individual Income	€2,427	€435	2.41	0.50
Household Income	€3,194	€742	2.05	0.51
Ind. Income - Ind. Earnings	€367	€195	0.83	0.60
Hhld. Income - Ind. Income	€492	€618	0.09	0.60
Wealth				
Net Wealth	€27,956	€7,010	2.57	0.56
Total Assets	€32,238	€7,898	2.35	0.54
Total Debt	€3,201	€2,440	1.06	0.68
Housing Assets	€16,121	€3,844	2.38	0.60
Financial Assets	€6,437	€2,122	2.06	0.56
Checking & Savings	€5,143	€1,398	2.03	0.55
Bonds & Stocks	€596	€762	2.23	0.79
1 if Has Bonds or Stocks	0.03	0.01		
Mortgage Debt	€1,183	€2,047	0.68	0.75

Table 2: Effects of second generation's PGI on second generation's outcomes (estimates of equation 5 for various outcomes $Y_i(t)$).

Our results show that the reference's PGI has a causal impact on their socioeconomic status in adulthood. On average, individuals with higher PGIs attain more education, earn higher incomes, and accumulate greater wealth than their peers. A one standard deviation increase in the reference's PGI leads to approximately 0.5 additional years of schooling and

increases the likelihood of college graduation by 7 percentage points. It also raises annual individual and household incomes by 6.4 and 3.8 log points (not shown in the table), respectively, moving them 2.4 and 2 percentiles up in these income distributions. Additionally, it raises net wealth by approximately €28,000 (corresponding to a 10% increase relative to the average net wealth of €278,747), moving them 2.6 percentiles up in the wealth distribution. This increase is driven by growth in the two major components of total assets: housing assets (up 6.3% relative to the average) and financial assets (up 9.5% relative to the average). We find no statistically significant effect on total debt or mortgage debt. This null effect may reflect opposing forces: on one hand, higher-SES individuals are less likely to experience financial distress, which could reduce debt; on the other hand, they may take on larger mortgages, as housing debt constitutes a major component of total debt.

These results preview one of the pathways through which the reference's genetics may influence their offspring's SES. Individuals with higher PGIs tend to attain more education, earn higher incomes, and accumulate greater wealth than their peers. As a result, their offspring are more likely to grow up in higher-SES family environments, which may, in turn, enhance the offspring's own SES outcomes–a mechanism we refer to as the *genetic nurture channel* (Kong et al. 2018). At the same time, children of high-PGI individuals inherit higher PGIs themselves, a mechanism we term the "genetic transmission channel." We proceed now to estimate the total effect of the reference's PGI on the SES of the next generation, without distinguishing between these two channels. Later in the paper, we will quantify their relative contributions.

5.2 Next-Generation Genetic Effects

Next, we examine how the reference's genetics affects the SES of the reference's offspring. Table 3 estimates equation (6). The left panel reports results with outcomes measured in levels, while the right panel presents results with outcomes measured in percentile ranks. Odd-numbered columns show estimates of β_1, the coefficient on the reference's PGI, while

even-numbered columns report standard errors–standard errors are clustered at the level of the reference. All regressions control for the sum of the PGIs of the reference's parents, as well as the reference's year of birth and gender, the gender of the reference's offspring, and year-of-measurement fixed effects for the time-varying variables. Only the coefficient on the reference's PGI is reported in the table.

	Levels		**Percentile Rank**	
	β_1	S.E.	β_1	S.E.
Education				
Years of Schooling	0.33	0.04		
College Graduate	0.07	0.01		
Income				
Individual Earnings	€1,968	€472	1.70	0.43
Individual Income	€2,010	€452	1.83	0.42
Household Income	€3,033	€777	1.77	0.43
Ind. Income - Ind. Earnings	€13	€83	-0.19	0.42
Hhld. Income - Ind. Income	€854	€543	0.47	0.44
Wealth				
Net Wealth	€7,171	€3,734	1.40	0.45
Total Assets	€12,062	€4,910	1.72	0.44
Total Debt	€4,510	€2,334	0.69	0.44
Housing Assets	€8,453	€2,794	1.26	0.49
Financial Assets	€2,898	€937	2.03	0.45
Checking & Savings	€2,558	€721	1.94	0.45
Bonds & Stocks	€189	€249	1.83	0.56
1 if Has Bonds or Stocks	0.021	0.006		
Mortgage Debt	€3,840	€2,144	0.49	0.49

Table 3: Estimates of the effect of the parent's PGI on SES-related outcomes of their children (estimates of equation 6 for various outcomes $Y_{i,k}^{child}$). All outcome variables are measured annually except years of schooling and being a university graduate, which are measured only once per child. Additional control variables include parent year of birth fixed effects, the gender of the parent of the child, and year fixed effects for variables that are measured annually. Standard errors are clustered at the level of the parent. In the columns "Percentile Rank", all income and wealth-related outcomes are measured as their within-year-of-birth percentiles.

