Understanding the intergenerational transmission of educational (under)achievement

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Introduction and Rationale

Educational attainment and achievement are key predictors of outcomes in later life, including financial security and social mobility, as well as physical and mental health. Across societies, children with less educated parents often face significant challenges in reaching levels of academic success compared to their peers whose parents have higher education. Research consistently shows that parental education is a primary predictor of children's educational attainment, with evidence of substantial genetic and environmental contributions.

Sstudies typically fail to consider that parents influence their children's educational attainment and achievements both environmentally, via their behaviour and resources, and through the transmission of their genes. Not considering these factors could potentially lead to biased conclusions.

Mothers and fathers each pass on half of their genetic material to their children (***genetic transmission***). We also know that parents' traits and behaviours can shape the child's rearing environment, which is called nurture (for example, reading habits or access to better resources). However, the non-transmitted genes influence the parents' traits and can influence the traits in their children through the parent's trait. In other words**, parental genetics can shape the environment they provide for their offspring, which, in turn, nurtures children's outcomes. This concept is called *genetic nurture***.

Recent advances in genetic epidemiology research have made it possible to integrate measured DNA with observed behaviour to **understand complex gene-environment interplay.** One such complex gene-environment interplay is the fact that biological parents provide both their genetic predisposition and – in most cases – a rearing environment to their children.

This project thus responds to the need for more nuanced insights into the relationship between genetics and environment in the intergenerational transmission of educational outcomes. By jointly accounting for genetic transmission and genetic nurture, **we aimed to provide a broader understanding of the pathways through**

which parental characteristics – including education, socioeconomic position, and health-related behaviours – **influence their children's outcomes.**

Project Questions

This project sought to answer the following questions:

1. **What is genetic nurture and why does it matter for education?**
   The concept of genetic nurture has gained considerable traction in research, highlighting how parental genetics influence a child's environment and development. This specific case of gene-environment interplay is likely to be relevant for researchers working in the field of education and other social scientists. In this first section, we introduce the concept of genetic nurture, and the methods we can use to estimate it.
2. **Are there robust genetic nurture effects in educational outcomes?**
   We wanted to establish whether there are robust genetic nurture effects for educational outcomes. For this purpose, we conducted a meta-analysis evaluating available evidence to date that used a trio design (mother–father–child genotyped data) to estimate the magnitude of genetic nurture versus genetic transmission.
3. **How can we explain genetic nurture effects for educational outcomes?**
   Recognising that parental education itself is intertwined with other traits, we examined additional parental characteristics – such as cognitive performance, lifestyle factors, non-cognitive components of educational attainment (for example: communication skills, self-regulation), and mental health – to identify new avenues for future research into transmission mechanisms and possible intervention targets to address underachievement.
4. **When does genetic nurture manifest in development?**
   We investigated how genetic and environmental influences unfold across different developmental stages, from early primary education through to late adolescence. Understanding when genetic transmission and environmentally mediated parental influences have greater impact in children's behaviour can

help to design more targeted research questions and therefore more effective interventions down the road.

Approach and Methodology

We adopted a combination of approaches designed to separate genetic transmission from environmental effects:

- **Systematic Review and Meta-Analysis**
  We identified and analysed all available published studies of genetic nurture in educational outcomes. This produced combined estimates of both genetic transmission and genetic nurture, enabling more robust conclusions about their relative influence.
- **Longitudinal and Trio Designs**
  Using polygenic scores for parents and children within the same families, we evaluated genetic transmission and genetic nurture effects on educational achievement at multiple developmental stages in large UK birth cohort studies.

  The polygenic scores capture an individual's genetic propensity for various traits (such as educational attainment or smoking behaviour), allowing us to model how parents' and children's genetic predispositions independently influence educational achievement measures and mental health.

It is necessary to stress **that polygenic scores capture genetic predispositions rather than actual behaviour and should not be considered a replacement for observed survey data**. An advantage of polygenic scores however is that they provide a standardised metric that proxy parental traits and removes some of the biases associated with self-reports and heterogeneity of measurements so common in developmental studies.

See Appendix for detailed information about the methodology.

Key Findings

1. **Robust evidence of genetic nurture**
   Across studies, we found that genetic nurture plays a clear role in children's educational outcomes. This means that parental genetics likely influence the home or broader family environment, which in turn affects the child's academic achievement. These findings still hold when we account for the substantial genetic transmission that is occurring.

2. **Socioeconomic position**
   When we accounted for family socioeconomic position and parental education, genetic nurture effects dropped considerably – approximately by three quarters – implying that a substantial portion of genetic nurture operates via socioeconomic resources and opportunities.

3. **Contribution of other parental traits**
   Beyond educational attainment, we identified multiple parental genetic predispositions that influence offspring educational outcomes, including personality, mental health, and reproductive behaviours. For these traits, environmental effects (genetic nurture) often surpassed genetic transmission, underscoring the significance of parental behaviours and the home environment.

4. **Timing of effects**
   Overall, genetic nurture effects are stronger in earlier childhood (around ages 7–11), whereas genetic transmission becomes more pronounced in adolescence. This pattern suggests that environmental factors in early life are especially important for children's initial academic success, while individual factors become more important as the child grows.

5. **Maternal and paternal influences**
   For most traits, we found similar magnitudes of genetic nurture in mothers and fathers. However, for certain variables – such as age at first birth, number of

children ever born, and smoking behaviour – maternal and paternal influences may differ.

Future Research and Recommendations

1. **Multi-agent, multi-stage approach**
   Our results suggest that interventions need to be tailored to developmental stages. Interventions focusing on the parents might be more effective earlier, while child-focused interventions might be more useful in adolescence.

   Researchers should consider the use of longitudinal designs with repeated measures to pinpoint the developmental windows when these interventions are most beneficial.

2. **Continued use of genetically informed approaches**
   Given the evidence of genetic influences in the intergenerational transmission of educational achievement, the use of genetically informed methods in developmental and educational research is warranted (e.g., use of polygenic scores in longitudinal trio designs). Such methods can shed light into the causal pathways between genetic predispositions, environment, and children's developmental trajectories.

3. **Socioeconomic pathways**
   Because our findings underscore the importance of family socioeconomic position in shaping children's rearing environments even after accounting for genetic transmission, research exploring which specific resource-based disparities drive educational underachievement is relevant and necessary.

4. **Maternal and paternal contributions**
   Maternal and paternal influences may operate through distinct mechanisms. Additional research is needed to identify where this is the case and better understand how maternal and paternal influences intersect, particularly in contexts that vary by culture, family structure, or parental involvement.

Conclusion

By jointly considering genetic transmission and genetic nurture, this project provides robust evidence that the environments parents create – shaped by their own genetics – play a significant role in the cycle of underachievement.

This project emphasises the need for a comprehensive approach that integrates genetic and environmental perspectives to educational research. By addressing the complex interplay of genetic influences and early childhood environments, future research and, in due course, intervention programmes, we may be better positioned to provide equitable educational opportunities for all children and foster social mobility.

We emphasise that genetic studies, including ours, cannot directly inform policy decisions due to the complexity of the causal chain from genes to behaviour and the many intervening factors that separate biological influences from policy-relevant outcomes. Ultimately, **genetic nurture research highlights that nature and nurture are not mutually exclusive** but rather deeply entwined processes that affect children's learning and development. Embracing this perspective in policy and practice may foster a more equitable educational landscape and help break the cycle of disadvantage across generations.

Depending on the target, no single intervention is likely to be effective across all developmental stages. Interventions focusing on the parents might be more effective earlier, while child-focused interventions might be more useful in adolescence.

PRIMARY REPORT

What is genetic nurture and why does it matter for education?

Educational attainment and achievement (see Glossary, page 31) are linked to major economic, social, physical and mental health milestones throughout life (Islam & Jaffee, 2023). **Parental education is consistently the strongest predictor of children's educational attainment and achievement across all modern societies** and positively influences their children's educational achievement (Björklund & Salvanes, 2011; Haveman & Wolfe, 1995; Hertz et al., 2008).

Educational underachievement hinders social mobility, leading to an intergenerational cycle of social inequalities. To break this cycle, we must understand how parental education influences their children's achievement. By understanding what leads children with less educated parents to do less well at school, we may be able to design better interventions at multiple stages in children's development.

