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Re-examining the relationship between education and adult mental health in the UK: A research note

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Abstract

Previous studies using variation in education arising from compulsory schooling laws have found no causal effects of education on mental health in the UK. We re-examine the relationship between education and mental health in the UK by taking a different approach: sibling fixed-effects with controls for polygenic scores (summary measures of genetic predisposition) for educational attainment and adult depressive symptoms. We find that higher educational attainment is associated with better adult mental health, that sibling controls reduce these associations by ~40–70% but important associations remain and find evidence for non-monotonic effects. We also find suggestive evidence that education partially "rescues" genetic predictors of poor mental health.

Keywords

Mental health; Education; Siblings fixed-effects; UK

1. Introduction

Mental health issues are one of the main causes of the overall disease burden, accounting for 21% of years lived with disability worldwide (Vos et al. 2015). About 3–4% of people worldwide suffer from anxiety and depression at any given time (James et al. 2018), and lost productivity due to anxiety and depression costs the global economy $1 trillion a year (The Lancet Global Health 2020). There are robust associations between higher educational attainment and better mental health (Lorant et al. 2003), and education is viewed as a fundamental cause of health disparities (Link and Phelan 1995). Policies to increase educational attainment may therefore equalize opportunities and reduce health inequalities. However, credibly identifying the true causal relationship is difficult because of other "third"

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Supplementary materials

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factors correlated with education and mental health (e.g., health endowments) and reverse causality (Fletcher 2008; Mikkonen et al. 2020).

Evidence on the causal relationship between education and mental health in the UK (and in other countries) is limited.[1] Three studies have used the 1972 Raising of the School Leaving Age (RoSLA) from 15 to 16 to isolate exogenous variation in education. Davies et al. (2018) estimated effects of education on several health measures including self-reported depression in the UK Biobank (UKB). Janke et al. (2020) combined UK Quarterly Labour Force Surveys and looked at self-report chronic conditions, which included indicators for (1) depression, bad nerves or anxiety and (2) mental illness, phobias, panics, other nervous disorders. Both studies found no evidence of a causal relationship. Avendano et al. (2020) estimated education-mental health gradients in the Annual Population Survey (APS), Understanding Society (UKHLS) and the UKB. They found, counterintuitively, that an extra year of education increased the probability of having depression/anxiety by 30% in the APS. Results in the UKHLS and UKB were imprecise, and signs were not consistent—education worsened mental health based on the general health questionnaire and SF-12 mental health component in UKHLS but improved mental health based on the patient health questionnaire (PHQ) and general anxiety disorder (GAD) scales in the UKB. They argue the negative effects of education arose because the RoSLA forced young people who did not want to stay in school, but rather go to the labor market, to continue their education. These young people may have been negatively affected by being forced to stay in a stressful academic environment in which they were less likely to succeed compared to their peers.[2]

We estimate education-mental health gradients in the UKB using sibling fixed-effects models with controls for polygenic scores (PGS; summary measures of genetic predisposition) for educational attainment and adult depressive symptoms. By taking a different approach, we offer alternative evidence from studies using the 1972 RoSLA for identification. We also depart from Avendano et al. (2020) by using a recently validated scale for recent depressive symptoms (RDS-4) which was administered to almost all UKB participants (whereas the PHQ and GAD scales are only available for about 30% of participants). We have a large sample of 13,577 sibling pairs, and find important protective associations of education—high school graduates have a RDS-4 score that is 13% lower compared to high school dropouts. Sibling fixed-effects do not control for sibling-specific differences that affect education and mental health, and so our estimates are not fully causal. Nevertheless, associations that remain after controlling for shared genetics, early-life factors, and individual genetic controls may increase the confidence of identifying causal relations. In supplementary materials we show that estimates of education on the PHQ and GAD

[1]Using compulsory schooling laws for identification Dahmann & Schnitzlein (2019) found no effect of education on mental health in West Germany, whereas Crespo et al. (2014) and Mazzona (2014) both found protective effects in Europe. Wang (2021) and Jiang et al. (2020) have found that education reduces depressive symptoms in China using the 1986 compulsory schooling law.

[2]Avendano et al. (2020) provide descriptive evidence that individuals who wanted to leave school at 15 (but could not due to the RoSLA) have worse mental health in adulthood than those who willingly stayed until 16. While they argue that the RoSLA may violate the exclusion restriction by affecting mental health through a “coercion effect”, there are alternative interpretations of the findings. First, individuals who report that they would have left school may not be the same individuals as the compliers with the reform. Second, the RoSLA could be a valid instrument if there are heterogenous effects of an additional year of education (protective effects for some individuals and small protective or null effects for other individuals).

scales are too imprecise to make meaningful conclusions, consistent with Avendano et al. (2020). The imprecision is due to having a smaller sample of 2200 sibling pairs.