The genetics of one generation causally influences the SES of the next. Just like their parents, the children of high-PGI parents attain more education, earn higher incomes, and accumulate greater wealth than their peers. A one standard deviation increase in the reference's PGI increases the offspring's education by a third of a year of schooling and their likelihood of college graduation by 7 percentage points. It also raises the offspring's annual individual and household incomes by 4.6 and 3 log points (not shown in the table), respectively, moving them up by 1.7-1.8 percentiles in these income distributions.

Table 3 also examines the effects of the reference's PGI on two additional income mea-

sures: the offspring's non-labor individual income (i.e., individual income minus individual earnings) and the total income of all other members of the offspring's household (i.e., household income minus the offspring's individual income). We find no effect on non-labor income, suggesting that the higher individual incomes of offspring with high-PGI parents are driven entirely by higher earnings. Additionally, we cannot reject the hypothesis that the reference's PGI has no effect on the income of other household members, indicating that the higher household incomes of offspring with high-PGI parents are primarily due to the offspring's own higher wages.

Finally, we examine the impact of the reference's PGI on the offspring's wealth. A one standard deviation increase in the reference's PGI raises the offspring's net wealth by €7,171, a 6.5% increase relative to the average net wealth of €110,182, moving them 1.4 percentiles up in the net wealth distribution. The point estimates suggest that, in percentile rank terms, the effect is larger for financial assets than for housing assets. A one standard deviation increase in the reference's PGI moves the offspring 1.3 percentiles up in the housing assets distribution and 2 percentiles up in the financial assets distribution. This difference is partly explained by the age at which we observe the offspring generation. Appendix Table XXXX re-estimates next-generation genetic effects, allowing them to vary with the offspring's age. The results indicate that the effect on housing assets increases significantly with age, while we cannot reject the hypothesis that the effect on financial assets is constant across age groups.

5.2.1 Assessing Magnitude of Next-Generation Genetic Effects

To understand the magnitudes of our estimates of next-generation genetic effects, we propose benchmarking them against existing estimates of the effects of family environment–in particular to estimates from studies that exploit the quasi-random assignment of adoptees to adoptive families, which arguably provide some of the most credible estimates of the causal effects of the family environment. The quasi-random assignment ensures that the

environment of these families is orthogonal to pre-adoption characteristics of the adoptees; any post-adoption differences between the adoptees can be attributed to the effects of the adoptive family's environment. Black et al. (2020) and Fagereng et al. (2021) estimate the impact of increasing an adoptive parent's rank in the (net) wealth distribution by 10 percentiles on their adopted children. To facilitate comparability with their estimates, we generate an alternative set of estimates where the reference's PGI is measured in percentile ranks rather than in standard deviation units. Additionally, we provide a complementary set of estimates that adjust for measurement error in the PGI.

Table 4 presents these two sets of estimates, along with the estimates from Black et al. (2020)'s and Fagereng et al. (2021). The first set of columns reports the causal effect of increasing the reference's PGI by 10 percentiles (while holding the sum of the PGIs of the reference's parents constant) on the SES of the reference's offspring. Column 1 presents estimates without adjusting for measurement error–these correspond to the same estimates shown in Table 3, but with the reference's PGI being expressed in percentile ranks instead of standard deviation units. Column 2 presents estimates that adjust for the measurement error in the PGI. The last two columns display the estimated causal effects of increasing the adoptive parent's net wealth by 10 percentiles on the SES of the adopted child. The penultimate column presents estimates from Black et al. (2020). The last column presents estimates from Fagereng et al. (2021). Each row corresponds to a different measure of offspring SES–though Fagereng et al. (2021) estimate only the effect on the adopted child's net wealth. Standard errors are shown in parentheses.

Overall, our estimates of genetic effects are comparable in magnitude to the effects of family environment. In fact, for education and income, our estimates suggest a larger impact of genetics than family environment. Black et al. (2020) estimate that increasing the adoptive parents' net wealth rank by 10 percentiles raises the education of the adopted children by 0.1 years and moves them 0.52 percentiles up in the distribution of individual income. In contrast, we estimate that increasing the reference's PGI rank by 10 percentiles raises their

	Genetic Effect		Family Environment Effect	
	Unadjusted	Adjusted for ME	Black et al.	Fagereng et al.
Next Generation's SES				
Years of Schooling	0.12	0.18	0.10	–
	(0.01)	(0.03)	(0.02)	
Individual Income	0.64	0.84	0.52	–
	(0.15)	(0.28)	(0.25)	
Net Wealth	0.49	0.68	1.99	1.67
	(0.16)	(0.30)	(0.27)	(0.21)

Table 4: The first set of columns reports the causal effect of increasing the reference's PGI by 10 percentiles (while holding the sum of the PGIs of the reference's parents constant) on the SES of the reference's offspring. Column 1 presents estimates without adjusting for measurement error–these correspond to the same estimates shown in Table 3, but with the reference's PGI being expressed in percentile ranks instead of standard deviation units. Column 2 presents estimates that adjust for the measurement error in the PGI. The last two columns display the estimated causal effects of increasing the adoptive parent's net wealth by 10 percentiles on the SES of the adopted child. The penultimate column presents estimates from Black et al. (2020). The last column presents estimates from Fagereng et al. (2021). Fagereng et al. (2021) estimate only the effect on the adopted child's net wealth. Standard errors are shown in parentheses.