The Department for Education's *Plan for improving social mobility through education* in 2017 highlighted the key role played by parental education in the processes underlying social mobility. However, for most traits, including educational outcomes, **children resemble their parents because of nature (the genes they inherit) and nurture (the environment they grow up in)**. Research shows that many behaviours linked to academic achievement are influenced by genetics (J. J. Lee et al., 2018; Okbay et al., 2022; Polderman et al., 2015), a finding that is often overlooked or arguably misinterpreted (Barlow, 2019; Harden, 2023; Heine et al., 2019). Educational achievement is shaped by a complex interplay of genetic predispositions and environmental influences that makes the "nature versus nurture" dichotomy obsolete and demands for more complex theories and methods (Barlow, 2019; Turkheimer, 2000).

Integrating genetic and environmental factors in our models can help us uncover how biological processes work alongside external influences, giving us a clearer picture of what drives differences in academic achievement and how to support every learner more effectively. Ignoring genetic influences in educational outcomes not only hinders rigorous aetiological research but might contribute to the very social inequality that we wish to address (Barlow, 2019; Hart et al., 2021; Turkheimer, 2000).

Genetic transmission and genetic nurture

One such complex gene-environment interplay is the fact that parents provide both their genetic predisposition and a rearing environment to their children. Mothers and fathers each pass on half of their genetic material to their children **(*genetic transmission*)**. However, even though the other half is not inherited, these non-transmitted genes continue to influence the parents' traits and can influence traits in their children.

This concept – **when parents' genes influence offspring outcomes by shaping the environment that they provide for them, above and beyond genetic transmission – is known as *genetic nurture*** *(Bates et al., 2018; Kong et al., 2018)*. It describes how parents' genes indirectly shape their children's characteristics via an environmental route.

For example, parents with a higher genetic predisposition for cognitive abilities may have a greater interest in activities such as reading and may engage in nurturing behaviour such as reading to their child, which, in turn, fosters learning in their offspring (see Figure 1). Despite the term 'nurture,' genetic nurture may exist without actual parent-offspring nurturing behaviour. Instead, it could work through other factors, inside or outside the home, which are linked to parental genetic factors, such as income, school quality or neighbourhood.

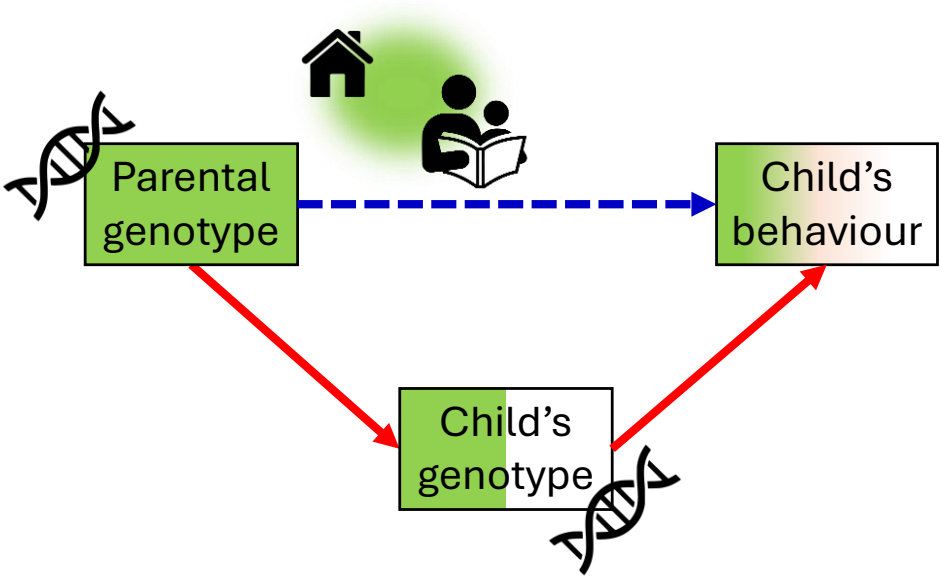

Figure 1. Graphical representation of genetic transmission, i.e., the direct inheritance of genetic material from parents to offspring (continuous line, in red) and genetic nurture, i.e., the influence of parental genes on the environment they provide, which indirectly affects offspring development (dotted line, in blue).

Perhaps counterintuitively, **evidence of genetic nurture is prime evidence that environmental pathways *do matter* when it comes to shaping children's educational outcomes**, **even after accounting for genetic transmission** (the genetic route of transmission whereby parents directly transmit their genes to offspring). That is, even if we are measuring genetics, by identifying genetic nurture effects we can test if the rearing environment of a child, independent from their own genetic predisposition, is influencing their behaviour (for example, educational achievement).

In fact, accounting for genetic transmission is key to provide evidence of environmental effects that are unbiased by genetic effects/confounding. Yet, until recently, **most educational research has failed to consider that parents influence their children's educational attainment and achievements both environmentally through their behaviour and resources, and through their genes**, potentially leading to biased conclusions (Hart et al., 2021). Failure to account for genetic transmission in the association between parental education, or any other parental or environmental factor, and offspring educational achievement hinders our ability to establish any causal links between them.

Polygenic scores and educational research

Since the seminal works by Bates et al. (2018) and Kong et al. (2018) on genetic nurture and education, several studies have aimed to estimate genetic nurture effects for educational attainment. In those studies, *polygenic scores* for educational attainment generated from genome-wide association studies (GWAS, see Glossary on page 31) where combined with family-based designs of educational attainment to disentangle genetic nurture from genetic transmission effects (described below).

A polygenic score can be described as the count of the number of trait-relevant genetic variants present in the person's DNA and it is expressed as a single score for each individual. Before adding them up, genetic variants are assigned a different weight based on the strength of the link between that variant and the outcome of interest that was reported in the sample where the GWAS was conducted. For a review on this topic, see Allegrini et al. (2022) and Pingault et al. (2022). For a lay overview, we refer the reader to the education tool developed by Lia Petronio in collaboration with the Broad Institute: https://polygenicscores.org/explained/.

Accounting for genetic confounding is critical in education research. Work by us and others has shown that polygenic scores - the individual scores summarising the effects of thousands of DNA variants - can explain over 12% of the individual differences in children's educational achievement (Allegrini et al., 2019) and over 13% in educational attainment in adults (Okbay et al., 2022).

It is particularly important to note that polygenic scores do not reflect any individual's innate potential for education. Rather, they capture genetic correlations that *also* reflect the sociocultural, familial, and historical contexts of education and aggregate the effect of hundreds or thousands of genetic variants. In turn, these genetic variants are linked to a host of intermediate traits, each influenced by many other genetic variants and intertwined with environmental conditions.

For example, **polygenic scores can reflect both direct genetic transmission and also social influences**, in particular environmentally transmitted intergenerational effects (Fletcher & Lehrer, 2011). In other words, part of the genetic influences on children's educational achievement reflects the environment that parents provide to their children (Bates et al., 2018; Belsky et al., 2018; Kong et al., 2018). To this purpose, the **polygenic scores for the child, the father and the mother are**

combined into a statistical model to estimate genetic and environmental routes of transmissions (see Figure 2 and Glossary, page 31).

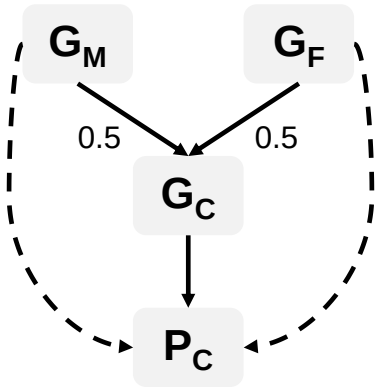

Figure 2. Associations between parental and child genotypes and offspring's phenotype in a trio model using polygenic scores. If associations between parental genotypes (G_M or G_F) and child phenotype (P_C) persist after accounting for child genotype (G_C), this indicates 'genetic nurture' effects. For mothers, for example, genetic nurture effects act via the pathway $G_M \rightarrow P_C$ after accounting for G_C, and 'genetic transmission' effects via the path $G_C \rightarrow P_C$. Note: C = child, F = father, G = genotype, M = mother, P = phenotype.

By identifying the extent to which each transmission route (genetic or environmental) influences offspring behaviour, genetic nurture studies may help researchers to evaluate and design more effective compensatory interventions. That is, once transmission routes are understood, follow-up research can search for distal (such as, parental education, income distribution, equal access to good quality schooling) and proximal targets for intervention programs (for example, parenting).

Therefore, by modelling genetic transmission and genetic nurture together using a genetically informed approach, this work provides answers to the following questions:

- ***Are there robust genetic nurture effects in educational outcomes?***
- ***How can we explain genetic nurture effects for educational outcomes?***
- ***When does genetic nurture manifest in development?***

Are there robust genetic nurture effects in educational outcomes?

