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A CHIP OFF THE OLD BLOCK? GENETICS AND THE INTERGENERATIONAL TRANSMISSION OF SOCIOECONOMIC STATUS

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

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ABSTRACT

Progress in understanding the role of genetics in intergenerational socioeconomic persistence has been hampered by challenges of measurement and identification. We examine how the genetics of one generation influences the SES of the next by linking genetic data from the Dutch Lifelines Cohort to tax records for 2006-2022. Our genetic measure is the polygenic index (PGI) for educational attainment. To isolate causal genetic effects, we exploit randomness in genetic transmission across generations. One generation's genetics impacts the education, income, and wealth of the next. A 10-percentile increase in one generation's PGI raises next generation's education by 0.11 years. "Next-generation genetic effects" are also large relative to "same-generation genetic effects": a 10-percentile increase in a person's PGI raises their income by 0.9 percentiles and their child's by 0.7 percentiles, indicating strong persistence across generations. We next turn to mechanisms: about half of next-generation genetic effects reflect direct genetic inheritance ("genetic transmission"). The remainder operates through environmental pathways ("genetic nurture"): one generation's genetics shapes the circumstances in which the next is raised. This environmental channel is reinforced by assortative mating: high-PGI individuals select more-educated, higher-earning partners. Our findings underscore that genetics is one of the forces anchoring SES across generations.

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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 (Holmlund et al., 2011; Dickson et al., 2016), higher family income (Page, 2024), attending higher-quality schools (Card et al., 2022), or living in lower-poverty neighborhoods (Chetty et al., 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—including their adult children. 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 6% of the cross-sectional variation in years of schooling. As a benchmark, in our data, parental household income explains about half as much of this variation.

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 this, 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. Specifically, 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 again controlling for the PGIs of the reference's 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 includes over 7,000 individuals in the "reference generation" and more than 14,000 in the "offspring generation", with the latter restricted to those who were aged 30 or older in 2022. These samples yield approximately 119,000 and 122,000 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 leads to a 0.11-year increase in their offspring's years of schooling—or 0.18 years when adjusting for measurement error in the PGI. These are sizable effects. To put them into perspective, a 10-percentile increase in the reference's net wealth is *associated with* a 0.23-year increase in their offspring years of schooling—an association that may even overstate the causal effect of parental net wealth.

We also document that the effects of "a genetic shock" are highly persistent across generations. To illustrate, moving a reference 10 percentiles higher in the PGI distribution

increases that reference's own income by 0.9 percentiles, and their offspring's' income by 0.7 percentiles. These findings 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.

We next examine the channels through which the genetics of one generation may influence the SES of the next, distinguishing between two key mechanisms. First, a reference's genetics can indirectly affect their offspring's SES through purely environmental pathways—a process known as *genetic nurture* (Kong et al., 2018; Nivard et al., 2024). Even genetic variants not transmitted to the offspring may influence how they are raised, by shaping the reference's characteristics and behaviors as a parent. Second, the genetic variants inherited by the offspring directly influence their SES through genetic effects, a process we refer to as *genetic transmission*. Assortative mating may modulate these two channels. If references choose genetically similar partners—a phenomenon known as *genotypic assortative mating*—this amplifies the genetic transmission channel, since offspring inherit half of their genes from each of their parents. Alternatively, if references select partners based on particular characteristics and behaviors, this may affect the environment in which their offspring is raised, thereby amplifying (or dampening) the genetic nurture channel.

We find that generally genetic transmission accounts for about half of the effect of the reference's PGI on their offspring's SES, 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 the reference's genetics on their offspring'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 enable 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 offspring'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 children'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 parental genetics in the intergenerational transmission of SES.

We find no evidence of genotypic assortative mating. The PGIs of the reference and their partner are positively correlated ($r = 0.1$), an estimate broadly consistent with prior reports (Okbay et al., 2022; Barban et al., 2019; Conley et al., 2016). However, this association disappears once we control for the sum of the PGIs of the reference's parents, suggesting that the raw correlation likely reflects shared socioeconomic or cultural background, or sorting on observable characteristics. Even under the most conservative scenario, we estimate that genotypic assortative mating would amplify the genetic transmission by no more than 5.1%.

If genetic transmission accounts for roughly 50% of the effect of the reference's genetics on their offspring SES—and genotypic assortative mating contributes little—then the remaining half must be attributed to genetic nurture. This inference is supported by two additional observations. First, this paper shows that the reference's genetics causally influence their own SES, and a large body of research has documented that parents' SES causally affect their children's SES. Second, more direct evidence of genetic nurture comes from studies showing that the PGIs of adoptive parents are positively associated with the SES outcomes of their adopted children—despite the absence of shared genetics (Domingue & Fletcher, 2020; Beauchamp et al., 2023).

Finally, we examine the contribution of assortative mating to genetic nurture. While assortative mating does not appear to modulate the genetic transmission channel, our findings indicate that it may substantially amplify the genetic nurture channel. For example, a one-standard deviation increase in the reference's PGI causes them to select a reproductive partner who, on average, has about 0.23 more years of schooling, compared to 0.46 years for the reference themselves (Collado et al., 2023; Abdellaoui et al., 2023).

This paper contributes to the economics literature on the intergenerational transmission

of SES (Bowles & Gintis, 2002; Björklund et al., 2006; Sacerdote, 2007; Black et al., 2020; Fagereng et al., 2021; Beauchamp et al., 2023; Collado et al., 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 design has greater generalizability—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, amplified by assortative mating, also plays a significant role.

The paper also contributes to the literature on assortative mating by offering new insights into how genetics influences partner selection. Prior work has documented that the PGIs of partners are positively correlated (Conley et al., 2016; Barban et al., 2019; Okbay et al., 2022). We show that this correlation disappears once we condition on the sum of the PGIs of the reference's parents. Our data are consistent with the *phenotypic assortment hypothesis*: individuals appear to select partners based on observable characteristics, such as education, rather than on genotype itself. Because these phenotypes are partially heritable, sorting on phenotype can induce a raw correlation in the PGIs of two partners—even in the absence of direct selection on genotype.

Finally, this paper speaks to a growing literature in social genomics that exploits exogenous genetic variation to study the causal effects of one's genetics on their own SES (Belsky et al., 2018; Burik et al., 2021; Barcellos et al., 2021; Okbay et al., 2022; Houmark et al.,

2024; Carvalho, 2024; Buser et al., 2024; Muslimova et al., 2024). It advances this work by examining how an individual's genetics affects their offspring's SES and by directly 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 2006–2022. Income data are available at both the individual and household levels. We also use information on annual individual earnings, which include both labor earnings and self-employed income. Data cover various components of net wealth, all measured at the household level. These include total assets, total debt, financial assets, housing assets, mortgage debt, and other debt. Financial wealth is further divided into (i) checking and savings balances and (ii) bonds and shares.[1] All monetary values are deflated using the consumer price index provided by CBS, with 2015 as the base year. In some analyses, we measure income and wealth in terms

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

of percentile ranks, calculated within calendar year and year of birth. When estimating effects on levels, we winsorize the top and bottom 1% of the data.

2.2 The Dutch Lifelines Biobank

Lifelines is a prospective cohort of 167,729 individuals who, at the time of recruitment, lived in the Northern Netherlands (i.e., in the provinces of Drenthe, Friesland, and Groningen).[2] 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.[3] 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 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 (see appendix A1).

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, and partners. Family members are identified using a parent-child linkage file, which records the legal parents of each person living in the Netherlands. The term "partner" 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). In the Netherlands, legal

[2]Lifelines is a multi-disciplinary prospective population-based cohort study examining in a unique three-generation design the health and health-related behaviors of 167,729 persons living in the North of the Netherlands. It employs a broad range of investigative procedures in assessing the biomedical, sociodemographic, behavioral, physical and psychological factors which contribute to the health and disease of the general population, with a special focus on multi-morbidity and complex genetics.

[3]GPs were instructed to exclude individuals with a terminal illness, a severe mental illness, those unable to visit the GP, those unable to complete questionnaires, or those unable to understand Dutch.

parenthood is a reliable predictor for biological parenthood, as adoption is rare (affecting <0.5% of the Dutch population (CBS, 2008, 2010)). Any misclassifications 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 also use 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.

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 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 a person inherits one copy from their mother and one copy from the father, 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). 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 s, 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).[4] In practice, this "Genome-Wide Association Study" (GWAS) involved running a series of regressions – one for each SNP – of years of schooling on the individual's number of reference alleles at that SNP, controlling for age, gender, and other covariates. Roughly speaking, the GWAS weights are the coefficients from these regressions—Appendix A2 provides more detail on how these weights were constructed. Following the standard practice in social genomics, the sample is restricted to participants with European genetic ancestries.[5] We standardize the PGI using its cross-sectional standard deviation.

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. The top panel presents the average years of schooling for each quintile, while the bottom panel 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 25 percentage points more likely to graduate from college. The PGI explains approximately 6% of the cross-sectional variation in years of schooling and 5% of the variation in college graduation. As a benchmark, in our data parental household income explains about 3.1% and 3.0% of these outcomes, respectively.

[4]Because effect sizes of each individual SNP are typically very small, a large GWAS discovery sample is critical for constructing a PGI with high predictive power. Okbay et al. (2022)'s GWAS 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.

[5]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 (∼0.3% of the total genotyped respondents).

However, Figure 1 cannot be interpreted causally, as it confounds genetic effects with environmental influences. In particular, the quintiles differ not only in their genetic makeup but also in their socioeconomic backgrounds. Figure 2 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.

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: the cross-sectional variance of the random component is half as large as the cross-sectional variance of the PGI itself (Wang & Xu, 2019).

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 3 plots the density of individuals' PGIs, G_i, separately for those whose parents have low, intermediate, and high PGIs.[6] 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 3 plots the density of the random component, δ_i, for the same three groups. They completely overlap, appearing virtually indistinguishable. Figure 3b 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.[7] We begin by documenting that an individual's PGI is associated with their parents' SES, reinforcing the pattern observed in Figure 2. Specifically, we estimate:[8]

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

where $Y_i^{parents}(t)$ represents the SES of individual i's parents in year t. The top panel of Figure 4 displays the coefficient on individual i's PGI, π_1. Each row corresponds to a different regression using a different indicator 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.

We proceed to examine how these relationships change once we control for the sum of

[6]This analysis uses a different sample from the main analysis, as it requires genetic data for the individual and both of their parents. As explained in Section 4.3, our main analysis only requires genetic data for the individual and either one of their parents or one of their full siblings. Another difference is that, unlike the main analysis, we impose no restriction on the individual's age here. See Appendix Table A1.

[7]As the individual ages, this random component may lead them to self-select into particular environments, which in our analysis will be considered part of the causal effect of the PGI.

[8]This analysis is limited to the 5,975 references (about 85% of the main sample) for whom these measures are available. We find no evidence that the reference's PGI causally affects the likelihood of inclusion in this subsample: a one standard deviation increase in the reference's PGI raises this likelihood by 0.9 percentage points ($p = 0.272$). Appendix Figure A1 repeats the balance test in the largest sample where we jointly observe an individual's PGI, the (imputed) sum of their parents' PGIs, and parental SES measures, and yields the same conclusion.

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 4 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 Empirical Specifications

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:[9]

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

[9]Crucially, the causal effects of a person's genetics on their own outcomes need not operate solely through biological channels but may also work through environmental pathways. As a result, the magnitude of these effects can vary across contexts—precisely the motivation for studying gene-by-environment interactions (Biroli et al., 2025).

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 biological mother, G_i^{dad} is the PGI of the reference's biological father, and $\mathbf{Z}_i(t)$ is a vector of controls. The parameter of interest in equation (5), α_1, is known in the social genomics literature as a "direct genetic effect". It estimates the causal impacts of an individual's genetics on their own outcome, holding constant any environmental factors determined before the individual's conception—including environmental circumstances into which they were born.[10],[11] Notice that the coefficient on the sum of the PGIs of the reference's parents, α_2, cannot be given a causal interpretation.

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:

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

where $Y_{i,k}^{child}(t)$ is the SES of the reference's k-th child in year t.[12] Notice that equations (5)

[10]One may wonder why conditioning on the PGIs of the reference's parents is sufficient for obtaining unbiased estimates—would this not require conditioning on the full vector of parental genetic variants? This follows from the result in Borusyak & Hull (2023), which considers a setting where treatment is a deterministic function of both random shocks and predetermined variables. In our context, the treatment—the reference's PGI—is determined by a combination of the parents' genotypes (the predetermined variables) and random Mendelian segregation (the shocks). The paper shows that when the treatment is generated in this way, unbiased estimation of its causal effect can be obtained by controlling for the expected treatment conditional on the predetermined variables. In our case, the expected treatment is the average of the PGIs of the reference's parents. Specifically, we have: $E[G_i|\boldsymbol{X}_i^{mom}, \boldsymbol{X}_i^{dad}] = E\left[\sum_s^S x_{i,s} w_s | \boldsymbol{X}_i^{mom}, \boldsymbol{X}_i^{dad}\right] = \sum_s^S E\left[x_{i,s}|\boldsymbol{X}_i^{mom}, \boldsymbol{X}_i^{dad}\right] w_s = \sum_s^S \frac{x_{i,s}^{mom}+x_{i,s}^{dad}}{2} w_s = \frac{1}{2}\left(\sum_s^S x_{i,s}^{mom} w_s + \sum_s^S x_{i,s}^{dad} w_s\right) = \frac{1}{2}\left(G_i^{mom} + G_i^{dad}\right)$, where $\boldsymbol{X}_i^{mom}$ and , $\boldsymbol{X}_i^{dad}$ are vectors with the genotypes of the reference's mother and father.

[11]Benjamin et al. (2024) show that α_1 can be interpreted as a weighted average of the causal effects of individual genetic variants. Let κ_1^s denote the causal effect of having an additional copy of the reference allele at SNP s: $Y_i(t) = \kappa_0^s + \kappa_1^s x_{i,s} + \kappa_2^s (x_{i,s}^{mom} + x_{i,s}^{dad}) + u_{i,s}(t)$, where $x_{i,s}$ is the reference's genotype at SNP s, and $x_{i,s}^{mom}$ and $x_{i,s}^{dad}$ are the genotypes of the reference's mother and father. The paper shows that $\alpha_1 = \frac{\Omega' \Sigma K}{\Omega' \Sigma \Omega}$, where Ω is the vector of weights used to construct the PGI, Σ is the variance-covariance matrix of the genotypic data, and K is the vector of SNP-specific causal effects κ_1^s.

[12]In this specification, older offspring and those from references with more children contribute more observations to the regression. Appendix Table A8 shows that our main results are similar when we collapse the data to one observation per reference, averaging first across the years of each offspring and then across all of a reference's children. Beyond being a robustness check, this exercise also highlights a possible mechanism: to the extent that high-PGI references have fewer children, part of the next-generation genetic effect may operate through fertility decisions.

and (6) share the same right-hand side. The only distinction between the two equations is that equation (6) replaces the reference's SES 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. This estimate reflects a particular channel through which the genetics of one generation affects the SES of the next: the PGI is constructed to predict the reference's years of schooling. There may be other pathways through which the reference's genetics affect their offspring's SES that are not captured by the PGI for educational achievement. Although large-scale GWAS for other SES-related outcomes exist, these have not been able to identify genetic variants that predict these outcomes but do not predict educational attainment.[13] As a result, controlling for the education PGI in regressions predicting income or wealth leaves little added predictive power for PGIs targeting those outcomes.