offspring's education by 0.12 years of schooling and moves them 0.64 percentiles up in the distribution of individual income. When we adjust for measurement error in the PGI, these estimates increase to 0.18 years and 0.84 percentiles, respectively. However, our estimates of the effect of genetics on wealth are smaller than existing estimates of the effect of family environment. While Black et al. (2020) and Fagereng et al. (2021) estimate that increasing an adoptive parents' net wealth rank by 10 percentiles moves their adopted children 2 percentiles and 1.67 percentiles up in the (net) wealth distribution, respectively, we find that increasing the reference's PGI rank by 10 percentiles moves their offspring only half a percentile up in the net wealth distribution (or two-thirds of a percentile when adjusting for measurement error). At face value, these findings align with Black et al. (2020)'s conclusion that genetics plays a more limited role in wealth transmission compared to family environment.

5.2.2 Persistence of Genetic Effects across Generations

Here, we contrast the effects of the reference's genetics on their offspring's SES with its effects on their own SES. This comparison provides insight into the persistence of the "shock" that originates in the reference's generation.

Figure 4 plots the next-generation genetic effect against the same-generation genetic

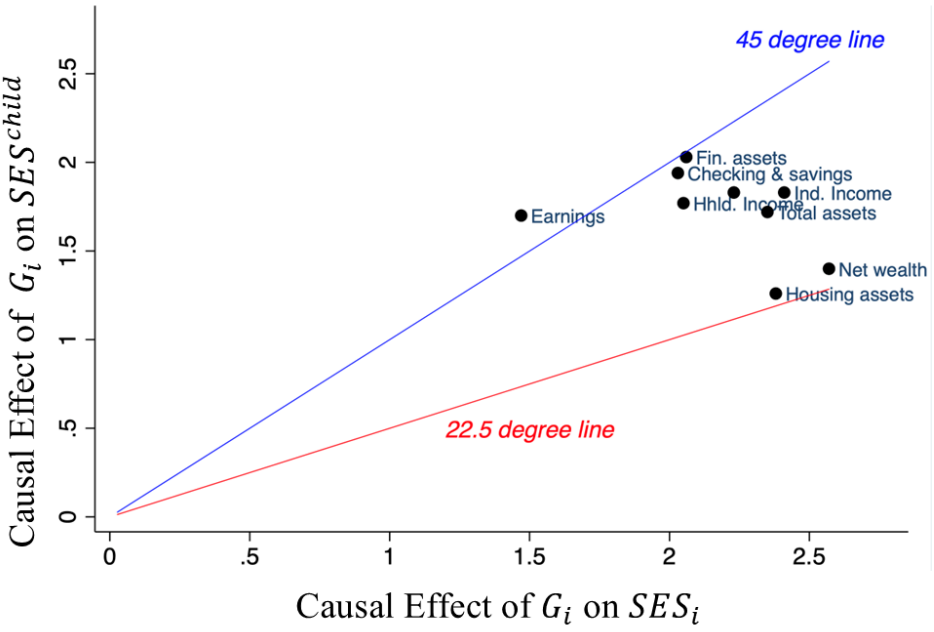


Figure 4: This figure compares point estimates of same-generation effects of the EA PGI on various SES-related outcomes (x-axis) to next-generation effects for the same SES-related outcomes (y-axis). When effects lie on the 22.5 degree line, they indicate a transmission of the genetic shock of 50%, consistent with genetic transmission being the only mechanism through which the effects of the genetic shock manifests in the child. When effects lie below the 22.5 degree line, they indicate a higher than 50% persistence, which can not be explained by parental genetic shocks operating through genetic transmission alone. Effects lying on the 45-degree line are consistent with a complete persistence of the genetic shock

effect for each SES measure. Each marker represents a different SES outcome, allowing us to visualize how the genetic influence on an individual's own SES compares to its influence on their offspring's SES for different outcomes. The figure also includes a 45-degree line and a 22.5-degree line. The 22.5-degree line represents a benchmark where the next-generation genetic effects are half as large as the same-generation genetic effects. This benchmark holds under two assumptions: (1) genetic transmission is the only channel through which one generation's genetics influences the next and (2) the same-generation genetic effects are identical across generations—that is, the causal effect of the offspring' PGI on their own SES is the same as the causal effect of the reference's PGI on the reference's own SES.

The figure indicates a high degree of persistence in the effects of the reference's genetics across generations. For most outcomes, the markers lie closer to the 45-degree line than to

the 22.5-degree line, suggesting that at least one of the two assumptions described above may not hold. Specifically, this pattern implies that either (i) genetic transmission is not the sole mechanism through which one generation's genetics affects the next, or (ii) same-generation genetic effects differ between the reference generation and the offspring generation. A caveat to the second assumption is that, as shown in Table 1, the two generations are observed at different stages of the life cycle. If the same-generation genetic effect on a given SES outcome varies with age, this could help explain deviations from the 22.5-degree line benchmark. This appears to be the case for the notable exceptions in the figure—housing assets and net wealth—which lie much closer to the 22.5-degree line. Indeed, we find that the effect of the reference's PGI on the offspring housing assets increases steeply with the offspring's age. A similar pattern holds for broader wealth measures that include housing, such as total assets and net wealth. In sum, the evidence suggests that the genetic transmission is not the only channel through which genetic effects operate across generations. In the next section, we turn to formally quantifying the contribution of the genetic transmission channel relative to other pathways. Regardless, the findings in Figure 4 reveal that a perturbation to the reference's genetics does not stop with them—it sends ripples across generations, shaping the lives of their descendants in the offspring generation and beyond.

