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Giorgia Menta
Luxembourg Institute of Socio-Economic Research (LISER)

Anthony Lepinteur
University of Luxembourg and IZA

Andrew E. Clark
Paris School of Economics and IZA

Simone Ghislandi
Bocconi University

Conchita D'Ambrosio
University of Luxembourg

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IZA – Institute of Labor Economics

Schaumburg-Lippe-Straße 5–9
53113 Bonn, Germany

Phone: +49-228-3894-0
Email: publications@iza.org

www.iza.org

ABSTRACT

Maternal Genetic Risk for Depression and Child Human Capital*

We here address the causal relationship between the maternal genetic risk for depression and child human capital using UK birth-cohort data. We find that an increase of one standard deviation (SD) in the maternal polygenic risk score for depression reduces their children's cognitive and non-cognitive skill scores by 5 to 7% of a SD throughout adolescence. Our results are robust to a battery of sensitivity tests addressing, among others, concerns about pleiotropy and dynastic effects. Our Gelbach decomposition analysis suggests that the strongest mediator is genetic nurture (through maternal depression itself), with genetic inheritance playing only a marginal role.

JEL Classification: I14, J24
Keywords: maternal depression, human capital, ALSPAC

Corresponding author:
Anthony Lepinteur
University of Luxembourg
2 Av. de l'Universite
4365 Esch-sur-Alzette
Luxembourg
E-mail: Anthony.lepinteur@uni.lu

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1. Introduction

Mental-health disorders have been rising steadily for over two decades (Stansfeld *et al.*, 2016), and these are now estimated to affect over 20% of the population in the UK (www.mind.org.uk) and the US (www.nami.org/mhstats). Depression is one of the most common of these disorders. A vast literature has documented worse outcomes for the depressed in terms of not only health, but also employment and earnings (Zimmerman and Katon, 2005; Fletcher, 2013; Banerjee *et al.*, 2017; Hakulinen *et al.*, 2019), productivity (Bubonya *et al.*, 2017), marital status and marital satisfaction (Gotlib *et al.*, 1998), and parenting style (Kiernan and Huerta, 2008). Major Depressive Disorder has been identified as the largest worldwide contributor to years lost to disability (Prince *et al*., 2007).

Depression is a complex trait that depends on a variety of behavioural and environmental factors (Assari, 2017; Zimmerman and Katon, 2005). Part of an individual's life-time risk of depression additionally depends on her genetic makeup and its interactions with the surrounding environment (Dunn *et al*., 2015). Recent advances in Molecular Genetics have helped to identify the diffused signal coming from a multitude of genetic variants that are correlated with depressive symptoms – variants that were shown to predict about 1% of the observed variability of depression in Turley *et al*. (2018). While relatively small in size, this figure is comparable to two-thirds of the combined predictive power of income, race, health, region, and demography on a similar measure of depressive symptoms (Zimmerman and Katon, 2005).

There is some work linking the genetic risk of depression to individual outcomes. For instance, Mendelian Randomisation studies (where genetic variants are used as instrumental variables) reveal that greater genetically-driven depression affects sociodemographic characteristics (Campbell *et al.*, 2022; Reed *et al.*, 2022) and predicts health issues such as cardiovascular diseases (de Geus, 2020) and Vitamin-D deficiency (Mulugeta *et al.*, 2021). Other research has investigated the direct link between the genetic risk of depression and health outcomes, such as white blood-cell count (Sealock *et al*., 2021) and patients' response to lithium as a treatment for bipolar disorder (Amare *et al*., 2021).

The adverse socio-economic consequences of depression likely spill over onto others (see Gotlib *et al*., 2020, for a recent summary), and onto children in particular (see Goodman *et al.*, 2011, and O'Hara and McCabe, 2013, for meta-analyses and reviews of the psychological literature). While the causal link between parental mental health and child outcomes is of primary policy importance, it is not in general particularly easy to establish. The interplay

between maternal mental health and child human-capital development is complex and subject to potential endogeneity concerns. For instance, poor child school performance or behavioural problems might themselves produce maternal depression; alternatively, environmental variables (shared by parents and children who live in the same household), such as local public goods or criminality, could feed through to both parental mental health and child outcomes. In both cases it is difficult to establish causality. Contrary to depressive symptoms over the life-course, the genetic propensity to be depressed is fixed at conception and can be taken as exogenous conditional on the parents' genes. Additionally, a person's genetic endowment can be seen as a 'circumstance' (as defined in the inequality of opportunity literature: see, for example, Roemer, 1998, and Atkinson, 2015) that can translate into a source of unfair advantage or disadvantage for them and their family later in life (Harden and Koellinger, 2020). As such, inequalities that stem from the genetic lottery at conception might be an object of interest to policy makers whose objective is to equalise opportunities (Joint Research Centre F7 - Knowledge Health and Consumer Safety, 2019). We are not aware of any work considering the role of parents' genetic risk for depression on child outcomes.

We here consider the human-capital consequences of growing up with a mother with a higher genetic propensity for depression, using data from a unique British birth-cohort survey. In particular, we estimate the effect of the maternal genetic risk of depression using a synthetic measure (the polygenic score) based on the mother's genetic variants that are robustly associated with the trait of depression. Our empirical analysis is based on genetic and socio-economic information on mother-child pairs from the Avon Longitudinal Study of Parents and Children (ALSPAC), a UK-based cohort study that recruited about 14,000 pregnant mothers in the early 1990s. The key explanatory variable is the polygenic score (PGS) for maternal depression, built from Genome-Wide Association Studies (GWAS) summary statistics from the depression meta-analysis in Turley *et al.* (2018). Following the human-capital development and skill-formation literature (see, for example, Cunha and Heckman, 2008), we consider child cognitive and non-cognitive skills as human-capital components. The cognitive element is given by the measurement of child skills and knowledge at different stages of compulsory education in the UK. We in particular analyse the child's average Key Stage test-scores at ages 11 and 14, and their total GCSE score at age 16 (at the end of compulsory education); all three of these test scores come from administrative data. Non-cognitive skills come from the child's scores using the questions in the Strengths and Difficulties Questionnaire (as reported by their principal carer) at child ages 11, 13 and 16.

We find that an increase of one standard deviation in the maternal PGS for depression has a persistent negative impact on both cognitive and non-cognitive skills, with an effect size of around 4.5% of a standard deviation for the former and 6.5% for the latter. Our conclusions remain unchanged when we (1) account for horizontal pleiotropy (that is, when one genetic variant is associated with multiple traits) and potential shared genetic aetiology between depression, cognitive and non-cognitive skills, (2) exclude genetic variants linked to any known biological pathways, (3) address dynastic effects, (4) attenuate assortative matching concerns regarding depression between parents, and (5) account for reporting biases by instead using teacher-reported scores for the Strengths and Difficulties Questionnaire. Last, we estimate the extent to which genetic inheritance (the transmission of genes from parent to child) and genetic nurture (the influence that parental genes have on child outcomes independent of genetic inheritance; see Kong *et al*., 2019) explain the negative impacts of a greater maternal genetic propensity for depression. With a decomposition-style exercise as in Gelbach (2016), we address the issue of the sequence sensitivity of covariates and find that the genetic nurture, through maternal depression, is the most important mediator. Genetic inheritance on the contrary plays only a marginal role.