There are additional reasons to interpret β_1 as a lower-bound estimate of the causal effect of one generation's genetics on SES of the next.[14] First, PGIs capture the effects of common genetic variants but omit rare variants. While this is in theory a limitation, in practice the aggregate influence of rare variants is likely modest.[15] Second, PGIs are measured with error due to estimation error in the GWAS-derived weights used to construct them (Becker et al., 2021; Sanz-de Galdeano & Terskaya, 2023; van Kippersluis et al., 2023). We address this issue in Table 4, using an Obviously Related Instrumental Variable approach to correct for measurement error (van Kippersluis et al., 2023). Third, GWAS weights themselves reflect

[13]Specifically, GWAS on occupational status (Akimova et al., 2025) and income (Kweon et al., 2025) all estimate a very large genetic correlation between these outcomes and educational attainment of 0.92 or more. A genetic correlation measures how SNP effects on one trait tend to align with SNP effects on another trait, by assessing how these effects co-vary across SNPs. This indicates that GWAS on SES-related phenotypes other than EA results in a very similar vector of weights. At the moment of writing, a large-scale GWAS of wealth does not yet exist.

[14]Similarly, α_1 can be interpreted as a lower-bound estimate of the causal effect of one generation's genetics on their own SES.

[15]For illustration, Wainschtein et al. (2022) show that the variance explained in height by common variants (i.e., SNP-genotyped data) is between 50%-56%, whereas the variance explained by whole-genome sequence data (which includes rare variants) is 68%. Similarly, for BMI it finds 16-21% for common variants and 30% based on whole-genome sequencing. We do not know of any current estimates, based on whole-genome sequencing, for educational attainment or any other SES-related outcome.

associations between genetic markers and outcomes, not causal effects. Ideally, PGIs would be constructed using weights that capture the true causal impact of each variant. Each of these sources of error likely attenuates our estimates toward zero.

4.3 Main Sample

To implement our empirical strategy, we require genetic data from the reference, their mother, and their father. However, Lifelines includes a small number of cases in which a participant and both biological parents were genotyped. Nevertheless, if genetic data are available not only for the participant but also for one of their parents or a full sibling, it is possible to impute the sum of the genotypes of the participant's parents using *Mendelian imputation*, a methodology recently developed by Young et al. (2022). Economists are often skeptical of imputation techniques because they typically rely on strong, untestable assumptions. In contrast, Mendelian imputation exploits the laws of genetic inheritance—namely, recombination and Mendelian segregation—to recover missing parental genotypes without relying on statistical modeling assumptions. Intuitively, if a full sibling of the participant carries 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. The more genetically distinct two full siblings are, the more information they collectively reveal about the alleles carried by their parents. Any remaining information about the participant's parental genotypes is imputed using population averages.

In practice, we estimate equations (5) and (6) by replacing the sum of the PGIs of the participant's father and mother with the corresponding imputed sum (using the actual sum when both parents are genotyped). Importantly, although the imputed parental PGI sum is a noisy measure of the true value, the estimated causal effect of the participant's PGI remains unbiased.[16] This is because failing to control for parental genotype introduces bias

[16]Despite the imputation, we calculate that the variance in the reference's "residual PGI"—that is, the component of the reference's PGI remaining after controlling for the imputed sum of the parental PGIs—is still about one third of the cross-sectional variance in the PGI.

from genetic variants shared between the participant and their parents—the very variants that can be imputed. In contrast, the portion that cannot be imputed consists of the parents' genetic variants that were not transmitted to the participant, and thus are uncorrelated with the participant's PGI. See Young et al. (2022) for a formal proof.

We can show that Mendelian imputation does not bias our estimates by estimating an alternative "sibling fixed effects" specification—more familiar to economists—in which the sum of the PGIs of the reference's parents in equation (6) is replaced with a fixed effect specific to the reference and their full siblings ($\mu_{f(i)}$):

$$Y_{i,k}^{child}(t) = \gamma_0 + \gamma_1 G_i + \mu_{f(i)} + \mathbf{Z}_i(t)\boldsymbol{\gamma}_2 + u_{i,k}^{child}(t) \tag{7}$$

Appendix Figure A4 illustrates that the results are similar whether we estimate equation (6) or equation (7). The former is our preferred specification because: (i) it is better powered (Young et al., 2022); (ii) it can be estimated even when genetic data for siblings are unavailable, as long as we have genetic data for at least one parent—including cases where the reference is an only child; (iii) it remains estimable even if one sibling has no children—in which case the dependent variable would be missing; and (iv) it avoids potential bias, as equation (7) would yield biased estimates if the genetics of the reference's sibling causally influenced either the reference or the reference's offspring (Young et al., 2022).[17]

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

[17]Specifically, in a family fixed effects design, the effect of the reference's genetics on their offspring cannot be separated from any influence of *the genetics of the reference's sibling* on those same offspring.

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.[18]

Our main sample includes 7,038 individuals from the "reference generation" and 14,117 individuals from the "offspring generation"—that is, the reference's children.[19] The number of annual observations for these two samples is 118,844 and 121,777, respectively, implying that each individual is observed, on average, for more than eight years. In addition, we will conduct a series of supplementary analyses using distinct subsamples, defined by specific requirements for genetic data availability and demographic criteria. Appendix Table A1 summarizes the datasets employed in our analyses.

4.4 Summary Statistics

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 offspring generation. Individuals in the reference generation were born between 1940 and 1969 (average birth year of 1957) while those in the offspring generation were born between 1964 and 1991 (average birth year of 1983.5). Naturally, the offspring generation is observed at a younger age than the reference generation: the average ages at the time of observation were 56.9 years and 37.1 years, respectively. In our sample, women are overrepresented in the reference generation, with approximately two-thirds of the reference individuals being female. Nevertheless, Appendix Table A2 shows that we cannot reject the hypothesis that the effect of the reference's PGI on their offspring's

[18] A potential concern with our design is that our age restriction (requiring offspring to be at least 30 years old) may induce selection on the reference's PGI. If the PGI affects the age at which individuals have their first child, then it could affect the likelihood that a reference and their offspring are included in our sample. Appendix Figure A3 reports how our main results vary with the choice of offspring age cutoff.

[19] For 706 individuals in the offspring generation, we observe the PGIs of both their biological father and mother, as well as the (imputed) sum of the PGIs of the individual's paternal and maternal grandparents. In these cases, we randomly selected either the individual's father or mother to serve as the "reference" and excluded the other parent when estimating equations (5) and (6).

SES is the same regardless of the reference's gender.[20] 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: 14.5 years vs 12.5 years of schooling.[21] While the younger generation earns more, the older generation has accumulated significantly more wealth, primarily due to age-related differences at the time of observation. Despite this, their portfolio compositions are broadly similar.

It is also useful to provide a sense of the magnitude of intergenerational SES transmission in our data. A one-year increase in the reference's education is associated with a 0.23-year increase in the offspring's schooling. Similarly, moving the reference 10 percentiles higher in the household income distribution is associated with a 2.2 percentile increase in the offspring's income, while a 10-percentile increase in parental net wealth is associated with a 3.5-percentile increase in the offspring's net wealth. These estimates are broadly consistent with prior work. For example, Black et al. (2020) report that in Sweden, a 10-percentile increase in parental net wealth is associated with a 3.4-percentile increase in offspring wealth. Van Elk et al. 2024 report the same estimate (2.2) for the intergenerational correlation in household income using a different subsample of the same administrative data employed here—their sample is representative of the population born in the Netherlands.

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.

[20] For some outcomes, the lack of statistical significance may reflect limited power, as the point estimates are economically meaningful.

[21] Because most individuals in the offspring generation did not participate in Lifelines, their education information comes from CBS (available for 89% of the offspring generation), while education data for the reference generation come from Lifelines (available for 98% of the reference generation).

Odd-numbered columns show estimates of α_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, and year-of-measurement fixed effects for the time-variant variables. Only the coefficient on the reference's PGI is reported in the table.

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 0.46 additional years of schooling and increases the likelihood of college graduation by 7 percentage points. It also raises annual individual and household incomes by 7.5 and 3.8 log points (not shown in the table), respectively, moving them 2.5 and 2 percentiles up in these income distributions. Additionally, it raises net wealth by approximately 19,000 (corresponding to a 7.7% increase relative to the average net wealth of €250,540), moving them 1.8 percentiles up in the wealth distribution. This increase is driven by growth in the two major components of total assets: housing assets (up 5.3% relative to the average) and financial assets (up 6.8% relative to the average). We find a small but statistically significant effect on total debt (up 3.0% relative to the average), but no significant effect on mortgage debt. The small effect on debt 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 findings are consistent with a growing body of work in economics examining the relationship between an individual's PGI for education and their SES. Barth et al. (2020) and Papageorge & Thom (2020) documented associations between the PGI and SES outcomes. More recent work has used within-family designs to estimate its causal effects on education (Rustichini et al., 2023; Sanz-de Galdeano & Terskaya, 2023; Muslimova et al., 2024), human capital formation (Houmark et al., 2024), and income (Barcellos et al., 2021; Carvalho, 2024;

Buser et al., 2024). Other relevant studies in social genomics include Belsky et al. (2018), Okbay et al. (2022), and Young et al. (2022). Our contribution to this literature is estimating the effects of the education PGI using rich administrative data on both income and wealth. While Buser et al. (2024) studied its effect on administrative income data, to our knowledge, no prior study has examined its effects on administrative wealth data.

The results in Table 2 preview one of the pathways through which the reference's genetics may influence their offspring's SES: *genetic nurture* (Kong et al. 2018). This mechanism captures the idea that an individual's genetics can affect their children's outcomes not only through genetic inheritance but also by shaping the environment in which those children are raised. Even genetic variants that are not transmitted from the individual to their offspring may still influence the offspring's SES indirectly, by affecting the parent's characteristics and behaviors and, in turn, the family environment. Specifically, individuals with higher PGIs for educational attainment tend to be more educated, earn more, and accumulate greater wealth—resulting in their children being raised in environments shaped by greater socioeconomic advantage. We now turn to estimating the total effect of the reference's PGI on the SES of the next generation, without yet disentangling the specific mechanisms. Later in the paper, we will decompose this effect to quantify the relative importance of the different channels.

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.

The genetics of one generation causally influences the SES of the next. Children of high-PGI parents—like their 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 approximately 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 2.9 log points, respectively (not shown in the table), moving them up by 1.8 percentiles in these income distributions.

We also examine the impact of the reference's PGI on the offspring's wealth.[22] A one standard deviation increase in the reference's PGI raises the offspring's net wealth by €6,098, a 5.6% increase relative to the average net wealth of €109,771, moving them 1.3 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 1.1 percentiles up in the housing assets distribution and 2 percentiles up in the financial assets distribution. This difference likely reflects the age at which we observe the offspring generation. Appendix Table A3 re-estimates these effects, allowing them to vary with the offspring's age. The results indicate that the effect on housing assets increases with age, while the effect on financial assets appears constant across age groups.

5.2.1 Assessing Magnitudes of Next-Generation Genetic Effects

To contextualize the magnitudes reported in Table 3, we benchmark them against the degree of intergenerational SES transmission observed in our data. We use the reference's percentile

[22] One might worry that, for offspring still living with the reference, the effects of the reference's PGI on offspring outcomes measured at the household level (e.g., household income or wealth) may partly capture the effects of the reference's PGI on their own outcomes. This concern is minimal: only 2.8% of offspring live with the reference, and the reference's PGI does not causally impact co-residence (a one-SD increase in the reference's PGI reduces the probability of co-residence by 0.12 percentage points, $p = 0.532$).

rank in the net wealth distribution as a proxy for the older generation's SES (i.e., the reference generation). Columns 1 and 2 of Table 4 report the change in offspring SES outcomes associated with a 10-percentile increase in the reference's net wealth rank. For example, a 10-percentile increase in parental net wealth corresponds to an increase of approximately one-fifth of a year in the offspring's educational attainment.

To facilitate direct comparison with these estimates, we generate an alternative set of estimates in which the reference's PGI—rather than being expressed in standard deviation units—is also expressed in percentile ranks. This allow us to contrast the estimated impact of moving the reference 10 percentiles higher in the *PGI distribution* with the impact of moving them 10 percentiles higher in the *net wealth distribution*—bearing in mind that the latter reflects an association rather than a causal effect.

We provide two sets of estimates of the impact of moving the reference 10 percentiles in the PGI distribution—they differ in whether we adjust for measurement error in the PGI or not. The two intermediate columns present 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.[23] The two last columns present estimates that adjust for the measurement error in the PGI.[24]

Our estimates of genetic effects are sizable when benchmarked against the intergenerational SES transmission observed in our data. The impact on offspring education of moving the reference 10 percentiles higher in the PGI distribution is approximately 50% as large as the impact of a comparable increase in the reference's net wealth. After adjusting for

[23]To obtain these estimates, we estimate a two-stage least squares (2SLS) model in which the endogenous variable is the reference's PGI, expressed in percentile ranks. We control for the sum of the parental PGIs, expressed in standard deviation units, and use the reference's PGI in standard deviation units as an instrument for the endogenous variable.

[24]We use a methodology that is akin to a split-sample IV measurement error correction (van Kippersluis et al., 2023). Specifically, the GWAS discovery sample is randomly divided into two subsamples. Separate GWASs are conducted on each subsample, producing two sets of GWAS weights, which are then used to construct two distinct PGIs. These two PGIs, which serve as noisy proxies for the "true PGI", are used to instrument for each other using Obviously Related Instrumental Variables (ORIV). We constructed the two PGIs using GWAS summary statistics on educational attainment, estimated separately in two halves of the UK Biobank, following van Kippersluis et al. (2023).

measurement error in the PGI, the estimated genetic effect rises to roughly 80% of the corresponding wealth effect. For offspring individual income, the genetic effect is about 40% as large as the wealth effect—rising to approximately 50% after correction. In contrast, the estimated genetic effects on offspring wealth are relatively smaller: about 15% the size of the wealth effect, increasing to 19% with adjustment for measurement error.[25] Taken together, these comparisons indicate that the next-generation genetic effects we estimate are substantial, especially since the intergenerational SES correlations reported here likely overstate the causal effects of increasing parental wealth.

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 5 plots the next-generation genetic effect against the same-generation genetic 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. One would expect this benchmark to hold 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's PGI on their own SES is the same as the causal effect of the reference's PGI on the reference's own SES.

[25] Another useful measure of intergenerational SES transmission is the change in offspring outcomes associated with moving the reference 10 percentiles higher in the household income distribution. Using this alternative measure, the estimated genetic effects on offspring wealth appear comparatively larger—see Appendix Table A4, which contrasts changes in offspring outcomes associated with moving the reference 10 percentiles higher in either the net wealth or household income distribution.

The figure indicates a high degree of persistence in the effects of the reference's genetics across generations. For example, moving a reference 10 percentiles higher in the PGI distribution increases that reference's own income by 0.9 percentiles, and their offspring's income by 0.7 percentiles. 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. Recall 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 two outcomes that lie closer to the 22.5-degree line: housing assets and net wealth. Indeed, Appendix Table A3 shows 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.

