Complementarities in human capital production:
Evidence from genetic endowments and birth order

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Abstract

On average, firstborns complete more education than their laterborn siblings. We study whether this effect is amplified by genetic endowments. Our family-fixed effects approach allows us to exploit exogenous variation in birth order and genetic endowments among N = 14,850 siblings in the UK Biobank. We find that those with higher genetic endowments benefit disproportionally more from being firstborn compared to those with lower genetic endowments, providing a clean example of how nature and nurture interact in producing human capital. Moreover, since parental investments are a dominant channel driving birth order effects, our results are consistent with complementarity between endowments and investments in human capital formation.

Keywords: Birth order, complementarity, gene-environment interaction, educational attainment, polygenic score/index

JEL codes: D15, I24, J24

I. Introduction

It is increasingly accepted that important life outcomes such as educational attainment are influenced by a complex interplay between nature (i.e., genetic variation) and nurture (i.e., environments in which one grows up; Rutter, Moffitt, and Caspi, 2006; Heckman, 2007). For example, the formation of skills relevant to educational attainment may result from complementarity between endowments and parental investments (Ben-Porath, 1967; Becker and Tomes, 1986; Cunha & Heckman, 2007). Empirically estimating the separate contributions of genetic endowments and environments, and their possible interaction, is however complicated by the endogenous nature of both. Indeed, environmental characteristics are partially heritable and typically cluster together; e.g., higher educated parents tend to have higher incomes. Hence, disentangling what drives the 'environment-effect' is not straightforward. Similarly, although several population studies have found specific genetic variants that are associated with human capital outcomes such as educational attainment (Lee et al., 2018; Okbay et al., 2016; Rietveld et al., 2013), genetic variation is only random conditional on parental genotypes (e.g., Lawlor, Harbord, Sterne, Timpson, & Davey Smith, 2008; Davey Smith & Ebrahim, 2003). Not controlling for the latter implies that the 'genetic-effect' may in fact reflect 'genetic nurture' – that is, the parental genotype can shape the environment in which children grow up, thereby producing a spurious association between the child's genetic variants and their outcomes (Belsky et al., 2018; Kong et al., 2018).

This paper exploits exogenous variation in both genetic endowments for education and the family environment to analyze the importance of gene-environment interactions for educational attainment. Genetic endowments are measured using a so-called "polygenic score" (PGS), also referred to as a "polygenic index". A PGS is a highly predictive index that can explain up to 13% of the variance in educational attainment, constructed as the sum of all measured genetic variants, weighted by the strength of their correlation with educational attainment (Dudbridge, 2013; Lee et al., 2018). Our measure of the environment is an individual's birth order, which is consistently negatively correlated with educational attainment in developed countries (see e.g. Bagger et al., 2013; Behrman et al., 1986; Black, Devereux, & Salvanes, 2005; Booth & Kee, 2009; De Haan, 2010; De Haan, Plug, & Rosero, 2014; Kantarevic

et al., 2006). [1]

We overcome endogeneity issues by exploiting within-family variation in both birth order and genetic endowments. Indeed, siblings' birth order is randomly determined within families (e.g., Damian and Roberts, 2015), and genetic variants are randomly assigned across siblings within a family according to Mendel's Law of Independent Assortment. Hence, genetic variants are unrelated to birth order by construction. [2] With both exogenous variation in genes and environments, this provides a compelling context in which we can fundamentally improve our understanding of the nature-nurture interplay in shaping life outcomes. Do advantageous environments complement genetic advantages? Answering this question constitutes our first main contribution.

We choose birth order as a measure of the environment not merely for being conveniently uncorrelated with genetic endowments. In fact, birth order has been shown to proxy for parental investments (see below). As such, economic theories of human capital production provide a specific hypothesis of the sign of the interaction term. Becker & Tomes (1986, eq. 4) specify a human capital production function of the form

$$H_t = f(x_{t-1}, s_{t-1}, E_t), \tag{1}$$

where H denotes human capital, x represents parental investments, s denotes public investments, and E refers to genetic and cultural endowments (with index t for time). A critical assumption made to explain intergenerational persistence of earnings and assets is that there are complementarities between endowments and investments (Becker & Tomes, 1986; eq. 5):

$$\frac{\partial^2 H_t}{\partial x_{t-1} \partial E_t} > 0.$$

In words, children with higher (genetic) endowments E benefit more from parental investments x_{t-1}.

The complementarity assumption is not specific to Becker & Tomes (1986). In fact, it is also embedded in so-called "Ben-Porath neutrality" where the stock of human capital raises the

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[1] To illustrate the importance of this environmental determinant, Handy & Shester (2020) estimate that the rise in the fraction of laterborn children was responsible for 20-35% of the stagnation in college completion among US baby-boom cohorts born between 1946 and 1974.

[2] A systematic relationship between birth order and genetic endowments could arise when parents base their fertility decisions on the observed genetic endowments of their offspring, i.e., a stopping rule depending on the "quality" of children (e.g., Eirnæs & Pörtner, 2004). We find no such evidence in our sample; we discuss this below.

productivity of investments in human capital (Ben-Porath, 1967; Heckman, 1976; Rosen, 1976). Moreover, it is also the key building block for the concept of dynamic complementarity in skill production (Cunha & Heckman, 2007), where skills θ produced at time t raise the productivity of investments I_t. That is, $\frac{\partial^2 \theta_{t+1}}{\partial \theta_t \partial I_t} > 0$. Hence, complementarity between the child's endowments and parental investments is a central assumption underlying seminal economic theories of human capital production.

We use an individual's birth order as an exogenous and highly predictive proxy for parental investments because we do not observe parental investments directly, and because realized parental investments are endogenous to the child's endowments (e.g., Almond & Mazumder, 2013; Becker & Tomes, 1986; Behrman, Pollak, & Taubman, 1982; Breinholt & Conley, 2019; Rosenzweig & Wolpin, 1995; Sanz-De-Galdeano & Terskaya, 2019). The theoretical literature on the 'quantity-quality trade-off' (Becker, 1960; Becker & Lewis, 1973; Becker & Tomes, 1976; Galor & Weil, 2000) shows that with each additional child, it is more expensive to maintain the same 'quality' children (i.e., with the same level of education or health), implying that parents invest less in laterborn children. Moreover, with parental preferences for fairness in investments over equality in outcomes (see e.g., Berry, Dizon-Ross, & Jagnani, 2020), parents tend to distribute their resources equally over their children, leading to natural dilutions in time investments for laterborn children compared to their firstborn siblings (Blake, 1989; Downey, 2001).

Indeed, firstborns have undivided attention until the arrival of the second child (Breining, Doyle, Figlio, Karbownik, & Roth, 2020), and the empirical literature suggests that a dominant channel through which birth order affects educational attainment is parental time investments in early life (see e.g., Birdsall, 1991; Black, Grönqvist, & Öckert, 2018; Breining et al., 2020; De Haan, 2010; Del Bono et al., 2016; Monfardini & See, 2012; Pavan, 2016; Price, 2008). Using the American Time Use Survey (ATUS), Price (2008) shows that firstborns receive 20-30 minutes more daily quality time compared to their younger siblings (see also Black et al., 2018; Monfardini & See, 2012), with the gap being largest at early ages. For laterborn children, mothers postpone prenatal care, breastfeed less, are more likely to smoke when not breastfeeding (Lehmann, Nuevo-Chiquero, & Vidal-Fernandez, 2018), and fathers take shorter periods of parental leave (Sundström & Duvander, 2002). Hotz and Pantano (2015) show that parents have less stringent parenting strategies for laterborn children. Hence, whilst we do not

dismiss other potential channels through which birth order may affect educational attainment,[3] parental investments are a prominent channel through which these effects arise, consistent with the evidence that parental investments are an important input into the child's human capital production (e.g., Del Boca, Flinn, & Wiswall, 2014).

Economic theories of human capital production, and in particular the central assumption of complementarity between endowments and investments, therefore, provides a clear prediction for the sign of the Gene-by-Environment (G×E) interaction term: in a sample of siblings, being firstborn should show a positive interaction with genetic endowments. Hence, apart from providing a rare context in which economic theory helps formulate hypotheses about fundamental interactions between genetic variation and the environment, the interaction between birth order and genetic endowments is also informative about the existence of complementarities in human capital production. This constitutes our second main contribution.

Our empirical analysis exploits data from 14,850 full siblings from the UK Biobank, a population-based sample from the United Kingdom (Fry et al., 2017). To measure participants' genetic endowments, we construct polygenic scores for educational attainment based on the results from our own tailor-made genome-wide association study (GWAS) that uses the UK Biobank sample but excludes all siblings and their relatives.[4] We adopt a family fixed effects approach to exploit within-family variation in genetic endowments and birth order to study the G, E and G×E effects on educational attainment, and we apply Obviously-Related Instrumental Variables estimation (ORIV; Gillen, Snowberg, & Yariv, 2019) to reduce random measurement error in the polygenic score. We mainly focus on firstborns versus laterborns, since the literature suggests birth order effects are particularly salient at this margin (e.g., Breining et al., 2020).

We confirm earlier findings that firstborns have a higher level of education than laterborns, and that one's genetic endowments are a strong predictor of educational attainment. We also confirm that genetic endowments do not differ systematically across birth order within a

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[3] Eirnæs & Pörtner (2004) distinguish three additional environmental channels through which birth order effects may arise: (i) younger children may benefit from the interaction with their older siblings; (ii) firstborns benefit from a lower maternal age and better maternal immune system (e.g., Behrman, 1988; Black et al., 2016); and (iii) in some societies the oldest son (or older children more generally) are favored as they are the first to become economically independent. Other channels could be that later born children are exposed to more family disruptions (Björklund, Ginther, & Sundström, 2021), or experiencing respiratory illnesses at younger ages due to older siblings bringing home viruses (Daysal, Ding, Rossin-Slater, & Schwandt, 2021).

[4] See Appendix A for definitions and explanations of the genetic terms used here.

family. This finding corroborates that birth order effects must be due to environmental influences (see also Isungset, Freese, Andreassen, & Lyngstad, 2021), such as greater parental time investments in firstborns. Our main finding is that birth order and genetic endowments interact: being firstborn and having higher genetic endowments for education exhibit a positive interaction, meaning that those with a higher polygenic score benefit disproportionally more from being firstborn compared to those with a lower polygenic score. This finding is a clean example of how genetic endowments and the environment interact in producing important life outcomes such as educational attainment. Moreover, our empirical results are consistent with the existence of complementarity between endowments and investments in human capital production: additional parental time investments associated with being firstborn are more 'effective' among those siblings who randomly inherited higher genetic endowments for educational attainment.