Finally, in addition to extending the evidence for education effects on adult mental health, we investigate whether education reduces the role played by genetic factors in determining mental health. The social-trigger model suggests that healthy environments protect against genetic risk of poor health (Ellis et al. 2011). Higher educational attainment could attenuate the impact of genetic propensities toward mental illnesses because of the social and behavioral mechanisms through which education affects mental health. Consider a high school and college graduate who both have the same genetic risk of depression. The college graduate may never be depressed despite having a high genetic risk because college graduation is associated with a higher income, leading a healthier lifestyle, and interacting more with other healthy peers. We find some suggestive evidence that relative to high school dropouts the depressive symptoms PGS exerts a smaller influence on RDS-4 for people with more education, suggesting that education may reduce the role played by genetic factors in mental health disparities (Boardman et al., 2014).

2. Data

The UKB is a population-based prospective study of 502,499 individuals aged 40–69 years in 2006–2010 from across the UK. The UKB is not representative of the wider UK population, with participants being healthier and more educated (Fry et al. 2017). At baseline, participants were asked over the past two weeks, how often have you (1) felt down, depressed or hopeless, (2) had little interest or pleasure in doing things, (3) felt tense, fidgety or restless, and (4) felt tired or had little energy. Possible responses are “not at all”, “several days”, “more than half days” and “nearly every day”. We sum up the responses to these questions assigning 0 to “not at all” to 3 for “nearly every day” to create the RDS-4 scale, as proposed and validated by Dutt et al. (2021). The RDS-4 questions correspond with several diagnostic criteria for major depressive disorder in the Diagnostic and Statistical Manual of Mental Disorders manual. The advantage of the RDS-4 is that it is available for almost everyone, whereas the GAD and PHQ are available for a substantially smaller subset, who are also more educated and healthier than the whole UKB cohort (Davies et al. 2020).[3] Education is measured by mapping educational qualifications to an International Standard Classification of Education category and imputing years of education. The qualifications (years of education) are: no qualification (7); CES/O levels/GCSEs (10); A/AS levels (13); other professional qualification (15); NVQ/HNC (19); college degree (20).

Siblings are not identified in the survey, but relatedness among individuals can be inferred from the kinship coefficient—the probability that a random allele from an individual is identical by descent with an allele at the same locus from the other individual. We identified siblings using the UKB provided kinship file, listing all pairwise kinships among 100,000 pairs in the sample of nearly 500,000 individuals. Following the literature (Fletcher et al. 2021), we first choose pairs with kinship >0.2, which reflects first-degree biological relatives (parents/siblings). We then choose the remaining pairs who are <13 years apart in age,

[3]Supplementary table S1 shows the number of observations for the individual RDS-4, GAD and PHQ components.

leaving ~22,000 sibling dyads.[4] We then chose to keep only one dyad from any family with more than one dyad, leaving ~17,600 dyads.

We constructed PGSs for education and depressive symptoms within the UKB using four-fold cross-validation. Cross-validation is required in cases where sample(s) in the Genome Wide Association Study (GWAS) overlap with the analysis sample used in polygenic score analysis, such as in our work. For each fold, we conducted a GWAS on 75% of UKB samples while adjusting for sex, age, and genetic principal components, and produced PGS on the 25% holdout samples. We clumped GWAS summary statistics using 1000 Genomes Project Phase III European samples as a reference for linkage disequilibrium. We used a pairwise R^2 threshold of 0.1 and a window size of 1 Mb. PGSs were standardized within each fold of cross-validation, and then combined for regression analysis.

We limit our analysis to respondents of European descent (n=408,956) due to the lack of portability of PGSs in non-European populations (Martin et al. 2017). We then drop individuals with missing data on education (n=3816) and RDS-4 (n=37,813) leaving a sample of 367,327 individuals. In total, we have 13,577 sibling pairs with data on education, PGSs and RDS-4.