To our knowledge, existing studies of natural experiments where one generation was directly exposed and the next was indirectly affected typically find less persistence than we do (Black et al., 2005; Holmlund et al., 2011; Lundborg et al., 2014). For instance, estimates by Holmlund et al. (2011) imply that the effect of a Swedish compulsory schooling reform on the children's education is less than one-tenth of its effect on the directly exposed parents. It is worth noting that some studies do find much higher degrees of persistence Galama et al. (2025). A distinctive feature of "genetic shocks" is that, on average, half of any genetic shock in one generation is biologically inherited by the next. Yet, for most outcomes, we estimate persistence substantially above 50%, suggesting that genetic transmission is not the only channel through which genetic effects operate across generations.

We formally quantify in the next section the contribution of the genetic transmission channel relative to other pathways. Regardless, the findings in Figure 5 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 6 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 begin by distinguishing between two conceptually distinct mechanisms which were briefly discussed in Section 5: *genetic nurture* and *genetic transmission*.

Genetic nurture refers to the idea that a person's genetics can influence their 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 the offspring's 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 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 dia-

gram).

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.

Notice that the diagram in Figure 6 assumes no interaction between genetic transmission and genetic nurture—that is, it assumes that the effect of the offspring's PGI on their SES does not vary with changes in the offspring's family environment induced by exogenous variation in the reference's genetics. Appendix table A5 provides empirical support for this

assumption. Specifically, we allow the effect of an individual's PGI on their own SES to vary with the SES of the individual's parents.[26] For most outcomes, we find no evidence of a significant interaction, and point estimates of the interactions are small, relative to the overall effect of one's PGI.[27]

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 "partner" to refer to the person with whom the reference has a biological child). The reference's partner selection can influence their offspring's SES through two distinct pathways: (1) The offspring inherits half of their genetics from the reference's partner (represented by the hollow red arrow), and (2) The characteristics and behaviors of the reference's partner will also contribute to shaping the environment in which their offspring is raised (represented by the hollow orange arrow). Pathway (1) can be seen as amplifying or dampening the genetic transmission, while pathway (2) operates similarly on the genetic nurture channel.

We proceed as follows. In Section 6.1, we quantify the genetic transmission channel and assess its contribution to the next-generation genetic effects. In Section 6.2 and Section 6.3.1, we examine assortative mating, analyzing pathways (1) and (2) separately.

6.1 Quantifying The Genetic Transmission Channel

In Section 5.2, we exploited the randomness in genetic transmission across generations to estimate our main effect of interest: the next-generation genetic effect. This randomness creates a "natural experiment" at every generation, which we can leverage not only to estimate the overall impact of one generation's genetics on the next generation's economic prospects but also to help identify the mechanisms behind this effect. Specifically, we used the random-

[26] In panel A, we interact the PGI with an indicator for whether the father's net wealth was in the top half of the distribution. In panel B, we interact the PGI with a continuous measure of SES: the father's net wealth percentile rank.

[27] The one exception may be in housing-related wealth outcomes. In Panel B, where we use a continuous measure of parental SES, the effects of the PGI on housing assets, total assets, mortgage debt, and total debt are smaller among individuals from more advantaged socioeconomic backgrounds.

ness in genetic transmission from the reference's parents to the reference to isolate exogenous variation in the reference's genetics and estimate its effects on their offspring's SES. Similarly, we can exploit the randomness in the transmission of genes from the reference and their partner to the reference's offspring to isolate exogenous variation in the offspring's genetics and estimate its effect on the offspring's SES. The contribution of the genetic transmission channel to the overall influence of the reference's genetics on their offspring's SES is equal to the effect of the offspring's genetics on the offspring's SES divided by two.

We can estimate this contribution by estimating the following equation:

$$Y_{i,k}^{child}(t) = \lambda_0 + \lambda_1 G_{i,k}^{child} + \lambda_2 (G_i + G_{i,k}^{partner}) + \mathbf{Z}_i(t)\boldsymbol{\lambda}_3 + v_{i,k}^{child}(t) \tag{8}$$

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 $G_{i,k}^{partner}$ is the PGI of the other biological parent of the reference's k-th child. Notice the similarity between equation (8) and equation (5). The key distinction is that equation (8) 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 (i.e., same-generation genetic effect for the offspring generation) and their parent's PGI (i.e., the next-generation genetic effect) 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 (8))[28], 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

[28]With genetic data on the reference and their offspring, we can impute the sum of the genotypes of the reference and their partner.

for estimating equations (6) and (8).

For this reason, we estimate equation (8) using a different sample from the one used to estimate equation (6). Specifically, we begin with the sample of 39,392 Lifelines respondents for whom both their PGIs and the sum of their parents' PGIs are available. To make this sample as comparable as possible to the offspring generation in our main sample, we take two steps: (1) we restrict this sample to 26,407 individuals born between 1963 and 1992, matching the birth years of the offspring generation in our main sample, and (2) we re-weight this sample to match the year-of-birth-by-gender distribution in the offspring generation of our main sample. See Appendix A3 for details about the construction of these weights.

Appendix Figure A2 compares this sample to the offspring generation in our main sample before and after reweighting.[29] The red circles plot the standardized mean differences prior to reweighting—that is, the absolute difference in group means divided by the pooled standard deviation. The blue circles show the same measure after applying the weights. Even before reweighting, 8 of the 15 variables had standardized differences below 0.1. After reweighting, the differences are further reduced—especially for net wealth, total assets, and its components—so that 13 of the 15 variables fall below the 0.1 threshold. This analysis alleviates, at least in part, concerns that differences between the two samples may bias our estimates of the relative contribution of the genetic transmission channel to the next-generation genetic effects.

Our estimates quantifying the genetic transmission channel are presented in Table 5.[30] 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 3 reports the estimated effect of the offspring's PGI on their own SES (i.e., the coefficient λ_1 from equation (8)), divided by 2. Column 4 presents the standard

[29] Appendix Table A6 shows the complete summary statistics of this same-generation sample, both with and without applying the weights.

[30] We conduct a similar exercise in Table A7, without re-weighting the sample. The results closely mirror those in Table 5 where weights are applied.

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, 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 or assortative mating. 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.[31] Second, genetic transmission appears to account for 74-78% 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 understated the broader ways in which genetics contributes to the intergenerational transmission of SES. If genetic transmission alone cannot account for

[31]In the Netherlands, wealthier parents have various opportunities to gift tax-free inter vivo transfers to their children. During the period that we are studying here, these policies included a one-off tax-free gift of up to €100,000 for children to finance their first house, prepayments, or home improvements (Li & Mastrogiacomo, 2022). Parents also have the opportunity to finance housing of their children as part of a family mortgage. Although parents need to charge their children market-based interest rates as part of this family mortgage, they have the option to gift this amount back to their children up to a tax-free threshold (€6,035 per year in 2023). Such family mortgages are common in the Netherlands, and their value is about 10% of all Dutch mortgage debt (Eijsink & Mastrogiacomo, 2023).

the observed effects, the remaining contribution must arise from genetic nurture or assortative mating. In the next section, we investigate the role played by genotypic assortative mating—that is, the tendency for individuals to choose genetically similar partners.

6.2 Genotypic Assortative Mating

We begin by documenting the degree of similarity between the reference and their partner in both observable characteristics and PGIs. This analysis requires genetic data from the reference, their parents, and their partner. To maximize statistical power, we conduct this analysis with a slightly different sample. Specifically, we do not impose age restrictions on the reference's offspring. However, we restrict the sample to reference individuals and partners who were at least 30 years old.

Table 6 presents the correlation between educational attainment and PGIs for the reference and their partner. The correlation in years of schooling between the reference and their partner is 0.4—commonly referred to in the social genomics literature as "phenotypic correlation". In contrast, the correlation in their education PGIs is substantially lower—about one-quarter as large—at 0.1, a quantity often referred to as as the "genotypic correlation". For comparison, in our sample the correlation between an individual's own PGI and their years of schooling is approximately 0.24.

Our estimates are broadly consistent with those reported in the existing literature. Okbay et al. (2022) estimate a phenotypic correlation of 0.43 and a genotypic correlation of 0.17.[32] Barban et al. (2019) report a phenotypic correlation of 0.56 and a genotypic correlation of 0.13. Conley et al. (2016) find a phenotypic correlation of 0.53 and a genotypic correlation of 0.13. Domingue et al. (2014) estimate that the genetic similarity between partners is at most one-third of the similarity in their educational attainment.

As illustrated, the literature has focused primarily on the raw correlation between the

[32]Okbay et al.'s (2022) genotypic correlation is likely a little higher than ours because their PGIs are more predictive: their PGIs were constructed using a larger GWAS with 23andMe data, which is unavailable to us.

PGIs of partners. However, this moment is not the right object for our goal—quantifying the contribution of genotypic assortative mating to the next-generation genetic effects—as this raw PGI correlation may also reflect shared socioeconomic or cultural background, or sorting on observable characteristics. Instead, we focus on the relationship between the exogenous component of the reference's PGI—specifically, the random deviation from the average PGI of their parents—and the PGI of their partner. If a one standard deviation increase in the reference's PGI causally leads them to select a partner whose PGI is σ standard deviations higher, the offspring would inherit 0.5 units more from the reference and an additional $0.5 \times \sigma$ units from their other biological parent. To estimate σ, we regress the partner's PGI on the reference's PGI, controlling for the sum of the PGIs of the reference's parents:

$$G_{i,k}^{partner} = \chi_0 + \chi_1 G_i + \chi_2 (G_i^{mom} + G_i^{dad}) + \varepsilon_{i,k}^{partner} \tag{9}$$

where $G_{i,k}^{partner}$ 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 own PGI. The coefficient χ_1 provides an estimate of σ.

We find that, once we control for the PGIs of the reference's parents, the correlation between the PGIs of the reference and their partner disappears, decreasing from 0.1 to -0.004 (SE = 0.028). This result suggests that genotypic assortative mating does not meaningfully contribute to the next-generation genetic effects estimated in Table 3. The upper bound of the 95% confidence interval implies that σ is less than 0.051—that is, genotypic assortative mating would amplify the genetic transmission channel by no more than 5.1%.

This finding raises the question of how to reconcile it with the phenotypic correlation shown in Table 6. A useful benchmark is the *phenotypic assortment hypothesis*, commonly assumed in the literature Okbay et al. (2022). This hypothesis posits that individuals select partners based on observable characteristics, such as educational attainment, rather than on genetics. However, because these *phenotypes* are partially heritable, sorting on phenotype can induce genetic similarity between partners, even in the absence of direct selection

on genotype. Under this hypothesis, we can predict how large we would expect σ to be.[33] When we do so, we find that our estimates are consistent with phenotypic assortment, providing a natural reconciliation between our estimate of σ and the phenotypic and genotypic associations reported in Table 6.

One potential concern is that our estimate of σ may be biased toward zero due to measurement error in the PGI. Two pieces of evidence argue against this explanation. First, adjusting for measurement error using ORIV yields similar results (a point estimate of 0.001, SE=0.055). Second, we reach a similar conclusion when examining the relationship between the PGIs of the reference and their offspring. We can also estimate σ indirectly by running the following regression:

$$G_{i,k}^{child} = \eta_0 + \eta_1 G_i + \eta_2 (G_i^{mom} + G_i^{dad}) + \varepsilon_{i,k}^{child} \tag{10}$$

where $G_{i,k}^{child}$ is the PGI of the reference's k-th child. Under this specification, an estimate of σ can be recovered by multiplying η_1 by two and subtracting one. Figure 7 presents the corresponding scatterplot, displaying both the fitted regression line and a benchmark line at $\eta_1 = 0.5$, which corresponds to $\sigma = 0$. The two lines are visually indistinguishable (p-value = 0.66), providing no evidence of genotypic assortative mating.

In sum, we find that genotypic assortative mating does not contribute meaningfully for the next-generation genetic effects. While the PGIs of the reference and their PGI are positively associated, this relationship disappears once we control for the sum of the PGIs of the reference's parents. While some may find this result surprising, we show that our results are consistent with the phenotypic assortment hypothesis, suggesting that the raw correlation between the PGIs of the reference and their partner may reflect sorting on phenotypes rather

[33]Formally, the hypothesis implies that the PGIs of the reference and their partner should be independent once we control for both partners' phenotypes. Under this assumption, the causal effect of the reference's PGI on their partner's PGI should equal the product of three terms: (i) the phenotypic correlation between the reference and their partner (0.4), (ii) the association between the partner's PGI and their own years of schooling (0.24), and (iii) the causal effect of the reference's PGI on their own years of schooling (0.19). This product is approximately 0.019. Our estimated effect of –0.004 (SE = 0.028) is statistically indistinguishable from this value (p-value = 0.791), and we therefore cannot reject the phenotypic assortment hypothesis.

than on genotypes.

6.3 Genetic Nurture

The results presented in this section (Table 5) suggest that, for most SES outcomes, the left-hand side of Figure 6 accounts for no more than half of the next-generation genetic effects. Genetic transmission explains about 50% of the total effect, while genotypic assortative mating contributes little. This implies that the remaining 50% must be attributed to genetic nurture—the influence of the reference's genetics operating through environmental pathways, even when these genetic markers are not transmitted to the offspring. The reference's genetics may affect the offspring's rearing environment through two pathways: directly, by shaping the reference's own characteristics and behaviors, and indirectly, by influencing partner selection, with the partner's characteristics and behaviors also contributing to the environment in which the offspring is raised.

This inference is consistent with two other pieces of evidence: (1) the reference's genetics causally influence their own SES—as shown in Table 2; and (2) parents' SES causally impacts their children's SES—as evidenced by a large literature (Björklund et al., 2006; Sacerdote, 2007; Holmlund et al., 2011; Chetty et al., 2016; Dickson et al., 2016; Black et al., 2020; Fagereng et al., 2021; Card et al., 2022; Beauchamp et al., 2023; Page, 2024). More direct evidence of genetic nurture comes from studies showing that the PGIs of adoptive parents are positively associated with the SES outcomes of their adopted children (Domingue & Fletcher, 2020; Beauchamp et al., 2023). Because adoptive parents and their adopted children do not share their genetics, any association between adoptive parents' PGIs and their children's SES must arise through environmental mechanisms. This interpretation is further reinforced by earlier evidence showing that genetic variants in biological parents that are not transmitted to their biological children still predict the children's SES (Kong et al., 2018).

Our findings stand in contrast to those reported by Nivard et al. (2024). They also use a within-family design that exploits the randomness of genetic transmission across generations.

Yet, they find no evidence of genetic nurture in the academic achievement of Norwegian children, specifically on standardized test scores administered in the 5th, 8th, and 9th grades. More recent work, using the same Norwegian data set and a similar approach, does detect evidence of genetic nurture when outcomes are students' GPAs measured at age 16 (Demange et al., 2025). One possible explanation for this discrepancy is that the standardized tests analyzed by Nivard et al. (2024) are low-stakes diagnostic assessments, rather than high-stakes instruments used for student tracking (Tveit, 2014). Indeed, if family environment plays a meaningful role in shaping children's outcomes—and if parents' genetics influence the environment they provide—it is difficult to see how genetic nurture would not emerge.