The remainder of this paper is organised as follows. Section 2 provides an overview of the genetic approach to our research question. Section 3 then describes the birth-cohort data that we use and outlines the empirical framework. The main results, a variety of robustness checks, and the mediation analysis appear in Section 4. Last, Section 5 concludes.

2. A Genetic Approach

2.1. From Mendel to Polygenic Scores

Establishing causality in non-experimental data is very often challenging, particularly so for variables that are unlikely to be targeted by policies or be subject to exogenous variation. Thanks to the quasi-experimental setting of the genetic lottery at conception, genetic data can help overcome some of these challenges. The biological rationale is rooted in Mendel's laws of genetic inheritance, which are involved in the formation of reproductive cells (*i.e.* gametes) through meiosis and ensure genetic variability across children of the same parents. Take, as an example, a trait that is regulated by only one gene; this is defined by two alleles (one inherited from each parent).[1] The Law of Segregation states that each individual has a 50% chance of

[1] Alleles are the possible nucleotide variations of a genetic variant. Typically, for a common genetic variant such as a Single-Nucleotide Polymorphism (SNP), there would be two possible nucleotide variations, hence two

inheriting each one of her mother's two alleles for a given gene, with the same reasoning applying for the father's alleles. The Law of Independent Assortment, on the other hand, ensures that alleles for different traits are passed on independently of each other.[2] As a result, conditional on the parental genotypes, the child's genotype can be seen as the outcome of a lottery.

Genetic variants can be used as a conditionally-exogenous source of variation in a variety of contexts, such as Mendelian Randomisation (see Koellinger and De Vlaming, 2019, and Hemani *et al.*, 2018, for reviews of some recent developments). While some traits can be linked to a clear small set of genetic variants through well-characterised biological pathways (this is the case for severe health problems, such as Huntington's disease), most traits that interest economists and other social scientists (e.g., socio-economic status, education and subjective well-being) involve a greater degree of genetic complexity and, as such, are highly polygenic. The burgeoning literature on large-scale GWASs, which aims to estimate the relationship between a given trait and known genetic variants (typically Single-Nucleotide Polymorphisms, or SNPs) in large samples, has brought about significant advances in the understanding of the genetic architecture of complex traits such as education (Lee *et al.*, 2018; Demange *et al.*, 2021), depression (Okbay *et al.*, 2016; Turley *et al.*, 2018) and risk behaviour (Karlsson Linnér *et al.*, 2019).

However, in practice, each single SNP identified in a GWAS of complex traits likely has only relatively little predictive power on its own. Polygenic scores have then come into widespread use as a single synthetic measure given by a linear combination of all of the relevant genetic markers (Appendix B provides more details on the PGS and its functional form), capturing a greater portion of trait variance as compared to single SNPs (DiPrete *et al.*, 2018; Davies *et al.*, 2015).

2.2. Conditional exogeneity

Due to Mendel's Laws of Inheritance, the mother's genotype can be considered as truly random only when conditioning on the maternal grandparents' genotype. In a parametric framework, this can be ensured by either controlling for the grandparents' genes or using maternal family fixed-effects (if we had information on the mother's siblings). In practice, for

possible alleles. See Appendix B for further details on the definition of this and other genetic concepts used throughout this paper.

[2] The Law of Independent Assortment does however come with a caveat: genes that are close to each other on a chromosome strand have a higher chance of being transmitted together. This leads to what is known as *linkage disequilibrium*: in a given population, alleles for different genes have higher association rates than those that would be predicted from random matching.

data-availability reasons, it is seldom possible to partial out the genes of the mother's parents. What are then the main challenges to identification, if within-family analysis is not a viable option?

A first concern comes from genetic ancestry, which can influence both cultural practices linked to the trait of interest and systematic drifts in allelic frequencies within a population group. This phenomenon is known as population stratification, and bias from this source is more severe in populations with high ethnical and cultural diversity. Some common established good practices in social science genetics are the restriction of the analysis to ethnically-homogenous groups, while controlling for residual population stratification,[3] and documenting the absence of systematic correlations between the mother's genetic variants and observable confounders (Smith *et al.*, 2007; Boef *et al.*, 2015).

On top of population stratification, another form of bias can arise from dynastic effects, that is the confounding role of the genotypes of one's parents and their ancestors. As the ancestors' genotypes are often unobserved, it is seldom possible to account for their effect directly. In the context of multivariate regressions, we can attenuate some of the concerns about dynastic effects by controlling for a selected set of grandparental traits and environmental characteristics. Consider, as an example, the potential confounding role of grandparental depression in the association between a mother's PGS for depression and her child's human capital. Depressed grandparents are first more likely to have genetic variants associated with depression: via genetic inheritance, their daughters will then also likely display a higher PGS for depression. In addition, grandparental depression can affect child outcomes directly: this could reflect, for example, the crowding-out effect of the time that mothers with depressed parents can dedicate to their children. Not controlling for grandparental genes and/or their associated traits could thus confound the association between the mother's PGS for depression and her child's outcomes. Introducing controls for the depression of both of the maternal grandparents, as well as for other grandparental traits and environmental characteristics, can help attenuate concerns about this potential source of bias.

Last, patterns of assortative matching might constitute another source of confounding – regardless of whether the empirical setup is within- or between-family. If partners match on the basis of genetic similarity, then a person's genotype might correlate with their partner's

[3] One popular solution, which is particularly well-suited in contexts of considerable geographical and ethnic diversity, is to control for the principal components derived from genotyped data. These account for systematic associations between the alleles in subsets of a given population that are produced, among other things, by within-group assortative-matching patterns.

genotype. Take, as an example, a couple formed by partner A (the mother) and partner B who matched assortatively on their depression-associated genotypes (so that there is a positive association between A and B's depression genotypes). Through his own increased probability of experiencing depression, partner B's depression genotypes will also likely affect partner A's own likelihood of being depressed. As such, partner B's depression genotypes are likely omitted variables in the association between partner A's genetic propensity to experience depression and a given trait (depression, or something else) that can affect her child. Accounting for linkage disequilibrium (see the definition in footnote 2) and cross-genotype assortative matching further complicates the picture. The first-best solution would be to control for the partner's genotypes in the empirical analysis. As, again, it might be hard to observe the DNA of both partners, a second-best solution is to control for the partner's observable traits that likely mediate most of the association between the partner's genotypes and the outcome of interest.