The impact of children's polygenic scores on children's achievement appears to be a combination of direct genetic transmission and the rearing environment (Bates et al., 2018; Belsky et al., 2018; Kong et al., 2018). To reiterate, such scores in children can reflect direct genetic transmission but also social influences, in particular environmentally transmitted intergenerational effects. In other words, part of the genetic influences on children's educational achievement reflects the environment that parents provide to their children.

To find robust evidence of genetic nurture effects in educational outcomes, we conducted a meta-analysis on all available publications on this topic to date (see Appendix A). We extracted all the effects from the various studies and combined them into overall estimates of genetic nurture and direct genetic effects.

Such combined estimates are more robust because they are estimated on larger and more varied samples, and they also help understand what factors may modify those estimates. For example, we investigated whether those estimates are larger as the GWAS used to generate the polygenic scores have increased, gathering millions of participants, or if maternal or paternal genetic nurture effects are similar or differ in size, among others.

Key Findings

Twelve studies met the inclusion criteria, comprising 38,654 individual offspring, each with at least one genotyped parent across eight cohorts from the United Kingdom, Australia, the United States, the Netherlands, and Iceland. Most of the studies used the trio approach (see Figure 2, page 13). In terms of the most common outcomes of interest, there was an even divide between educational attainment and educational achievement.

Genetic nurture had a small but consistent effect on children's educational outcomes ($\beta = 0.08$, 95% CI [0.07, 0.09]). In practice, this means a small effect of

genetic nurture on educational outcomes, equivalent to around two months of schooling for every standard deviation change in parental polygenic scores for educational attainment (Wang et al., 2021).

Across studies, we found robust evidence that genetic nurture plays a significant role in children's educational outcomes. The effect size was consistent across studies. After accounting for genetic nurture, we also observed substantial genetic transmission effects (direct genetic inheritance) on offspring education.

The effect of genetic transmission on children's educational outcomes was stronger than the effects of genetic nurture (β = 0.17, 95% CI [0.13, 0.20]), comparable to approximately four months of schooling gained for every standard deviation change in parental polygenic scores for educational attainment.

The effects of genetic nurture and genetic transmission were similar whether the genetic scores came from mothers, fathers, or a combination of both. This means that there was no evidence that genetic nurture effects were larger for mothers compared to fathers.

When accounting for the role of environmental factors like measured parents' education or socioeconomic position, genetic nurture effects were significantly reduced. This indicates, as expected, that these factors play a significant role in how genetic nurture affects children's educational attainment and achievement.

Implications of Findings

Overall, our findings support the hypothesis that parental phenotypes matter for their offspring's educational outcomes, even after accounting for genetic transmission. Overall, our results provide compelling evidence that education disparities reflect a combination of endogenous (such as individual genetic factors) and exogenous influences, including parental genetic nurture and broader family characteristics like socioeconomic position. These endogenous and exogenous sources of educational inequalities are largely beyond a child's responsibility or

control, and each may therefore further motivate compensatory interventions (Bann, 2021).

There is significant genetic confounding in the intergenerational transmission of education. Therefore, **social scientists and educational researchers aiming to identify environmental targets for intervention programs should account for genetic factors** prior to inferring any causal relationship between a risk or protective factor and educational outcomes. This is an especially critical point, given that aetiological and intervention research that fails to account for genetic factors may be subject to bias.

The finding that genetic nurture effects are of similar magnitude in mothers and fathers suggests that **influences from both parents on their offspring behaviour are similar in magnitude. However, parents might contribute in different ways** (for example, one parent might increase family income or read to the child). Genetic nurture may also operate through the broad family-level environments shared by both parents (such as neighbourhood).

Accounting for observed measures of parental education or family socioeconomic position decreased the effect of genetic nurture by three quarters. This suggests that **a substantial amount of genetic nurture effects may be attributed to environmental pathways related to parental education, occupation, and income**. It echoes the evidence that children's educational outcomes are influenced by the availability of resources in their family, indicated either by socioeconomic background or the education of their parents (Morris et al., 2018).

Our finding that broad family-level socio economic characteristics largely explains genetic nurture effects does not rule out proximal factors such as parenting in the chain of factors leading to educational outcomes. Future investigations should explore specific family-level pathways through which genetic nurture operates to inform compensatory interventions (for example, financial support versus schooling access).

Future research should also examine genetic nurture effects in alternative family arrangements (such as single-parent families) and in families with varying levels of parental involvement. In the presence of genuine nurturing effects, we would expect genetic nurture effects on educational outcomes to vary accordingly

(for example, be stronger for the most involved parent), which could help shed further light on environmental factors mediating genetic nurture effects.

How can we explain genetic nurture effects for educational outcomes?

Previous studies suggest that many parental characteristics are associated with child educational outcomes, including cognitive and non-cognitive skills (Dickson et al., 2016; Taylor et al., 2004) (McGue et al., 2020; Vanzella-Yang et al., 2024), socioeconomic position (SEP) (Engle & Black, 2008; Sirin, 2005), psychopathology (Ranning et al., 2018; Shen et al., 2016), personality and wellbeing (Steinmayr et al., 2010), or fertility and risk behaviours (Batstra et al., 2003; Downey, 1995; Levine et al., 2001; Sayal et al., 2014).

Parental education is likely to also influence key aspects of child development strongly associated with the child's ability to thrive in the educational system, such as experiences of mental health difficulties, and thus contribution to the intergenerational transmission of underachievement (Esch et al., 2014; Kulkarni et al., 2021).

Having established the significance of genetic nurture effects on education, we wanted to broaden the question to examine a wider range of parental traits and child outcomes and investigate how these genetic nurture effects manifest themselves across various dimensions of child development.

We therefore conducted a series of studies in two UK-based longitudinal cohorts of families with genotype and phenotype (i.e., individual's observable traits and behaviour, see Glossary) data available using polygenic score analysis and a trio approach (see Appendices B and C for detailed description of samples and methodology).

First, we investigated how parents' genetic predisposition for 25 traits, including educational attainment, influence their offspring educational achievement via genetic transmission and genetic nurturing. Predictors include polygenic scores (PGS) in the domains of education, socioeconomic position (SEP), personality, and psychiatric disorders.

Then, we investigated which routes of transmission underlie the association between educational attainment, the cognitive and non-cognitive components of educational attainment (see Glossary, page 31), and two types of mental health difficulties: *externalising* mental health difficulties (such as aggression, hyperactivity, and impulsivity) and *internalising* difficulties (such as anxiety, low mood, or social withdrawal).

The data supporting the following findings originated from mother-father-child trios recruited by the ALSPAC and MCS cohorts (see Annexes B and C). The sample consisted of 1,377 children (47.0% female) and parents from ALSPAC and 3,228 children (49.3% female) and their parents from MCS.

Key Findings

We found evidence of genetic transmission and genetic nurture effects of parental genetic predisposition for educational attainment on offspring educational achievement. These estimates were comparable to the pooled estimates identified in our meta-analysis (Wang et al., 2021).

In contrast to the findings from the meta-analysis – where genetic effects originating from mothers and fathers were of similar magnitude**, we did find significant differences between maternal and paternal effects** for educational attainment at different developmental stages of educational attainment.

Genetic transmission effects were **larger than genetic nurture effects for educational attainment** and **cognitive performance.** Interestingly, we found the **opposite trend for** the **non-cognitive component** of educational attainment.

We also found that **parents' genetic predisposition for eight out of the 24 traits other than educational attainment influence their offspring educational achievement** (measured by their performance at Key Stages 1-5 of education in the UK at approximately 7, 11, 14, 16 and 18 years old). This includes 'age at first birth', 'number of children ever born', 'subjective well-being', 'ever (being a) smoker', 'bipolar disorder', 'cognitive performance', 'non-cognitive component of educational attainment' (see Glossary), and 'household income' (see Figures 4 and 5 in pages 25 and 26).