6.3.1 Contribution of Assortative Mating to Genetic Nurture

In contrast, less is known about the indirect pathway, through which the reference's genetics influence partner selection and, in turn, shape the offspring's rearing environment. To investigate this mechanism, we examine whether the exogenous variation in the reference's PGI is associated with the education and income of the reference's partner. Specifically, we estimate:

$$Y_{i,k}^{partner} = \theta_0 + \theta_1 G_i + \theta_2 (G_i^{mom} + G_i^{dad}) + \xi_{i,k}^{partner} \tag{11}$$

where $Y_{i,k}^{partner}$ is the SES measured in year t of the other parent of the reference's k-th child.

For this analysis, we return to the main sample. Table 7 presents the corresponding results. Columns 1 and 2 report the effects of the reference's PGI on the partner's education and income. To gauge the magnitudes of these effects, it is useful to compare them to the impact of the reference's PGI on their own outcomes: Columns 3 and 4 report the effects of the reference's PGI on their own education and income. These latter estimates are similar to those in Table 2, with one key difference: to ensure comparability with the first two columns, the sample is restricted to references for whom partner SES measures are available. We do not estimate the effects of the reference's PGI on the partner's household income or on the

partner's wealth, as these outcomes may partly reflect direct genetic effects of the reference's PGI on their own economic outcomes. Even the estimates of the effects of the reference's PGI on the partner's individual earnings and on the partner's individual income should be interpreted with caution, given that partners may make joint labor supply decisions.

The findings indicate that assortative mating may substantially amplify genetic nurture. A one-standard deviation increase in the reference's PGI causes them to select a reproductive partner who, on average, has about a quarter of a year more of schooling, is 5 percentage points more likely to have graduated from college, and ranks approximately 0.9 percentile points higher in the distribution of individual income. All three estimates are statistically significant at the 5% level.[34] In the same sample, a one-standard deviation increase in the reference's PGI raises the reference's education by about half a year of schooling and their likelihood of college graduation by 8 percentage points, and moves them 2.4 percentiles up in the distribution of individual income. Abdellaoui et al. (2023) find similar results in Norway.

At face value, the finding that the exogenous variation in the reference's PGI is associated with the partner's education—but not with the partner's PGI—may appear contradictory. After all, more-educated individuals tend to have higher PGIs. If an increase in the reference's PGI causes them to select a more-educated reproductive partner, one might expect that the partner would also have, on average, a higher PGI. Building on this intuition, we can predict the magnitude of the effect that the reference's PGI should have on their partner's PGI. A one standard deviation increase in the reference's PGI increases the partner's education by 0.23 years of schooling, and each additional year of schooling is associated with an increase of 0.07 standard deviations in the partner's PGI. Taken together, these estimates imply that a one standard deviation increase in the reference's PGI should be associated with a 0.016 standard deviation increase in the partner's PGI. In practice, we estimate that a one standard deviation increase in the reference's PGI is associated with a -0.0037 (SE = 0.0291)

[34]While the point estimates suggest that the reference's PGI may also affect the partner's individual earnings, these effects are not statistically significant. One potential explanation for that is that 28% of partners have zero earnings due to retirement.

standard deviation increase in the partner's PGI. These two estimates are not statistically distinguishable (p-value = 0.71), suggesting that the association between the reference's PGI and the partner's education is fully consistent with the lack of association between the reference's PGI and the partner's PGI. Our results are also consistent with Collado et al. (2023), who find that although their data imply a high rate of assortative matching, the similarity of partners is nearly exclusively due to non-genetic factors.

In sum, these patterns suggest that the reference's genetics shape the environment in which their offspring are raised in two ways: directly, through the reference's own behaviors and characteristics, and indirectly, through their choice of a reproductive partner—who also contributes to the child's rearing environment.

7 Conclusion

Designing effective policies to promote economic mobility requires a deep understanding of how SES advantages are transmitted across generations. Arguably, any account of this phenomenon that overlooks genetics can only ever offer an incomplete picture. This study helps fill that gap by linking genetic data from the Dutch Lifelines Biobank with administrative longitudinal tax records and leveraging the randomness in genetic transmission across generations.

Our findings underscore that genetics is one of the forces anchoring SES across generations: one generation's genetics causally influences the education, income, and wealth of the next. Much of the concern over intergenerational persistence of SES focuses on the perceived unfairness that an individual's economic prospects depend on their socioeconomic background. Our results highlight a parallel, less-discussed source of inequality: children of parents with specific genetic variants also enjoy a head start.

We further document that the effects of "a genetic shock" are highly persistent across generations. To illustrate, moving a generation 10 percentiles higher in the PGI distribution

raises that generation's own income by 0.9 percentiles, and the next generation's by 0.7 percentiles. If the effect of one generation's genetics on the next were mediated exclusively through the inheritance of genetic material from parent to offspring ("genetic transmission"), standard assumptions would predict the latter effect would be half the size of the former.

The high degree of persistence points to the importance of "genetic nurture." One generation's genetics can indirectly affect the next generation's SES through purely environmental pathways. We estimate that for most outcomes roughly half of the effect of one generation's genetics on the next can be attributed to this mechanism. Yet, this channel has been largely overlooked in economics. Prior work has tended to equate intergenerational genetic effects with the inheritance of genetic markers, neglecting the fact that even genes not transmitted from parent to child can shape the environment in which the child is raised, by influencing the parent's traits, behaviors, and resources.

Assortative mating amplifies this genetic nurture channel. We find that a parent's genetics causes them to choose more-educated, higher-earning partners, who in turn contribute to shaping the child's environment. In this way, a parent's genetics affects a child's SES not only through the parent's own characteristics and behaviors but also through this parent's partner selection.

In contrast, we find no evidence of genotypic assortative mating: individuals do not appear to select partners based directly on genotype. Instead, they sort on observable characteristics, such as education, which are themselves partially heritable. This sorting creates raw correlations in the PGIs of partners even without direct selection on genotype.

One of this paper's key contributions is to disentangle the mechanisms through which genetics influences intergenerational socioeconomic persistence, a distinction with important policy implications. The genetic nurture channel operates through the environments parents create for their children and is therefore similar in principle to other environmental factors: policies that reduce disparities in resources and opportunities could, at least in part, mitigate its effects.

The genetic transmission channel, by contrast, is fundamentally different. While families can decide which values and resources to transmit to the next generation and governments can try to influence some of these transfers—for example, through taxation—neither can influence the biological inheritance of genetic material from parent to child (while recent advances in assisted reproductive technologies, such as embryo selection in IVF, have introduced some degree of choice, these remain rare and ethically contested).

Crucially, however, the genetic transmission channel is not immutable. While the inheritance of genetic markers is biologically determined, the effects these markers have on the offspring SES work at least partly through environmental channels and therefore can in principle be modified by policy. Genetic effects do not represent destiny; rather, they depend on social, economic, and institutional contexts.

Future research should further investigate the mechanisms underlying same-generation genetic effects and how policy can influence these effects. Unpacking the complexity of the genetic nurture channel is another priority. Conventional environmental interventions typically alter a single dimension of experience—for example, raising the school-leaving age keeps students in school for an extra year, while holding most other factors constant. By contrast, the genetic differences that lead a student to remain in school longer may also influence a host of other characteristics and behaviors of this individual.

Looking forward, our findings may also be relevant in light of emerging embryo selection technologies that allow parents to screen embryos based on polygenic indexes, including for educational attainment. If access is concentrated among high-SES families with the means to afford them, such technologies could entrench socioeconomic stratification across family lines, further reinforcing intergenerational SES persistence.

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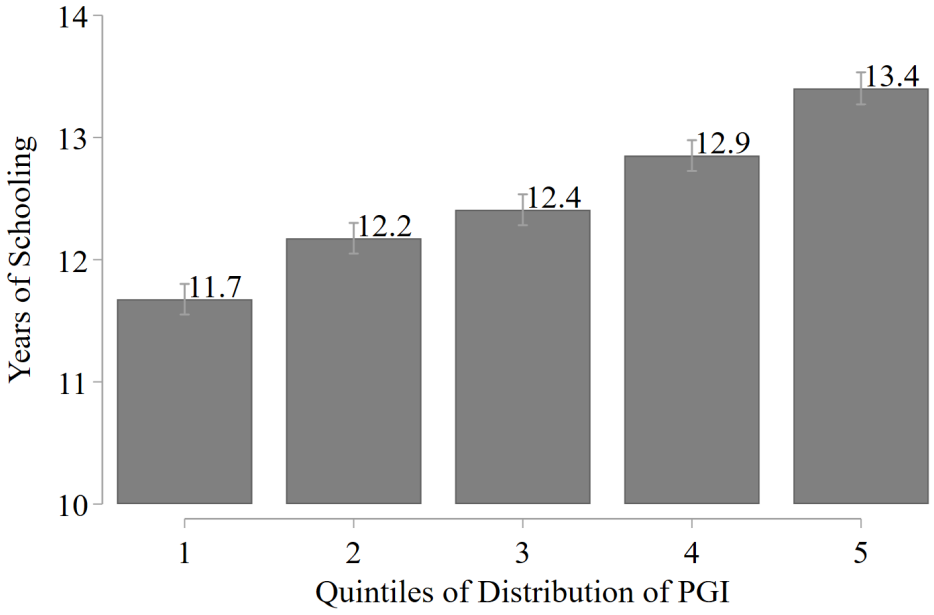
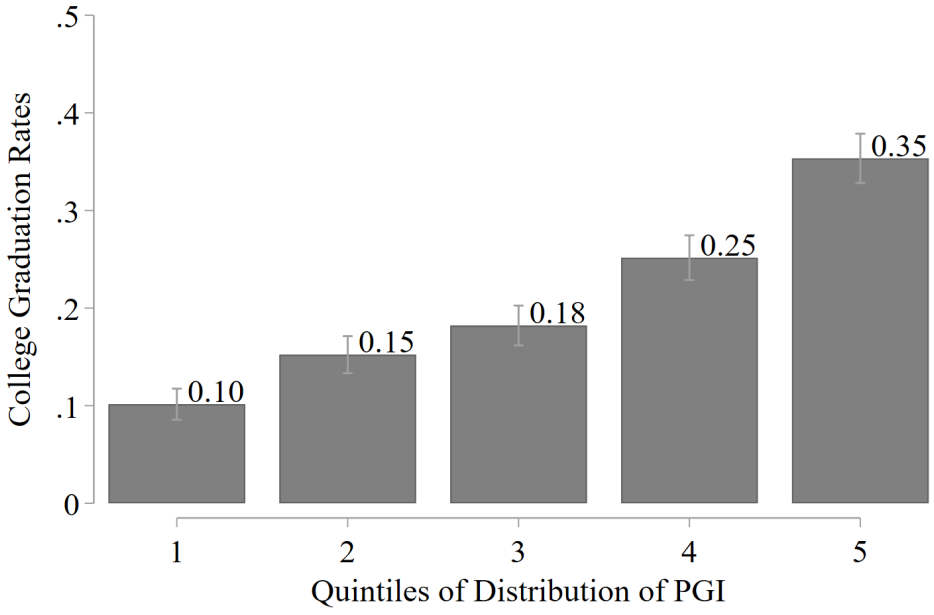
Figure 1: Polygenic Index for Education Predicts Years of Schooling and College Graduation



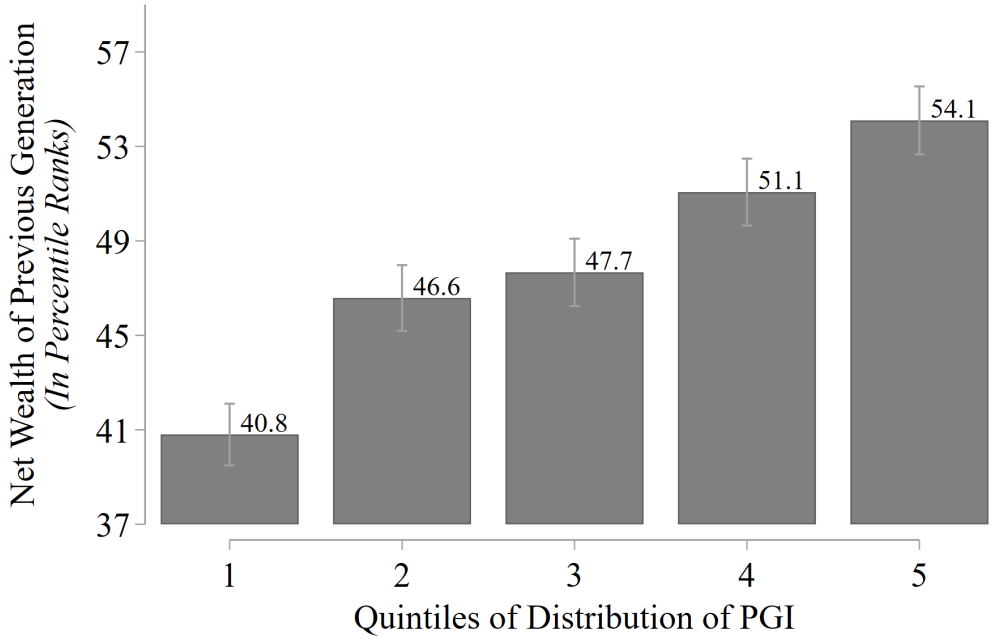
(a)



(b)

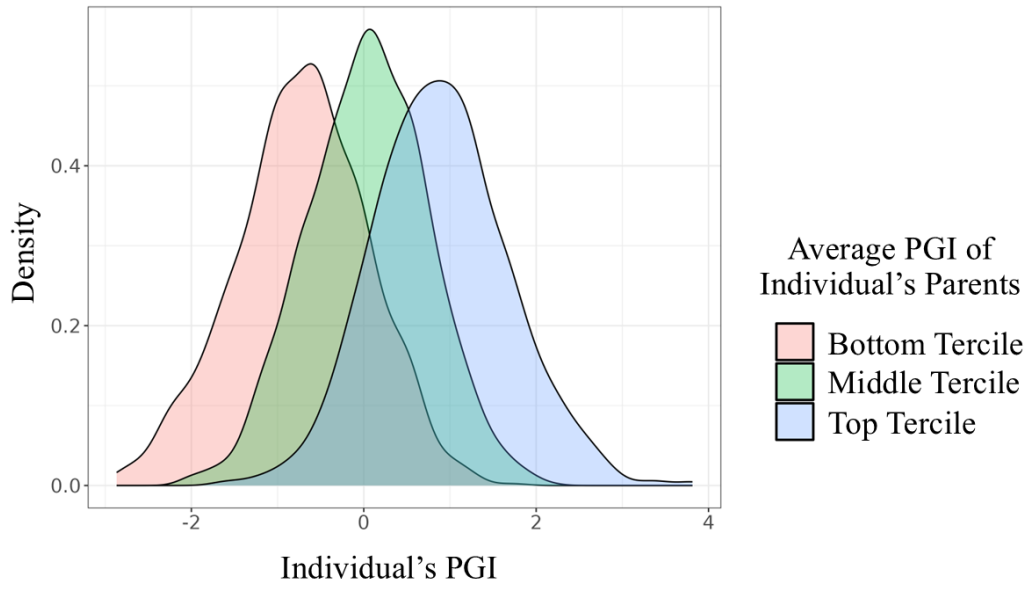
Notes: The sample is divided into quintiles based on the PGI distribution. Panel (a) displays the average years of schooling within each quintile, while Panel (b) presents the college graduation rate by quintile. Brackets correspond to 95% confidence intervals. $N = 6,893$