Our paper speaks to three main literatures. First, we contribute to an emerging literature on gene-environment interactions (G×E), which addresses how the environment moderates the effect of genetic variants, and vice versa. Previous studies have typically examined interactions between polygenic scores and endogenous environments such as socio-economic status (e.g., (Barth, Papageorge, & Thom, 2020; Bierut, Biroli, Galama, & Thom, 2018; Ronda et al., 2020), childhood trauma (e.g., Mullins et al., 2016; Peyrot et al., 2014), or a partner's death (e.g., Domingue, Liu, Okbay, & Belsky, 2017). The interpretation of these findings is complicated, because individuals with certain genetic predispositions may self-select into different environments (known as gene-environment correlation, or *rGE*; see Jencks, 1980; Schmitz & Conley, 2017a). In these analyses, therefore, the 'environmental effect' could be reflecting the effect of one's genotype through *rGE*, and the 'genetic effect' could be reflecting the rearing environment shaped by parental genotype (i.e., genetic nurture). A handful of studies use exogenous variation in environments to study G×E in educational attainment. For example, Conley & Rauscher (2013) analyze how random differences in the prenatal environment alter the genetic effects on education, depression, and delinquency. Schmitz & Conley (2017b) use the Vietnam War conscription as a natural experiment, Barcellos, Carvalho, & Turley (2021) use a UK compulsory schooling reform, and von Hinke & Sorensen (2022) explore early life

pollution exposure to study G×E in individuals' educational attainment. [5] We push this literature one step further by not only considering exogenous variation in the environment, but also in genetic endowments by exploiting within-family variation in polygenic scores.

A second strand of literature that we speak to is the literature on birth order effects. This literature consistently finds that in developed countries, laterborn children have lower educational attainment. Birth order effect have also been found for other outcomes, though sometimes with mixed results, such as early life cognitive skills and intelligence (Black, Devereux, & Salvanesz, 2011; Fenson et al., 1994; Keller, Groesch and Trob, 2015), health (Black, Devereux, & Salvanes, 2016; Pruckner et al., 2019), personality and leadership skills (Black et al., 2018), and delinquency (Breining et al., 2020). We contribute to this literature by studying heterogeneity in the birth order effect on educational attainment with respect to genetic endowments. The potential interaction between birth order and genetic endowments is not merely an important source of heterogeneity in the treatment effect, but one that – if present – carries over to the next generation, potentially exacerbating intergenerational inequalities (Barclay, Lyngstad, & Conley, 2021; Havari & Savegnago, 2022).

Finally, our study offers new insights on human capital production (e.g., Becker and Tomes, 1986; Cunha & Heckman, 2007; Cunha et al., 2010; Todd & Wolpin, 2003). Empirically testing complementarity in human capital formation requires independent variation in initial endowments and subsequent investments (Almond & Mazumder, 2013; Johnson & Jackson, 2019), and is therefore extremely challenging (Almond, Currie, & Duque, 2018). Indeed, the previous literature has almost exclusively relied on early-life outcomes such as birthweight as a measure of endowments (e.g., Datar, Kilburn, & Loughran, 2010; Figlio, Guryan, Karbownik, & Roth, 2014). However, such early life outcomes are affected by prenatal investments (Aizer & Cunha, 2012; Rosenzweig & Schultz, 2015), meaning they partially capture parental choices and are therefore endogenous. Furthermore, parents respond to children's endowments (e.g., Adhvaryu & Nyshadham, 2016; Aizer & Cunha, 2012; Almond & Mazumder, 2013; Becker & Tomes, 1986; Bharadwaj, Eberhard, & Neilson, 2018; Datar et al., 2010; Frijters, Johnston, Shah, & Shields, 2013; Giannola, 2020; Hsin & Felfe, 2014), with recent studies suggesting that parental investments also respond to the genetic endowments of children (Breinholt & Conley,

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[5] Studies with other outcomes that exploit exogenous environments include, e.g., Barban, Cao, & Francesconi (2021), Barcellos, Carvalho, & Turley (2018), Pereira, van Kippersluis, & Rietveld (2020), and Schmitz & Conley (2016).

2019; Fletcher, Wu, Zhao, & Lu, 2020; Houmark, Ronda, & Rosholm, 2020; Sanz-de-Galdeano & Terskaya, 2019). Hence, measures of children's endowments are rarely clean of parental investments, and parental investments are rarely independent of endowments, posing a formidable empirical challenge to accurately identify complementarities in human capital production.[6]

We contribute to this literature by exploiting random within-family variation in genetic endowments, and birth order induced reductions in parental investments. Indeed, our measure of endowments is randomly assigned within families and fixed at conception. It is therefore clean from parental investments. Furthermore, we proxy for parental investments using individuals' birth order, which is strongly associated with parental investments, but uncorrelated with genetic endowments. Essentially, employing birth order as a proxy for parental investments exploits the natural reduction in the time and money available with the arrival of a laterborn child, and is thus independent of the child's endowments. There could be other channels through which birth order may impact educational attainment, such as through interaction with younger siblings (Eirnæs & Pörtner, 2004). However, unless these other channels have completely opposite interaction effects with genetic endowments, birth order and genetic endowments provide a promising setting to empirically test *a necessary condition for* complementarity in human capital production.

Finding support for complementarity between endowments and investments is important for understanding the nature of the human capital production function. The production function of the child's human capital is – next to a parental budget and time constraint – an important input into the broader optimization problem where parents decide between own consumption and investments in their children. By informing the shape and properties of the production function, our analysis is an important precursor to a structural model of parental investment decisions, estimation of which is beyond the scope of this paper (see Houmark et al. (2020) for a recent application that incorporates polygenic scores into a dynamic latent factor model of skill formation). Evidence in support of complementarity also speaks to whether later-life investments can reduce or eliminate damage originating early in life (Almond et al., 2018), and

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[6] A recent set of studies has examined rare cases where exogenous variation exists in both initial endowments as well as later-life investments, with mixed evidence. Some studies find evidence consistent with complementarity (Adhvaryu et al., 2019; Duque et al., 2018; Gunnsteinsson et al., 2014; Johnson & Jackson, 2019), whereas others find weaker evidence or even substitutability between endowments and investments (Lubotsky & Kaestner, 2016; Malamud et al., 2016; Rossin-Slater & Wüst, 2020). See Appendix B for a detailed overview.

emphasizes the importance of early-life investments being followed-up by later-life investments to reap the full benefits in terms of human capital outcomes (e.g., Cunha & Heckman, 2007).

More generally, this paper shows how economic theory can inform empirical G×E analyses, and how genetic data can be leveraged to test economic theories. We show that the analysis of G×E within-family, exploiting exogenous G as well as E, provides a way to test for complementarities more generally, which is not restricted to birth order effects, but extends to other (exogenous) parental investments and policy changes (e.g., on student-teacher ratios, minimum school leaving ages, etc.). Our findings further provide one of the first pieces of causal evidence of how genetic variation (here measured by the polygenic score for years of education) and the environment (here measured by birth order) jointly shape and interact in producing important life outcomes such as educational attainment. While this finding was long anticipated by numerous scholars (e.g., Heckman, 2007; Rutter et al., 2006), finding credible and independent sources of variation in genes and environments is rare given how tightly genetic and environmental influences are entangled (e.g., Koellinger & Harden, 2018). Showing evidence of an interaction between genetic variation and environments is therefore a leap forward in our fundamental understanding of how nature and nurture jointly shape human capital, and may also provide an antidote against arguments of genetic or environmental determinism.

II. Empirical Strategy

We analyze gene-environment (G×E) interactions between genetic endowments for educational attainment and birth order. The empirical specification is rooted in the human capital production function (1), where we take adult educational attainment as a proxy for human capital by the end of childhood, as in Cunha & Heckman (2008) and Cunha et al. (2010).

Following Todd & Wolpin (2003) and Cunha & Heckman (2008), we specify a linear production function, where years of completed education for individual *i* of family *j* (denoted by Y_{ij}), is a function of initial endowments, parental investments, and unobserved parental characteristics. Empirically, we measure initial endowments by the polygenic score for educational attainment (denoted by G_{ij})[7], we proxy parental investments by an indicator for

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[7] For ease of interpretation, we standardize G_{ij} to have mean zero and standard deviation one. See Section 3 and Appendix A for more information about its construction.

being firstborn (E_{ij}), and assume parental characteristics are fixed and therefore subsumed into the family fixed effect (δ_j). We then allow for an interaction term between G_{ij} and E_{ij}, leading to the following specification:

$$Y_{ij} = \alpha_1 + \alpha_2 G_{ij} + \alpha_3 E_{ij} + \alpha_4 (G_{ij} \times E_{ij}) + \alpha_5 X_{ij} + \delta_j + \xi_{ij}, \qquad (1)$$

where X_{ij} is a set of individual level controls, including gender, month and year of birth dummies (Black et al., 2005; Handy & Shester, 2020). It also includes the vector of the first 40 principal components (PCs) of the genetic relatedness matrix.[8] Finally, ξ_{ij} is the error term; we use heteroskedasticity-robust standard errors, clustered at the family level. The parameter α_2 captures the association between the standardized polygenic score for education and years of schooling, whilst α_3 estimates the average advantage in years of schooling for firstborn children compared with their laterborn siblings. α_4 shows the extent to which the polygenic score and being first born complement each other's effect on education and is therefore informative about the existence of putative G×E effects and complementarity between endowments and investments.[9]

Following Black et al. (2005, 2011), Heiland (2009), and Lehmann, Nuevo-Chiquero, & Vidal-Fernandez (2018), we compare within-family and between-family specifications. The inclusion of family fixed effects theoretically ensures that variation in the polygenic score and birth order is random, ensuring polygenic score and birth order are orthogonal to each other, and we verify this empirically in Section 4.5. Hence, we avoid endogeneity concerns arising from omitted variables by comparing siblings within the same family. Similarly, the within-family analysis ensures that the child's polygenic score does not capture any parental genetic (nurture) effects.

Our polygenic score incorporates two sources of measurement error. First, since the GWAS underlying the construction of a polygenic score is based on a finite sample (see Section 3), our estimated polygenic score that is based upon the GWAS estimates is a noisy proxy for the true (latent) polygenic score (e.g., Benjamin, Cesarini, Laibson, & Turley, 2020; Van Kippersluis et

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[8] Genetic principal components (PCs) can be used to control for subtle forms of population stratification (i.e., correlations between allele frequencies and environmental factors across subpopulations in the sample) in a between-family (population-level) analysis (Price et al., 2006; Rietveld et al., 2014). Although not strictly necessary to include in the within-family analysis due to the inclusion of family fixed effects, we keep the PCs in all specifications to facilitate a clean comparison between the between-family and within-family results.