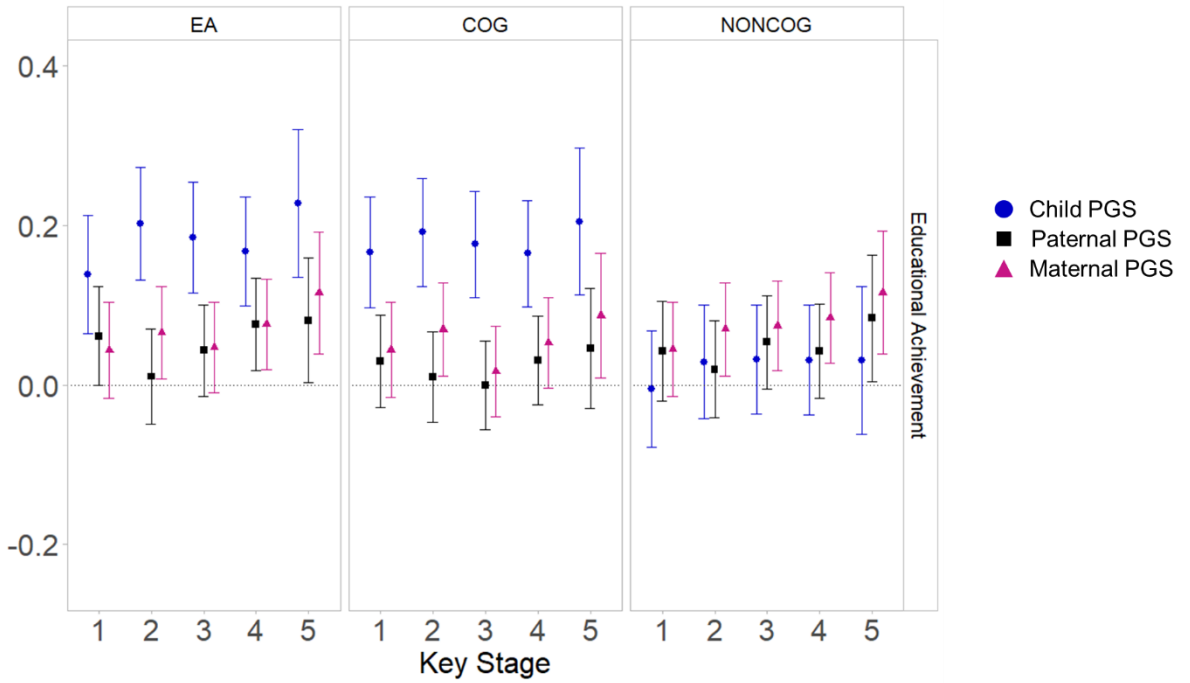

Figure 3. Beta estimates with 95% confidence intervals for polygenic scores (PGSs) associated with genetic transmission (child) and parent-specific (maternal and paternal) genetic nurture effects for educational attainment (EA), cognitive (COG), and non-cognitive components (NONCOG) on educational achievement of offspring measured at Key Stages 1-5. Note: child and parental PGSs are modelled jointly.

Overall, parental polygenic scores for personality and health behaviours were more associated with educational achievement in offspring than the child's own genetic predisposition for the same traits. This suggested **genetic nurture effects played a larger role in educational achievement than genetic transmission for personality and health behaviours**. For example, through ages 7 to 18, we found genetic nurture effects of 'age at first birth' while genetic transmission effects were only significant at one time point.

We found significant differences between maternal and paternal genetic influences for 'age at first birth,' 'number of children ever born,' and 'ever (being a) smoker,' with

maternal effects being larger overall[1]. We did not find differences between maternal and paternal influences in the domains of psychiatric diagnoses, cognition, or SEP traits. This is consistent with the findings of the meta-analysis (see '*Are there robust genetic nurture effects in educational outcomes*' section).

Finally, we found that **parental educational attainment is associated with less mental health difficulties in childhood and adolescence** and that this association operates **both via genetic transmission and genetic nurture** pathways. These influences are different for externalising (which includes aggression, hyperactivity, and impulsivity difficulties) and internalising difficulties (which includes difficulties such as anxiety, low mood, or social withdrawal). For externalising difficulties, genetic transmission effects increased from ages 3 to 14 years, while genetic nurture effects decreased. Both types of genetic influences remained constant across childhood and adolescence for internalising difficulties.

Implications of Findings

A key strength of these studies is that they integrate genetic and phenotypic data to examine genetic transmission and genetic nurture across a wide range of parental traits, extending beyond educational attainment.

Our results indicate that **several parental traits, previously associated with their offspring educational achievement, show evidence of both genetic transmission and genetic nurture effects**. These findings stress the need to combine genetic and environmental data when investigating the intergenerational transmission of educational achievement in more traits than just educational attainment.

The finding that genetic nurture effects (i.e., for personality and health behaviour traits) were larger than genetic transmission effects **highlights the importance of the rearing environment and suggests** that **specific family-level characteristics**

[1] It is important to consider the potential for *index event bias* in the cases of 'age at first birth' and 'number of children ever born', as analyses are conditioned on individuals who have had children. However, we argue that this does not undermine our conclusions, as polygenic influences on reproductive behaviour are likely to be present across the entire population.

(e.g., ‘subjective well-being’) may underpin the intergenerational cycle of underachievement. These results give us insight into what aspects of parental characteristics might be driving the genetic nurture effects of parental genetic predisposition to educational attainment.

Such findings (e.g., ‘age at first birth’, ‘number of children ever born’) **raise the question of what these genetic predispositions are acting as proxy for**, and what intermediate factors could explain this relationship, such as childhood socioeconomic circumstances or risk tolerance, in the case of reproductive traits (Mills et al., 2021). These findings help researchers design more granular questions regarding specific factors in a child’s environment that could underlie the intergenerational association of parental education and offspring educational achievement, beyond genetic confounding.

We did not find parent-specific effects for psychiatric diagnoses, cognition, or SEP traits, other than educational attainment. It is possible that both parents contribute equally to shaping the environment that influences educational achievement, albeit through different mechanisms - such as distal factors like increased family income or proximal factors like shared activities (such as reading to the child). This aligns with behavioural studies showing that parental involvement is equally influential on children’s educational outcomes regardless of whether it comes from mothers or fathers (Barger et al., 2019; Kim & Hill, 2015).

We did find significant differences between maternal and paternal genetic influences for ‘age at first birth’, ‘number of children ever born’, and ‘ever (being a) smoker’ which might indicate that **some maternal and paternal influences may operate through distinct mechanisms**, reinforcing the need for future research to delineate these pathways.

When does genetic nurture manifest in development?

Genetic nurture reflects the parental nurturing environment in the home, which is likely to be more important earlier in life than later (Briley & Tucker-Drob, 2017; Tucker-Drob et al., 2014). To investigate the developmental timing of genetic nurture and genetic transmission effects, we examined the timing of genetic transmission and genetic nurture effects using longitudinal data and estimated both types of genetic effects at five different points of children's academic journey (i.e., Key Stages 1-5 of education in the UK, at approximately 7, 11, 14, 16 and 18 years).

We did this for 25 traits including educational attainment, the two components of educational attainment (cognitive and non-cognitive), socioeconomic position (SEP), personality, and psychiatric disorders.

In addition, we also investigated if there were age-specific differences in the association between parental genetic predisposition to educational attainment, and two types of mental health difficulties in offspring: *externalising* mental health difficulties (such as aggression, hyperactivity, and impulsivity) and *internalising* difficulties (such as anxiety, low mood, or social withdrawal).

The data supporting the following findings originated from mother-father-child trios recruited by the ALSPAC and MCS cohorts (see Appendices B and C). The sample consisted of 1,377 children (47.0% female) and parents from ALSPAC and 3,228 children (49.3% female) and their parents from MCS.

Key findings

A key strength of these studies is that they **integrate genetic and phenotypic data** to examine genetic transmission and genetic nurture **across a wide range of parental traits, extending beyond educational attainment**, and are **developmentally sensitive** with measures of educational achievement throughout childhood and early adolescence.

We found that **genetic transmission and genetic nurture effects** of parental genetic predisposition to educational attainment on measures of offspring educational achievement **were of significantly different magnitude across developmental stages but followed the same trend during the offspring's educational journey** (see Figure 3). Both genetic transmission and genetic nurture pathways played a bigger role in offspring educational achievement later in life (Key Stages 4 and 5, henceforth referred to as KS).

Maternal genetic nurture effects were larger than paternal effects, and they either both followed the same trajectory (they had an increased effect by adolescence), or maternal effects became more important with age while paternal effects remained stable.

For example, by age 18, a one standard deviation higher child educational attainment PGS was associated with a 0.23 standard deviation higher KS5 standardised scores, indexing the strength of direct genetic transmission (β=0.23, 95% CI [0.14, 0.32]). In comparison, a one standard deviation higher *mothers'* educational attainment PGS was associated with a 0.12 higher KS5 score (indexing genetic nurture), while one standard deviation higher *fathers*' educational attainment PGS was associated with a 0.08 higher KS5 score (β=0.12, 95% CI [0.04, 0.19] and β=0.08, 95% CI [0.00, 0.16], respectively).