In Section 3.3 below we will discuss how we address the conditional exogeneity of the PGS in our empirical context.

3. Data and Empirical Strategy

3.1. The Avon Longitudinal Study of Parents and Children

We will use mother's genetic information to establish the causal effect of her genetic propensity for depression on her child's cognitive and non-cognitive outcomes. The data requirements to carry out this analysis are stringent. We need information on both the mother's and the child's genotype, as well as child outcomes. Few datasets contain these and other information on family characteristics. One that does is the Avon Longitudinal Study of Parents and Children (ALSPAC) survey, also known as 'The Children of the 90s'.

ALSPAC is an English birth-cohort study designed to investigate the influence of environmental, genetic, and socio-economic variables on health and development over the life course. Over 14,000 pregnant women who were due to give birth between April 1991 and December 1992 in the county of Avon (Bristol and its surrounding areas) were recruited. These women and their families have been followed ever since, even if they move out of the original recruitment area (see www.bristol.ac.uk/alspac/). The pregnancy outcomes of the participants resulted in a total of 14,062 live births, with 13,988 children surviving their first year. The sample is broadly representative of the early 1990s UK population of mothers with children under age one, although higher socio-economic status groups as well as Whites are over-represented (see Fraser *et al.*, 2013, and Boyd *et al.*, 2013, for a full description of the cohort

profile). The study includes detailed information about the family environment, as well as indicators of child development, wellbeing and skills over time, and rich information on the parents' characteristics and background.[4] Biological samples from the children and their parents were collected at different points in time, allowing for DNA genotyping. We here use imputed genotype data from around 9,000 children and their mothers (Taylor *et al.*, 2018, provide technical details on the genotyping technology, imputation, and quality control in ALSPAC).

Our child non-cognitive skill measures come from the Strengths and Difficulties Questionnaire (SDQ) (as used in Flèche, 2017; Briole *et al.*, 2020; and Clark *et al.*, 2021). The SDQ is a 25-question behavioural-screening tool for children, including questions on whether the child is considerate of others, and her concentration span, worries and fears, degree of obedience, and social isolation (Goodman, 1997). The full list of the SDQ items appears in Appendix Table A1. The main carer (this is the mother in the vast majority of cases) was asked to rate the child's SDQ seven times between child ages 4 and 16. We will relate the mother's depression PGS to the child's subsequent SDQ scores at ages 11, 13 and 16.[5]

The 25 SDQ items are split up into five sub-scales covering emotional problems, peer problems, conduct problems, hyperactivity/inattention and pro-social behaviour. Consistent with Goodman *et al*. (2010) and the SDQ scores produced by ALSPAC, our main analysis will use the total SDQ score, which is the sum of the first four sub-scales. We code total SDQ so that higher values represent better outcomes (*i.e.* strengths rather than difficulties). In the robustness checks (Section 4.4.2), we will consider additional non-cognitive skill measures to test for convergent validity (teacher-reported SDQ scores, and an alternative measure of non-cognitive skills from the Short Moods and Feelings Questionnaire, SMFQ, reported by the main carer).

Child cognitive development is measured by their national exam results in linked administrative data from the UK National Pupil Database. We use the average Key Stage fine-grading test-scores at ages 11 and 14 and the total General Certificate of Secondary Education (GCSE) score in all of the exams that the child took at the end of compulsory education at age 16.[6]

[4] The study website contains details of all the data that is available through a fully searchable data dictionary and variable search tool: http://www.bristol.ac.uk/alspac/researchers/our-data/.

[5] We focus on the total SDQ score measured between age 11 and 16 because this is the only period for which cognitive skills are available in our dataset. However, we will discuss the effect of the maternal PGS for depression on the total SDQ scores at younger ages in our robustness checks.

[6] At the end of Key Stages 2 (age 11) and 3 (age 14), children's progress in Mathematics, Science and English is assessed using National Curriculum tests. While these tests do not produce exit certificates, the national exams

3.2. The PGS for Depression

Thanks to the availability of imputed genotyped data for ALSPAC mothers, we are able to compute a PGS for depressive symptoms using the command-line program PLINK 1.9, with summary statistics from the single-trait depression meta-analysis GWAS in Turley *et al.* (2018). 68 of the 88 SNPs identified in the GWAS are genotyped in ALSPAC participants and were used in the PGS: see Appendix B for the details of the calculation. As noted above in Section 2.2, a PGS based on such a general-population GWAS remains exposed to issues such as population stratification and dynastic effects. One potential solution would be to use a PGS based on summary statistics from a within-family GWAS, such as the within-sibship GWAS of Howe *et al*. (2022). Within-family GWASs help to identify cleaner SNP-trait associations, net of assortative matching, population stratification and dynastic effects. As such, the relevant SNPs and their weights would arguably reflect true genetic associations rather than environmental and ancestry confounding. However, discovery sample sizes for these GWASs are still comparatively small due to the relative scarcity of genetic family data. These GWASs are then still unable to capture sufficiently-precise associations. Despite this being the largest currently-available within-family GWAS for depressive symptoms, none of the SNPs identified in Howe *et al*. (2022) is significant at the commonly-used genome-wide significance threshold of $p<5\times10^{-8}$). Even so, we have calculated a set of PGSs based on the sibship GWAS and the general-population GWAS of Howe *et al*. (2022), using more-liberal p-value thresholds for the selection of relevant SNPs (see Appendix B for details of these calculations).

We compare the performance of the PGSs based on Turley *et al*. (2018) and Howe *et al*. (2022) in Appendix Figure A1. In the first column, the PGS from the summary statistics of Turley *et al*. (2018) is systematically associated with depressive symptoms in ALSPAC, regardless of how these latter are measured (25 out of the 28 associations that we tested are positive and significant at conventional levels).[7] Strong positive associations with depressive symptoms are also seen in columns two and three of Appendix Figure A1 for other versions of the Turley *et al*. (2018) depression PGS that take into account concerns about horizontal pleiotropy (see Section 4.2. for more details on the calculation of these PGSs). This is however

taken at the end of Key Stage 4 (at age 16), GCSEs, do produce qualification certificates. Students in the UK typically take at least 5 GCSEs (one per subject), with Mathematics, Science and English being compulsory.