6 Mechanisms

In this section, we study the channels through which the genetics of one generation may affect the economic prospects of the next. Figure 5 offers a framework that clarifies and contextualizes the findings shown in Table 3. We consider the effects of an exogenous shock to the reference's genetics caused by the randomness in the genetic transmission from the reference's parents to the reference. We distinguish between two conceptually-distinct mechanisms which were briefly discussed in Section 5.1: *genetic nurture* and *genetic transmission*.

Genetic nurture refers to the idea that a person's genetics can indirectly influence their

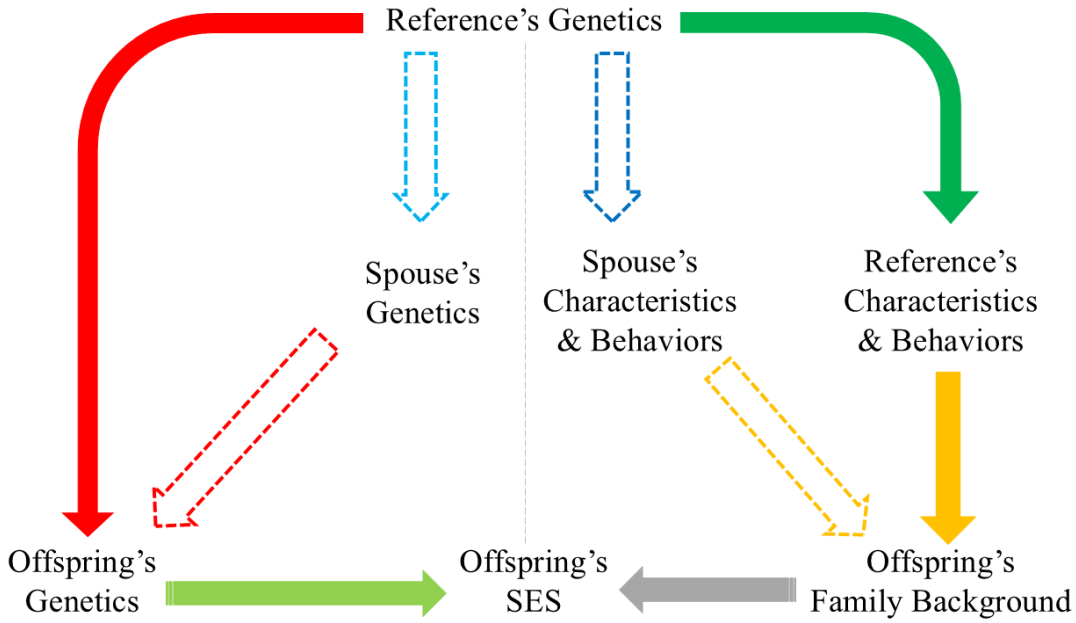


Figure 5: The figure illustrates the channels through which the reference's genetics may affect the economic prospects of their offspring. The path on the left-hand side of the diagram formed by solid arrows illustrates the effects of the genetic variants transmitted from the reference person to the offspring: the solid red arrow illustrates the transmission of these genetic variants and the solid light green arrow illustrates the effects these variants have on the offspring's SES. The path on the right-hand side of the diagram formed by solid arrows illustrates effects working through the characteristics and behaviors of the reference person. The reference's genetics will also influence their characteristics and behaviors (solid dark green arrow on the right-hand side of the diagram), shaping the environment in which their offspring grows up (solid orange arrow). The gray arrow represents environmental effects. The paths formed by dashed arrows capture the contribution of assortative mating, since the reference's genetics may influence their choice of reproductive partner, influencing spuose's genetics and other characteristics and behaviors of the spouse, which influence the offspring's genetics and family background.

offspring through purely environmental pathways (Kong et al. 2018). Specifically, a person's genetics shapes their characteristics and behaviors, which in turn influence the family environment in which their children are raised. Notably, even genetic variants that a person does *not* pass down to their offspring may still impact their offspring's SES by shaping their upbringing. This genetic nurture effect applies even to adopted children. Although adoptive parents and their adopted children do not share their genetics, the adoptive parents' genetics can still affect the adopted children's SES by influencing the family environment in which the adopted children are raised (Domingue & Fletcher 2020; Beauchamp et al. 2023).