[9] In section 4.5, we study the interaction effect using more flexible approaches than the linear interaction presented here.

al., 2020). Second, GWASs typically do not control for parental genotypes. Trejo & Domingue (2019) show that not controlling for parental genotypes causes the polygenic scores to be measured with error. Both sources of measurement error lead to an attenuation bias in the coefficient of the polygenic score in within-family analyses.[10] While we cannot solve the attenuation bias arising from the omission of parental genotype in the GWAS, we follow DiPrete, Burik, & Koellinger (2018) and Van Kippersluis et al. (2020) in applying Instrumental Variables (IV) to tackle the classic measurement error problem. More specifically, we split our discovery GWAS sample into two equal halves and construct two polygenic scores based on the two discovery samples. To ensure that these subsamples are unrelated, we randomly select only one individual from each cousin cluster. Even though the two polygenic scores individually have lower predictive power, the measurement error in the two is plausibly orthogonal and so they can be used as instrumental variables for each other. Using these two polygenic scores, we apply Obviously-Related Instrumental Variables (ORIV; Gillen et al., 2019). See Appendix C for more details.

III.   Data

We use data from the UK Biobank (2006-2010), a population-based cohort with approximately 500,000 individuals aged between 40-69 at the time of interview and living within a radius of 40 km from one of the 22 assessment centers in England, Wales, and Scotland (Fry et al., 2017). It contains survey data, biomarker and DNA samples, physical measurements, and linkage to inpatient registers and death records (Sudlow et al., 2015). Because participation in the UK Biobank is voluntary, it is not a representative sample of the UK population (see Fry et al. (2017) for a detailed analysis).

We apply the following sample selection criteria. The original data include 502,498 consented individuals. We follow the literature and remove those of non-European descent (92,892 observations), as well as twins and multiple births (9,310 observations), and individuals with

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[10] The reason why classical measurement error as a result of finite-sample bias leads to attenuation bias is well known. It is more subtle why the exclusion of parental genotype in the discovery GWAS leads to an attenuation bias in within-family studies. The reason is that a polygenic score constructed on basis of a GWAS that did not control for parental genotype will reflect both direct genetic effects arising from the individual's genotype as well as indirect genetic effects arising from the omitted parental genotypes. The latter effects are known as 'genetic nurture' (e.g., Kong et al., 2018). When applied within families, the differences in the polygenic score arising from parental genotype are spurious since parental genotype is the same across siblings. Hence, part of the differences across siblings in the polygenic score reflect genetic nurture (i.e., indirect genetic effects) and can be considered measurement error attenuating the resulting within-family estimates (Trejo & Domingue, 2019).

missing or conflicting information regarding the number of siblings and/or family size (3,801 observations). In doing so, we arrive at a sample of 396,494 individuals. We further restrict this sample to individuals with at least one sibling[11] in the UK Biobank and without missing values on any of the variables included in our analysis (i.e., years of education, birth order, family size, year and month of birth, principal components of the genetic relatedness matrix, gender, and the polygenic score for education). Since the UK Biobank did not specifically target families, this leads to a final sample size of N = 14,850 individual siblings.

Table 1. *Descriptive statistics analysis sample (N = 14,850).*

Variable	Mean	S.D.	Min.	Max.
Years of education	15.058	4.951	7.000	20.000
Firstborn (1/0)	39.4%			
PGS for years of education	0.000	1.000	-4.049	3.912
Birth order	1.913	0.998	1.000	5.000
Second born	41.50%			
Third born	11.18%			
Fourth born	4.33%			
Fifth- or laterborn	3.60%			
Family size	2.987	1.530	2.000	14.000
Last child (1/0)	36.9%			
Male (1/0)	42.5%			

Notes: S.D. = Standard deviation; Min. = Minimum; Max. = Maximum.

We follow the literature (see e.g., Lee et al., 2018; Okbay et al., 2016; Rietveld et al., 2013) and convert individuals' qualifications to equivalent years of education using the International Standard Classification of Education (ISCED).[12] The average years of education for the sibling sample is 15 years (see Table 1). We construct individuals' birth order based on their response to a question of how many older siblings they have. If a respondent reports zero older siblings, the birth order is set to one. For individuals with missing information on the number of older siblings, we determine birth order based on family size and birth year of the individual and his/her siblings if all of them are present in the UK Biobank. This adds information on birth order for 1,752 siblings in our analysis sample. The remainder of the individuals with missing

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[11] Siblings are identified based on the genetic data because there is no direct information about sibling status in the UK Biobank. We also use the genetic data to identify other relatives up to the third degree. See Appendix B for the full procedure followed to identify siblings and relatives.

[12] Years of education ranges from 7 to 20, where College or University degree is equivalent to 20 years, National Vocational Qualification (NVQ), Higher National Diploma (HND), or Higher National Certificate (HNC) to 19 years, other professional qualifications to 15 years, having an A or AS levels or similar to 13 years, O levels, (General) Certificate of Secondary Education ((G)CSE) to 10 years, and if none of the above to the lowest level of 7 years.

birth order or any other information used in the analysis are excluded from the analysis sample. Table 1 shows that 39.4% of our sample are firstborns, with an average birth order of 1.91 (where we have censored birth order at 5 for the 245 respondents with birth order beyond 5). Around 37% of our sample is lastborn, and the average family size is 3 (i.e., the average number of siblings is 2).

Our measure of genetic endowment for education is the polygenic score for education. A polygenic score is a weighted sum of genetic variants called Single Nucleotide Polymorphisms (SNPs, see Appendix A for details). The SNP weights are determined by the association between a SNP and years of education (Dudbridge, 2013) in an independent (discovery) sample:

$$PGS_i = \sum_{j=1}^{J} \beta_j x_{ij}, \qquad\qquad (2)$$

where PGS_i is the value for the polygenic score for individual i, β_j is the regression coefficient of SNP j ($j = 1, \dots, J$) from our own tailor-made GWAS (see below), and x_{ij} is the genotype of individual i for SNP j (coded as 0, 1 or 2, indicating the number of "effect" alleles). We use the LDpred software (Vilhjálmsson et al., 2015) to correct for the correlation structure across SNP (i.e., "linkage disequilibrium"). The polygenic scores are standardized within the sibling sample to have mean 0 and standard deviation 1.

The polygenic score measures the genetic predisposition towards educational attainment within the environmental context and demographic characteristics of the discovery GWAS sample (Domingue, Trejo, Armstrong-Carter, & Tucker-Drob, 2020). It is therefore preferable to select discovery and analysis samples from the same environmental context, especially when analyzing gene-environment interactions. At the same time, the discovery sample should be independent of the analysis sample to avoid overfitting (Dudbridge, 2013). As an optimal balance, we therefore construct the polygenic score by using the weights from our own tailor-made GWAS that uses the UK Biobank sample without siblings and their relatives. The GWAS discovery sample comprises 389,419 individuals from the UK Biobank; we use the summary statistics from these analyses to create the polygenic scores on the sample of 14,850 siblings. This tailor-made polygenic score alleviates the differences between the discovery and the analysis samples in terms of demographics and environmental context, as well as measurement (i.e., the variables of interest are measured in the same way (Elam, Clifford, Shaw, Wilson, & Lemery-Chalfant, 2019; Tropf et al., 2017). Moreover, running our own GWAS enables the construction of two independent polygenic scores, obtained by splitting the

discovery GWAS sample into two equal halves, which can be used in ORIV. This approach has been shown to outperform a single polygenic score that is based on meta-analyzing multiple cohorts (Van Kippersluis et al., 2020).[13]

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[13] To check if our results are sensitive to the choice of polygenic scores, we constructed a polygenic score based on the meta-analyzed GWAS results of 23andMe summary statistics and our own UK Biobank discovery sample GWAS. As expected, this polygenic score is more predictive for educational attainment than the polygenic score constructed on the basis of the UK Biobank alone. However, this polygenic score is based on several discovery cohorts from very different environmental contexts and does not allow us to use ORIV since we do not have access to all underlying samples to allow us to create multiple polygenic scores. The results are very similar, with the interaction term being slightly smaller but not significantly different from our main results (see Appendix F). We obtain similar results when employing the polygenic score for educational attainment from the Polygenic Index Repository (Becker, Burik, et al., 2021).

IV.   Results

4.1. Predictive power of the polygenic score for educational attainment

Figure 1 shows that our polygenic score for years of education is approximately normally distributed. To create this figure, we divided the polygenic score in 200 bins; the dots represent the average years of education for each bin. The line through the dots is obtained from a local polynomial regression of years of education on our polygenic score. In line with the literature (Lee et al., 2018; Okbay et al., 2016; Rietveld et al., 2013), the polygenic score is positively correlated with years of education ($r = 0.23$, $p < 0.001$). The average difference between those two standard deviations below the mean of the polygenic score, and those two standard deviations above the mean is almost 4 years of completed education, highlighting the substantial predictive power of the polygenic score. Furthermore, Figure 1 suggests the relationship is approximately linear, with little suggestion of any strong non-linearities between years of education and the polygenic score.

Figure 1. The relationship between the standardized polygenic score and years of education in the analysis sample.
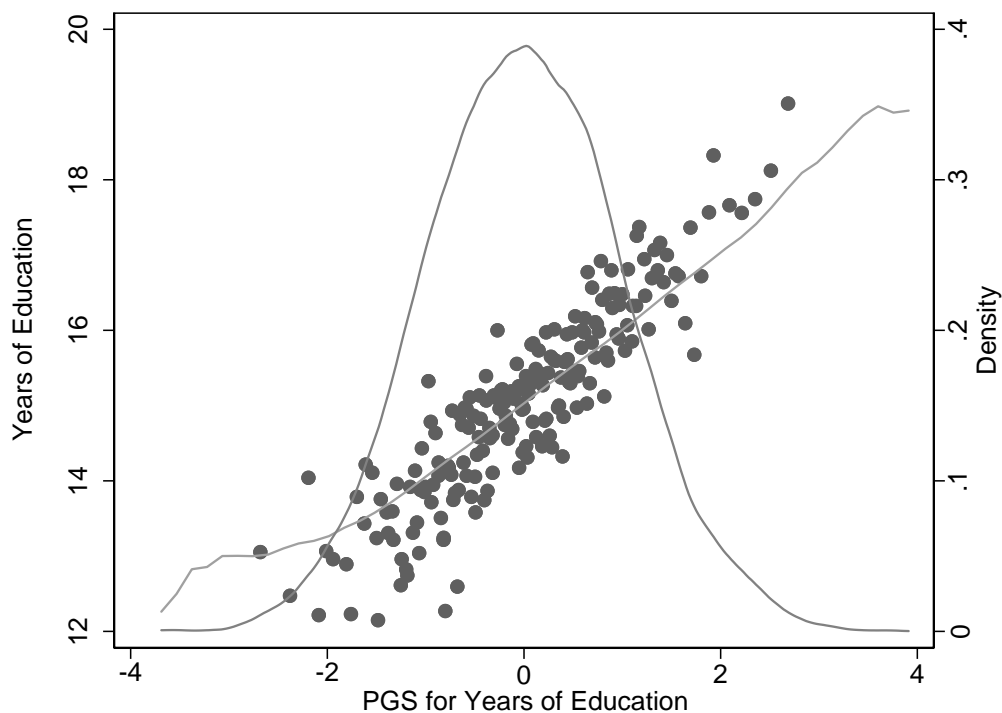
Notes: Plotted using 200 bins of polygenic score.