[7] The measures of maternal depression displayed in rows of Figure A1 are defined as follows: "Feeling depressed (*t*)" indicates a dummy for the mother having felt depressed between period *t* and *t-1* (where *t-1* refers to the previous interview date); "Episodes of depression (*s-t*)" is the sum of all depression episodes in the time period between *s* and *t*; "Saw doctor for depression (*t*)" is a dummy for the mother having consulted a doctor after feeling depressed between period *t* and *t-1*; and "EPDS (*t*)" is the value of the Edinburgh Postnatal Depression Score in period *t*.

not the case for the PGSs based on Howe *et al*. (2022): the PGSs in the last four columns of Figure A1 display almost no significant associations with the measures of depression available in ALSPAC (details on these new four PGSs, based on the within-family and general-population summary statistics of Howe *et al*., 2022, appear in Appendix B). As the within-sibling discovery cohorts are still relatively small (16,782 siblings for the depression sibship GWAS, compared to the half-million participants in Turley *et al*., 2018), the PGSs calculated from Howe *et al*. (2022) systematically fail to predict depressive symptoms in our ALSPAC sample, despite the use of more-liberal p-value thresholds to detect the relevant SNPs. We therefore carry out our main analysis using the PGSs constructed on the basis of Turley *et al*. (2018), directly addressing in our empirical strategy the issues linked to using general-population GWAS summary data.[8]

3.3. Empirical Strategy

We estimate the effect of the maternal genetic propensity for depression on child human capital via the following OLS regression:

$$HK_C^t = \alpha_1 PGS_M^D + \alpha_2 X_C + \alpha_3 PC_M + \epsilon_M . \quad (1)$$

In Equation (1), the outcome HK_C^t is successively different measures of child C's human capital at age t: the fine-grading average Key Stage test-scores at ages 11 and 14, the total GCSE score at age 16, and total (carer-reported) SDQ at child ages 11, 13 and 16.[9] We standardise the different HK_C^t variables for comparison purposes, as they are not measured on the same scale.

PGS_M^D is the maternal polygenic score for depression, computed on the basis of 68 SNPs from the single-trait depression meta-analysis GWAS in Turley *et al.* (2018). We standardise the PGS_M^D values, as polygenic scores have no natural scale. Given that the polygenic scores are based on genetic variants that are determined at conception, the variation in PGS_M^D is fixed prior to the child's birth: this rules out reverse-causality concerns.

[8] We have replicated our main analysis using the different PGSs calculated from Howe *et al.* (2022) for completeness: the results appear in Appendix Table A4. We find almost no significant associations between the mother's depression PGS and her child's outcomes. The finding of significance would actually be puzzling here: as the PGSs are not strongly correlated with the depression measures, any significant effect would imply that the depression PGSs affect child outcomes only via non-depression pathways.

[9] Unlike the average Key Stage test-scores at ages 11 and 14, the total GCSE score at age 16 includes not only the exam results in English, Mathematics and Science but also those in the other subjects chosen by the student. We could harmonise the exam results over the different ages were we to have information on the separate age-16 results in English, Mathematics and Science: this is unfortunately not the case. As such, the cognitive-skills results at age 16 reflect both differences in scores within-subject across pupils, and differences in the selection of subjects.

As explained in Section 2.2., the role of PGS_M^D in Equation (1) cannot directly be read as causal: we need to address concerns such as population stratification and dynastic effects. We attenuate population stratification first by excluding mothers of non-European descent: Hansell *et al.* (2015) find no evidence of any remaining population stratification in ALSPAC after this selection and other standard quality-control (QC) procedures (see Taylor *et al.*, 2018, for a complete overview of the QC procedures that were applied to ALSPAC data prior to its release). While the documented lack of stratification provides evidence in favour of the independence assumption in our context, we always control for 10 ancestry-informative principal components PC_M (as in von Hinke *et al.*, 2016). As argued by Abdellaoui *et al.* (2019), however, there may still be residual forms of population stratification in the UK in the form of geographic clustering, even after controlling for ancestry-informative principal components. Given the geographically-circumscribed nature of the ALSPAC dataset (only covering residents of the former county of Avon), we believe this to be a second-order issue in our empirical application. We last exploit the rich information contained in ALSPAC to partly address the concerns of dynastic effects and assortative matching by controlling for, respectively, grandparental characteristics and the partner's depression (see Section 4.2.2).

In the main specification set out in Equation (1), we control for a limited set of exogenous, time-invariant child traits X_C: gender, birth year and birth order. We do not include maternal controls or time-variant child characteristics as it can be argued that some of these are bad controls, being themselves potentially influenced by the maternal genetic propensity to be depressed (PGS_M^D).

Our estimation sample consists of observations with non-missing values for mothers' genetic information and the child time-invariant controls. As there are only 1,065 families with non-missing information on all six human-capital measures, we here use a different estimation sample for each dependent variable to maximise statistical power. Our final samples consist of between 2,036 and 2,993 observations per equation estimated. Due to attrition, the size of the non-cognitive skills estimation samples falls naturally with child age (from 2,989 to 2,073 observations). For cognitive skills, the estimation samples consist of 2,825 observations at age 16 (GCSE), 2,598 observations at age 11 (KS2) and 2,034 at age 14 (KS3). Note that the mother's PGS for depression is not predictive of attrition (with the exception of the KS2

sample: the probability to be in the GCSE sample but not in the KS2 sample increases with mother's PGS for depression).[10]

Appendix Figure A2 displays the distribution of the maternal PGSs for depression in the non-cognitive skills estimation sample at age 11. As is common in the literature, this is normally distributed. This is also the case for the other estimation samples. The distribution of the measures of children's human capital is depicted in Appendix Figure A3, and the complete descriptive statistics are listed in Tables A2 (cognitive skills) and A3 (non-cognitive skills).

4. Results

4.1. Main Results

Table 1 presents the OLS estimates from Equation (1) of the effect of the mother's genetic propensity for depression on the different measures of her child's human capital during adolescence. The top half of the table shows the coefficients on the maternal PGS for depression in the test-scores regressions, while the bottom half shows those in the SDQ regressions. The child time-invariant characteristics (gender, birth order, and birth year) are held constant in the even-numbered columns only.

From Table 1, all of the estimated coefficients are negative and statistically different from zero at the 5% level at least. The estimated coefficients are robust to the introduction of the exogenous controls, as we would expect under the assumption that these exogenous child controls are orthogonal to the maternal PGS. A one standard-deviation (SD) rise in the mother's PGS for depression reduces child test-scores by 4.5% SD on average and total SDQ by around 6.5% SD.