The genetic nurture channel is illustrated on the right-hand side of the diagram. First, the reference's genetics influences their characteristics and behaviors (represented by the dark green arrow in the top right corner of the diagram), as documented in Table 2. These characteristics and behaviors shape the family environment in which their children are raised (represented by the solid gold arrow in the right-hand side of the diagram), which in turn impacts the children's SES (represented by the gray arrow in the bottom right of the diagram), as evidenced by adoption studies showing how the family environment provided by adoptive parents affects the SES of their adopted children (Björklund et al. 2007; Sacerdote 2008; Black et al. 2020; Fagereng et al. 2021; Beauchamp et al. 2023).

In contrast, the genetic transmission channel captures the direct impact of genetic variants passed from parents to offspring. When there is an exogenous shock to the reference's genetics, half of this shock is inherited by the offspring on average. This genetic inheritance then causally influences the offspring's SES through genetic effects. Children given up for adoption are also useful for thinking about this channel. While biological parents do not shape the environment in which their adopted children are raised, they still pass down their genetic makeup, which influence the child's SES through genetic effects.

The genetic transmission channel is illustrated on the left-hand side of the diagram. The solid red arrow in the top left corner of the diagram represents the transmission of genetic material from the reference to the reference's offspring. This genetic inheritance

then influences the offspring's SES through genetic effects. The light green arrow in the bottom left of the diagram represents the causal impact of the offspring's genetics on their own SES. The magnitude of the genetic transmission channel is equal to this effect divided by two, reflecting that, when there is a shock to the reference's genetics, on average, half of this shock is inherited by the offspring. We will quantify this channel in the next section. It is important to note that the light green arrow in the bottom left corner also represents a form of "same-generation genetic effect". However, it is distinct from the effects estimated in Table 2. While Table 2 examined how the reference's genetics influenced the reference's SES, the light green arrow represents the impact of the offspring's genetics on the offspring's SES. In other words, Table 2 estimates same-generation genetic effects for the reference generation, while the light green arrow represents same-generation genetic effects for the offspring generation.

Finally, both the genetic transmission and genetic nurture channels may be modulated by assortative mating. The exogenous variation in the reference's genetics may affect their choice of reproductive partner (for simplicity, we use the term "spouse" to refer to the person with whom the reference has a biological child). The reference's partner selection can influence the offspring's SES through two distinct pathways: (1) The offspring inherits half of their genetics from the reference's spouse, which is represented by the hollow red arrow and (2) The characteristics and behaviors of the reference's spouse will also contribute to shaping the environment in which their offspring is raised, which is represented by the hollow orange arrow. We return to this in Section 6.2.

6.1 Quantifying The Genetic Transmission Channel

As discussed above, the genetic transmission channel is equal to the effect of the PGI of the reference's offspring on the offspring's own SES, divided by two. In principle, we could estimate this effect by running the following regression:

$$Y_{i,k}^{child}(t) = \lambda_0 + \lambda_1 G_{i,k}^{child} + \lambda_2 (G_i + G_{i,k}^{spouse}) + \mathbf{X}_i(t)\boldsymbol{\lambda}_3 + v_{i,k}^{child}(t) \tag{7}$$

where $Y_{i,k}^{child}(t)$ is the SES of the reference's k-th child (measured in year t), $G_{i,k}^{child}$ is the PGI of the reference's k-th child, G_i is the reference's PGI, and $G_{i,k}^{spouse}$ is the PGI of the other biological parent of the reference's k-th child. Notice the similarity between equation (7) and equation (5). The key distinction is that equation (7) estimates same-generation genetic effects for the offspring generation, whereas equation (5) estimates same-generation genetic effects for the reference generation.

To study how the offspring's SES is causally impacted by both their own PGI and their parent's PGI (i.e., the reference's PGI) using the same sample, we would need genetic data from three generations of a family: the reference's parents (which could be imputed), the reference, and the reference's offspring. The genetic data of the reference and their offspring are necessary to estimate the impact of the offspring's PGI (i.e., equation (7)), while the genetic data of the reference and their parents are needed to estimate the impact of the reference's PGI (i.e., equation (6). However, restricting the analysis to families with genetic data from three generations would result in a sample too small to provide sufficient statistical power for estimating equations (6) and (7).

For this reason, we estimate equation (7) using a different sample from the one used to estimate equation (6). To make this sample as comparable as possible to the reference generation in our main sample, we take two steps: (1) we restrict this sample to individuals born between 1964 and 1991, matching the birth years of the reference generation in our main sample and (2) wed re-weigh this sample to match the year-of-birth-by-gender distribution in the reference generation in our main sample.

Our estimates from the decomposition exercise are presented in Table 5. The first two columns reproduce results from Table 3, estimating the total effect of the reference's PGI on the SES of their offspring. Column 1 reports the point estimate, while Column 2 presents the standard errors. The next two columns quantify the genetic transmission channel. Column

	Total Effect		Genetic Transmission		Ratio	
	β_1	S.E.	$\lambda_1/2$	S.E.	$\lambda_1/2\beta_1$	S.E.
Education						
Years of Schooling	0.33	0.04	0.16	0.01	0.48	0.05
College Graduate	0.07	0.01	0.03	2.3E-03	0.45	0.06
Income						
Individual Earnings	1.70	0.43	1.41	0.13	0.83	0.30
Individual Income	1.83	0.42	1.44	0.13	0.79	0.24
Household Income	1.77	0.43	0.94	0.13	0.53	0.19
Wealth						
Net Wealth	1.40	0.45	0.42	0.14	0.30	0.23
Total Assets	1.72	0.44	0.59	0.12	0.35	0.14
Financial Assets	2.03	0.45	1.09	0.13	0.53	0.17
Housing Assets	1.26	0.49	0.43	0.14	0.35	0.34