Table 2 shows that the incremental R^2 of the polygenic score (i.e., the additional variance explained by the polygenic score after controlling for gender, month, and year of birth, and the first 40 principal components of the genetic relatedness matrix) is 5.4% in the between-family analysis (i.e., 0.113-0.059=0.054; Columns 1 and 2). In the specifications with family fixed effects (Columns 3 and 4), the incremental (within) R^2 for the polygenic score is reduced to 1%. This reduction in predictive power when moving from between-family to within-family estimates is well-established in the literature (see e.g., Koellinger & Harden, 2018; Kong et al., 2018; Lee et al., 2018; Rietveld et al., 2013; Selzam et al., 2019), and reflects the fact that family fixed effects account for the shared family environment and parental genotype, which was not accounted for in the between-family specification. In terms of the effect sizes, we observe that a one standard deviation increase in the polygenic score is associated with an increase of 1.155 years of education. With family fixed effects, the effect size is reduced to 0.646.

Table 2. *Results of the regressions of years of education on the polygenic score (PGS).*

	Between-family analysis		Within-family analysis	
	(1)	(2)	(3)	(4)
PGS for years of education		1.155***		0.646***
		(0.038)		(0.071)
Constant	15.889***	15.249***	13.757***	13.628***
	(1.690)	(1.666)	(1.685)	(1.821)
R^2	0.059	0.113	0.046	0.056
N	14,850	14,850	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors.

4.2. The relationship between birth order and educational attainment

Figure 2 shows differences in years of education (residualized with respect to year and month of birth, family size, gender, and 40 first principal components of the genetic relatedness matrix) by birth order (panel A). Without taking into account family fixed effects and other control variables, we see that the average levels of education of children with higher birth order are clearly lower (albeit with higher variances) than that of the children born first. Given that laterborn children have similar levels of education on average, when pooling all laterborns together (panel B), the difference between firstborns and laterborns becomes even more pronounced.

Figure 2. *The relationship between birth order and years of education in the analysis sample.*

Notes: 95% confidence intervals. The variable years of education is residualized with respect to year and month of birth, family size, gender, and 40 first principal components of the genetic related matrix.

Table 3. *Results of the regressions of years of education on different specifications of birth order.*

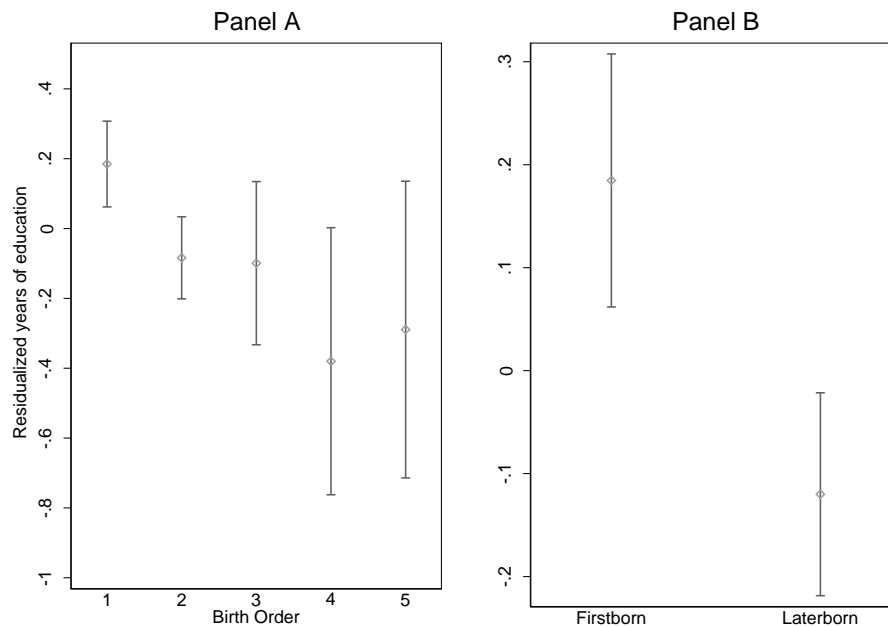|	Between-family analysis		Within-family analysis	
	(1)	(2)	(3)	(4)
Firstborn	0.357***		0.418***	
	(0.087)		(0.109)	
2nd born		-0.310***		-0.450***
		(0.090)		(0.124)
3rd born		-0.431***		-0.743***
		(0.146)		(0.245)
4th born		-0.822***		-0.808**
		(0.228)		(0.366)
5th born		-0.892***		-0.463
		(0.279)		(0.498)
Constant	16.258***	16.473***	12.711***	13.002***
	(1.746)	(1.737)	(1.681)	(1.678)
R^2	0.069	0.069	0.048	0.048
N	14,850	14,850	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix)) are not displayed, but available upon request from the authors.

Table 3 confirms the birth order effects in the specifications with and without family fixed effects, and shows that conditional on the control variables, the differences become more salient. We observe a consistent gap of 0.3-0.4 years of schooling between first- and laterborn children. The direction and magnitude of the effect is robust to using the binary indicator or the categorical variable for birth order. The within-family effect sizes for birth orders higher than four do not reach statistical significance due to the relatively small number of observations (see Table 1).

4.3. The relationship between birth order and the polygenic score for years of education

Figure 3. The relationship between birth order and the polygenic score for years of education in the analysis sample.
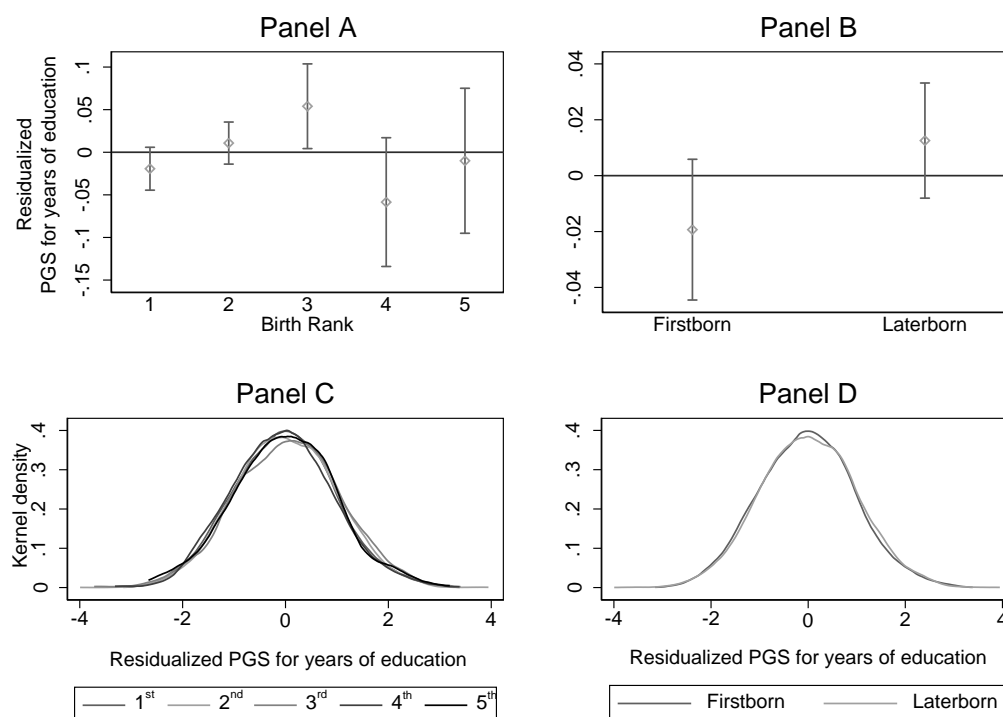
Notes: 95% confidence intervals. The polygenic score (PGS) for years of education is residualized with respect to year and month of birth, family size, gender, and 40 first principal components of the genetic related matrix.

To measure causal gene-environment interactions, it is important to verify that birth order is orthogonal to the polygenic score. Figure 3 provides the first impression of the relationship between the two measures, where polygenic score for years of education is resizualized for our standard set of controls: year and month of birth, gender, family size, and 40 first principal

components of the genetic relatedness matrix.. Panel A illustrates that educational attainment polygenic scores do not depict any particular pattern across different birth order of children. Although panel A suggests that 3rd born children have somewhat higher genetic endowments, this difference is not confirmed in further analysis. The same holds when focusing the comparison on firstborns versus laterborns in Panel B: the observed small differences are not statistically significant. Furthermore, the distributions of the polygenic score by birth order are the same (Panels C and D).

Table 4. Results of the regressions of polygenic score for educational attainment on birth order.

	Between-family analysis		Within-family analysis	
	(1)	(2)	(3)	(4)
Firstborn	-0.037**		-0.002	
	(0.018)		(0.018)	
2nd born		0.034*		0.008
		(0.019)		(0.020)
3rd born		0.078**		0.009
		(0.031)		(0.039)
4th born		-0.036		0.018
		(0.045)		(0.057)
5th born		-0.013		0.061
		(0.057)		(0.080)
Constant	0.648*	0.605	0.207	0.226
	(0.356)	(0.355)	(0.377)	(0.378)
R^2	0.017	0.018	0.013	0.013
N	14,850	14,850	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix)) are not displayed, but available upon request from the authors.

Consistent with the graphical evidence, Table 4 shows a slight difference of 0.04 standard deviations in the polygenic score between firstborn and laterborn children in the between-family analysis. However, the difference becomes neither economically nor statistically significant within families. These results establish that firstborns on average do not have different genetic endowments than their laterborn siblings (as expected, based on Mendel's Law of Independent Assortment). To check if there are birth order related systematic differences in other, possibly related, polygenic scores, we analyzed polygenic scores from the Polygenic Index Repository (Becker et al., 2021) for anthropometric, health, health behaviour, and personality traits. We find no systematic difference in birth order among any of the

available anthropometric, health, and personality related polygenic scores (see Appendix G for details). These results also corroborate that there is no gene-environment correlation (*rGE*) between genetic endowments and birth order.