3. Methods

We relate the RDS-4 score of sibling i in family f (RDS_{if}) to education (Edu_{if}), basic control variables (X_{if}) consisting of age, sex, and the first 20 genetic principal components, and an error term (v_{if}).

$$RDS_{if} = \beta_0 + \beta_1 Edu_{if} + X_{if}'\theta + \upsilon_{if} \quad (1)$$

OLS estimates of β_1 that use between-family variation do not control for unobserved genetic and family factors that can confound estimates. We control for shared genetic and family factors by estimating the sibling fixed-effect regression (Eq. 2) which contains indicator variables for each family (μ_f). We additionally include PGSs for education (PGS_Edu_{if}) and depressive symptoms (PGS_DepSym_{if}) to control for measured genetic differences between siblings. We include controls for the "fold" of the individual from the cross-validation step as well.

$$RDS_{if} = \beta_0 + \beta_1 Edu_{if} + X_{if}'\theta + \mu_f + \beta_2 PGS\_Edu_{if} + \beta_3 PGS\_DepS_ym_{if} + \upsilon_{if} \quad (2)$$

Note that within-sibling variation in PGSs is quasi-exogenous because differences in genotypes of full biological siblings are the outcomes of a genetic lottery (Fletcher and Lehrer 2011). Sibling fixed-effect estimates control for population stratification (the

[4]The probability of classifying a dyad as first-degree (parents/siblings) when the dyad is actually unrelated is essentially zero. We refer readers to Fig. 1 in the original KING paper (Manichaikul et al. 2010) which shows that the distribution of kinship coefficients for unrelated and sibling pairs do not overlap, indicating there should be no misclassification error. However, it is difficult to separate parents from siblings since both have a kinship coefficient >0.2. There is some risk of classifying dyads as siblings who may be parent-child if there is a 13-year age difference between the parent and child. We do not use dyads with kinship >0.2 and age difference >13 years because we cannot be certain whether they are siblings or parent-child.

nonrandom patterning of alleles across global populations), parental genetics, and any parental, neighborhood, or school factors that are shared by siblings, and are more likely to represent causal impacts of genetic factors than OLS estimates.

4. Results

Summary statistics are shown in Table 1. In the full estimation sample (column 1) the average age is 56.83 years and 54% are female. Average years of education is 14.94, 17% are high school dropouts (7 years of education, no qualifications), and 32% are college graduates (20 years of education). The average RDS-4 score is 1.54. Summary statistics for the sibling pairs subsample (column 4) are similar to those for the full sample.

Main results are presented in Table 2. OLS estimates in columns 1 for the full sample indicate large mental health differences between high school dropouts (individuals with 7 years of education) and those with higher educational qualifications. Individuals with 10 years of education (individuals with GCSEs, equivalently high school graduates) have a RDS-4 score that is 0.367 (24% relative to the sample mean) lower than high school dropouts. The RDS-4 score of individuals with 20 years of education (college graduates) is 0.616 (40%) lower compared to high school dropouts. Adding the PGSs as controls in column 2 does not substantially attenuate the OLS estimates. For example, the difference in the RDS-4 score between high school graduates and high school dropouts only falls by 6% to 0.346. OLS estimates for the sibling sample (column 3) are similar to those for the full sample. In the sibling sample, there is a difference of 23% (34%) between high school dropouts and high school (college) graduates. Controlling for the education and depressive symptoms PGSs in column 4 does not affect these OLS gradients. The gradients though are substantially attenuated with sibling controls. The difference in the RDS-4 score between high school (college) graduates and high school dropouts is now 14% (16%) in column 5. There are no significant differences between the point estimates on the years of education indicators. This suggests the main effect of education comes from completing high school, whereas additional education does not lead to significant improvements in mental health. Column 6 controls for the education and depressive symptom PGSs. Sibling fixed-effect estimates also show that a one standard deviation (SD) increase in the depressive symptom PGS is associated with 0.181 (12%) increase in the RDS-4 score. The education PGS does not predict mental health within siblings. Similar to findings from the OLS regressions, adding the PGSs as controls in sibling fixed-effect regressions does not attenuate the education-mental health gradients. The difference in the RDS-4 score between high school graduates and dropouts drops slightly to 13%, while the difference between college graduates and high school dropouts remains at 16%.

Table 3 examines the evidence for gene-environment (GxE) interactions between education and the depressive symptoms PGS. Columns 1 and 2 look at whether the depressive symptoms PGS predicts education. If the depressive symptom PGS predicts education, then this would imply an important GxE correlation where the PGS affects both the environment and outcome. OLS estimates in column 1 show that a one SD increase in the depressive symptom PGS is associated with a 0.071 decrease in years of education. However, the sibling fixed-effect estimate—where the genetic variation is quasi-exogenous