Table 5: This table shows the extent to which the effect of parental PGI on socioeconomic outcomes of children occurs through a genetic transmission channel: the transmission of genetic material from parent to child, at conception of the child. The left column shows the effect of the parental PGI on the socioeconomic outcomes of the child (as previously shown in table 3). The middle columns show the part of this total effect that can be attributed to the genetic transmission channel. To estimate it, we restrict the effect of assortative mating to be zero, and estimate κ by estimating the effect of one's PGI on one's *own* outcome in the direct effect sample, with inverse probability weights applied to make this sample comparable to the third generation of the main sample. Finally, the right column shows the ratio of the genetic transmission channel to the total effect. When it is lower than one, this implies the remainder of the total effect operates through the family background of the child.

3 reports the estimated effect of the offspring's PGI on their own SES (i.e., the coefficient λ_1 from equation (7)), divided by 2. Column 4 presents the standard errors for these estimates. The last two columns assess the proportion of next-generation genetic effects explained by the genetic transmission channel. Column 5 reports the ratio of Column 3 to Column 1, while Column 6 presents the standard errors around this estimate. If we reject the hypothesis that the estimate in Column 5 is zero, it suggests that the genetic transmission channel contributes at least partly to the next-generation genetic effects. If we reject the hypothesis that the estimate in Column 5 is one, it indicates that genetic transmission alone cannot fully explain the next-generation genetic effects–suggesting that genetic nurture or assortative mating may also play a role.

We find that genetic transmission accounts for approximately 50% of the next-generation genetic effects on education, implying that the remaining 50% must be driven by genetic nurture and assortative mating. Similarly, genetic transmission explains about half of the next-generation genetic effects on household income and financial assets. However, it explains a smaller fraction – approximately one-third – of the next-generation genetic effects on

housing assets and broader wealth measures that include housing assets, such as total assets and net wealth. This finding is consistent with housing policies in the Netherlands that previously enabled higher-SES parents to financially assist their children in purchasing their first homes. In contrast, genetic transmission appears to account for approximately 80% of the next-generation genetic effects on individual earnings and individual income. We speculate that this is because while higher-SES parents can give their children a leg up in educational attainment and homeownership, they may have less direct influence over their offspring's success in the labor market.

6.2 Assortative Mating

We begin by examining whether there is evidence of genotypic assortative mating—that is, whether an individual's genetics causes them to choose a genetically-similar person as a reproductive partner. To investigate this , we test whether exogenous variation in the reference's PGI is associated with the PGI of their spouse. We proceed as follows. First, we estimate the following two regressions:

$$G_{i,k}^{spouse} = \chi_0 + \chi_1(G_i^{mom} + G_i^{dad}) + \varepsilon_{i,k}^{spouse} \tag{8}$$

$$G_i = \psi_0 + \psi_1(G_i^{mom} + G_i^{dad}) + \varepsilon_i \tag{9}$$

where $G_{i,k}^{spouse}$ is the PGI of the other biological parent of the reference's k-th child, G_i^{mom} and G_i^{dad} are the PGIs of the reference's mother and father, and G_i is the reference's PGI. Next, we produce a scatterplot by plotting the fitted residuals from equation (8), $\hat{\varepsilon}_{i,k}^{spouse}$, against the fitted residuals from equation (9), $\hat{\varepsilon}_i$. This is equivalent to estimating the regression:

$$G_{i,k}^{spouse} = \gamma_0 + \gamma_1 G_i + \gamma_2(G_i^{mom} + G_i^{dad}) + u_{i,k}^{spouse} \tag{10}$$

The result is shown in Figure 6a. The fitted regression line is completely flat, with a slope