4.4. Gene-environment interaction: complementarity in human capital production

Table 5 presents the gene-environment interaction results in the between-family and within-family analyses. Comparison of Columns 1 and 4 in Table 5 to the estimates presented above (Table 2 and Table 3) shows that the addition of the educational attainment polygenic score does not change a lot the direct effect of birth order on years of schooling. This comparison confirms once again that the polygenic score and birth order are independent. A one standard deviation increase in the polygenic score is estimated to raise years of education by 1.14 years (between-family analysis) and 0.64 years (within-family analysis). Furthermore, firstborns enjoy on average $0.40 - 0.43$ extra years of schooling compared with laterborns.

Table 5. Results of the regressions of years of education on the gene-environment interaction.

	Between-family analysis			Within-family analysis		
	(1)	(2)	(3)	(4)	(5)	(6)
	OLS	OLS	ORIV	OLS	OLS	ORIV
Firstborn	0.400***	0.400***	0.428***	0.419***	0.415***	0.428***
	(0.078)	(0.078)	(0.078)	(0.109)	(0.109)	(0.109)
PGS for years of education	1.144***	1.099***	1.376***	0.646***	0.567***	0.757***
	(0.040)	(0.049)	(0.063)	(0.071)	(0.078)	(0.102)
Firstborn × PGS for years of education		0.119*	0.222**		0.204**	0.285***
		(0.072)	(0.097)		(0.080)	(0.102)
Constant	15.516***	15.517***	14.742***	12.578***	12.544***	12.184***
	(1.710)	(1.716)	(1.762)	(1.819)	(1.838)	(4.419)
R^2	0.121	0.121		0.058	0.040	
Cragg-Donald *F*-statistic			3568.570			2971.412
N	14,850	14,850	14,850	14,850	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors. We do not report the R^2 for the ORIV specifications in Column (3) and (6) given the differences in its interpretation and computation for the instrumental variable type of regressions.

The between-family design shows a positive interaction between the polygenic score and being firstborn of $\hat{\beta} = 0.119$ (Column 2). Yet, it is borderline statistically significant at conventional thresholds. When we use Obviously-Related Instrumental Variables (ORIV) regression (Column 3), the interaction term becomes larger in magnitude and statistically significant at 5%

threshold, suggesting that measurement error in the polygenic score attenuated the main effect of the polygenic score and the interaction term. In the family fixed effects specification, we find that the interaction effect is significant in both the OLS and the ORIV specifications. The measurement error correction again strengthens the interaction term in terms of magnitude and precision ($\hat{\beta} = 0.204$ vs. 0.285).

As a complementary approach to test the statistical significance of our main results, we also conduct permutation inference by employing 10,000 permutations of the polygenic score and birth order, each time re-estimating the within-family specification (Fisher, 1935; Rosenbaum, 2002). This approach does not rely on repeated sampling of a hypothetical population and enables gauging how rare the size and significance of our interaction effect is for other permutations of the polygenic score and birth order. Figure 4 provides the distribution of *t*-statistics for all 10,000 permutations with the *t*-statistic of the actual estimate shown as a solid vertical line. The *t*-statistic of the estimated interaction term has an exact *p*-value (i.e., the proportion of *t*-statistics more extreme than our estimate) of 0.04.

The positive and statistically significant interaction term provides strong evidence for gene-environment interactions in education and is consistent with the existence of complementarity between endowments and investments in human capital production: the effect of being firstborn (associated with more parental investments) is complementary to a higher value for the polygenic score for years of education. In other words: those with a higher polygenic score benefit more from the higher parental investments associated with being firstborn. The magnitude of the coefficients suggests that for those with a polygenic score two standard deviations below the mean, there is no advantage of being firstborn. In contrast, for those with a high polygenic score, being firstborn increases one's years of education. For example, firstborns with a polygenic score two standard deviations above the mean completed on average 0.82 (OLS) to 1 (ORIV) more years of education compared to their laterborn siblings with a similar polygenic score.

Figure 4. *Distribution of the t-statistic for the G×E interaction term on basis of permutation inference.*
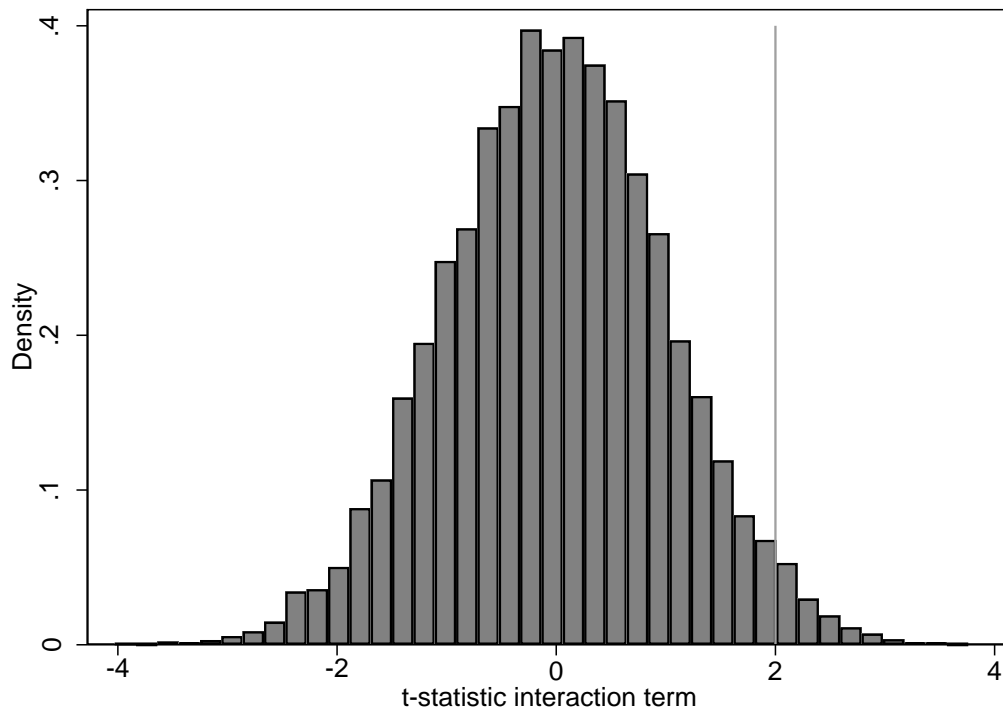
4.5. Robustness checks

In this section, we check the robustness of our results against potential non-linearities in the functional form of the polygenic score as well as birth order, against the addition of further control variables, and against fertility choices. While the linear form adopted in section 4.4 seems justified based on the visual inspection of Figure 1, we explore robustness of our results by allowing for possible non-linearities. Table 6 compares the within-family specification in continuous form (replicated in Column 1 for comparison), with those where we specify the polygenic score in binary form (above and below the mean, Column 2), in quartiles (Column 3) and in squared form (Column 4). We observe positive interaction terms across all specifications. In line with our main findings, the effect of being firstborn is insignificant for those with a lower polygenic score, and the effects are concentrated among those in the upper half of the polygenic score distribution. We also find that the main result in Column 1 is robust to the inclusion of a quadratic version of the polygenic score.

Table 6. Results of the regressions of years of education on the gene-environment interaction; Robustness to non-linearities in the polygenic score.

	Within-family analysis			
	(1)	(2)	(3)	(4)
Firstborn	0.415***	0.239*	0.186	0.437***
	(0.109)	(0.145)	(0.198)	(0.125)
PGS for years of education	0.567***			0.566***
	(0.078)			(0.078)
Firstborn × PGS for years of education	0.204**			0.205**
	(0.080)			(0.081)
PGS for years of education (>mean)		0.588***		
		(0.136)		
Firstborn × PGS for years of education (>mean)		0.356**		
		(0.169)		
PGS for years of education (2nd quartile)			0.439**	
			(0.190)	
PGS for years of education (3rd quartile)			0.810***	
			(0.197)	
PGS for years of education (4th quartile)			1.230***	
			(0.208)	
PGS for years of education (2nd quartile) × Firstborn			0.155	
			(0.276)	
PGS for years of education (3rd quartile) × Firstborn			0.190	
			(0.255)	
PGS for years of education (4th quartile) × Firstborn			0.595**	
			(0.232)	
PGS for years of education (squared)				0.018
				(0.047)
PGS for years of education (squared) × Firstborn				-0.022
				(0.061)
Constant	12.544***	12.087***	11.821***	12.518***
	(1.838)	(1.796)	(1.868)	(1.838)
R^2	0.059	0.053	0.057	0.059
N	14,850	14,850	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family; * $p<0.10$, ** $p<0.05$, *** $p<0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors.

Table 7 reports the sensitivity of the results to an alternative specification of birth order. Column 1 replicates the main result from Table 5 for comparison. In Column 2, we include dummies for each birth rank with firstborns as the reference category. Hence, we expect the effects to be reversed as compared to Column 1. All point estimates of the main effects and the interaction terms are in line with the birth order literature and consistent with

complementarity: on average, laterborns have lower educational attainment compared to firstborns and benefit less from having a high polygenic score. The estimates for rank 4 and higher are rather imprecisely estimated due to the relatively small sample sizes with a birth rank of 3 or higher, but they are in line with our main results.

Table 7. Results of the regressions of years of education on the gene-environment interaction; Robustness to non-linearities in birth order.

	Within-family analysis	
	(1)	(2)
PGS for years of education	0.567***	0.767***
	(0.078)	(0.086)
Firstborn	0.415***	
	(0.109)	
Firstborn × PGS for years of education	0.204**	
	(0.080)	
2nd born		-0.450***
		(0.124)
3rd born		-0.741***
		(0.244)
4th born		-0.832**
		(0.362)
5th born		-0.470
		(0.499)
2nd born × PGS for years of education		-0.212**
		(0.083)
3rd born × PGS for years of education		-0.152
		(0.148)
4th born × PGS for years of education		-0.316
		(0.254)
5th born × PGS for years of education		-0.089
		(0.300)
Constant	12.544***	12.882***
	(1.838)	(1.842)
R^2	0.059	0.060
N	14,850	14,850

Notes: Robust standard errors in parentheses, clustered by family; * $p<0.10$, ** $p<0.05$, *** $p<0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from

In Table 8, we explore robustness of our results to endogenous fertility decisions. A possible correlation between our measure of endowments and birth order could arise when fertility decisions are based on the genetic endowments of the children, known in the literature as the "child stopping rule" (Black et al., 2005; Pavan, 2016). Whereas Section 4.3 shows that such a correlation does not exist, here we explicitly control for a possible child-stopping rule by

including a dummy variable for being lastborn, which is set to one if an individual's birth order is equal to the total number of children in his/her family. To facilitate an easy comparison, Column 1 in Table 8 replicates the results from Column 5 in Table 5. As can be seen from Column 2, the lastborn dummy is not statistically significant and does not meaningfully affect our results, suggesting that potential endogenous fertility decisions do not change any of our conclusions. In Columns 3 and 4, we report the within-family results based on data from the families with at most three siblings (N = 11,364) and two siblings only (N = 7,918) to check if our results are sensitive to the exclusion of relatively large families with possibly different characteristics (see Figure 2). Even though this sample restriction is endogenous, it is reassuring that our results are robust to this sample restriction. Finally, in Column 5 we investigate any gender differences in the interaction of interest. While we do find that gender is an important predictor of educational attainment, with men having 1.46 more years of education than women, the interaction term does not differ significantly by gender.