Figure 2: Net Wealth of the Previous Generation, by Quintile of the Current Generation's PGI Distribution

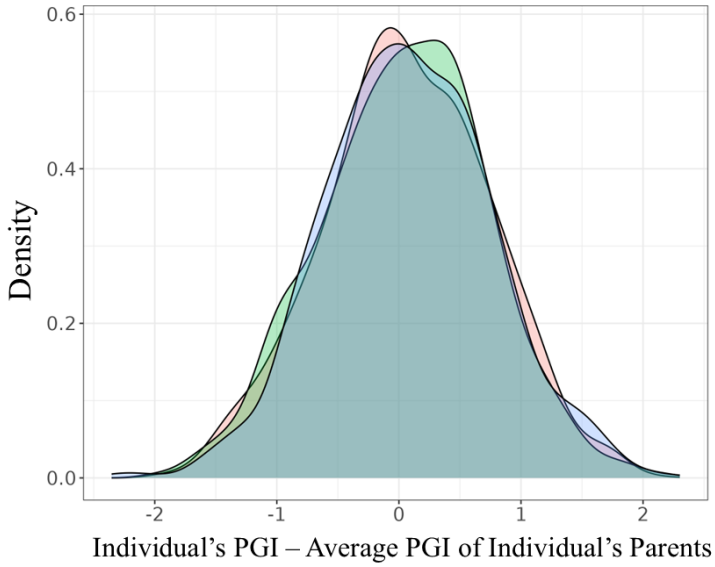


Notes: It displays the average net wealth *of the previous generation* by quintile of the PGI distribution. Brackets correspond to 95% confidence intervals. $N = 5,954$

Figure 3: Distribution of the Current Generation's PGI by Tercile of the Previous Generation's PGI



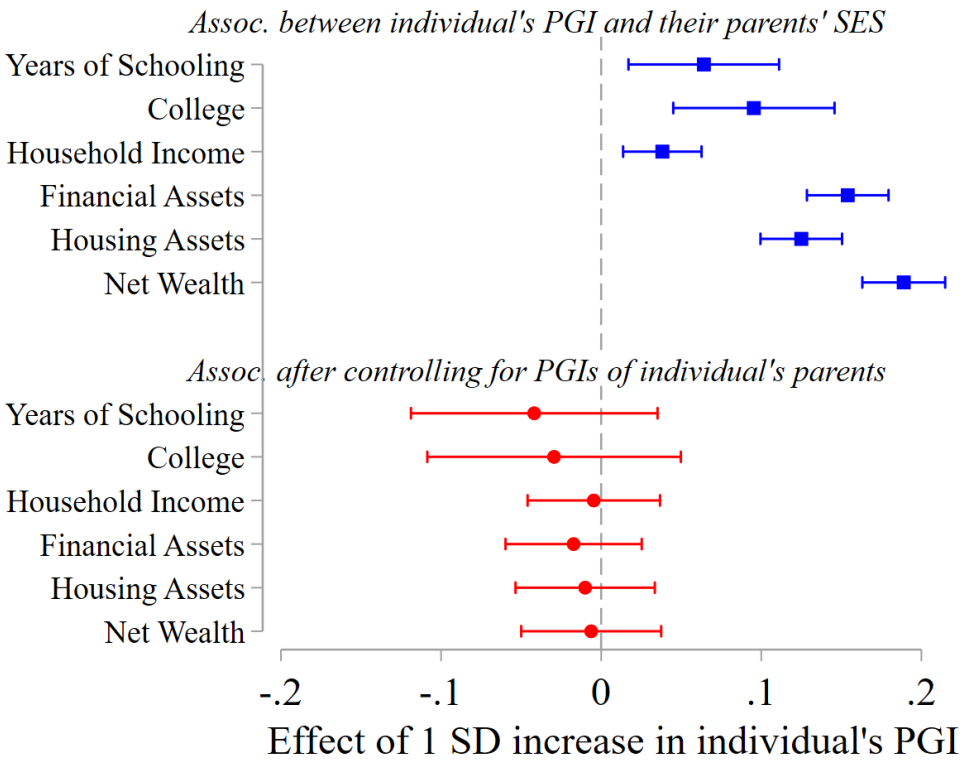
(a)



(b)

Notes: It shows densities separately for three groups of individuals: those whose parents have low PGIs, intermediate PGIs, and high PGIs. Panel (a) displays the distribution of individuals' "raw" PGIs (i.e., G_i in equation 2) across these three groups. As expected, individuals with higher-PGIs parents tend to have higher PGIs themselves. Panel (b) shows the distribution of the deviation of an individual's PGI from the average PGI of their parents (i.e., δ_i in equation 2). The densities in Panel (b) completely overlap, illustrating that individuals with different genetic ancestries have equal chances in this "genetic lottery". N = 3,282.

Figure 4: Balance Test—The Association between the Current Generation's PGI and the Previous Generation's SES

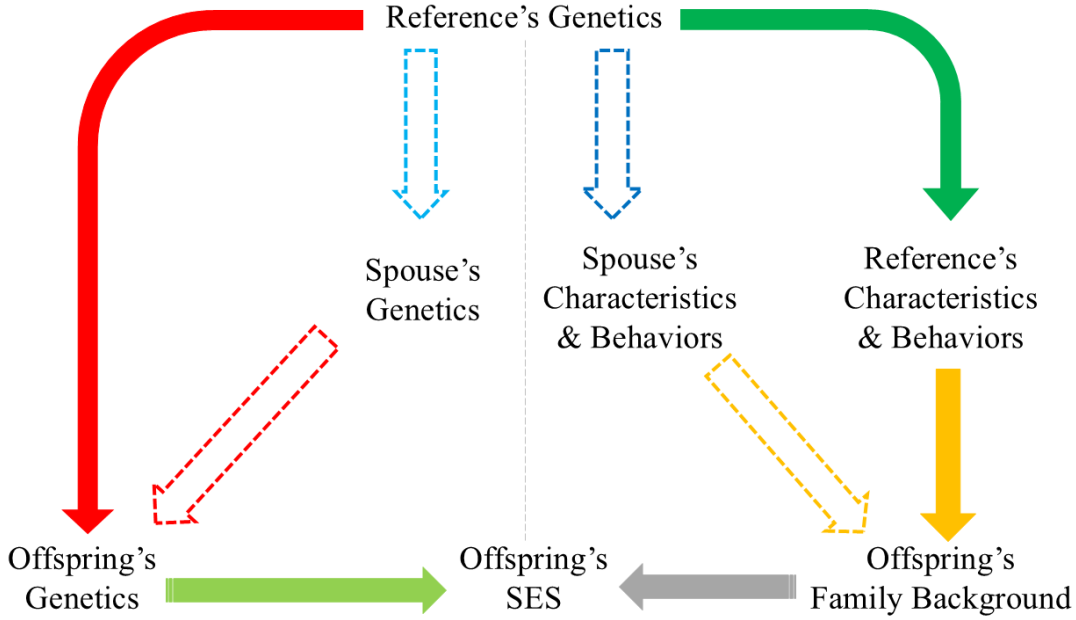


Notes: It examines the relationship between an individual's PGI and their parents' SES, showing that the association disappears once we control for the sum of the parents' PGIs. Each row corresponds to a regression of a parental SES measure (indicated by the row label) on the individual's PGI. Regressions in the bottom panel control for the sum of the parents' PGIs; those in the top panel do not. Markers show the coefficient on the individual's PGI: specifically, estimates of π_1 from equation (3) in the top panel and ϕ_1 from equation (4) in the bottom panel. Brackets display 95% confidence intervals. Standard errors are clustered at the level of the individual. For household income N *individuals* = 5,975 and N *observations* = 85,479. For assets and wealth N *individuals* = 5,474 and N *observations* = 67,257. For Years of Schooling and College, N *individuals* =N *observations* = 1,956, due to missingness in years of schooling in the administrative data for especially older generations.

Figure 5: Comparison of The Effects of The Reference's PGI on Their Offspring's SES and Their Own SES

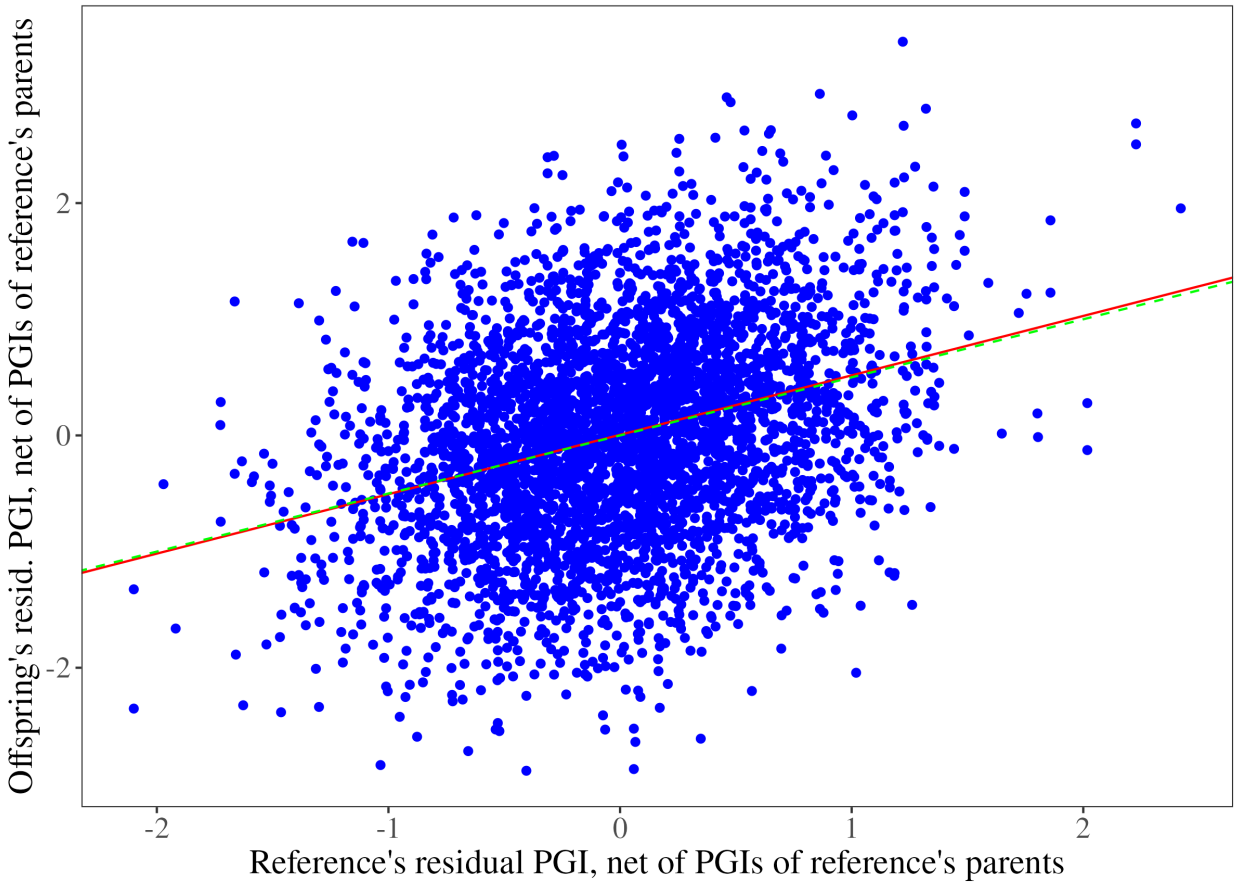
Notes: This figure compares the causal effect of increasing the reference's PGI rank by 10 percentiles on their own SES (x-axis) with its effect on their offspring's SES (y-axis). Each marker represents a different SES outcome. The figure includes a 45-degree line and a 22.5-degree line for reference. The 22.5-degree line represents a theoretical benchmark under which the next-generation genetic effect is exactly half the size of the same-generation genetic effect—consistent with genetic transmission being the sole mechanism through which the reference's genetics influences their offspring's SES. Markers that lie closer to the 45-degree line suggest greater persistence of genetic effects across generations, potentially reflecting additional pathways such as genetic nurture or assortative mating.

Figure 6: The Channels Through Which The Reference's Genetics May Affect The Offspring's SES



Notes: The diagram illustrates the channels through which the reference's genetics may affect their offspring's SES. The path formed by the solid arrows on the right-hand side of the diagram captures the *genetic nurture* channel—the idea that the reference's genetics can indirectly influence their offspring's SES through purely environmental pathways: the reference's PGI affects the reference's characteristics and behaviors, which in turn shape the environment in which their children are raised. The path formed by the solid arrows on the left-hand side of the diagram illustrates the *genetic transmission* channel, whereby the genetic variants inherited by the offspring directly influence their SES through genetic effects. The dashed arrows in the center of the diagram represent the contribution of assortative mating. The hollow red arrow indicates that the offspring inherits half of their genetics from their other biological parent (i.e., the reference's partner), while the hollow orange arrow reflects that the environment in which the offspring is raised is also shaped by the characteristics and behaviors of the reference's partner.

Figure 7: The Causal Effect of The Reference's PGI on Their Offspring's PGI



Notes: It presents a scatterplot of the PGI of the reference's offspring against the PGI of the reference. Both variables are residualized with respect to the PGIs of the reference's parents, so the plot corresponds to a regression of the offspring's PGI on the reference's PGI, controlling for the PGIs of the reference's parents. The figure displays both the fitted regression line and a benchmark line that reflects the theoretical expectation under random mating: namely, that a one-unit (exogenous) increase in the reference's PGI leads to a 0.5-unit increase in the offspring's PGI. The two lines are visually indistinguishable (p-value of 0.66), providing no evidence of genotypic assortative mating. $N = 3{,}852$.

Table 1: Summary Statistics

	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	56.9	8.3	37.1	5.9
Education				
Years of Schooling	12.5	2.5	14.5	2.2
University Graduate	0.21	0.41	0.53	0.50
Income				
Individual Earnings	€28,947	€30,364	€46,157	€29,490
Individual Income	€36,157	€28,203	€48,125	€28,307
Household Income	€85,036	€46,777	€91,239	€47,583
Wealth				
Net Wealth	€250,540	€311,029	€109,771	€235,884
Assets	€373,518	€349,299	€289,241	€292,317
Debt	€118,888	€127,139	€178,134	€143,336
Housing Assets / Total Assets	0.68	0.33	0.67	0.38
Fin. Assets / Total Assets	0.23	0.29	0.26	0.35
Checking & Savings / Fin. Assets	0.90	0.22	0.94	0.17
Stocks & Bonds / Fin. Assets	0.09	0.20	0.06	0.16
Owns Bonds and/or Stocks	0.27	0.44	0.20	0.40
Number of Individuals	7,038		14,117	
Number of Annual Observations	118,844		121,777	

Notes: It reports summary statistics (means and standard deviations) separately for the reference generation and the offspring generation. Educational attainment are available for 6,893 individuals (98% of the total of 7,038) in the reference generation and for 12,625 individuals (89% of the total of 14,117) in the offspring generation.