In our main model, we make the implicit assumption that the mother's PGS for depression affects child human capital linearly. We can test this assumption by replacing the continuous depression PGS by a series of dummy variables for the quartiles of the depression PGS distribution. The results appear in Appendix Table A5. In all cases, children whose mothers have a PGS at the top of the distribution have worse cognitive and non-cognitive abilities than

[10] The discrepancy between the sample sizes at age 16 and earlier child ages reflects that the average KS2 and KS3 grades are retrospectively matched when the child takes her GCSE exams at age 16. 10% of the 227-observation difference between the GCSE and KS2 samples is due to either missing values in the school and academic year identifiers or in the grades, while the remaining 90% is due to the NPD data-cleaning process. For the gap between the GCSE and KS3 samples, 258 observations are missing for these two reasons, while the remaining 533 are due to the KS3 grades of ALSPAC children taking their GCSE in academic year 2008-09 no longer being collected. Technical details about the NPD cleaning process and the collection of the KS3 average grades we use here can respectively be found at http://www.bristol.ac.uk/media-library/sites/cmpo/migrated/documents/ ks5userguide2011.pdf and https://find-npd-data.education.gov.uk/en/data_elements/11e50a8a-78d6-425c-871d-9d9fd3330dd9.

children whose mothers' PGS is in the bottom quartile. The estimated coefficients on the second and third quartile PGS variables are for the most part negative and of smaller size. It could be argued that these results suggest some degree of non-linearity. We test for this parametrically by means of a quadratic term for the PGS and report the results in Appendix Table A6. The squared value of the mother's PGS never attracts a significant estimate. We take this as evidence in support of Equation (1)'s assumption of a linear effect of the PGS for depression on child human capital.

To see whether the negative effect of the mother's PGS for depression affects different dimensions of academic performance equally, we re-estimate our main model using the single-subject test-scores in English, Mathematics and Science at ages 11 and 14 as dependent variables (we unfortunately do not have access to the fine-graded point scores for the separate subjects in the GCSE exams at age 16). In a similar spirit, total SDQ can be split into two finer subscales: internalising SDQ (emotional health: the sum of 'peer problems' and 'emotional problems') and externalising SDQ (behavioural issues: the sum of 'hyperactivity/inattention' and 'conduct problems'). The results appear in Appendix Table A7. In the top half of the table, the cognitive-skills penalty from the mother's depression PGS is the same for English and Maths, but insignificant (although still negative) for Science. In the bottom half of the table, the estimated coefficients for internalising SDQ look a little larger than those for externalising SDQ, although the gap between the two is not statistically significant.

We might also ask whether the effect of the maternal genetic propensity to depression is moderated by the child's objective characteristics. Appendix Table A8 re-estimates our main model separately for boys and girls, and then for firstborns and others. We find no systematic differences between these groups in panels A to D.

4.2. Robustness Checks

4.2.1. Horizontal Pleiotropy

As noted above, the PGSs are based on genetic variants that are determined at conception. However, a genetic variant may be associated with more than one trait: this phenomenon is known as horizontal pleiotropy. We may then confound the effect of the maternal PGS for depression with those of other traits that share, at least in part, a common genetic aetiology and are also correlated with child human capital. While not a risk to identification *per se*, pleiotropy does affect the interpretation of the effect of interest, as some of the genetic variants used in the depression PGS can be linked to other traits (say, maternal education) that may in turn feed through to the child's accumulation of cognitive and non-cognitive skills. In our

intergenerational context, pleiotropy can play a role in two ways. The first is through genetic inheritance: the mother-transmitted allele that a child displays for a depression SNP can be in pleiotropy with the child's own human capital (or with other child traits that are associated with her human capital). The second is through genetic nurture (Kong *et al*., 2019): the influence that parental genes can exert on the child's outcomes through environmental channels. This is the case, for instance, when a mother's depression SNP is in pleiotropy with another maternal trait that can shape her child's nurturing environment and play a role in human-capital formation.

We here address pleiotropy in two ways. We first compute the mother's PGS for both cognitive and non-cognitive skills based on the GWAS summary statistics in Demange *et al.* (2021) and introduce these as controls in our main specification. By holding these PGSs constant in our main model, we reduce the probability that the depression PGS will capture the effect of genetic variants that, if transmitted, could have a direct impact on our dependent variables. The results appear in panel B of Table 2 (where panel A reproduces the baseline estimates for comparison purposes). Holding constant maternal genetic variation in cognitive and non-cognitive skills makes very little difference to the estimated mother's PGS coefficients.

Second, we investigate the known biological functions that are linked to the 68 SNPs used in the mother's PGS for depression. We do so using the NHGRI-EBI online GWAS Catalog to review all of the biological functions associated with our SNPs. In line with von Hinke *et al.* (2016), we then calculate a new PGS discarding the six lead SNPs linked to either the cognitive or non-cognitive outcomes.[11] Panel C of Table 2 lists the OLS estimates with this restricted PGS: these are very similar to those in the baseline.

We then go one-step further and calculate the mother's PGS for depression excluding the lead SNPs that predict any trait other than depression, even those that may appear unrelated to human capital (e.g. bone density). The results in panel D of Table 2 continue to be very similar to those in the baseline. The associations between these two restricted PGSs and depressive symptoms in ALSPAC are shown in columns 2 and 3 of Figure A1. Last, we construct a PGS that excludes not only the SNPs that are associated with other cognitive and non-cognitive skills, but also those that are in LD with the genetic variants that are associated with these traits. We define SNPs in a window of 500k base-pairs to be in LD if they display a squared pairwise

[11] At the time of writing, these are the following: rs10514301, rs10789340, rs10045971, rs11876620, rs12958048 and rs174548.

correlation of at least 0.6. The estimates for this PGS are displayed in Panel E of Table 2. Although they remain similar in sign, the point estimates are somewhat smaller than that of Panel A. This is not surprising: by excluding genetic variants from the PGS, we weaken its predictive power over depression (especially over clinical depression, as revealed by Appendix Figure A1) and we should expect the effect of the PGS to converge towards zero. In an alternative specification, we excluded all genetic variants correlated with and/or in LD with an augmented set of traits (cognitive and non-cognitive abilities, BMI, and smoking). When we do so, the PGS we calculated no longer predicts depression and we find no effect on child cognitive and non-cognitive skills (results available upon request).

Additional evidence against pleiotropy appears in Appendix Table A9, which shows the bivariate associations between the PGS for depression and a variety of maternal traits measured between the child's birth and her third birthday. None of these is significantly associated with the PGS for depression. We also looked at whether the PGS for depressions predicts maternal traits at later ages, and found similar results. While we cannot entirely rule out an effect of the genetic variants in the mother's PGS on other unobserved traits involved in child human-capital development, the lack of any correlation with the observed traits is reassuring.

4.2.2. The Influence of Maternal Grand-Parents and the Partner

As our subsample of ALSPAC participants is both geographically-circumscribed and ethnically-homogeneous, and we always control for 10 ancestry-informative principal components, we have little reason to believe that residual population stratification is a major problem. We can nevertheless not *a priori* rule out that the PGS for depression reflects some residual ancestry, dynastic effects, and assortative matching. In particular, the mother's PGS might partly capture the effect of her parents' genotypes, with the latter also potentially being correlated with the unobserved confounders. While we cannot control for the genetic variants of the maternal grandparents, nor use a within-family design, we do have data on a set of grandparental traits: their education, social status, and a dummy for at least one of the maternal grandparents having had a severe mental illness prior to the birth of the child. The results controlling for these variables appear in Panel F of Table 2. The OLS estimates are virtually unchanged from those in the baseline, suggesting that dynastic effects do not play a major role.