Table 8. Results of the regressions of years of education on the gene-environment interaction; Robustness to fertility choices.

	Within-family analysis				
	(1)	(2)	(3)	(4)	(5)
Firstborn	0.415***	0.392***	0.439***	0.466***	0.278**
	(0.109)	(0.128)	(0.132)	(0.178)	(0.134)
PGS for years of education	0.567***	0.568***	0.589***	0.570***	0.628***
	(0.078)	(0.078)	(0.090)	(0.109)	(0.092)
Firstborn × PGS for years of education	0.204**	0.203**	0.197**	0.183*	0.114
	(0.080)	(0.080)	(0.087)	(0.102)	(0.115)
Lastborn		-0.049			
		(0.136)			
Male	1.265***	1.265***	1.429***	1.571***	1.460***
	(0.099)	(0.099)	(0.109)	(0.130)	(0.148)
Male × PGS for years of education					-0.149
					(0.124)
Firstborn × PGS for years of education × Male					0.207
					(0.188)
Constant	12.544***	12.509***	13.065***	12.578***	12.695***
	(1.838)	(1.840)	(2.106)	(2.148)	(1.846)
R^2	0.059	0.059	0.066	0.073	0.060
N	14,850	14,850	11,364	7,918	14,850

Notes: Column 1 replicates our main results from Table 5; Column 2 additionally controls for a dummy indicating whether the individual is the lastborn; Column 3 restricts the sample to families with less than four siblings; Columns 4 restricts the sample to families with two siblings only; Column 5 explores differences in the interaction term by gender. Robust standard errors in parentheses, clustered by family; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, family size, and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors.

V.   Discussion

A large literature shows consistently higher educational attainments for firstborn children. Using within-family data we move beyond the existing literature by showing that children benefit disproportionally from being firstborn when they have relatively high genetic endowments. More specifically, firstborns with an average polygenic score enjoy 0.415 years (≈ 5 months) of additional schooling compared to their laterborn siblings, on average. However, firstborns with a polygenic score that is one standard deviation above the mean enjoy an additional 0.204 years (≈ 2.5 months) of education, compared to their laterborn siblings with the same genetic endowment. In contrast, for individuals with below-average polygenic scores, being firstborn does not provide an advantage in terms of educational attainment. Since we provide evidence that genetic endowments are orthogonal to birth order, and previous literature suggests that birth order effects on children's education are mainly driven by parental investments, we interpret the positive and significant interaction term as providing support for the existence of the complementarity between endowments and investments in human capital production.

An alternative interpretation of our finding that birth order effects are concentrated among those with higher polygenic scores could be that the additional investments associated with being firstborn are higher for those with higher polygenic scores. That is, the positive interaction effect could also be explained by parents investing more in the firstborn, or altering fertility decisions, when the firstborn child has a higher polygenic score. While we cannot fully rule out this explanation, we believe this explanation is less plausible for three reasons. First, Breinholt & Conley (2019) and Houmark et al. (2020) show that parenting during infancy is not driven by genetic make-up because these endowments are not clearly expressed yet, and parental investment responses to polygenic scores do not arise before age six. This is long after the typical arrival of subsequent children, and so the most precious time of undivided attention for the firstborn is unlikely to be influenced by – at that time unobserved – differences in polygenic scores.[14] Second, whereas it is established that parents do respond to the polygenic scores of children at later ages (e.g., Sanz-De-Galdeano & Terskaya, 2019), we control for this with the main effect of the polygenic score in our specification. Only when – for some reason

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[14] For the few early-life parental investments we observe in our data, we do not find evidence of any response to the polygenic score. Appendix D shows that maternal smoking around pregnancy and whether the child was breastfed are all unrelated to the firstborn's polygenic score. If anything, the age gap between first- and secondborns is slightly lower if the firstborn has a higher polygenic score.

– parents respond more to the polygenic score *when the child is firstborn*, our interpretation of the interaction term would be challenged. We cannot rule this out, but deem it less plausible, as the additional investments associated with being firstborn decrease with age (Price, 2008), and become increasingly modest at the ages where genetic endowments are clearly expressed (Breinholt & Conley, 2019). Third, recent evidence exploiting within-family differences in polygenic scores for educational attainment suggests that parents compensate for genetic differences in education between siblings. More specifically, Fletcher et al. (2020) show that the association between the education polygenic score and educational attainment is stronger for siblings with the lower polygenic score. This is consistent with parental preferences for equality among siblings, and suggests the differences are due to compensating parental investments. As birth order as well as genetic variation is random in a within-family design, this does not support the alternative interpretation that parents invest more in the firstborn only when the firstborn has a higher polygenic score. Our findings therefore suggest that the additional investments associated with being firstborn are driven by less restrictive time and budget constraints and are independent of the child's genetic endowment. The appearance of a positive interaction between endowments and being firstborn is therefore consistent with complementarity in human capital production.

Another potential explanation for our findings is that other mechanisms through which birth order effects arise (e.g., parental age at birth, interactions with younger siblings) could interact with genetic endowments. It is reassuring that additionally accounting for parental age at birth and other potential confounders does not affect our results (see Appendix E). Unfortunately, we cannot test alternative explanations regarding sibling interactions directly because the UK Biobank is very limited in measures of parental and sibling interactions. Still, *Table 7* suggests that our findings are mainly driven by a distinction between first- and all laterborns, with significant differences appearing prominently between the first and secondborns. If interactions with younger siblings would be driving our results, one would expect a more gradual decrease in the magnitude of the interaction terms with rising birth rank, as some second- and thirdborns also benefit from interactions with younger siblings. Moreover, even if birth order only partially captures parental investment – a premise that should not be controversial given the overwhelming evidence in the literature – then unless these other channels exhibit completely opposite interaction effects, a necessary (but not sufficient) condition for complementarity would be a positive interaction between birth order and genetic

endowments. This is indeed what we find.

A number of limitations should be acknowledged. First, our specification may not be the perfect empirical translation of the human capital production function. In particular, we do not measure skills or human capital directly. Instead, we follow Cunha & Heckman (2008) and Cunha et al. (2010) who specify adult human capital as a combination of skills accumulated by the end of childhood, and employ a commonly used and convenient proxy: years of education. Moreover, we do not measure parental investments directly, and use an environmental variable closely related to parental investments: birth order. The upside of using birth order rather than a direct measure of parental investments is that birth order is randomly assigned within families, whereas parental investments are known to be endogenous to offspring endowments. Moreover, whereas birth order cannot distinguish between early-life and later-life investments, it does capture a persistent difference across siblings rather than a one-time shock in investments that many other papers rely on (see Almond et al., 2018), and Appendix B).

A second limitation is that our measure of genetic endowments is imperfect. In particular, a polygenic score captures only common genetic variations in the human genome, and even within the realm of common variations the measure is subject to measurement error. While the use of ORIV reduces concerns about classical measurement error, our family fixed effects estimates of the polygenic score are still subject to attenuation bias due to genetic nurture. However, since the sign of the bias arising from genetic nurture is known to be negative, our effect size represents in fact a conservative estimate.

The polygenic score should also not be interpreted narrowly as a measure of immutable biological endowments. While within-family analyses allow us to interpret the effect of the polygenic score as a causal effect of genetic variation, it is well-established that the environment may mediate this effect (e.g., Breinholt & Conley, 2019; Houmark, Ronda, & Rosholm, 2020), including the family environment (Fletcher et al., 2020). Hence, a polygenic score measures education-enhancing endowments, and will reflect how *on average* in the discovery sample environments (including parental investments) respond to differences in genetic endowments. Importantly though, since the measure is fixed at conception and orthogonal to birth order in the within-family analysis, the measure does not reflect parental investments *of the child's own parents*, nor does it reflect parental genetic (nurture) effects.

As a result, the inclusion of environmental responses to genetic variation into the construction of the polygenic score is not a source of concern for our identification strategy, but does imply that our finding of complementarity between genetic endowments and birth order is not an immutable property of the production function for human capital and may be specific to the context studied.

A related limitation regards the external validity of the empirical findings. As mentioned in the data section, there is sample selection into the UK Biobank, with a bias towards healthier and higher-educated individuals (Fry et al., 2017). On top of this, we focus on European-ancestry individuals and the coincidental sampling of siblings even though these were not specifically targeted, further reducing the representativeness of the sample. Finally, we construct our polygenic score on basis of a tailor-made GWAS, again on basis of the same UK Biobank excluding the siblings and their relatives. While the latter choice helps to maintain the same environments across discovery and prediction sample, it may further increase the likelihood that our results are specific to the UK Biobank. Future research should replicate our findings, but in light of our evidence in support of economic theories of human capital production undergirding our results, we have good reasons to be positive about their replicability in comparable contexts.

VI.   References

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VII. Appendix

A. Genetic data, GWAS, and Polygenic scores

Genetic Data. A complete human genome consists of 23 pairs of chromosomes, from which the 23rd pair determines the biological sex of a person. One of each pair of chromosomes is inherited from the father, and the other is inherited from the mother. A chromosome is composed of two intertwined strands of deoxyribonucleic acid (DNA), each made up of a sequence of four possible nucleotide molecules: adenine, cytosine, thymine, and guanine. Adenine (A) on one strand is always paired with thymine (T) on the other strand, and cytosine (C) is always paired with guanine (G). These pairs are called base pairs. Every human genome consists of approximately 3 billion base pairs and stretches of base pairs coding for proteins are called genes. There are approximately 20,000 genes in the human genome, with varying lengths in terms of base pairs (Ezkurdia et al., 2014).

Two unrelated human beings share approximately 99.6% of their DNA, and most genetic differences across humans can be attributed to single nucleotide polymorphisms (SNP) (Auton et al., 2015). A SNP is a locus in the DNA at which two different nucleotides can be observed in the population. Each of the two possible nucleotides is called an allele for that SNP. An individual's genotype is coded as 0, 1, or 2, depending on the number of "effect" alleles present. In the human genome, there are at least 85 million SNPs with a "minor" allele prevalence of at least 1% (Auton et al., 2015).