Table 2: Same-Generation Genetic Effects—The Effect of The Reference's PGI on Their Own SES

	Levels		Percentile Rank	
	α_1	S.E.	α_1	S.E.
Education				
Years of Schooling	0.46	0.05		
College Graduate	0.07	0.01		
Income				
Individual Earnings	€2,410	€410	1.88	0.42
Individual Income	€2,803	€408	2.53	0.41
Household Income	€3,548	€696	2.00	0.42
Wealth				
Net Wealth	€19,213	€5,722	1.76	0.46
Total Assets	€24,714	€6,544	1.81	0.44
Total Debt	€4,659	€2,175	0.95	0.51
Housing Assets	€13,748	€3,150	1.85	0.48
Financial Assets	€4,056	€1,087	1.66	0.45
Checking & Savings	€3,658	€1,098	1.65	0.44
Bonds & Stocks	€683	€575	1.79	0.64
1 if Has Bonds or Stocks	0.02	0.01		
Mortgage Debt	€2,150	€1,839	0.58	0.56

Notes: It estimates the causal effect of the reference's genetics on the reference's own SES—see equation (5). The first two columns report results with outcomes measured in levels, while the last two columns present results with outcomes measured in percentile ranks. Odd-numbered columns show point estimates, 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, 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. Percentile ranks are calculated within calendar year and year of birth (of reference). For income and wealth outcomes, N *individuals* = 7,038 and N *observations* = 117,172-118,844. For education, N = 6,893.

Table 3: Next-Generation Genetic Effects—The Effect of the Reference's PGI on Their Offspring's SES

	Levels		Percentile Rank	
	β_1	S.E.	β_1	S.E.
Education				
Years of Schooling	0.32	0.04		
College Graduate	0.07	0.01		
Income				
Individual Earnings	€2,007	€472	1.74	0.43
Individual Income	€2,056	€453	1.87	0.42
Household Income	€2,995	€782	1.75	0.44
Wealth				
Net Wealth	€6,098	€3,669	1.34	0.45
Total Assets	€10,377	€4,821	1.58	0.43
Total Debt	€4,043	€2,308	0.64	0.44
Housing Assets	€7,760	€2,749	1.13	0.49
Financial Assets	€2,658	€917	2.01	0.44
Checking & Savings	€2,398	€706	1.94	0.44
Bonds & Stocks	€162	€248	1.79	0.55
1 if Has Bonds or Stocks	0.02	0.01		
Mortgage Debt	€3,447	€2,125	0.41	0.49

Notes: It estimates the causal effect of the reference's genetics on their offspring's SES—see equation (6). The first two columns report results with outcomes measured in levels, while the last two columns present results with outcomes measured in percentile ranks. Odd-numbered columns show point estimates, 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, the reference's year of birth and gender, the gender of the reference's offspring, and year-of-measurement fixed effects for the time-variant variables. Only the coefficient on the reference's PGI is reported in the table. Percentile ranks are calculated within calendar year and year of birth (of the offspring). For income and wealth outcomes, N *individuals* = 14,117 and N *observations* = 118-850-120,191. For educational attainment, N = 12,315.

Table 4: Assessing Magnitudes of Next-Generation Genetic Effects

Effect on Offspring of Moving Reference 10 Percentiles Up in the Distribution...	**...of Net Wealth**		**...of the PGI**			
	Estimate	*S.E.*	*Unadjusted*	*S.E.*	*Adjusted for ME*	*S.E.*
Education						
Years of Schooling	0.23	0.01	0.11	0.01	0.18	0.03
College Graduate	0.047	0.003	0.024	0.003	0.038	0.006
Income (in Percentile Ranks)						
Individual Earnings	1.89	0.11	0.64	0.14	0.84	0.28
Individual Income	1.79	0.11	0.67	0.14	0.91	0.28
Household Income	1.86	0.11	0.64	0.14	0.83	0.29
Wealth (in Percentile Ranks)						
Net Wealth	3.54	0.10	0.55	0.15	0.67	0.30
Total Assets	3.20	0.99	0.59	0.14	0.56	0.29
Financial Assets	3.55	0.98	0.77	0.15	0.97	0.30
Housing Assets	2.65	0.12	0.44	0.16	0.29	0.33

Notes: We contextualize the magnitudes of the next-generation genetic effects by benchmarking them against the degree of intergenerational SES transmission observed in our data. Columns 1 and 2 report the change in offspring SES outcomes associated with a 10-percentile increase in the reference's net wealth rank. To facilitate direct comparison with these estimates, the other columns show alternative estimates of the next-generation genetic effects in which the reference's PGI is also expressed in percentile ranks. This allow us to contrast the estimated impact of moving the reference 10 percentiles higher in the PGI distribution with the impact of moving them 10 percentiles higher in the net wealth distribution. We provide two sets of estimates of the impact of moving the reference 10 percentiles in the PGI distribution—they differ in whether we adjust for measurement error in the PGI or not. The two intermediate columns present 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. The two last columns present estimates that adjust for the measurement error in the PGI.

Table 5: Contribution of Genetic Transmission to Next-Generation Genetic Effects

	Total Effect		**Genetic Transmission**		**Ratio**	
	β_1	S.E.	$\lambda_1/2$	S.E.	$\lambda_1/2\beta_1$	95% CI
Education						
Years of Schooling	0.32	0.04	0.16	0.01	0.49	[0.46–0.78]
College Graduate	0.07	0.01	0.032	0.003	0.46	[0.41–0.74]
Income						
Individual Earnings	1.74	0.43	1.36	0.13	0.78	[0.51–1.70]
Individual Income	1.87	0.42	1.39	0.13	0.74	[0.29–1.10]
Household Income	1.75	0.44	0.89	0.13	0.51	[0.49–1.43]
Wealth						
Net Wealth	1.34	0.45	0.45	0.20	0.33	[0.10–0.92]
Total Assets	1.58	0.43	0.61	0.12	0.38	[0.21–0.92]
Financial Assets	2.01	0.44	1.09	0.13	0.54	[0.35–0.91]
Housing Assets	1.13	0.49	0.42	0.14	0.37	[0.09–2.99]

Notes: It quantifies the contribution of the genetic transmission channel to the next-generation genetic effects. The first two columns reproduce results from Table 3, estimating the total effect of the reference's PGI on their offspring's SES: Column 1 reports the point estimate, and Column 2 reports the corresponding standard errors. The two middle columns quantify the genetic transmission channel: Column 3 presents the estimated effect of the offspring's PGI on their own SES (i.e., the coefficient λ_1 from equation (8)), divided by 2, and Column 4 reports the corresponding standard errors. The final two columns assess the share of the next-generation genetic effects attributable to genetic transmission: Column 5 displays the ratio of Column 3 to Column 1, and Column 6 show bootstrapped 95% confidence level intervals for this proportion, based on 500 iterations. λ_1 was estimated in the weighted same-generation genetic effect sample ($N = 26,407$)

Table 6: Assortative Mating

	Reference's Years of Schooling	Spouse's PGI
Spouse's Years of Schooling	0.40	0.24
	(0.015)	(0.016)
Reference's PGI	0.26	0.10
	(0.016)	(0.016)

Notes: It shows the correlation between educational attainment and PGIs for the reference and their partner. Standard errors between parentheses. *N observations = 3,788.*

Table 7: Association between the Random Component of Reference's PGI and their Partner's Education and Income

	Effect on Spouse		**Effect on Reference**	
	θ_1	S.E.	β_1	S.E.
Education				
Years of Schooling	0.23	0.06	0.46	0.05
College Graduate	0.05	0.01	0.08	0.01
Income				
Individual Earnings in Levels	€837	€507	€2,418	€425
Individual Earnings in Percentile Ranks	0.52	0.49	1.79	0.44
Individual Income in Levels	€1,045	€471	€2,737	€422
Individual Income in Percentile Ranks	0.89	0.42	2.37	0.42

Notes: It shows that the random component of the reference's PGI is positively associated with the education and with the income of their partner, presenting estimates of equation (11). Columns 1 and 2 report the effects of the reference's PGI on the partner's education and income. For comparison, Columns 3 and 4 report the effects of the reference's PGI on their own education and income. These latter estimates are similar to those in Table 2, with one key difference: to ensure comparability with the first two columns, the sample is restricted to references for whom partner SES measures are available. In some cases, the reference is linked to multiple partners, reflecting instances where they had children with more than one partner. Standard errors are clustered at the level of the reference. Number of partners N = 4,996 (education) and 7,191 (income and wealth). *N references* = 4,715 (education) and 6,754 (income and wealth). *N annual observations for income and wealth* = 113,798 (partner) and 110,376 (reference).

A1 Quality control procedures of genetic data

In Lifelines, Study participants were genotyped using blood samples drawn during the first assessment visit. Lifelines genotyped data was released for two subsamples. The first is the GWAS CytoSNP cohort which consists of 15,400 unrelated respondents that were 18 years or older. The second is the UGLI cohort which consists of 64,589 respondents aged 8 or older.

Genotypes of the CytoSNP cohort were measured using the Illumina CytoSNP-12v2 array, measuring $\sim$ 300,000 SNPs. Genotypes of the UGLI cohort were measured using the Infinium Global Screening Array®(GSA) MultiEthnic Disease Version, measuring 691,072 SNPs. Both genetic data cohorts were subject to strict quality control procedures prior to release.[35] Further, missing SNPs not measured by the genotyping arrays were imputed using the dense reference panels Genome of the Netherlands and 1000 Genomes. As a result, $\sim$40,000,000 loci are assessed in both subcohorts.[36]

To construct well-estimated PGIs, we performed various quality control (QC) procedures: ensuring only well-estimated SNPs and respondents with reliable genetic data were included. Most of these are recommended by Marees et al. 2018. We only use data on the first 22 chromosomes, ignoring the sex chromosome, which is the smallest. Restricting to the first 22 chromosomes ensures that the EA PGI will not be artificially higher in any of the sexes.

First, we treated the CytoSNP and UGLI cohorts as separate cohorts. Within each cohort, we dropped multiallelic SNPs and loci with a minor allele frequency of $< 1\%$ ($\sim$33.5 million in CytoSNP, $\sim$27.2 million in UGLI). We further dropped SNPs with low imputation quality, as determined by an info score of < 0.8 (744,661 SNPs in CytoSNP in 333,409 in

[35]The quality control reports for CytoSNP and UGLI are available from `http://wiki.lifelines.nl/doku.php?id=gwas` and `http://wiki.lifelines.nl/lib/exe/fetch.php?media=qc_report_ugli_r1.pdf`, respectively.

[36]Reliable imputation is feasible because SNPs are inherited in chunks (called haplotype blocks). This implies that SNPs that are closely located to one another in the genome are highly correlated (R^2 values > 0.99 are not uncommon). To save costs, genotyping arrays are designed to only measure a subset of SNPs in a given genomic region, knowing that reliable imputation can be used to map out the non-measured nearby SNPs.

UGLI). Last, we dropped individuals with excess homozygosity rates (3 standard deviations above or below the average), removing 126 respondents in CytoSNP and 529 in UGLI.

Further, we dropped SNPs that were not in Hardy-Weinberg Equilibrium (with p-value threshold 10^{-6}) (1,163 SNPs in CytoSNP, 22,549 in UGLI)[37])

1,289 respondents in the CytoSNP cohort were also part of the UGLI cohort. We removed these respondents from the CytoSNP cohort to avoid double counting. After all QC steps, there were 6,789,250 SNPs present in CytoSNP, and 7,000,369 in UGLI. We restricted both data sets to the 6,408,251 SNPs that they both had in common, and combined them using the –bmerge command in PLINK.[38]

A2 Details on estimation of PGI weights

We calculated polygenic indices on the genetic data in the Lifelines as follows. First, we used MetaSubtract to correct the publicly available summary statistics in the most recent GWAS on years of education for the inclusion of the Lifelines CytoSNP cohort in GWAS discovery Nolte (2020); Okbay et al. (2022). To implement Metasubtract, we first replicated Okbay et al.'s 2022 GWAS on years of education in the CytoSNP cohort: we first residualized years of education in this cohort from the first 10 principal components of the genetic data, a cube in age, a sex dummy, and an interaction of this sex dummy with the cube in age. We next used plink2 to perform a GWAS on this residualized years of education variable. We verified the validity of this GWAS analysis by checking the genetic correlation between Okbay et al.'s GWAS summary statistics and our own summary statistics conducted within the Lifelines CytoSNP cohort, and found no significant difference from one, as expected.[39]

[37]In both data cohorts, the data providers already performed this quality check prior to releasing the data, but in CytoSNP, they used a more lenient threshold of $P < 0.0001$

[38]The QC report of the first release of the UGLI cohort includes 606 respondents that were also genotyped using the CytoSNP genotyping array. The UGLI Quality Control report assessed that the concordance within individuals of the genotypes assessed using both arrays was extremely high, being 99.82% in the respondent with lowest concordance

[39]Genetic correlations were calculated using LD-score regression Bulik-Sullivan et al. (2015): $r_g = 1.07$ $(s.e. = 0.10)$.

Finally, we used Metasubtract to process the summary statistics of Okbay et al. 2022 prior to PGI construction. Metasubtract analytically subtracts the GWAS summary statistics of CytoSNP from Okbay et al.'s 2022 summary statistics, using inverted versions of the formulas used to meta-analyze GWAS summary statistics. The resulting processed versions of Okbay et al.'s 2022 summary statistics are therefore independent from the CytoSNP cohort.

Using these processed EA GWAS summary statistics, we next constructed PGIs for EA in the Lifelines genetic data, using SbayesR Lloyd-Jones et al. (2019). This algorithm uses Bayesian regression to correct GWAS summary statistics for linkage disequilibrium, using a mixture of normal distributions as the prior. We use linkage disequilibrium scores included in the SBayesR software, estimated on respondents of the UK Biobank.

A3 Construction of inverse probability weights

Weights were calculated inversely proportional to the probability of inclusion in the same-generation sample (used to estimate the effects of the offspring PGI on their own SES). Frequency cells by sex, year of birth, and year of observation were first computed using the offspring generation in the main sample (the sample used to estimate next-generation genetic effects in Table 3). The same-generation sample was then constructed from all genotyped Lifelines respondents born between 1963 and 1992 for whom parental PGI sums could be imputed (see Appendix Table A1). Frequency cells were computed for this sample as well, and weights were assigned to each individual as the ratio of main-sample to same-generation frequencies, normalized to have mean one. To limit imprecision from sparse cells, weights were trimmed at the 1st and 99th percentiles.

A4 Additional Tables

Table A1: Overview of Subsamples Used in the Analysis

Sample	Genetic Data Requirements	Demographic Restrictions	Used In	Main Purpose It Is Used For	Sample Size
(1)	Reference and their parents (imputed)	Reference's offspring older than 30	All analyses except those listed below	Estimate the impact of reference's PGI on their own SES and on their offspring's SES	7,038 in reference generation / 14,117 in offspring
(2)	Individual and their biological parents	None	Figure 3	Show that the deviation of an individual's PGI from the average PGI of their parents is orthogonal to the latter	3,282
(3)	Individual and their parents (imputed)	Individuals born between 1963 and 1992—the birth years of the main sample's offspring generation	Equation (8) and Table 5	Estimate the impact of the offspring's PGI on their own SES	26,407
(4)	Reference, their partner, and their parents (imputed)	Reference and their partner older than 30	Equation (9) and Table 6	Investigate the relationship between exogenous variation in the reference's PGI and their partner's PGI	3,788
(5)	Reference, their offspring, and their parents (imputed)	None	Equation (10) and Figure 7	Investigate the relationship between exogenous variation in the reference's PGI and their offspring's PGI	3,852

Notes: This table summarizes the distinct subsamples employed across our analyses. Sample (1) serves as the main sample and is used in most of the primary analyses. With the exception of Sample (2), it is sufficient to have imputed genetic data for an individual's parents. In contrast, Sample (2) requires the observed genetic data of both biological parents. Sample (1) cannot be used to estimate how the offspring's PGI affects their own SES; for this purpose, we rely on Sample (3). Finally, while the estimates reported in Table 6 do not strictly require genetic data for the reference's parents, we restrict the analysis to the same sample used in equation (9) for consistency.