When investigating developmental effects in other traits, **both genetic transmission and genetic nurture effects varied across time for 'age at first birth', 'number of children ever born', 'subjective well-being', 'ever (being a) smoker', 'bipolar disorder', 'cognitive performance', 'non-cognitive factor, and 'household income'** (i.e., at approximately 7, 11, 14, 16 and 18 years old).

For most traits, both genetic transmission and genetic nurture estimates appear to increase with age. For example, standardised beta estimates of maternal genetic nurture and genetic transmission effects for 'age at first birth' at Key Stage 1 were β=0.09 [0.02, 0.15] and β = −0.03 [−0.09, 0.06], while at Key Stage 5 they were β= 0.13 [0.05, 0.21] and β=0.07 [−0.02, 0.16], respectively (see Figures 4 and 5).

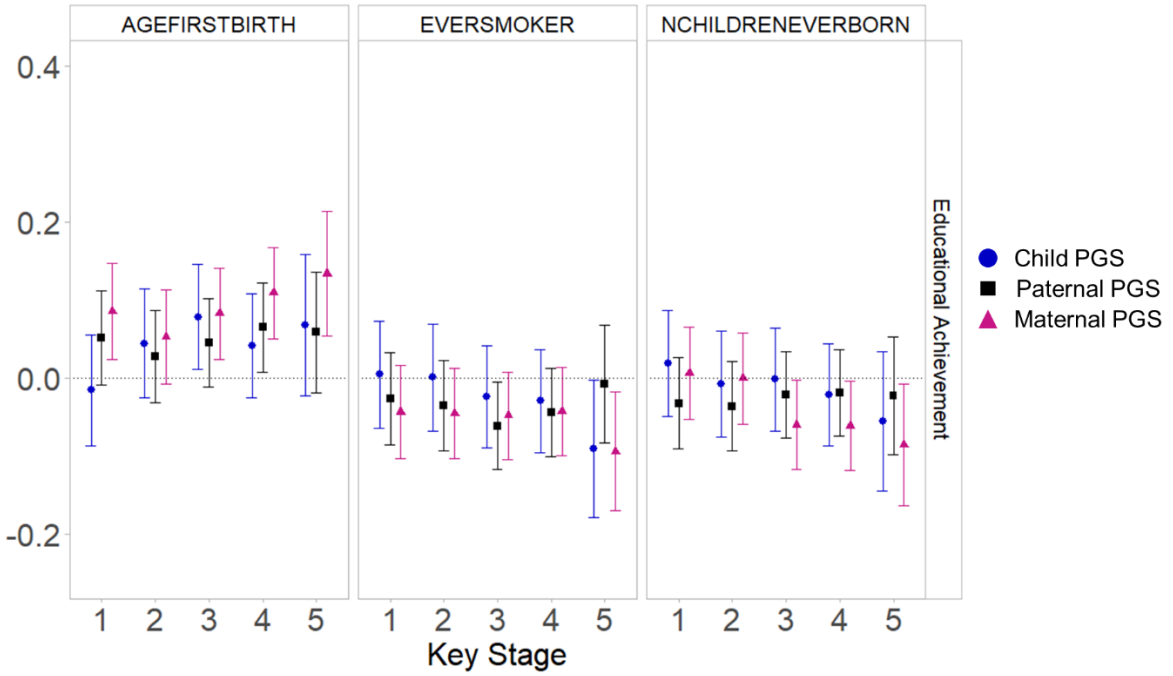

Figure 4. Beta estimates with 95% confidence intervals for polygenic scores (PGSs) associated with genetic transmission (i.e., child polygenic scores, PGS) and genetic nurture effects (i.e., parent-specific PGS) for age at first birth, ever smoker, and number of children ever born (NCHILDREN) on educational achievement from best fitting model. Note: Child and parental PGSs are modelled jointly.

Genetic transmission estimates for the **non-cognitive factor** remained non-significant across all ages, but **genetic nurture effects became more important with age**. Genetic nurture effects for the non-cognitive factor were larger than for cognitive performance, especially at earlier ages (see Figure 3, page 20).

Finally, we found that the association between parental educational attainment and externalising difficulties in childhood and adolescence changed over time. At age 3, genetic transmission and genetic nurture effects were of similar magnitude. However, by age 14, genetic nurture effects had faded while genetic transmission effects had nearly doubled. We found no age differences for genetic transmission and genetic nurture effects on internalising difficulties and interestingly, genetic nurture effects were larger than genetic transmission effects. The association between educational attainment and mental health difficulties was different for cognitive and non-cognitive genetic components of the parental genetic predisposition to educational attainment.

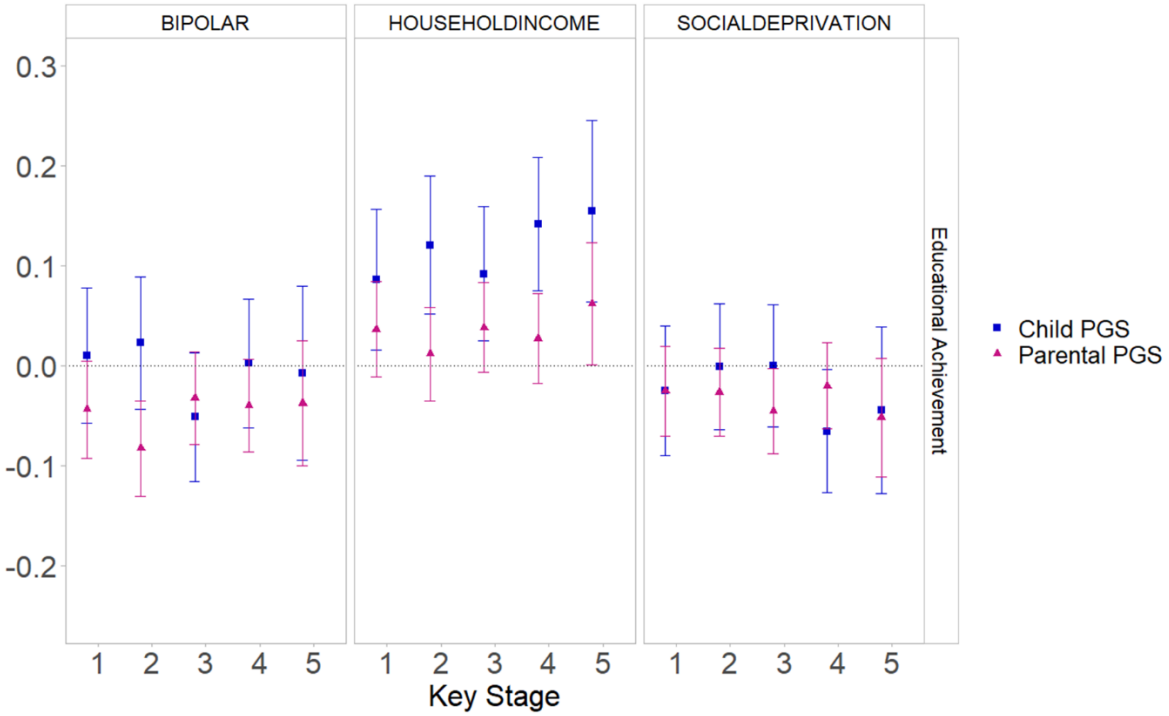

Figure 5. Beta estimates with 95% confidence intervals for polygenic scores (PGSs) associated with genetic transmission (i.e., child polygenic scores, PGS) and genetic nurture effects (i.e., parental PGS) for bipolar disorder, household income, and social deprivation, on educational achievement from best fitting model. Note: Child and parental PGSs are modelled jointly.

Implications of Findings

Our findings suggest substantial differences in the role of genetic transmission and genetic nurture in the intergenerational transmission of educational achievement **across childhood and adolescence**, as well as some environmentally mediated genetic nurture effects. The increasing influence of both effects on educational achievement during later stages (KS4 and KS5) **highlights the necessity of modelling age-specific differences** when considering intergenerational transmission of educational achievement.

In addition, **these findings suggest the existence of specific factors in the rearing environment of children contributing to their educational achievement**. These factors operate **both via the cognitive and non-cognitive components** of educational attainment. In addition, intervention programmes targeting parents might

prove more effective when focusing on non-cognitive aspects of education and parent-child interaction.