Another source of confounding may arise from assortative matching between the child's parents: depressed mothers might choose their partners according to certain traits (depression itself, and/or other traits), which may in turn affect child human capital. We can first show that having a partner, and the partner's working status and education are not systematically explained by the mother's PGS for depression (see Table A7). While this alleviates concerns

about cross-trait assortative matching, mothers with a higher genetic risk of being depressed might be more likely to have a depressed partner. Mothers in ALSPAC are asked to report whether their partner has had depression or has felt depressed in the following time intervals: from the child's birth to child age 8 months; from 8 months to 2 years; from 3 to 4 years; from 5 to 6 years; and from 7 to 9 years. Similar to the mother's own depression episodes (see Section 4.3), we summarise this information on the partner's experiences of depression as a count variable ranging from 0 (never felt depressed) to 5 (felt depressed in all time intervals). While the unconditional correlation between the partner's depression and maternal depression (measured similarly as the number of depressive episodes from the child's birth to child age 12: see Section 4.3) is relatively high (0.34) and significant, its correlation with the PGS for maternal depression is small and not statistically different from zero in both bivariate and multivariate analyses (ranging from 0.02 to 0.04 depending on the estimation sample). Introducing partner's depression as a control makes little difference to our main results: see the last panel of Table 2.

4.2.3. The Measurement of Non-Cognitive Skills

The SDQ measure of non-cognitive skills we use is reported by the mother. As depressed mothers may over- or under-estimate their children's non-cognitive skills (Del Bono *et al.*, 2020), the genetic likelihood of being depressed could also be correlated with a reporting bias. We thus turn to teacher-reported SDQ (which is only available when the child was aged 8 and 11). In the first two columns of Appendix Table A10, a higher maternal PGS for depression continues to reduce total SDQ with an effect size that is statistically similar to that in Table 1. We also test for convergent validity using the SMFQ measure of non-cognitive skills (reported by the main carer – see the questionnaire in Appendix Table A11) in columns (3) to (5) of Appendix Table A10: the resulting estimates are not significantly different from those in the baseline (except for that at age 16, which is not statistically significant).

Additionally, as with any parental characteristics, the effect of the mother's genetic propensity for depression may arguably change over time. This is not what we see in Table 1: the estimates attracted by the mother's PGS are remarkably stable over time. While we do not have access to fine-grading test-scores measured at earlier ages, carer-reported SDQ scores are also available at child ages 4, 6, 8 and 9. We have replicated our main analysis using total SDQ at these ages as dependent variables: see Appendix Table A12. The negative impact of the maternal PGS for depression appears at age 6 and remains qualitatively similar from then on. The PGS coefficient for total SDQ at age 4 is not statistically different from zero, which we take as a first piece of evidence that the relationship between the mother's depression PGS and

her child's human capital is not congenital but rather emerges due to environmental exposure (we will explore the mediating role of environmental factors in Section 4.3).

4.3. Mechanisms

In our main results, a one SD increase in the mother's PGS for depression reduces her child's measures of cognitive and non-cognitive abilities by 4.5 and 6.5% of a SD respectively. In order to establish public policies aimed at mitigating the effect of the maternal PGS for depression on child human capital, we need to identify the underlying mediators. From a theoretical point of view, we expect two mediating channels: genetic inheritance (the direct transmission of genes from mother to child) and genetic nurture.

From genetic inheritance, we can expect a 50% correlation between the mother's and the child's PGSs for depression. This figure might be even higher if parents match assortatively on the basis of their genetic variants for depression, as the child would then have a greater likelihood of inheriting depression-linked alleles from both parents. If the versions of the depression genetic variants the child inherits are also associated with child human capital due to horizontal pleiotropy and/or LD, we can then expect them to mediate part of the effect of the mother's depression PGS. We here again use the summary statistics from the depression meta-analysis GWAS in Turley *et al.* (2018) to calculate a PGS for depression in children. As expected, the raw correlation between the mother's and the child's PGS ranges between 49 and 51% according to the estimation sample, which is consistent with evidence against genetic single-trait assortative matching in the parents as shown in the last panel of Table 2. We additionally use the GWAS-by-subtraction summary data from Demange *et al.* (2021) to compute the child PGSs for cognitive and non-cognitive skills.[12]

Genetic nurture refers to the fact that a child's outcomes can be affected by her parents' genes though environmental pathways. Both transmitted and non-transmitted alleles can in fact have an indirect impact on child outcomes via their effect on the parental outcomes (e.g. parenting practices) and family environment (Kong *et al.*, 2018). We can identify two sub-channels of genetic nurture here. The first is maternal depression: the PGS for depression by

[12] We also used alternative summary statistics from other GWASs (Benke *et al*., 2014; Pappa *et al*., 2016; Middeldorp *et al*., 2016) to calculate alternative polygenic scores for non-cognitive skills, but none of these alternatives was significantly correlated with total SDQ. These results are available upon request. Note that the discovery sample of these additional GWASs coming from the EAGLE consortium include the ALSPAC cohort, so using summary statistics from these GWASs, while more appropriate in terms of both trait definition and age of the discovery cohorts, comes at the risk of inducing spurious correlations between the child PGS and total SDQ.

definition captures a higher genetic probability of suffering from depression, and a number of contributions have underlined a negative link between maternal depression and child outcomes (Perry, 2008; Dahlen, 2016; von Hinke *et al.*, 2022). In ALSPAC, we measure maternal depression as follows. When the child was aged 8 months and 2, 3, 4, 5, 6, 9, and 12 years, their mothers were asked whether they had experienced depression since the last interview in which they were asked about their health (or since the birth of the child, for the 8-months questionnaire).[13] Although the wording of the question changed slightly across waves, the potential responses were the same: "Yes and consulted a doctor", "Yes but did not consult a doctor" and "No". We consider a mother to have experienced an episode of depression between two periods if she replied "Yes and consulted a doctor" or "Yes but did not consult a doctor". We combine these eight reported depression scores to produce two indices of reported maternal depression from the child's birth to the child's 12th birthday (with index values running from zero to eight): one for episodes that were followed by a visit to the doctor and one for episodes that were not. Since these indices may not fully capture all the experiences of maternal depression, we also make use of a diagnostic tool, the Edinburgh Postnatal Depression Scale (EPDS). This is measured at child ages 8 months and 2, 3, 5, 6, 8, and 11 years, and we take its average value for mothers between the child's birth and child age 11. We then carry out a factor analysis on the two indices of self-reported depression episodes and the average maternal EPDS score. This produces two factors, the first loading more heavily on depression episodes that were not followed by a doctor consultation, and the second loading more intensively on EPDS and depression with a doctor consultation. We interpret the first factor as capturing 'lighter' forms of depressive symptoms, and the second as reflecting more-severe symptoms or clinical depression. By using these two factors, we reduce the issue of multicollinearity of the depression measures while preserving the variability coming from different measurement approaches and scales.