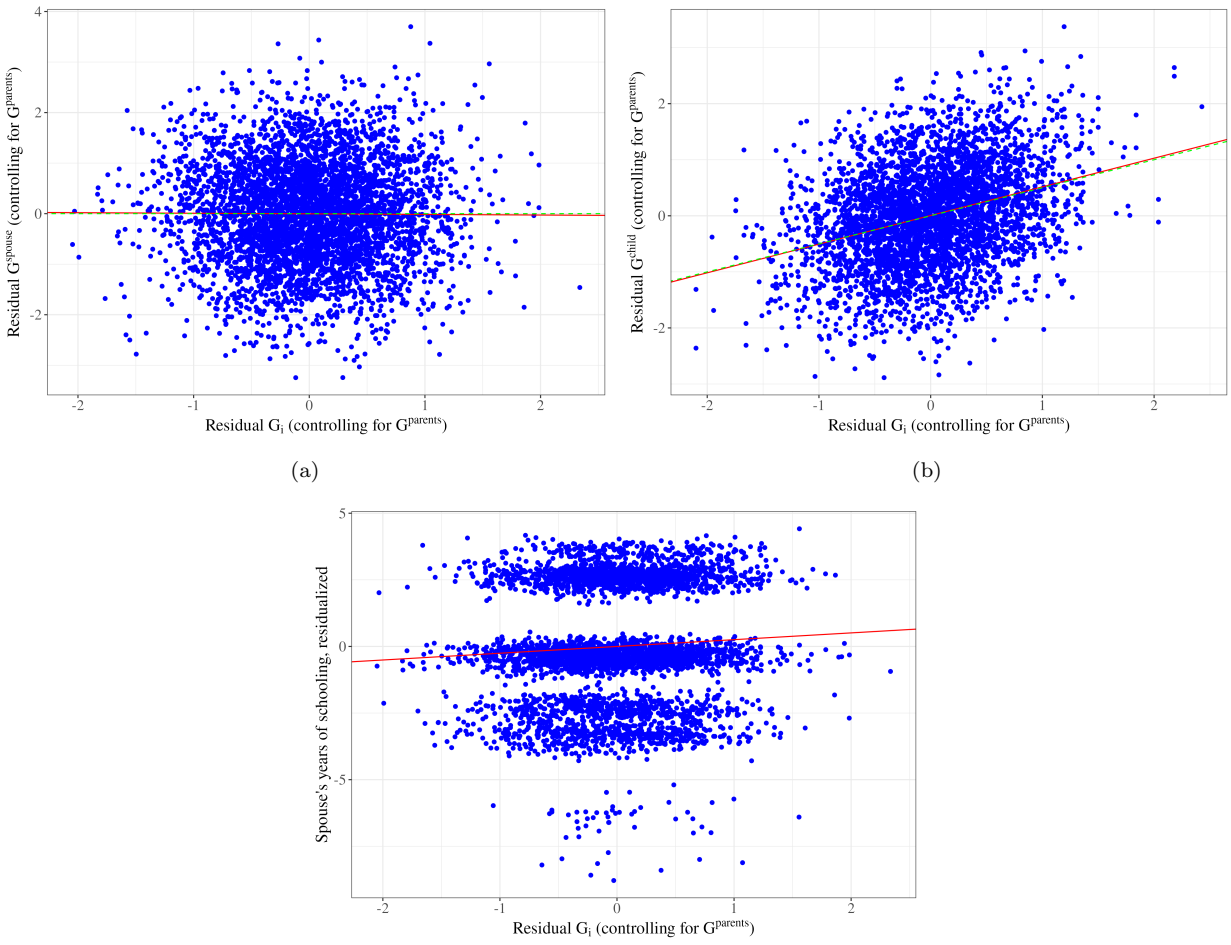


Figure 6: Figure 6a shows the relationship between the reference's PGI and the PGI of the reference's spouse, conditional on the sum of the PGIs of the reference's parents. It shows no evidence of assortative mating on the genetic predisposition for educational attainment. Figure 6b shows the relationship between the reference's PGI and the PGI of the reference's offspring, conditional on the sum of the PGIs of the reference's parents. It also shows no evidence of assortative mating on the genetic predisposition for educational attainment—the slope of 0.5 if consistent of random mating. Figure 6c shows the relationship between the reference's PGI and education of the reference's spouse, conditional on the sum of the PGIs of the reference's parents. It provides evidence that an exogenous increase in an individual's PGI causes them to select a more-educated reproductive partner.

of -0.012 (SE = 0.026), indicating no evidence of genotypic assortative mating. Although the reference's PGI is positively associated with their spouse's PGI – as documented in prior studies – this relationship disappears after controlling for the sum of the PGIs of the reference's parents. This suggests that the unconditional correlation between spouses' PGIs may be driven by shared genetic ancestry rather than active mate selection based on genetic predisposition for educational attainment.

One concern is that this null result could be due to measurement error in the PGI. However, three pieces of evidence reject this explanation. First, adjusting for measurement error in the PGI yields similar results (slope = -0.027, SE = 0.05). Second, the relationship between the reference's PGI and their offspring's PGI is also consistent with the hypothesis of no genotypic assortative mating. Specifically, if an exogenous increase in the reference's PGI caused them to choose a partner with a higher (lower) PGI, we would expect that a one standard deviation increase in the reference's PGI would increase their offspring's PGI by more (less) than 0.5. To investigate this, we estimate the following regression:

$$G_{i,k}^{child} = \varphi 0 + \varphi_1 (G_i^{mom} + G_i^{dad}) + \varepsilon_{i,k}^{child} \tag{11}$$

where $G_{i,k}^{child}$ is the PGI of the reference's k-th child. We the plot the fitted residuals from this regression, $\varepsilon_{i,k}^{child}$,against the fitted residuals from equation (9), ε_i. This is equivalent to estimating:

$$G_{i,k}^{child} = \eta_0 + \eta_1 G_i + \eta_2 (G_i^{mom} + G_i^{dad}) + u_{i,k}^{child} \tag{12}$$

The results are shown in Figure 6b. We cannot reject the hypothesis that $\eta_1 = 0.5$, which is again consistent with random mating with respect to genetic predisposition for educational attainment. Third, we extend this analysis to a PGI for height, a more statistically powerful PGI than the PGI for educational attainment. While the PGI for educational attainment explains about 7% of the cross-sectional variation in years of schooling, the PGI for height

explains approximately 18% of the cross-sectional variation in height. Height is also an observable physical trait, making it easier for individuals to sort on. Nevertheless, despite confirming a positive association between the reference's height and their spouse's height, our results are consistent with random mating with respect to genetic predisposition for height (Appendix Figure XXX).