For example, early parental interventions focused on non-cognitive traits may be particularly useful, while child-focused interventions addressing both cognitive and non-cognitive traits may be more beneficial during adolescence.

The strength of this approach lies in our ability to disentangle the complex interplay between inherited traits and environmental influences on educational outcomes, offering a more nuanced understanding of development. Using longitudinal data and within-family studies allows us to control for shared family environments. In this way, we can obtain less biased estimates of genetic transmission and genetic nurture effects of parental education-related traits on offspring educational achievement over time.

Furthermore, our findings regarding intergenerational influences of parental education on offspring's mental health difficulties suggest that **the effectiveness of interventions might depend on the type of mental health difficulty and the developmental stage**. For externalising difficulties in early childhood (rule-breaking, hyperactivity, aggression), programs promoting non-cognitive skills in parents (e.g., socio-emotional skills) might prove fruitful, while later in life they might focus on the youth's and their environment outside of home. For internalising difficulties (emotional and peer problems), a focus on both the family environment, targeting non-cognitive skills, and the child, targeting cognitive behavioural skills, may provide significant opportunities for additional research and, potentially, intervention.

Conclusions, Limitations and Future Directions

This project underscores the intricate interplay between genetic and environmental factors in shaping educational outcomes across generations. Our findings confirm that both **genetic transmission** (direct inheritance of genes) and **genetic nurture** (environment shaped by parental genetics) influence children's educational attainment and achievement. While **genetic inheritance** does play a role in educational outcomes, it does not imply that carrying specific genetic variants causes the behaviour; rather, it **reflects a complex chain of events and interactions, including how genes interplay with environmental factors and broader developmental processes**.

We show that the rearing environment – influenced by parental genetic predisposition to educational attainment, personality and health behaviour traits, or non-cognitive aspects of educational attainment – **plays a significant role in educational achievement and mental health of children and young adolescents**. These results reinforce the importance of integrating genetic and social science perspectives to better understand intergenerational educational transmission.

A substantial amount of genetic nurture effects may be attributed to environmental pathways related to family socioeconomic position. This supports the claim that children's educational outcomes are influenced by the availability of resources in their family, indicated either by socioeconomic background or the education of their parents. Future investigations should explore specific family-level pathways through which genetic nurture operates to inform compensatory interventions (for example, financial support versus schooling access).

Our findings suggest that, overall, **for parental traits other than educational attainment, genetic nurture effects play a bigger role during early childhood**, while **genetic transmission effects become more pronounced in adolescence**, emphasising the developmental sensitivity of these influences. This trajectory suggests research focusing on environmental factors in early childhood might prove especially fruitful when it comes to understanding the aetiology of learning and

emotional difficulties and the role of parenting, family environment and socioeconomic in educational outcomes.

Our results suggest that **no single intervention is likely to be effective for all family members independent from the developmental stage.** Interventions focusing on the parents might be more effective earlier, while child-focused interventions might be more useful in adolescence.

For most traits under study, we found that genetic nurture effects are of similar magnitude in mothers and fathers. Incorporating data from mothers and fathers can therefore help us increase the robustness of our findings and for certain traits might support the use of imputation techniques for cases when data from both biological parents is not available, therefore increasing sample size and improving the precision of our estimates.

Additionally, **non-cognitive factors**, such as motivation and self-regulation, **emerge as good candidates for researchers interested in individual-level factors likely to influence the rearing environment of children, their educational outcomes and mental health**. Research targeting non-cognitive factors in early childhood might identify interventions that improve educational outcomes and bolster mental health, preparing children to overcome future challenges. In addition, our **findings regarding the influence of parental traits beyond educational attainment – such as lifestyle choices** (for example smoking, age at first birth) **and psychological factors** (such as bipolar disorder) – further underscore the value of research partnerships between educational and health researchers to identify risk and protective factors that could improve both educational outcomes and mental health in youth.

It is necessary to stress **that polygenic scores capture genetic predispositions rather than actual behaviour and should not be considered a replacement for observed survey data**. An advantage of polygenic scores however is that they provide a standardised metric for parental traits that removes some of the biases associated with self-reports and heterogeneity of measurements so common in developmental studies.

Finally, **there are several caveats to the findings presented in this report**. First, technical biases may give rise to genetic nurture in our models even in the absence

of true genetic nurture (e.g. assortative mating or population stratification – see Glossary, page 31). Second, our samples primarily comprise participants of European ancestry, which introduces a Eurocentric bias and limits generalisability to non-European populations. Third, missing data are a common challenge in longitudinal studies, although we used estimation methods specifically designed to address this issue. Fourth, within-family trio analyses of single cohorts may lack sufficient power, highlighting the need for replication in larger, more diverse cohorts. Fifth, different definitions of educational achievement at different developmental stages might be driving some of the differences observed across developmental points.

Looking ahead, we argue that **the evidence supporting the presence of genetic nurture in educational outcomes supports further research into the mechanisms of genetic nurture.** It would be particularly interesting to study how specific family-level pathways – such as parental mental health, income, or parenting styles – mediate its effects.

In conclusion, this project underscores the necessity of a comprehensive approach to breaking cycles of educational disadvantage, one that integrates genetic and environmental perspectives. By addressing the complex interplay of genetic influences and early childhood environments, future research and, in due course, intervention programmes, can be better positioned to provide equitable educational opportunities for all children, fostering resilience, reducing inequalities, and promoting social mobility across generations.

Glossary

Note: These definitions were elaborated by the research team. For additional terms, we refer the reader to the Talking Glossary of Genomic and Genetic Terms developed by the National Human Genome Research Institute (https://www.genome.gov/genetics-glossary).

Term	Definition
Assortative mating	The tendency of individuals to pair with others who share similar traits, such as height, education, or social background, influencing population genetics and increasing family-level similarity in those traits.
Cognitive component	The cognitive component of educational attainment refers to the subset of genetic variants for educational attainment that operates through or strongly overlaps with measured cognition.
Confidence interval (CI)	A confidence interval is a range of values around an estimate (e.g. an estimate of genetic nurture). It provides a sense of how precise that estimate is; the narrower the interval, the more confident we are in the estimate.
Educational achievement	A person's performance at school – the grade they achieve (A, B, C, etc.)
Educational attainment	The highest education level a person reaches (GCSE, A level or master's degree for example)

Genetic nurture	The process by which parents' genes indirectly influence outcomes for their offspring through their rearing environment beyond direct genetic transmission. In the genetics literature, several other terms have been used to describe this including 'dynastic effects,' 'genetic nurture,' 'familial genetic effects,' and 'indirect genetic effects.'
Genome-wide association study (GWAS)	Genetic studies that examine millions of genetic variants to identify those that are associated with a trait of interest (e.g. educational attainment).
Heritability	Metric that represents how much of the individual differences we see in a trait (like height or educational attainment) can be explained by genetic differences for a given sample at a given time. Heritability does not apply to one individual, but to trait variation in a group of individuals. When estimating heritability in a group of people, it does not necessarily translate to other groups (e.g. groups living in other environments).
Meta-analysis	A statistical method for combining estimates (e.g. genetic nurture estimates) from multiple studies.
Non-cognitive component	The non-cognitive component of educational attainment refers to another portion of the genetic signal underlying educational attainment that is not captured by cognition in GWAS studies. It might tap on traits such as diligence, grit, or self-control. These traits also promote educational success but are not fully accounted for by intelligence measures.

Phenotype	The observable traits or characteristics of an organism e.g., educational attainment.
Polygenic scores	An individual score summarising the effects of thousands of DNA variants that contribute to individual differences on a trait. Also referred to in the genetics literature as “polygenic risk score,” “polygenic index.”
Population stratification	Bias in genetic analyses due to systematic differences in allele frequencies among subpopulations e.g., ancestry or other factors. If not accounted for, it can confound genetic association studies, creating misleading links between genetic variants and traits.
Statistical control	Method to account for “confounding” variables, i.e. variables that can create apparent associations between a risk factor and an outcome when there is no true causal effect. Statistical techniques such as regression analysis aim to account for those confounding variables to minimise confounding and get closer to the true effect.
Systematic review	A review of the scientific literature using a structured and transparent method to collect, assess, and synthesise the evidence.
Trio design or within-family trio design	Genetic epidemiology method which involves using the child’s and both of their biological parents’ polygenic scores to predict a specific phenotype of interest. Combining those three polygenic scores in a single model allow us to statistically control the effect of each polygenic score for the two others. We can then isolate (i)

	direct genetic transmission, which is indexed by the controlled effect of the child polygenic score in that joint model; (ii) and genetic nurture effect which is indexed by the effect of the parental polygenic scores in that joint model, which controls for genetic transmission via the child polygenic score. Also referred to as the ‘statistical control approach.’
Virtual parent	A numerical score based on the set of alleles that were not passed on to the offspring from a specific parent, therefore capturing genetic nurture effects.