The second sub-channel of genetic nurture is the remainder of the family environment. We measure this via a rich vector of maternal characteristics when the child is aged nine: age at the birth of the child and dummies for being employed, having at least an A-level,[14] having a partner, having a partner with at least an A-level, having an employed partner, five normalised

[13] When looking at child outcomes at age 11, considering maternal episodes of depression up to age 12 might induce concerns about reverse causality. We hence replicate all of the analyses in Table 3 using only episodes of depression up to child age 9. We have also replicated all the analyses excluding the episodes of depression reported after child age 6 due to the change in the interval between two interviews (one year *vs.* three years). The results, available upon request, are unchanged.

[14] An A-level (Advanced-Level) qualification is a subject-based school-leaving certificate that is typically obtained at the end of Upper-Secondary School at around age 18.

factors for parental time investments (playing, school and homework, sports and outdoors activities, tending to basic needs, and tending to emotional needs), the number of additional children, and banded household income (the latter two variables are measured at child age 8). We additionally include a 'life-event score' (measured at child age 5) – an index calculated by the data producer that measures the study child's intensity of exposure to 18 different arguably-traumatic events (such as being sexually abused or the parents separating). This ranges from 0 to 72, with higher values reflecting greater exposure to disruptive events.

To estimate the extent to which genetic inheritance and genetic nurture mediate our main results, we use the conditional decomposition method of Gelbach (2016). This method compares the coefficient on a variable of interest (in our case, the mother's PGS) in a 'base' model specification and in a 'full' model specification (where potential mediators are included as covariates), allowing for the assessment of the contribution of each potential mediator to the difference in the coefficients across the two empirical models. Doing so additionally allows us to avoid the problem of sequence sensitivity of these estimated contributions that arises when the covariates that are being tested in the model are correlated between themselves.

As noted above, genetic inheritance is measured via the child's own PGS for depression and, to account for potential genetic overlap and linkage disequilibrium between depression and the outcomes, her PGSs for cognitive and non-cognitive skills (the results do not however depend on the inclusion of these latter two PGSs). Genetic nurture is instead here measured by two components: the two normalised factors for maternal depression and the set of family characteristics described above.

The results from this mediation exercise appear in Table 3. Overall, our mediators have greater explanatory power in the SDQ regressions than in the test-scores regressions (they explain, respectively, about 45% and 15% of the baseline estimates). Of the covariates that contribute to the explained portion of the baseline estimates, genetic nurture is the strongest mediator, with maternal depression being the only factor to explain a significant share of the baseline estimates in most cases. The mediating role of maternal depression, however, seems to fade away over time: this could reflect that, as children grow older, they become more independent from the mother having other resources to count upon, such as friends; or that we are not able to measure new episodes of maternal depression in ALSPAC (maternal depression is no longer consistently recorded after child age 12). The Gelbach estimates of the depression

channel are thus likely attenuated at later ages due to this missing information on the most-recent depression episodes.[15]

With respect to the channel of genetic inheritance, Table 3 shows that the transmission of genetic variants linked to either depression, cognitive or non-cognitive skills does not explain the negative effect of the maternal PGS for depression. As such, the negative relationship between a mother's genetic propensity to be depressed and her child's human capital does not seem to transit via the child's own genetic propensity for depression, but rather through the worsening in the childhood family environment from having a mother with depressive symptoms.[16]

5. Conclusion

Using data from a British birth-cohort study, we have illustrated how genetic variants for depression can be used to estimate the effect of a mother's genetic propensity for depression on her child's human capital. We first show that a one-SD increase in the mother's polygenic score for depression (a synthetic linear combination of the genetic variants for depression) predicts lower child cognitive and non-cognitive skills of about 4.5% and 6.5% of a SD respectively. Our conclusions are robust to a battery of checks addressing, among others, issues of pleiotropy, shared genetic aetiology and dynastic effects.

Given the importance of human-capital accumulation during childhood (Heckman *et al.*, 2006; Heckman *et al*., 2018; Clark *et al.*, 2018; Clark and Lepinteur, 2019), policy-makers may wish to reduce the inequalities between children caused by their mother's genetic propensity for depression. However, the possibility of public action here depends on the factors that lie behind the negative effect of the mother's genetic propensity to be depressed. If this is mostly explained by genetic inheritance, then public policies should focus on reducing the human-

[15] We have carried out what could be considered as a falsification test, and re-estimated our regressions separately for depressed and non-depressed mothers (we used the factors derived from our factor analysis to split the sample into roughly equal-sized parts). If maternal depression is really the main channel behind our results, we would expect to only find a significant coefficient on maternal depression PGS for mothers who experienced depression. This is what we see in Appendix Table A13 for five out of the six outcomes. The different result for the total SDQ score at age 16 may reflect the lack maternal-depression information after child age 12 (so that mothers we classify as less-depressed symptoms might have experienced substantial depression after their child turned 12) or selective attrition (more-depressed mothers are more likely to drop out of the sample for SDQ at age 16). However, it is important to bear in mind that the stratification is endogenous, and that the inferences drawn from Appendix Table A13 should be treated with caution.

[16] Measurement error in the child PGS would imply that we incorrectly estimate the contribution of genetic inheritance. We have addressed this issue via the disattenuation formula of Becker *et al.* (2021). The results from this procedure are consistent with those from the classic Gelbach decomposition for non-cognitive skills for any plausible correction. However, the greater the measurement error in the child PGS, the greater the contribution of genetic inheritance for cognitive skills (although the child's depression PGS is never significantly correlated with the child's test-scores). These results are available upon request.

capital inequalities stemming from genetic differences between children. On the contrary, were the main role to be played by genetic nurture, family policies could help level-up children's nurturing environments. Our decomposition exercise suggests that genetic inheritance does not play a significant role, while genetic nurture, through maternal depression, does appear to be a statistically-important mediator. From a policy perspective, the prevention and treatment of maternal depression will then likely not only benefit mothers, but also improve their children's human capital. As revealed by the evaluation of the Improving Access to Psychological Therapies programme in the UK in Clark (2018), the costs of effective treatments for depression are very low compared to their expected benefits. If treatment also produces positive spillovers on children, the benefit-cost ratio will be even higher, making treatment more attractive.