Table A2: Do Next-Generation Genetic Effects Vary with The Reference's Gender?

	$\mathbf{PGI^{2nd} \times Male^{2nd}}$		$\mathbf{PGI^{2nd}}$		$\mathbf{Male^{2nd}}$	
	β	S.E.	β	S.E.	β	S.E.
Education						
Years of Schooling	-0.03	0.08	0.33	0.05	-0.05	0.05
College Graduate	0.003	0.018	0.068	0.01	-0.015	0.01
Income (percentiles)						
Individual Earnings	-0.25	0.93	1.81	0.51	-0.95	0.56
Individual Income	-0.31	0.92	1.97	0.50	-1.07	0.55
Household Income	-0.90	0.94	2.04	0.53	-0.36	0.46
Wealth (percentiles)						
Net Wealth	-0.38	0.93	1.47	0.56	-0.21	0.57
Assets	-1.18	0.91	1.95	0.53	0.58	0.55
Debt	-1.16	0.92	1.01	0.54	0.98	0.54
Real Estate	-1.10	1.03	1.49	0.60	0.78	0.61
Financial Assets	-0.48	0.93	2.17	0.55	0.17	0.58
Checkings & Savings	-0.48	0.93	2.10	0.55	0.30	0.57
Bonds & Stocks	-0.57	1.18	1.96	0.67	-1.39	0.70
1 if Has Bonds or Stocks	-0.005	0.013	0.022	0.007	-0.017	0.008
Mortgage (Own Home)	-1.14	1.03	0.78	0.61	0.61	0.62

Notes: This table examines whether the effects of a reference's genetics on their offspring's SES differ by the reference's gender. We regress the offspring's outcome on the reference's PGI, an indicator for whether the reference is male, and their interaction. Income and wealth are expressed in percentile ranks (except for the indicator for bond or stock ownership). We cannot reject the null hypothesis that the effect of the reference's PGI on offspring SES is the same for men and women. However, some point estimates are economically meaningful, and the lack of statistical significance may reflect limited statistical power.

Table A3: Do Next-Generation Genetic Effects Vary with The Offspring's Age?

	Reference PGI × Offspring Age		**Reference PGI**		**Offspring Age**	
	β	S.E.	β	S.E.	β	S.E.
Education						
Years of Schooling	0.01	0.01	0.32	0.04	-0.05	0.01
College Graduate	0.00	0.001	0.07	0.01	-0.01	0.001
Income						
Individual Earnings	-0.01	0.06	1.67	0.42	-0.33	0.07
Individual Income	0.00	0.05	1.80	0.42	-0.34	0.07
Household Income	0.03	0.06	1.70	0.43	-0.29	0.07
Wealth						
Net Wealth	0.10	0.06	1.29	0.45	-0.27	0.07
Total Assets	0.14	0.06	1.50	0.43	-0.41	0.07
Total Debt	0.06	0.06	0.59	0.44	-0.31	0.07
Housing Assets	0.19	0.07	1.14	0.48	-0.02	0.07
Financial Assets	0.03	0.06	1.89	0.44	-0.47	0.07
Checkings & Savings	0.03	0.06	1.81	0.44	-0.59	0.07
Bonds & Stocks	-0.05	0.09	1.81	0.54	0.03	0.08
1 if Has Bonds or Stocks	0.00	0.00	0.02	0.01	0.00	0.00
Mortgage Debt	0.12	0.07	0.45	0.49	0.18	0.08

Notes: This table examines whether the effects of a reference's genetics on their offspring's SES vary with the offspring's age. We regress the offspring's outcome on the reference's PGI, the offspring's age, and their interaction. Income and wealth are expressed in percentile ranks (except for the indicator for bond or stock ownership). The results show that the effect of the reference's PGI on offspring housing assets rises sharply with age. A similar pattern emerges for broader wealth measures that include housing, such as total assets and net wealth.

Table A4: Intergenerational Transmission of SES: Associations of Parental Wealth and Income with Offspring Outcomes

	Effect on Offspring of Moving Reference 10 Percentiles Up in the Distribution			
	. . . of Net Wealth		**. . . of Hhld. Income**	
	Estimate	S.E.	Estimate	S.E.
Education				
Years of Schooling	0.23	0.01	0.24	0.01
College Graduate	0.047	0.003	0.049	0.003
Income (in Percentile Ranks)				
Individual Earnings	1.89	0.11	2.04	0.12
Individual Income	1.79	0.11	2.04	0.12
Household Income	1.86	0.11	2.17	0.12
Wealth (in Percentile Ranks)				
Net Wealth	3.54	0.10	0.81	0.12
Total Assets	3.20	0.99	1.64	0.12
Financial Assets	3.55	0.98	1.56	0.12
Housing Assets	2.65	0.12	1.53	0.13

Notes: This table summarizes the degree of intergenerational SES transmission observed in our data. Columns 1-2 present the changes in offspring outcomes associated with moving the reference 10 percentiles higher in the net wealth distribution, while Columns 3-4 present the changes in offspring outcomes associated with moving the reference 10 percentiles higher in the household income distribution. Odd columns report post estimates, even columns robust standard errors.

Table A5: Gene-by-Environment: Do Same-Generation Genetic Effects Depend on Previous Generation's SES?

	Individual's PGI × SES of Individual's Parents		**Individual's PGI**		**SES of Individual's Parents**	
	β	S.E.	β	S.E.	β	S.E.
Panel A						
Education						
Years of Schooling	0.03	0.04	0.36	0.04	0.52	0.03
College Graduate	0.01	0.01	0.07	0.01	0.10	0.01
Income						
Individual Earnings	-0.34	0.47	3.03	0.35	3.14	0.28
Individual Income	-0.21	0.47	3.07	0.35	2.95	0.28
Household Income	0.11	0.49	1.92	0.37	3.80	0.32
Wealth						
Net Wealth	-0.31	0.51	0.79	0.39	12.12	0.30
Total Assets	-0.61	0.47	1.70	0.35	9.90	0.27
Total Debt	-0.76	0.46	1.53	0.37	3.02	0.29
Housing Assets	-0.97	0.52	1.55	0.39	7.42	0.36
Financial Assets	-0.36	0.48	2.15	0.37	11.38	0.32
Checkings & Savings	-0.26	0.48	1.93	0.37	11.09	0.28
Bonds & Stocks	0.36	0.69	1.63	0.49	7.78	0.49
1 if Has Bonds or Stocks	0.00	0.01	0.02	0.01	0.09	0.01
Mortgage Debt	-0.73	0.54	1.59	0.41	0.58	0.32
Panel B						
Education						
Years of Schooling	0.001	0.001	0.38	0.02	0.014	0.001
College Graduate	0.0003	0.0002	0.08	0.01	0.0026	0.0001
Income (percentiles)						
Individual Earnings	-0.006	0.010	2.80	0.23	0.086	0.006
Individual Income	-0.004	0.010	2.93	0.24	0.081	0.006
Household Income	-0.006	0.010	1.94	0.24	0.108	0.006
Wealth (percentiles)						
Net Wealth	-0.016	0.010	0.61	0.25	0.336	0.006
Total Assets	-0.026	0.010	1.32	0.23	0.281	0.006
Total Debt	-0.026	0.011	1.14	0.25	0.002	0.006
Housing Assets	-0.034	0.011	0.96	0.25	0.198	0.006
Financial Assets	-0.014	0.010	1.94	0.23	0.368	0.006
Checkings & Savings	-0.012	0.010	1.76	0.23	0.297	0.006
Bonds & Stocks	0.010	0.015	1.86	0.35	0.227	0.009
1 if Has Bonds or Stocks	0.0001	0.0002	0.021	0.004	0.0025	0.0001
Mortgage Debt	-0.030	0.012	1.14	0.27	0.002	0.007

Notes: This table investigates whether the effect of an individual's PGI on their own SES varies with the SES of the individual's parents. Each outcome is regressed on the individual's PGI, a measure of the SES of the individual's parents, and their interaction, controlling also for the sum of the PGIs of the individual's parents and its interaction with the parents' SES. In Panel A, parental SES is measured by an indicator for whether the father's net wealth was above the median; in Panel B, it is the father's net wealth percentile rank (demeaned). For both Panels A and B, if the father's net wealth was unavailable, we used the mother's net wealth rank instead. The number of observations are: For income and wealth outcomes, $Nindividuals = 25,892$ and $Nobservations = 335,439$; for educational attainment $Nobservations = 25,727$.

Table A6: Summary statistics of the same-generation sample, before and after weighting

	Which Effect is the Sample Used to Estimate?		
	Next-Generation Genetic Effect	Same-Generation Genetic Effect	
		Unweighted	*Weighted*
Demographics			
Male	0.51	0.39	0.46
	(0.50)	(0.49)	(0.50)
Birth Year	1983.5	1976.1	1982.9
	(6.90)	(8.44)	(6.84)
N	14,117	26,407	
Education			
Years of Schooling	14.5	14.6	14.8
	(2.20)	(2.17)	(2.00)
College Graduate	0.53	0.55	0.56
	(0.50)	(0.50)	(0.49)
N	12,625	20,306	
Income			
Individual Earnings	€46,157	€44,648	€47,259
	(29,490)	(28,735)	(27,424)
Individual Income	€48,435	€46,398	€48,731
	(28,307)	(28,030)	(26,654)
N	121,575	343,655	
Wealth			
Net Wealth	€109,772	€160,047	€122,249
	(235,884)	(275,806)	(244,539)
Total Assets	€289,241	€335,965	€302,372
	(293,217)	(312,110)	(283,388)
Total Debt	€178,193	€173,368	€178,428
	(139,317)	(137,622)	(129,719)
Housing Assets	€213,944	€242,050	€216,249
	(183,233)	(179,994)	(176,692)
Financial Assets	€34,491	€44,338	€37,208
	(56,335)	(66,874)	(56,057)
Checkings & Savings	€29,492	€37,067	€32,166
	(42,743)	(50,211)	(43,352)
Bonds & Stocks	€4,147	€6,148	€4,281
	(18,262)	(22,891)	(18,245)
Mortgage Debt	€165,452	€162,547	€167,522
	(132,927)	(121,853)	(120,021)
N	120,366	343,655	

Notes: When quantifying the relative contribution of the genetic transmission channel in Table 5, we use two different samples: the same-generation sample (used to estimate the effects of the offspring PGI on their own SES) and the next-generation sample (used to estimate the next-generation genetic effects in Table 3). To improve comparability, we re-weight the same-generation sample to match the year-of-birth-by-gender distribution in the next-generation sample. Column 1 reports averages for the next-generation sample, while Columns 2 and 3 report averages for the same-generation sample before and after reweighting, respectively. Standard deviations in parentheses. Both samples are restricted to individuals born between 1963 and 1992. See Appendix Table A1 for details on the eligibility criteria of the two samples: the next-generation sample corresponds to Sample (1), and the same-generation sample corresponds to Sample (3).

Table A7: Contribution of Genetic Transmission to Next-Generation Genetic Effects—No Weighting

	Total Effect		**Genetic Transmission**		**Ratio**	
	β_1	S.E.	$\lambda_1/2$	S.E.	$\lambda_1/2\beta_1$	95% CI
Education						
Years of Schooling	0.32	0.04	0.19	0.01	0.58	[0.49–0.82]
College Graduate	0.07	0.01	0.039	0.003	0.56	[0.47–0.82]
Income						
Individual Earnings	1.74	0.43	1.38	0.12	0.80	[0.52–1.67]
Individual Income	1.87	0.42	1.45	0.12	0.77	[0.52–1.49]
Household Income	1.75	0.44	0.95	0.12	0.54	[0.33–1.11]
Wealth						
Net Wealth	1.34	0.45	0.24	0.14	0.18	[-0.02–0.60]
Total Assets	1.58	0.43	0.61	0.12	0.38	[0.20–0.90]
Financial Assets	2.01	0.44	0.90	0.12	0.48	[0.28–0.82]
Housing Assets	1.13	0.49	0.44	0.13	0.39	[0.12–2.74]

Notes: Table 5 in the main text quantifies the contribution of the genetic transmission channel to the next-generation genetic effects. This table replicates the exercise without the re-weighting discussed in Section 6.1. The first two columns reproduce results from Table 3, estimating the total effect of the reference's PGI on their offspring's SES: Column 1 reports the point estimate, and Column 2 reports the corresponding standard errors. The two middle columns quantify the genetic transmission channel: Column 3 presents the estimated effect of the offspring's PGI on their own SES (i.e., the coefficient λ_1 from equation (8)), divided by 2, and Column 4 reports the corresponding standard errors. The final two columns assess the share of the next-generation genetic effects attributable to genetic transmission: Column 5 displays the ratio of Column 3 to Column 1, and Column 6 show bootstrapped 95% confidence level intervals for this proportion.