Appendix A: Meta-analytical approach

This systematic review and meta-analysis was performed in line with well-known quality guidelines as reported in detail in the published paper (Wang et al., 2021). We conducted a literature search in July 2020 using following several literature databases.

To estimate genetic nurture effects on educational outcomes, we included articles estimating genetic nurture in parent(s)-offspring samples using polygenic scores (PGS) for educational attainment (EA) since 2013, when the first EA genome-wide association study (GWAS) became available. Two authors independently screened titles and abstracts of all articles retrieved during the search before reviewing the full text of potentially eligible studies.

To be included in the meta-analysis, studies had to meet the following criteria:

- They assessed offspring educational attainment (for example, years of education or highest degree obtained) or educational achievement (national test scores or levels, school grades) in the general population.
- Genetic factors were measured using PGSs for educational attainment.
- They derived estimates for genetic nurture effects on education from, either studies using either the virtual parent (testing whether the PGSs calculated from parents' non-transmitted alleles predict offspring educational outcomes) or trio design (calculating genome-wide polygenic scores and correcting for the effect of child's genotype by analysing parental and children polygenic scores in the same statistical model).

Quality assessment, data extraction, and effect size calculation

Two of the authors independently assessed the methodological quality of each included study using an adapted version of the Newcastle-Ottawa scale (NOS21). Authors gave each study a score that reflected overall study quality, ranging from 0 to 9 (see supplemental note 2.3 of the main paper for detailed scoring criteria and Table S3 for scores of included studies). Two of the authors independently extracted data for each study, including:

- Publication characteristics (study name, first author, year).
- Sample characteristics (cohort name, sample size, population source, ethnicity, sex distribution).
- Study design (virtual parent or trio design).
- Calculation of PGSs (the GWAS used to derive the PGS, PGS threshold, source/parent of origin of genotype, whether standardised).
- Education-related outcomes assessed (educational outcome, outcome type, age at assessment, whether standardised).

- Effect size (estimation type, estimation, 95% CI or standard error of the estimation)
- Confounding variables adjusted for.

Where information was missing, original study authors were contacted to request the information.

The two authors who extracted and coded the data had a high level of agreement (92.6% on quality assessment and 97.8% on data extraction).

Statistical analysis

Analyses were conducted in a statistical software called R with Metafor, a dedicated package for meta-analysis. To account for the fact that some studies reported multiple results originating from the same cohorts, thereby creating dependencies between estimates, we implemented adequate statistical models (Multilevel Random-Effects).

Next, we went a step further and used a specific technique, meta-regression, to evaluate if certain characteristics (called moderators) of each study, like study design or how educational attainment was defined, might affect the overall results.

We tested four main categorical moderators:

1) Whether the parental PGS was constructed based on maternal, paternal, or the mixture of both parents' genotypes,
2) The type of analytic method used to estimate the genetic nurture effects (virtual parent, partial or full statistical control),
3) The type of educational outcome assessed (educational attainment or educational achievement), and
4) The specific GWAS summary statistics used to calculate the PGSs.

We also evaluated how study characteristics might have influenced the effect sizes reported in the literature (i.e., methodological quality, sample size, and attrition in cohorts). To investigate the possible environmental pathways through which genetic nurture works, we examined how much the effects of genetic nurture were reduced when adjusting for parents' educational levels and family socioeconomic position (SEP).

Appendix B: Analysis of the ALSPAC cohort

This study used data from the Avon Longitudinal Study of Parents and Children (ALSPAC), an ongoing population-based birth cohort study which follows pregnant women recruited in Avon (southwest England), their partners and their offspring, between 1991-1992 (Boyd et al., 2013; Fraser et al., 2013; Northstone et al., 2019).

We are extremely grateful to all the families who took part in the ALSPAC study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists and nurses.

Genome wide genotyping data was generated by Sample Logistics and Genotyping Facilities at Wellcome Sanger Institute and LabCorp (Laboratory Corporation of America) using support from 23andMe.

The UK Medical Research Council and Wellcome (Grant ref: 217065/Z/19/Z) and the University of Bristol provide core support for ALSPAC. This publication is the work of the authors and will serve as guarantors for the contents of this paper

Pregnant women resident in Avon, UK with expected dates of delivery between 1st April 1991 and 31st December 1992 were invited to take part in the study. Out of initial 14,541 pregnancies, 13,988 children were alive at 12 months and 85% of the eligible expectant mothers participated. Offspring and their primary carers were genotyped when children were 5 years old. At 7 years, eligible cases who had failed to join the study at the beginning were recontacted, which increased the sample size by 913 more children. The total sample size for analyses using any data collected after the age of seven is therefore 15,447 pregnancies, resulting in 15,658 foetuses. Of these 14,901 children were alive at 1 year of age.

Additional information:

- 14,203 unique mothers were initially enrolled in the study.
- 14,833 unique women (G0 mothers) enrolled in ALSPAC as of September 2021.
- 12,113 G0 partners have been in contact with the study by providing data and/or formally enrolling when this started in 2010.
- 3,807 G0 partners are currently enrolled.

Ethical approval

Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. The approval code for this project is B3496.

Informed consent for the use of data collected via questionnaires was obtained from participants following the recommendations of the ALSPAC Ethics and Law

Committee at the time, together with consent for biological samples in accordance with the Human Tissue Act (2004).

At age 18, study children were sent 'fair processing' materials describing ALSPAC's intended use of their health and administrative records and were given clear means to consent or object via a written form. Data were not extracted for participants who objected, or who were not sent fair processing materials.

Phenotype Definitions

The main outcomes of the study were offspring educational achievement throughout childhood and adolescence measured by their performance at five major Key Stages (1-5) of education in the UK at approximately 7, 11, 14, 16 and 18 years. Fine graded point scores of Key Stages were obtained from the UK National Pupil Database (NPD) through data linkage to the ALSPAC cohort.

These official records represent the most accurate record of individual educational achievement available in the United Kingdom during compulsory schooling.

To maximise consistency and sample size, we selected the following phenotypes as outcome:

- For Key Stage 1, we used the prorated summary scores in reading, writing and mathematics.
- For Key Stage 2, we calculated the sum score of the total marks achieved by the student in English, Maths and Science.
- For Key Stage 3, we used the total point score.
- For Key Stage 4, we used the total point score from General Certificate of Secondary Education (GCSE)/General National Vocational Qualification (GNVQ).
- For Key Stage 5 we used the points score contribution made by A Levels.

Scores were standardised to a mean of zero and a variance of one within each Key Stage. Please note that 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/.

Genotyping, Imputation, and Polygenic Scoring

Genotype data was uploaded to the Michigan Imputation Server and imputed using 1000G Phase 1 version 3 as the reference panel. Imputed genotypes were then filtered with PLINK 1.9 (Chang et al., 2015): duplicated SNPs, SNPs with missingness > 2% or minor allele frequency < 1%, or located in non-autosomal regions were removed.

Individuals with missingness > 2%, HWE $p < 1\times10^{-6}$, heterozygosity > mean ± 3SD were also removed. We identified related individuals up to third-degree identified using KING 2.2.7 (Manichaikul et al., 2010) and randomly excluded one individual from each pair of relatives (e.g. siblings in the parental or offspring generation). The

sample was further filtered to only include families with genetic data available for complete trios (i.e., mother, father and child), resulting in a final sample of 3,228 trios. Self-reported ethnicity at age 14 indicated that the majority of the sample identified as 'white' (87.5%). No trios were removed due to ancestry. All analyses were controlled for population stratification by including the first ten principal components of ancestry. The sample was filtered to include families with genetic data available for complete trios (i.e., mother, father, and child), resulting in a sample of 1,377 trios with genotype data available. No trios were removed due to ancestry.