—shows no association between the depressive symptom PGS and years of education in column 2.[5] Column 3 gives results from interacting the years of education indicators with the depressive symptoms PGS. The coefficient on the depressive symptoms PGS indicates that a one SD increase is associated with a 0.253 (17%) increase in the RDS-4 score for high school dropouts (individuals with 7 years of education). The interaction terms between the depressive symptom PGS and indicator variables for years of education are all negative, indicating a smaller impact of genetics at higher levels of educational attainment. For example, a one SD increase in the depressive symptom PGS is associated with a 0.155 (0.253–0.098) increase in the RDS-4 score individuals with individuals with 10 years of education (high school graduates), and a 0.139 increase for individuals with 13 years of education. However, all the interactions terms (apart from years of education=15) are statistically insignificant. While one might expect college education to substantially moderate the effect genetics, this is not the case. For college graduates (individuals with 20 years of education) a one SD increase in the depressive symptom PGS is associated with a 0.172 increase in the RDS-4 score. The difference in the effect of the depressive symptom PGS between college graduates and high school dropouts is not statistically significant.

Three additional analyses are provided in the supplementary materials. Supplementary table S2 provides results for the individual components (mood, disinterest, tenseness, tiredness) of the RDS-4. The sibling fixed-effect estimates show that high school graduates have better mental health on all four components relative to high school dropouts. The largest difference between high school graduates and high school dropouts is for disinterest, where the disinterest score is 19% lower for high school graduates. The smallest gradient is for tiredness, where the tiredness score is 10% lower for high school graduates. The interaction estimates between the years of education indicators and the depressive symptoms PGS are negative, suggestive of smaller effects of genetics at higher levels of education but are imprecise. Supplementary table S3 provides results for the PHQ and GAD scales. Consistent with Avendano et al. (2020), estimates are imprecise. Supplementary table S4 gives associations between years of education and the RDS-4, PHQ and GAD scales. An extra year of education is associated with less than a 1% decrease in the RDS-4 score with sibling and genetic controls. Estimates for the PHQ and GAD are also small in magnitude and imprecise.

5. Summary

Does education protect against mental health problems? The current causal evidence is limited and mixed. British studies have found no protective effects of education and Avendano et al. (2020) even find some evidence that more education worsens mental health. We provide new evidence on education-mental health gradients in the UK, extending previous work by taking a different approach: sibling fixed-effects with measured genetic controls for education and depressive symptoms. We uncover evidence of non-monotonic effects with high school graduates having a RDS-4 score that is 13% lower compared to high

[5]The within-r square for the sibling fixed-effect regression model in Table 3 column 2 is 0.021. The within-r square from the same regression after excluding the education and depressive symptoms PGSs and genetic principal components is 0.012. This suggests that the education PGS explains about 1% of the within-sibling variation in years of education.

school dropouts. We also find suggestive evidence that relative to high school dropouts, the effect of the depressive symptom PGS on RDS is smaller for people with more education. This suggests that education may “rescue” genetic propensities toward poor mental health (Cook and Fletcher 2015).

Our study has limitations. First, sibling fixed-effect estimates cannot fully control for sibling-specific factors that are correlated with education and mental health. Second, our results may not be generalizable. Our analysis does not contain diverse populations; it is limited to European ancestry individuals due to poor portability of PGSs in non-European populations. The UKB cohort is healthier and more educated than the wider UK population. This means that less educated people who likely have worse mental health are under-represented which would attenuate associations to null. Despite the limitations, our results indicate there may be a possible causal relationship between higher educational attainment and better mental health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data availability

Code to replicate analysis is available upon request.

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Table 1

Summary Statistics.

	Full Sample			Sibling Subsample		
	Mean (SD) (1)	Min (Max) (2)	Obs (3)	Mean (SD) (4)	Min (Max) (5)	Obs (6)
Demographics						
Age	56.83 (7.99)	39 (73)	367,327	57.07 (7.25)	40 (70)	27,154
Female	0.54 (0.50)	0 (1)	367,327	0.57 (0.49)	0 (1)	27,154
Educational Attainment						
Years of Education	14.94 (5.07)	7 (20)	367,327	14.80 (5.08)	7 (20)	27,154
Years of Education=7	0.17 (0.37)	0 (1)	367,327	0.17 (0.38)	0 (1)	27,154
Years of Education=10	0.17 (0.38)	0 (1)	367,327	0.18 (0.38)	0 (1)	27,154
Years of Education=13	0.05 (0.23)	0 (1)	367,327	0.05 (0.23)	0 (1)	27,154
Years of Education=15	0.12 (0.33)	0 (1)	367,327	0.13 (0.34)	0 (1)	27,154
Years of Education=19	0.16 (0.37)	0 (1)	367,327	0.16 (0.37)	0 (1)	27,154
Years of Education=20	0.32 (0.47)	0 (1)	367,327	0.30 (0.46)	0 (1)	27,154
Mental Health						
RDS-4	1.54 (2.04)	0 (12)	367,327	1.51 (2.02)	0 (12)	27,154
Genetics						
Standardized Education PGS	0.012 (0.99)	-4.50 (5.61)	366,677	0.028 (1.00)	-3.76 (4.04)	27,154
Standardized Depression PGS	-0.009 (0.99)	-4.68 (4.51)	366,677	-0.018 (0.99)	-3.75 (4.20)	27,154

Notes: RDS-4: 4 item Recent Depressive Symptoms score.