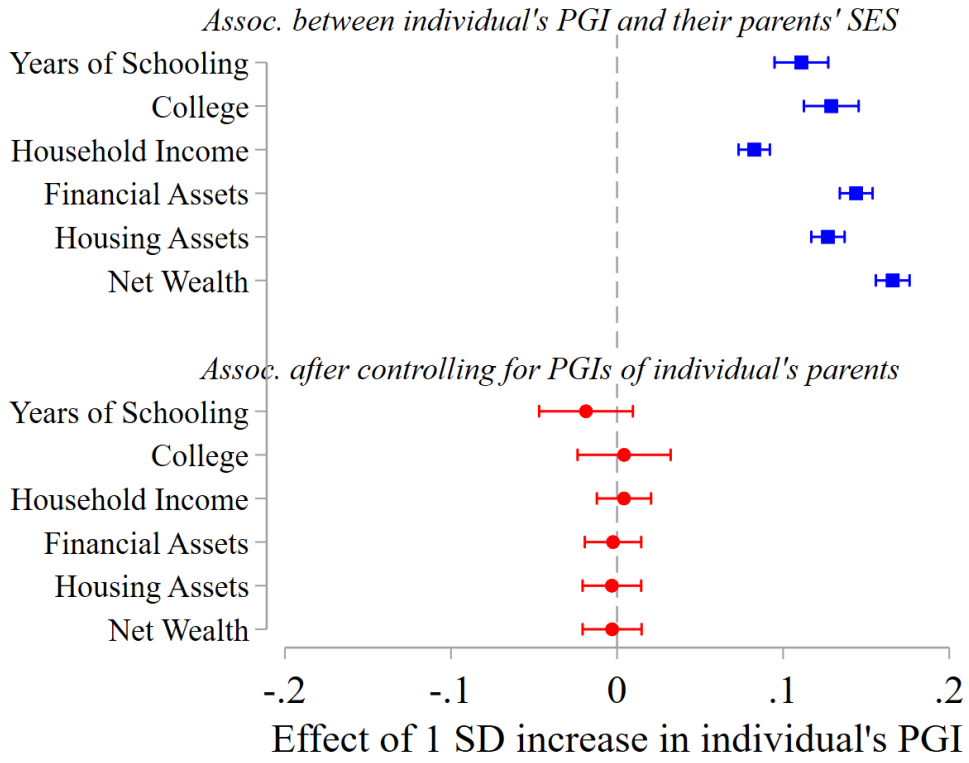
Table A8: Sensitivity of Results to Unequal Number of Observations per Reference

	Levels		Percentile Rank	
	β_1	S.E.	β_1	S.E.
Education				
Years of Schooling	0.32	0.04		
College Graduate	0.07	0.01		
Income				
Individual Earnings	€1,857	€399	1.77	0.41
Individual Income	€1,842	€373	1.87	0.40
Household Income	€2,163	€668	1.34	0.40
Wealth				
Net Wealth	€5,489	€3,021	0.87	0.41
Total Assets	€7,353	€3,953	0.92	0.39
Total Debt	€1,931	€1,927	0.32	0.40
Housing Assets	€4,627	€2,523	0.25	0.48
Financial Assets	€2,073	€702	1.67	0.41
Checking & Savings	€1,872	€557	1.63	0.41
Bonds & Stocks	€220	€200	1.51	0.51
1 if Has Bonds or Stocks	0.02	0.01		
Mortgage Debt	€761	€1,805	-0.25	0.51

Notes: In our main specification (i.e., Table 3 and equation (6)), some references contribute more observations than others—either because a reference has multiple children or because offspring from earlier birth cohorts contribute more years of data. To test the sensitivity of our results to this uneven weighting, this table collapses the data to a single observation per reference. Specifically, we first average outcomes across the years observed for each offspring, and then average across all of a reference's children. We then re-estimate the causal effect of the reference's genetics on their offspring's average SES. The first two columns report results with outcomes measured in levels, while the last two columns present results with outcomes measured in percentile ranks. Odd-numbered columns show point estimates, while even-numbered columns report robust standard errors. All regressions control for the sum of the PGIs of the reference's parents, the reference's year of birth and gender, and the (average) gender of the reference's offspring. Only the coefficient on the reference's PGI is reported in the table. Percentile ranks are calculated within calendar year and year of birth (of the offspring). *N observations* = 6,831 for education and 7,038 for income and wealth.

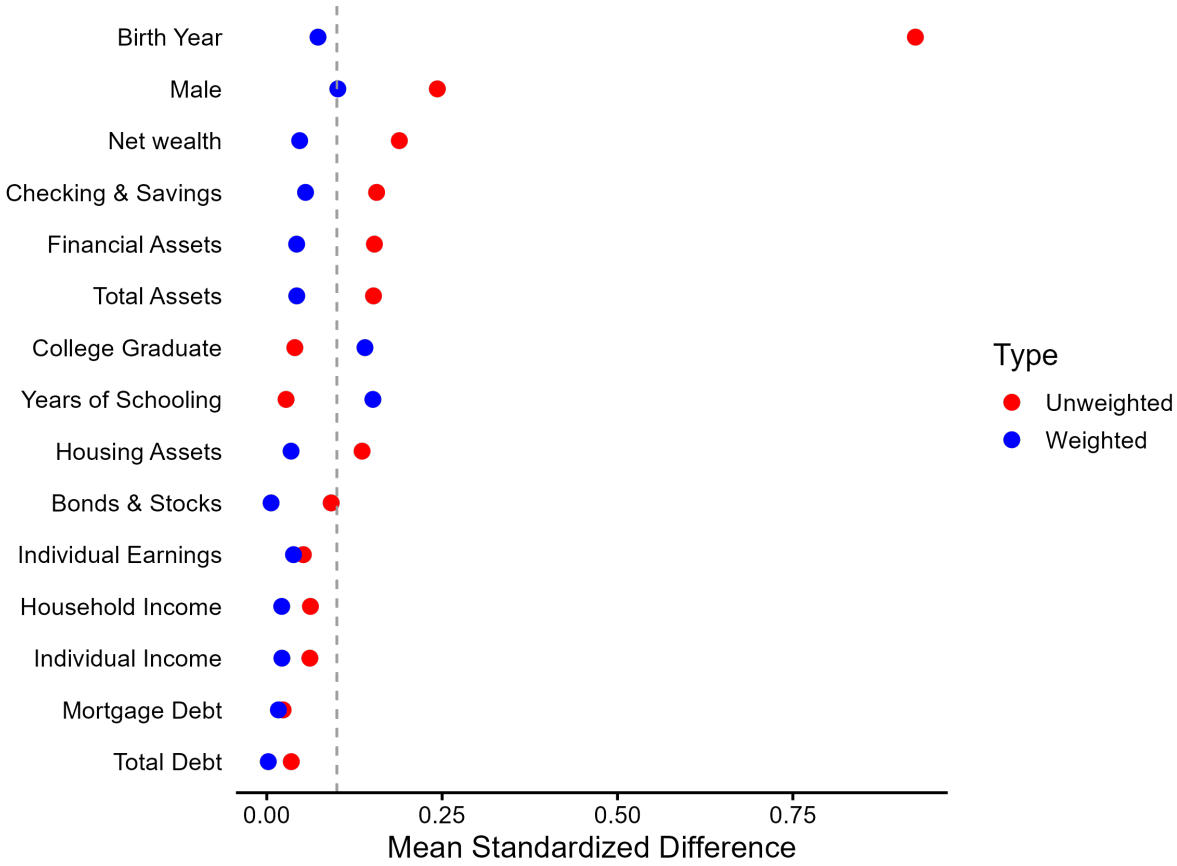
A5 Additional Figures

Figure A1: Balance Test—The Association between the Current Generation's PGI and the Previous Generation's SES



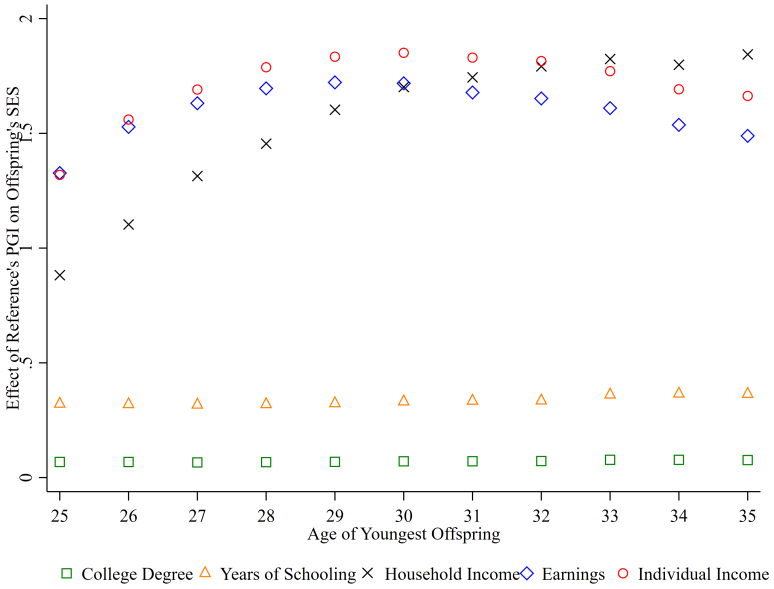
Notes: The analysis in Figure 4 is limited to the references for whom measures of their parents' SES are available. This figure repeats the same exercise in the largest sample for which we jointly observe an individual's PGI, the (imputed) sum of their parents' PGIs, and parental SES measures—see Figure 4 for details on construction. These results lead to the same conclusion. For household income N *individuals* = 32,195 and N *observations* = 586,580. For assets and wealth, N *individuals* = 31,461 and N *observations* = 484,843. For Years of Schooling and College, N *individuals* =N *observations* = 14,677, due to missingness in years of schooling in the administrative data for especially older generations.

Figure A2: Comparing The Two Samples Used for Estimating The Genetic Transmission Channel

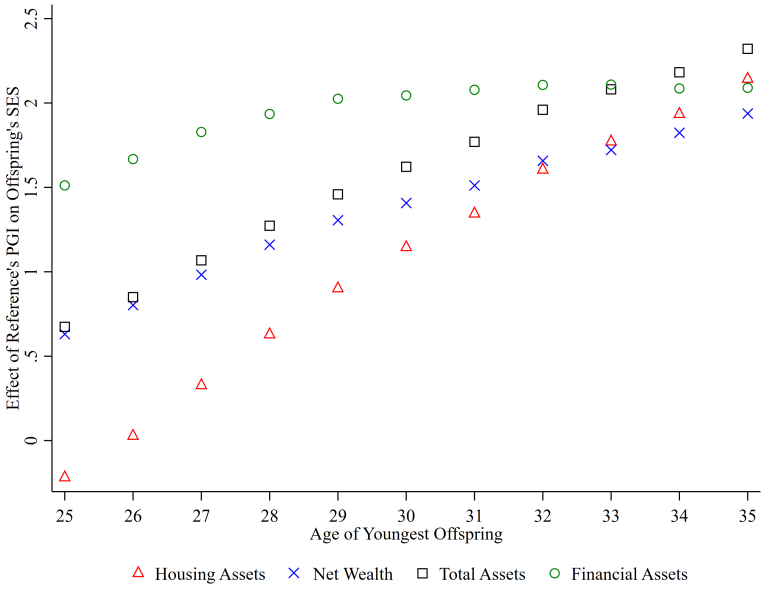


Notes: When quantifying the relative contribution of the genetic transmission channel in Table 5, we use a different sample to estimate the effects of the offspring PGI on their SES than the one used to estimate the next-generation genetic effects in Table 3. To make the former sample as comparable as possible to the latter, we re-weight it to match the year-of-birth-by-gender distribution in the offspring generation of our main sample. This figure compares these two samples before and after reweighting. The red circles plot the standardized mean differences prior to reweighting (the absolute difference in group means divided by the pooled standard deviation), while the blue circles show the same measure after applying the weights. Although both samples are restricted to the same birth-year range, their distributions differ before reweighting. The sample used to estimate the next-generation genetic effects is younger on average because, in addition to the PGI of one parent, we also require the (imputed) sum of the PGIs of that parent's parents—see Appendix Table A1.

Figure A3: Next-Generation Genetic Effects: Sensitivity to Minimum Offspring Age



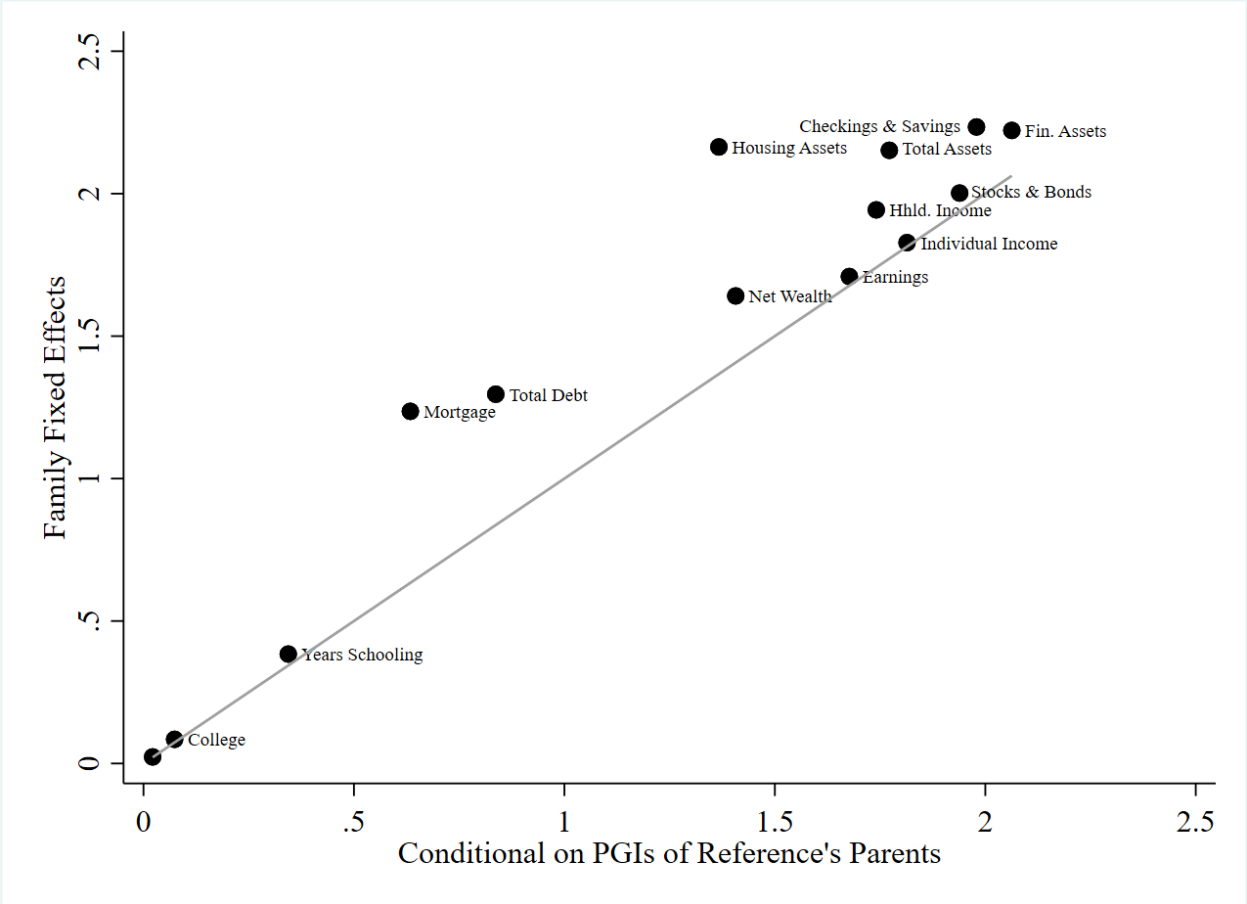
(a) Education and income-related outcomes



(b) Wealth-related outcomes

Notes: A potential concern with our design is that our age restriction (requiring offspring to be at least 30 years old) may induce selection on the reference's PGI. If the PGI affects the age at which individuals have their first child, then it could influence the likelihood that a reference and their offspring are included in our sample. This figure shows how the next-generation genetic effects in Table 3 change as we vary the minimum offspring age cutoff. The x-axis indicates the age of the youngest offspring included. For example, the first markers plotted at 25 correspond to estimates obtained when the sample includes offspring ages 25 and older. Each series of markers traces the estimated effect for a specific outcome across different age cutoffs.

Figure A4: Comparing Estimates of Next-Generation Genetic Effects from Two Alternative Specifications



Notes: This figure compares estimates of next-generation genetic effects obtained from two alternative specifications: one that controls for the sum of the PGIs of the reference's parents (i.e., equation (6), and another that includes a fixed effect for the reference and their full siblings (i.e., equation (7). Estimates from the former are plotted on the horizontal axis, and those from the latter on the vertical axis. Each marker represents the estimated effects for a specific outcome. For any given outcome, both specifications are estimated on the same set of observations. Sibling Fixed effects estimates are based on 4,897 families with at least two genotyped siblings who have children aged above 30, with outcomes available in the administrative data.