Genome-Wide Association Studies (GWASs) aim to identify genetic variants that are associated with a particular trait of interest by relating each variant to the trait in a hypothesis-free approach. Stringent significance thresholds are used to identify variants that are robustly associated with the trait, with other independent samples used for replication. Using the GWAS approach, thousands of genetic discoveries have been made (Visscher et al., 2017).

Individual SNPs typically explain less than 0.02% of the variance in a behavioural outcome (Chabris, Lee, Cesarini, Benjamin, & Laibson, 2015; Visscher et al., 2017). It is therefore common to combine multiple SNPs into a polygenic score (Dudbridge, 2013), constructed as a weighted sum of SNPs. Through increases in GWAS sample sizes, the predictive power of the polygenic score for education has increased from 2-3% (Rietveld et al., 2013), to 6-8% (Okbay et al., 2016), to 11-13% (Lee et al., 2018). In terms of biological pathways, there is evidence that many of the identified genes associate with health, cognitive, and central nervous system

traits (Rietveld et al., 2013). Likewise, the majority of the significant SNPs in Okbay et al. (2016) and Lee et al. (2018) relate to genomic regions responsible for gene expression in a child's brain during the prenatal period.

Methods. *Relatedness.* As a first step, we identify siblings and their relatives using the kinship matrix provided by the UK Biobank. The kinship matrix is based on genetically identified relatedness and contains relatives of third degree and closer identified using the KING software (Manichaikul et al., 2010). The UK Biobank does not have information about self-reported relatedness (Bycroft et al., 2018). The degree of relatedness between the pairs of individuals is based on the combination of the kinship coefficient and genetic similarity in terms of the identity by state (IBS_0) coefficient. IBS_0 measures the fraction of markers for which the related individuals do not share alleles. We follow the KING manual regarding the thresholds for how to determine family relationship (see Table A.1). The identified number of pairs per relationship type differs slightly from that of Bycroft et al. (2017), because some UK Biobank participants withdrew their consent to analyse their data since then.

Table A.1. Thresholds used to determine relatedness between individuals in the UK Biobank.

	Duplicate / Monozygotic twins	1st degree / Parent-child	1st degree siblings	2nd -3rd degree relatives / cousins	Total
Kinship coefficient	>0.3540	0.1770– 0.3540	0.1770– 0.3540	0.0442– 0.1770	
IBS_0		<0.0012	>0.0012		
N (pairs)	179	6,271	22,659	78,038	107,147

For our analyses, we go one step further by separating those who are related to the siblings up to the 3rd degree (kinship coefficient ≥ 0.025), i.e., siblings, parents of siblings, cousins of siblings (See Table A.2). In this way, our holdout sample for polygenic score construction and prediction (i.e., the sibling subsample) is unrelated to the GWAS discovery sample which is used to calibrate the SNP weights that are used to construct the polygenic score.

Table A.2. Relatedness to the individuals in the siblings' subsample of UK Biobank.

Relationship to siblings	Unrelated to siblings	Full siblings	2nd-3rd relative of siblings	Parent or child of siblings	Total
N (individuals)	91,055	41,498	10,207	4,740	147,500

Notes: Relatedness to siblings is computed based on the relatedness classification as reported in Table A.1.

GWAS. Our tailor-made GWAS is performed using the fastGWA protocol for Genome-wide Complex Trait Analysis (GCTA) developed by Jiang et al. (2019). fastGWA applies mixed linear modelling (MLM) to the genetic data of the UK Biobank. fastGWA requires the following steps. First, we generate a sparse genetic relatedness matrix (GRM) using the family relatedness file from the UK Biobank based on the KING software output. Next, we perform an MLM-based GWAS using the SNP data, the sparse GRM, the phenotype file and the minor allele frequency (MAF) filter of 0.001. The phenotype file provides the data on individual years of education residualized with respect to birth year, gender, interaction of birth year and gender, batch, and the first 40 principal components (PCs) of the genetic relatedness matrix. For quality control reasons, some individuals were not included in the GWAS.[15] The eventual GWAS discovery sample includes 389,419 individuals.

We further quality control the resulting GWAS summary statistics using EasyQC tool (Winkler et al., 2014) and meta-analyse our tailor-made GWAS weights with the summary statistics from Okbay et al. (2016). We use these for constructing an alternative polygenic score that is used in the robustness analysis (see footnote 13). Meta-analysis is conducted using the software package METAL (Willer, Li, & Abecasis, 2010).

Polygenic scores. The polygenic scores are constructed while accounting for linkage disequilibrium between SNPs using LDpred (Vilhjálmsson et al., 2015), version 1.06, and Python, version 3.6.6. Linkage disequilibrium pertains to the non-random correlations between SNPs at various loci of a single chromosome. LDpred is a software package based on Python that adjusts the GWAS weights for LD using a Bayesian approach. We follow the steps as outlined in Mills, Barban, & Tropf (2020), including the coordination of the base and target files, computing the LD adjusted weights, and then applying them for polygenic score construction using PLINK (Purcell et al., 2007). We re-weight the SNP effects on the basis of LD and the supposed fraction of causal SNPs, which we set to 1, as is standard practice for behavioural traits (Cesarini & Visscher, 2017). Our hold-out sample for constructing polygenic scores consists of 49,866 siblings and their relatives, where the final analysis sample with observations for all variables available is 14,850 individual siblings. The polygenic scores

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[15] More specifically, we exclude individuals who withdrew consent, have missing gender or whose self-reported gender does not match the genetic sex, are of other than European ancestry, have bad genotyping quality, putative sex chromosome aneuploidy, whose second chromosome karyotypes are different from XX or XY, with outliers in heterozygosity, or have missing information on any of the former criteria.

include all SNPs, that is 1,065,078 SNPs after filtering for HapMap3 SNPs at the coordination step. For the split sample GWAS, we first remove all remaining parent-child pairs (N = 5,134) and cousins except one from each cousin cluster (N = 45,099) and split the unrelated discovery sample with all control variables available (N = 340,009) randomly into two samples of 170,005 and 170,004 individuals each and use the same fastGWA procedure as for the full UKB GWAS to obtain SNP weights. The removal of parent-child pairs and cousins ensures that two subsamples do not contain related individuals and are thus independent from each other. We proceed by using LDpred to construct two polygenic scores based on the two sets of summary statistics. Likewise, we include all SNPs (1,065,146 after filtering for HapMap SNPs at the coordination step).

B. Empirical evidence on complementarity in human capital production

Testing complementarity between children's endowments and parental investments is challenging, since it requires independent variation in initial endowments and later-life investments, or alternatively exogenous variation in multiple investments over time (Almond & Mazumder, 2013; Johnson & Jackson, 2019). Cunha & Heckman (2007) and Cunha et al. (2010) adopt a structural approach, modelling both skills as well as parental investments as low-dimensional latent variables, and find evidence consistent with (dynamic) complementarity. A number of studies have examined whether the effect of specific interventions or investments varies by initial skills. Aizer & Cunha (2012) correct early life health measures for certain prenatal investments, and find that pre-school enrolment is more productive for children with higher levels of this residualized measure of endowments. Lubotsky & Kaestner (2016) use entrance-age in kindergarten as plausibly exogenous variation in initial cognitive skills, and find some evidence for complementarity, although the effect dies out after the first grade.

A recent set of papers have examined rare cases where there exists exogenous variation in both initial endowments as well as later-life investments. For example, Malamud et al. (2016) study the interaction between exogenous variation in access to better schools and variation in family backgrounds induced by access to abortion in Romania. Their findings do not suggest a meaningful interaction between initial endowments and later-life investments. Rossin-Slater & Wüst (2020) exploit a nurse home visiting program as an exogenous shock to endowments, and staggered access to high quality preschool childcare in Denmark as an exogenous shock to investment, and find that these interventions are substitutes rather than complements. Gunnsteinsson et al. (2014) exploit a unique combination where a tornado struck an area of Bangladesh that was coincidentally involved in a randomized experiment on vitamin A supplementation. Their findings are consistent with complementarity since children treated with Vitamin A supplements were better protected from the consequences of the earthquake. Adhvaryu et al. (2019) exploit local rainfall in the year of birth as exogenous variation in endowments, and randomized cash incentives from Progresa as an exogenous shock to investment. Their main finding is that children from families who received cash transfers were protected better against adverse endowments, consistent with complementarity. Similarly, Duque et al. (2018) also use a combination of adverse weather shocks and conditional cash transfers in Colombia to show that children born under normal weather conditions benefit

more from the cash transfers. Finally, Johnson & Jackson (2019) exploit the rollout of Head Start and the implementation of court-ordered school finance reforms (SFRs) that increased spending at public K-12 schools as two exogenous shocks to human capital investment, again finding evidence in favour of complementarity.

The papers above therefore suggest that an increasing number of papers explore variation in both initial endowments and later-life investments to investigate complementarity. However, the evidence from this literature is mixed, with some studies supporting the existence of complementarity, and others finding weaker evidence or even substitutability between endowments and investments.

C. Obviously-Related Instrumental Variable (ORIV) regression

In this section, we explain the technique of Obviously-Related Instrumental Variable (ORIV; Gillen et al., 2019) regression. Suppose we would like to predict an outcome variable of interest, Y, using a polygenic score, i.e., estimate the following model:

$$Y = \alpha + \beta PGS^* + \varepsilon, \qquad (C.1)$$

where α is a constant, β is the effect of a true polygenic score PGS^* and ε is the error term. We have two estimates of the true polygenic score: $PGS_1 = PGS^* + \vartheta_1$ and $PGS_2 = PGS^* + \vartheta_2$. The covariance between the two measurement error terms ϑ_1, ϑ_2 is zero, $Cov(\vartheta_1, \vartheta_2) = 0$, and they have the same relative variance of the measurement errors ϑ_1, ϑ_2. That is:

$$\frac{\sigma_{\vartheta_1}^2}{\sigma_{PGS_1}^2} = \frac{\sigma_{\vartheta_2}^2}{\sigma_{PGS_2}^2} = \frac{\sigma_{\vartheta}^2}{\sigma_{PGS}^2}, \qquad (C.2)$$

where $\sigma_{\vartheta_1}^2$ and $\sigma_{\vartheta_2}^2$ are the variances of the measurement errors ϑ_1, ϑ_2 respectively, and $\sigma_{PGS_1}^2$ and $\sigma_{PGS_2}^2$ are the variances of respective polygenic scores. If we use PGS_2 as an instrumental variable for PGS_1, the following applies:

$$plim\ \beta_{IV} = \frac{Cov(Y,PGS_2)/V(PGS_2)}{Cov(PGS_1,PGS_2)/V(PGS_2)} = \frac{Cov(\alpha+\beta PGS^*+\varepsilon, PGS^*+\vartheta_2)}{Cov(PGS^*+\vartheta_1, PGS^*+\vartheta_2)} = \beta\frac{\sigma_{PGS^*}^2}{\sigma_{PGS^*}^2} = \beta. \quad (C.3)$$

ORIV regression as developed by Gillen et al. (2019) estimates a 'stacked' model:

$$\begin{pmatrix} Y \\ Y \end{pmatrix} = \begin{pmatrix} \alpha_1 \\ \alpha_2 \end{pmatrix} + \beta \begin{pmatrix} PGS_{1+} \\ PGS_{2+} \end{pmatrix} + \varepsilon, \qquad (C.4)$$

where one instruments the stack of estimated polygenic scores $\begin{pmatrix} PGS_{1+} \\ PGS_{2+} \end{pmatrix}$ with $\begin{pmatrix} PGS_{2+} & 0_N \\ 0_N & PGS_{1+} \end{pmatrix}$, where N is the sample size and 0_N is a Nx1 vector with zero's. We include a family-stack fixed effect to conduct the within-family comparisons within a stack of the data. Standard errors are clustered at both the family and individual level following Correia (2017, 2019).