Table 2

OLS and Sibling Fixed-Effects Associations Between Educational Attainment and Recent Depressive Symptoms.

Sample	**Full**	**Full**	**Sibling**	**Sibling**	**Sibling**	**Sibling**
Outcome	**RDS-4**	**RDS-4**	**RDS-4**	**RDS-4**	**RDS-4**	**RDS-4**
Outcome Mean	**1.54**	**1.54**	**1.51**	**1.51**	**1.51**	**1.51**
Fixed-Effects	**None**	**None**	**None**	**None**	**Sibling**	**Sibling**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
Years of education=10	−0.367*** (0.012)	−0.346*** (0.012)	−0.354*** (0.042)	−0.336*** (0.042)	−0.209*** (0.066)	−0.200*** (0.066)
Years of education=13	−0.449*** (0.017)	−0.413*** (0.017)	−0.450*** (0.060)	−0.409*** (0.060)	−0.160* (0.086)	−0.147* (0.086)
Years of education=15	−0.517*** (0.013)	−0.484*** (0.013)	−0.455*** (0.045)	−0.423*** (0.045)	−0.243*** (0.070)	−0.239*** (0.070)
Years of education=19	−0.375*** (0.012)	−0.354*** (0.012)	−0.307*** (0.043)	−0.293*** (0.043)	−0.162** (0.068)	−0.159** (0.068)
Years of education=20	−0.616*** (0.010)	−0.562*** (0.011)	−0.509*** (0.038)	−0.456*** (0.039)	−0.244*** (0.068)	−0.235*** (0.068)
Education PGS		−0.029*** (0.003)		−0.022* (0.013)		−0.008 (0.025)
Depressive Symptoms PGS		0.179*** (0.003)		0.203*** (0.012)		0.181*** (0.023)
N	367,327	367,327	27,154	27,154	27,154	27,154

Notes: Reference group is years of education=7 (no qualifications/high school dropouts). All regressions additionally control for age, gender, and the first 20 genetic principal components. Columns 2,4, and 6 additionally control for the “fold” of the individual from the cross-validation step in constructing the PGSs.

PGSs are standardized to have mean 0 and standard deviation of 1. Standard errors clustered at the family level in sibling samples.

*** significant at 1%

** significant at 5%

* significant at 10%.

RDS-4: 4 item Recent Depressive Symptoms score.

Interactions between Education and the Depressive Symptom PGS on Recent Depressive Symptoms.

Sample	**Sibling**	**Sibling**	**Sibling**
Outcome	**Years of Education**	**Years of Education**	**RDS-4**
Outcome Mean	**14.80**	**14.80**	**1.51**
Fixed-Effects	**None**	**Sibling**	**Sibling**
	(1)	**(2)**	**(3)**
Years of education=10			−0.196*** (0.066)
Years of education=13			−0.145* (0.086)
Years of education=15			−0.236*** (0.070)
Years of education=19			−0.155** (0.067)
Years of education=20			−0.231*** (0.068)
Education PGS	1.086*** (0.030)	0.482*** (0.053)	−0.009 (0.025)
Depressive Symptoms PGS	−0.071** (0.030)	0.055 (0.051)	0.253*** (0.049)
Depressive Symptoms PGS *			
Years of education=10			−0.098 (0.060)
Years of education=13			−0.114 (0.074)
Years of education=15			−0.111* (0.064)
Years of education=19			−0.060 (0.063)
Years of education=20			−0.081 (0.057)
N	27,154	27,154	27,154

Notes: Reference group in column 3 is years of education=7 (no qualifications/ high school dropouts). All regressions additionally control for age, gender, the first 20 genetic principal components, and for the "fold" of the individual from the cross-validation step in constructing the PGSs PGSs are standardized to have mean 0 and standard deviation of 1. Standard errors clustered at the family level.

*** significant at 1%

** significant at 5%

* significant at 10%.

RDS-4: 4 item Recent Depressive Symptoms score.