For the ALSPAC cohort, we calculated 25 polygenic scores (PGS) for traits in the domains of education, cognition, and socioeconomic position (Davies et al., 2018; Demange et al., 2021; Hill et al., 2019; J. J. Lee et al., 2018; Trampush et al., 2017); personality, reproductive and risk behaviours traits (Barban et al., 2016; Karlsson Linnér et al., 2019; Nagel et al., 2018; Turley et al., 2018); and psychiatric disorders (Demontis et al., 2019) (Grove et al., 2019; Howard et al., 2019; Pardinas et al., 2018; Purves et al., 2019; Stahl et al., 2019). See Supplementary Note for complete description of the summary statistics used to calculate each PGS. Polygenic scores were computed using *PRSice-2* for each trait (Choi & O'Reilly, 2019). SNPs were clumped to obtain variants in linkage equilibrium with an $r^2 > 0.1$ within a 250 KB window and PRS were constructed across a range of p_t (p_t = 5e-8, 1e-6, 1e-5, 1e-4, 1e-3, 0.01, 0.05, 0.1, 0.2, 0.3, 0.4, and 0.5). We then performed a principal component analysis (PCA) on the resulting polygenic scores and used the first principal component in subsequent association tests. The PCA approach outperforms arbitrarily chosen p-value thresholds (Coombes et al., 2020). Polygenic scores were adjusted for population stratification by including the first ten principal components of ancestry and sex as fixed effects in the model.

Statistical Analysis

First, we assessed if the genetic factors for each parental trait (either via direct transmission or genetic nurture) played a role at all in children's educational achievement across Key Stages 1-5. Next, we tested if maternal and paternal could be assumed to be equal in size, and if genetic effects varied across childhood and adolescence. The genetic effect of each parental trait was tested independently. We did this by defining different structural equation models and perform a stepwise model comparison. To make sure our findings were reliable, we used a statistical correction method due to multiple comparisons and chose the best fitting, most parsimonious model.

Appendix C: Analysis of the MCS cohort

This study used data from the Millennium Cohort Study (MCS), a longitudinal study that follows a nationally representative cohort of children born in the UK between September 2000 and January 2002.

The Economic and Social Research Council funds the Centre for Longitudinal Studies (CLS) Resource Centre (ES/W013142/1) which provides core support for the CLS cohort studies. While the CLS Resource Centre makes these data available, CLS does not bear any responsibility for the analysis or interpretation of these data by researchers.

The CLS cohorts are only possible due to the commitment and enthusiasm of their participants, their time and contribution is gratefully acknowledged.

The first data collection took place when cohort members were approximately 9 months old. Cohort members and their primary carers were genotyped at age 14. In our study, we used questionnaire data collected when participants were 3, 5, 7 and 14 years old, and genotype data from mother-father-child trios.

To be entered into our analysis, participants must have phenotype data available for at least one sweep as well as genotype data. The numbers of trios at each wave with complete data were 2,907, 3,055, 3,051, and 3,203, respectively.

The data collection for the MCS was approved by the UK National Health Service Research Ethics Committee. Written consent was obtained from all parents in the MCS at each survey. For more details, see Morosoli et al. (2024).

Phenotype Definitions

The two phenotypes and main outcomes of the study were externalising and internalising problems in childhood and adolescence as measured by the Strengths and Difficulties Questionnaire (SDQ; Goodman, 1997). The SDQ comprises 25 items grouped into four “difficulties” domains (hyperactivity/inattention, emotional, conduct, and peer problems) and one “strength” domain (prosocial behaviour), with scores having the same range across all age groups. The conduct and hyperactivity scales, and the emotional and peer problems scales can be combined to obtain a single externalising and internalising score, respectively (Goodman et al., 2010). These two sum scores were used as indicators of externalising and internalising difficulties. The SDQ was answered by parents at approximate ages 3, 5, 7, and 14 years.

Genotyping, Imputation, and Polygenic Scoring

Genotyping was performed using the Infinium Global Screening Array-24 v1.0. For more details on the collection of samples, DNA extraction methods and laboratory procedures see Shireby et al. (2024). Prior to imputation, single-nucleotide polymorphisms (SNPs) with high levels of missing data (>5%), Hardy-Weinberg equilibrium (HWE) $p < 1e\text{-}6$ or minor allele frequency <1% were excluded. Genotype

data was uploaded to the Michigan Imputation Server and imputed using 1000G Phase 1 version 3 as the reference panel. Imputed genotypes were then filtered with PLINK 1.9 (Chang et al., 2015): duplicated SNPs, SNPs with missingness > 2% or minor allele frequency < 1%, or located in non-autosomal regions were removed.

Individuals with missingness > 2%, HWE $p < 1\times10^{-6}$, heterozygosity > mean ± 3SD were also removed. We identified related individuals up to third-degree identified using KING 2.2.7 (Manichaikul et al., 2010) and randomly excluded one individual from each pair of relatives (e.g. siblings in the parental or offspring generation). The sample was further filtered to only include families with genetic data available for complete trios (i.e., mother, father and child), resulting in a final sample of 3,228 trios. Self-reported ethnicity at age 14 indicated that the majority of the sample identified as 'white' (87.5%). No trios were removed due to ancestry. All analyses were controlled for population stratification by including the first ten principal components of ancestry.

To generate two quasi-independent components of educational attainment, we calculated polygenic scores for *educational attainment* and *cognitive performance* (henceforth *Cog*) using GWAS summary statistics from James J. Lee et al. (2018), and the *non-cognitive component* of educational attainment (henceforth *NonCog*) from Demange et al. (2021), mirroring the procedure followed by Demange and collaborators, which supports the logic of the present study of investigating subcomponents of educational attainment. That is, summary statistics for NonCog were generated by conducting a GWAS by subtraction removing the effect of cognitive performance and educational attainment, interpreting the residuals as noncognitive skills (Demange et al., 2021).

Polygenic scores were calculated using LDPred2 (Privé et al., 2020), a Bayesian method to derive polygenic scores using information on the genetic architecture of a trait, and on Linkage Disequilibrium (LD) obtained from a reference panel. We followed recommended quality control guidelines for LDPred2 and variants were restricted to those included in an extended HapMap3 set (Privé et al., 2023). The reference LD panel for the calculation of polygenic scores was UK Biobank and we used the precomputed LD matrices provided by Privé et al. (2023). Polygenic scores were generated by using the option 'LDpred2-auto' and standardised.

Statistical Analysis

As a preliminary step, we tested for differences across sex and ages at data collection for each outcome using independent samples t-tests and within-subjects one-way ANOVA as implemented in the *lme4* package (Bates et al., 2015) in R v4.4.1 (R Development Core Team, 2024). For the polygenic score analyses, externalising and internalising sum scores were standardised within age groups. We modelled the effect of parental and child polygenic score effects on offspring's outcomes using structural equation models implemented in the *lavaan* R-package (Rosseel, 2012). We modelled the effect of each family member's polygenic score on

the phenotype jointly, thus decomposing associations between parental polygenic scores on offspring phenotypes into genetic transmission or genetic nurture effects (see Figure 2, page 13).

Polygenic scores were adjusted for population stratification by including the first ten principal components of ancestry, and for sex including them as fixed effects. Missing data were handled using Full Information Maximum Likelihood estimation and additional power calculations were conducted for our polygenic score analyses.

We followed a three-step process to address our research questions. First, we estimated two models where the genetic transmission and genetic nurture effects of (a) *educational attainment*, or (b) *Cog* and *NonCog* on externalising and internalising sum scores – modelled together to account for their correlated developmental trajectories – were allowed to differ for mother and fathers and time points (*base model*). Note that Cog and NonCog polygenic scores were modelled together to account for the residual correlation between the two polygenic scores.

We then tested if both genetic transmission and genetic nurture estimates effects could be set to zero (i.e., *null model*) without a significant worsening of model fit, providing an omnibus test for genetic effects. Second, base models were compared against two restricted models, where either (a) all coefficients were allowed to vary across time but maternal and paternal coefficients were constrained to be equal (Model 1 or *time-varying model*); and (b) coefficients for maternal and paternal genetic nurture effects were allowed to differ but all coefficients were constrained to be equal across time points (Model 2 or *parent-specific model*).

Model comparison was conducted separately for each outcome using likelihood-ratio tests. We corrected for false discovery rate due to testing multiple hypothesis (i.e., model comparison) using the Benjamini-Hochberg procedure as implemented in the *stats* package (R Development Core Team, 2024) with a significance threshold q = 0.05. Multiple testing correction was applied within each set of comparisons: 5 tests for educational attainment, and 9 tests when Cog and NonCog were combined. In the absence of statistically significant differences in goodness of fit, the most parsimonious model was preferred.

Finally, we calculated standardised beta coefficients and 95% confidence intervals for genetic transmission and genetic nurture effects for educational attainment, Cog and NonCog on externalising and internalising sum scores for the best fitting model.

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