Section 6 discussed another pathway through which assortative mating may affect the next generation's SES: an individual's genetics may cause them to choose as a reproductive partner someone with specific characteristics and behaviors, which in turn could shape the environment in which the offspring is raised. We proceed conduct two exercises to investigate this hypothesis. First, we study the relationship between the exogenous variation in the reference's PGI and the education of the reference's spouse. Specifically, we estimate:

$$EA_{i,k}^{spouse} = \varsigma 0 + \varsigma_1(G_i^{mom} + G_i^{dad}) + \xi_{i,k}^{spouse} \tag{13}$$

where $EA_{i,k}^{spouse}$ is the years of schooling of the other biological parent of the reference's k-th child. We the plot in Figure 6 the fitted residuals from this regression, $\xi_{i,k}^{spouse}$, against the fitted residuals from equation (9), ε_i. This is equivalent to estimating:

$$EA_{i,k}^{spouse} = \tau_0 + \tau_1 G_i + \tau_2(G_i^{mom} + G_i^{dad}) + u_{i,k}^{spouse} \tag{14}$$

Figure 6c shows that an exogenous increase in the reference's PGI causes them to select a more-educated reproductive partner. Finally, we examine whether the exogenous variation in the reference's PGI is associated with the SES of the reference's parents-in-law, using their income and wealth as proxies. These results are presented in Table 6. For context, the first two columns reproduce select results from Table 2, showing the effect of the reference's PGI on their own SES. The final two columns display the association between the exogenous variation in the reference's PGI and the SES of their parents-in-law. While most estimates in these final columns are not statistically significant, the magnitudes of the point estimates are

	β_1	S.E.	θ_1	S.E.
Income				
Household Income	1.77	0.43	1.78	0.63
Wealth				
Net Wealth	2.05	0.51	1.17	0.69
Housing Assets	2.38	0.60	1.47	0.93
Financial Assets	2.06	0.56	0.58	0.69

Table 6: This table shows estimates of the reference person's PGI on socioeconomic outcomes of the reference's parents in law.

sizeable, providing suggestive evidence that individuals with higher PGIs choose reproductive partners from higher socioeconomic backgrounds.

7 Conclusion

We have shown that the genetic makeup of parents does not only affect their own socioeconomic outcomes but also those of their children once these children have reached adulthood. Parental genetics, as measured through a polygenic index for educational attainment, have a substantial influence on the educational attainment, income, and wealth of their children. We find that generally genetic transmission accounts for about half of the effect of one generation's PGI on the SES of the next, implying that the remaining 50% must be driven by genetic nurture and assortative mating. Evidence for the existence of "genetic nurture" for educational attainment has been mixed (Kong et al., 2018; Nivard et al., 2022). For our wealth-related estimates, the effect of the parental PGI operating through family background trumps the genetically transmitted effect. This reiterates the point made by Black et al. (2020) that the intergenerational transmission of wealth is different than that of education and income, and thus needs to be studied separately.

Overall, our results imply that the effects of genetics should not be studied in a vacuum and that strong genetic effects are compatible with strong environmental mediation. Our results imply that the intergenerational transmission of SES is partially formed because parents transfer their genetic makeup conducive to obtaining certain values of SES to their children at conception. This genetic makeup, in turn, affects the SES of children throughout

their life cycle. It is important to note that such direct effects driven by the child's genetic makeup should *not* be interpreted as evidence for genetic determinism. Studies have shown that PGI effects can interact with the environment, and are thus modifiable by, for example, policy. For example, in the UK, the effect of the EA PGI on wages increased after the school leaving age was raised (Barcellos et al., 2021). The genes included in the EA PGI may operate through a wide variety of behaviors and environments, and, for example, induce people to move to higher educated areas (Abdellaoui et al., 2019). The PGI may also influence various traits that are irrelevant to an individual's educational merit, but that still affect one's education due to social structure or discrimination (Jencks et al., 1972; Coop & Przeworski, 2022). Although we estimate our results in individuals of White European background only, and control for gender, effects of the PGI could still arise due to various sorts of discrimination. Importantly, the extent to which intergerational persistence of SES is genetically driven puts no upper bound on the extent to which policy can attenuate (or even eradicate) this persistence (Goldberger, 1979). Instead, our findings are useful to understand how a specific measure of genetics affects socioeconomic inequality given how the society of the Netherlands is structured. Our results are thus of relevance to policy makers: knowing the extent of genetic transmission of socioeconomic outcomes is vital to knowing *which* policy is most effective in decreasing socioeconomic inequality of subsequent generations.

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