With the increasing diffusion of polygenic scores into social-science research, it is however important to keep in mind that the genetic component of complex traits, such as mental health, is far from deterministic. The same polygenic score can be found in individuals with a very wide range of values of the trait of interest, reflecting that genetic architecture predicts outcomes partly via individuals' reactions to their environment. This opens an additional door to policy intervention: while genes are fixed, the environment is not. Future research on which stressors are the most important in this context will help advance our understanding of the sign and size of causal relationships that can serve as inputs to public-policy debate.

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Tables:

Table 1: Mother's PGS for Depression and Child Human Capital: OLS Results

	Test-scores					
	Age 11		Age 14		Age 16	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's PGS	-0.043**	-0.041**	-0.046**	-0.042**	-0.055***	-0.049***
	(0.020)	(0.020)	(0.022)	(0.021)	(0.018)	(0.018)
Observations	2598	2598	2034	2034	2825	2825
Controls	No	Yes	No	Yes	No	Yes
	Total SDQ					
	Age 11		Age 13		Age 16	
	(1)	(2)	(3)	(4)	(5)	(6)
Mother's PGS	-0.067***	-0.065***	-0.072***	-0.070***	-0.060***	-0.061***
	(0.018)	(0.018)	(0.020)	(0.020)	(0.022)	(0.022)
Observations	2989	2989	2581	2581	2073	2073
Controls	No	Yes	No	Yes	No	Yes

Notes: Standard errors appear in parentheses. All dependent variables are standardised. The controls are the child's gender, birth year and birth order. All regressions include ancestry-informative principal components. All of the regressions using test-scores as the dependent variable also include school and school-year fixed effects. *** p<0.01, ** p<0.05, * p<0.1.

Table 2: Mother's PGS for Depression and Child Human Capital: Robustness Checks

	Test-scores			Total SDQ		
	Age 11	Age 14	Age 16	Age 11	Age 13	Age 16
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Baseline Estimates						
Mother's PGS	-0.041**	-0.042**	-0.049***	-0.065***	-0.070***	-0.061***
	(0.020)	(0.021)	(0.018)	(0.018)	(0.020)	(0.022)
Panel B: Accounting for Shared Genetic Aetiology with Cog. and Non-Cog. Skills						
Mother's PGS	-0.038*	-0.039*	-0.047***	-0.065***	-0.071***	-0.061***
	(0.020)	(0.021)	(0.018)	(0.018)	(0.020)	(0.022)
Panel C: Excluding Genetic Variants Linked to Cog. and Non-Cog. Skills						
Mother's PGS	-0.038*	-0.038*	-0.043**	-0.060***	-0.063***	-0.053**
	(0.020)	(0.021)	(0.017)	(0.018)	(0.019)	(0.022)
Panel D: Excluding Genetic Variants Linked to Any Known Biological Pathways						
Mother's PGS	-0.040*	-0.033	-0.050***	-0.059***	-0.062***	-0.050**
	(0.020)	(0.021)	(0.017)	(0.018)	(0.020)	(0.022)
Panel E: Excluding Genetic Variants Linked to and in LD with Cog. and Non-Cog. Skills						
Mother's PGS	-0.027	-0.016	-0.024	-0.035*	-0.031	-0.038*
	(0.020)	(0.021)	(0.017)	(0.018)	(0.019)	(0.022)
Panel F: Accounting for Grand-Parental Characteristics						
Mother's PGS	-0.044**	-0.042**	-0.051***	-0.061***	-0.070***	-0.055**
	(0.020)	(0.020)	(0.017)	(0.018)	(0.020)	(0.022)
Panel G: Accounting for Partner's Depression						
Mother's PGS	-0.041**	-0.041*	-0.049***	-0.060***	-0.067***	-0.058***
	(0.020)	(0.021)	(0.018)	(0.018)	(0.020)	(0.022)
Observations	2598	2034	2825	2989	2581	2073

Notes: Standard errors are in parentheses. All dependent variables are standardised. The controls are the child's gender, birth year and birth order. All regressions include ancestry-informative principal components. All of the regressions using test-scores as the dependent variable also include school and school-year fixed effects. *** p<0.01, ** p<0.05, * p<0.1.

Table 3: Mother's PGS for Depression and Child Human Capital: Gelbach Decomposition

	Test-score (Age 11)			Test-score (Age 14)			Test-scores (Age 16)		
	Specification			Specification			Specification		
	Base	Full	Explained	Base	Full	Explained	Base	Full	Explained
Mother's PGS	-0.041**	-0.036*	-0.005	-0.042**	-0.036*	-0.005	-0.049**	-0.055***	0.006
	(0.020)	(0.020)	(0.012)	(0.021)	(0.021)	(0.013)	(0.018)	(0.018)	(0.010)
Covariates:									
Genetic Inheritance			0.002			0.005			0.007
			(0.009)			(0.010)			(0.008)
Maternal Depression			-0.002			-0.007***			-0.001
			(0.001)			(0.002)			(0.001)
Family Environment			-0.005			-0.004			-0.000
			(0.006)			(0.008)			(0.006)
Observations	2598	2598		2034	2034		2825	2825	
	Total SDQ (Age 11)			Total SDQ (Age 13)			Total SDQ (Age 16)		
	Specification			Specification			Specification		
	Base	Full	Explained	Base	Full	Explained	Base	Full	Explained
Mother's PGS	-0.065***	-0.028	-0.037***	-0.070***	-0.044**	-0.025**	-0.060***	-0.045*	-0.015
	(0.018)	(0.020)	(0.011)	(0.020)	(0.021)	(0.011)	(0.022)	(0.024)	(0.013)
Covariates:									
Genetic Inheritance			-0.012			0.001			-0.002
			(0.009)			(0.009)			(0.011)
Maternal Depression			-0.021***			-0.022***			-0.012**
			(0.005)			(0.005)			(0.005)
Family Environment			-0.004			-0.005			-0.001
			(0.004)			(0.004)			(0.004)
Observations	2989	2989		2581	2581		2073	2073	

Notes: Standard errors are in parentheses. All dependent variables are standardised. The controls in the base model are the child's gender, birth year and birth order. The controls in the full model also include the variables in the vectors "genetic inheritance" (child PGSs for depression, cognitive and non-cognitive skills), "maternal depression" (the two normalised factors from the factor-analysis combining the number of undiagnosed and diagnosed episodes of maternal depression from the child's birth to age 12 and the average maternal EPDS from the child's birth to age 11) and "family environment" (mother's traits when the child was aged nine - age at the birth of the child and dummies for being employed, having at least an A-level, having a partner, having a partner with at least an A-level, having an employed partner, five normalised factors for parental time investments, a life-event score at child age 5, and the number of additional children and banded household income, both measured at child age 8). All regressions include ancestry-informative principal components. All of the regressions using test-scores as the dependent variable also include school and school-year fixed effects. *** $p<0.01$, ** $p<0.05$, * $p<0.1$.