D. Early-life parental investments

The only early-life parental investments observed in the UK Biobank are whether the child was breastfed and whether the mother smoked around birth. We also observe the age gap between subsequent siblings. Table D.1 reports the results of regressions explaining the few early-life parental investment as a function of being firstborn, the polygenic score for education, and their interaction. This shows whether mothers change their behaviour depending on whether it is their first- or laterborn child and the polygenic score of their children. The results show that the probability of being breastfed (Column 1) and the likelihood of maternal smoking around pregnancy (Column 2) are similar between first- and laterborns. Furthermore, the coefficient of the polygenic score for (the child's) education is not significantly different from zero, and we find no evidence of any differences in maternal investments around pregnancy by the firstborn's polygenic score.

Next, we explore the relationship between the polygenic score and the age gap between siblings. Column 3 presents the estimates from a regression of the age gap in months between every two consecutive siblings on the polygenic score of the older sibling in the pair. These between-family estimates suggest that birth spacing is one month shorter for every one standard deviation increase in the polygenic score of the older sibling. However, note that these results derive from a between-family comparison, and therefore should not be interpreted causally. For families for whom we observe three consecutive siblings or more (*N* = 2,542), we can check this result in a within-family specification. The within-family estimates (Column 4) are very noisy because of the small sample size, and the wide confidence intervals do not allow drawing any firm conclusions here.

Table D.1. Results of regressions of early life parental investments on the gene-environment interaction.

	Breastfed	Mother smoked around birth	Age gap	
			Between-family analysis	Within-family analysis
	Within-family analysis			
	(1)	(2)	(3)	(4)
Firstborn	0.006	0.006		
	(0.010)	(0.006)		
PGS for years of education	-0.001	0.001		
	(0.007)	(0.004)		
Firstborn × PGS for years of education	-0.011	0.004		
	(0.007)	(0.004)		
PGS for years of education of the older sibling			-0.970***	6.417*
			(0.331)	(3.466)
Constant	0.705***	0.270***	50.444***	-24.447
	(0.067)	(0.040)	(6.934)	(31.458)
R^2	0.042	0.020	0.067	0.376
N	11,818	13,156	6,501	2,542

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis of columns 1-2 and 5; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender, family size, and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors. Sample sizes vary depending on the availability of early life parental investments and the number of siblings included in the analyses.

E. Additional confounders

In Table E.1. we present further analyses to investigate robustness of our results against the inclusion of additional confounders. Column 1 reproduces our main results (Table 5, Column 5). In Column 2, we employ the correction for missing confounders in gene-environment interaction analyses suggested by Keller (2014). Specifically, we interact both the dummy for being firstborn and the polygenic score for education with year of birth, month of birth, gender, and first 40 principal components of the genetic relatedness matrix, and include all these interactions as additional control variables in the analysis. The direct effect sizes of being firstborn and the polygenic score for years of education are now relative to the reference categories of the control variables (born in 1937, born in January, being female). Although the standard error of the interaction term increases due to the much larger number of regressors, the magnitude of the interaction effect is robust to this specification.

We also explore whether our results are robust against the inclusion of parental age as an additional control, as this may be one mechanism through which birth order effects arise (Eirnæs & Pörtner, 2004). In our sample, we have only 5,601 individuals with information about the age of the mother and 3,085 individuals with information about the age of the father (both measured at time of birth of the child). The inclusion of these variables as control variables in Column 3 and Column 4 respectively of Table E.1. and the subsequent drop in the sample size decreases the statistical power of our tests. Indeed, the interaction term loses statistical significance when controlling for father's age at birth. However, the effect sizes of the interaction term are similar to our main results.

Table E.1. Results of the regressions of years of education on the gene-environment interaction and additional control variables.

	Within-family analysis			
	(1)	(2)	(3)	(4)
Firstborn	0.415***	-10.488***	0.199	0.021
	(0.109)	(3.777)	(0.174)	(0.220)
PGS for years of education	0.567***	-4.870***	0.576***	0.528***
	(0.078)	(1.286)	(0.127)	(0.155)
Firstborn × PGS for years of education	0.204**	0.150	0.265**	0.235
	(0.080)	(0.092)	(0.125)	(0.160)
Mother's age at birth			0.099	
			(0.088)	
Father's age at birth				-0.028
				(0.148)
Constant	12.544***	25.753***	12.376***	6.177***
	(1.838)	(3.019)	(2.976)	(2.278)
R^2	0.059	0.076	0.062	0.093
N	14,850	14,850	5,601	3,085

Notes: Robust standard errors in parentheses, clustered by family; * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components of the genetic relatedness matrix) are not displayed, but available upon request from the authors. Column 1 replicates our main results for comparison. Column 2 includes as additional control variables the interactions between firstborn and the polygenic score with year of birth, month of birth, gender and the first 40 principal components. Column (3) and (4) include controls for maternal age at birth and paternal age at birth, respectively.

F. Replication of main results with two different polygenic scores for educational attainment

Columns 1-3 of Table F.1 replicate the main analyses using the polygenic score based on the meta-analysis of the GWAS summary statistics of our own UK Biobank GWAS described in Section 3 and the GWAS summary statistics of 23andMe (Okbay et al., 2016). The procedure for constructing the polygenic score is identical to the one used in the main analysis. Columns 4-6 replicate the main analyses with the polygenic score for educational attainment from the Polygenic Index Repository (Becker, Burik, et al., 2021). From Columns 1-2 and 4-5, we can see that the incremental R^2 of both scores are quite comparable, 1.1% and 1.2% respectively. The GxE interaction terms are slightly smaller than in the main analyses, but they are not significantly different from our main results.

Table F.1. Results of the regressions of years of education on the gene-environment interaction with meta-analyzed polygenic scores (PGS).

	Within-family analysis			Within-family analysis		
	(1)	(2)	(3)	(4)	(5)	(6)
	OLS	OLS	OLS	OLS	OLS	OLS
Firstborn			0.436***			0.448***
			(0.109)			(0.109)
PGS for years of education (meta)		0.664***	0.616***			
		(0.069)	(0.077)			
Firstborn × PGS for years of education (meta)			0.136*			
			(0.079)			
PGS for years of education (repository)				0.662***	0.625***	0.625***
				(0.069)	(0.077)	(0.077)
Firstborn × PGS for years of education (repository)						0.113
						(0.079)
Constant	13.757***	13.408***	12.348***	13.747***	13.549***	12.438***
	(1.685)	(1.823)	(1.838)	(1.690)	(1.771)	(1.777)
R^2	0.046	0.057	0.060	0.046	0.058	0.060
N	14,850	14,850	14,850	14,835	14,835	14,835

Notes: Robust standard errors in parentheses, clustered by family in the within-family analysis; * $p<0.10$, ** $p<0.05$, *** $p<0.01$; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components) are not displayed, but available upon request from the authors; The PGS for years of education in Columns 1-3 is based on a meta-analysis of summary statistics from our own UK Biobank GWAS and 23andMe; The PGS for years of education in Columns 4-6 has been taken from the Polygenic Index Repository (Becker et al., 2021).

G. Systematic differences in polygenic scores for other traits by birth order.

In this section, we test if there are any systematic differences in polygenic scores for anthropometric, health, and personality traits by birth order. Polygenic scores for these analyses are taken from Batch 1 of the Polygenic Index Repository which excludes the siblings sample of the UK Biobank (for further technical details, see Becker et al.(2021)). Table G.1 reports the results of regressions of the selected single-trait polygenic scores on a dummy for being firstborn and control variables for year and month of birth, gender, the first 40 principal components of the genetic relatedness matrix, and family fixed effects. After Bonferroni correction for multiple testing (0.05/28), being firstborn is not significantly associated with any of the selected polygenic scores.

Table G.1. Results of the regressions explaining selected single-trait polygenic scores from the Polygenic Index Repository (Becker et al., 2021) on being firstborn.

Trait	Coefficient	SE
Anthropometric		
1. Body mass index	0.002	(0.019)
2. Height	-0.018	(0.018)
Health and health behaviours		
3. Alcohol misuse	0.050	(0.019)
4. Asthma	-0.021	(0.019)
5. Asthma/eczema/rhinitis	-0.016	(0.019)
6. Attention deficit hyperactivity disorder (ADHD)	0.000	(0.019)
7. Cannabis use	0.045	(0.019)
8. Cigarettes per day	0.002	(0.019)
9. Depressive symptoms	0.004	(0.019)
10. Drinks per week	0.026	(0.019)
11. Ever smoker	0.026	(0.019)
12. Hayfever	0.002	(0.019)
13. Migraine	0.012	(0.019)
14. Nearsightedness	0.006	(0.019)
15. Physical activity	0.039	(0.019)
16. Self-rated health	0.001	(0.019)
Personality and well-being		
17. Adventurousness	0.001	(0.019)
18. Extraversion	-0.009	(0.019)
19. Left out of social activity	-0.014	(0.019)
20. Life satisfaction, family	-0.026	(0.019)
21. Life satisfaction, friends	-0.004	(0.019)
22. Morning person	-0.000	(0.019)
23. Narcissism	-0.030	(0.019)
24. Neuroticism	0.007	(0.019)
25. Openness	-0.014	(0.019)
26. Religious attendance	-0.023	(0.019)
27. Risk tolerance	0.049	(0.019)
28. Subjective well-being	-0.020	(0.019)

Notes: N=14,835; Robust standard errors (SEs) in parentheses, clustered by family in the within-family analysis; Coefficients for the control variables (year and month of birth, gender and the first 40 principal components) are not displayed, but available upon request from the authors.