Causal Effect of Adult Children's Education on Parental Longevity in the US. An Intergenerational Mendelian Randomization Approach*

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Extended Abstract for the 2020 EPC

Abstract

Having better educated adult children is associated with living longer and being healthier. Yet, causal tests of this association are still rare. Using the Health and Retirement Study (HRS), we propose a novel, intergenerational Mendelian Randomization approach (IGMR) to investigate the causal relationship between the education of HRS respondents and their parents' longevity. We are able to show that children's education is indeed associated with greater longevity for parents. When we instrument the endogenous education variable using genetic dispositions for educational attainment conditional on parental education and genetic predispositions for various health outcomes, the education effect remains strong and statistically significant for mothers, suggesting a causal effect of children's education on parental health in the US. We discuss substantive implications of our findings and investigate potential limitations of our new approach, including biological pleiotropy and family size.

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Introduction

Parents with better-educated adult children are healthier and live longer, a well-established recent finding that has become know as the 'social foreground' hypothesis (Torssander, 2013). But is parental health and longevity a consequence of children's education, or do healthier parents pass on traits to their children that are beneficial for the children's education? A recent review article established that this research area is understudied and particularly causal evidence is needed (De Neve and Kawachi, 2017). Potential pathways for a causal effect are that better-educated children are better able to help their parents navigate the health care system or provide better care, better support their parents economically, might induce better health behaviors in their parents, or might cause their parents less stress. Yet, it might also be that parents who live longer might have passed on traits to their children that are also beneficial to the children's education. Such an endogenous pathway would be in line with an association between adult children's education and parental longevity, yet would have completely different implications, e.g. for policymakers interested in potential benefits of investing in children's education.

Existing studies on the causal relationship make use of schooling reforms and reach mixed results. Particularly for the US, causal evidence is still lacking. Our study addresses the causal dimension of a children's education–parental mortality link with a innovative, genetically informed approach. Using data from the Health and Retirement Study (HRS) and a novel Intergenerational Mendelian Randomization (IGMR) approach, we examine in how far the spillover effect from children's education to parental longevity can be seen as causal.

Previous research

The evidence for an association between children's education and parental health and longevity is strong (Brooke *et al.*, 2017; De Neve and Harling, 2017; Elo *et al.*, 2018; Friedman and Mare, 2014; Jiang, 2019; Lee, 2018; Lee *et al.*, 2017; Meyer *et al.*, 2019; Sabater and Graham, 2016a,b; Sabater *et al.*, 2019; Torssander, 2013, 2014; Wolfe *et al.*, 2018a,b; Yahirun *et al.*, 2016, 2017, 2019; Yang *et al.*, 2016; Zimmer *et al.*, 2016, 2002, 2007), covering a wide range of health outcomes (including self-reports, biomarkers, and mortality) and stemming from a wide range of societies (ranging from rural South Africa to the US and Nordic welfare states).

Evidence for a causal effect of children's education on parental health is mixed. A number of studies support the notion of an intergenerational spillover effect. One mechanism might be economic support provided by better-educated children to their parents. For instance, a study of Tanzanian data exploited quasi-experimental variation created by an educational reform, showing that

the reform increased educational attainment by more than a year and reduced parental mortality risks (De Neve and Fink, 2018). Another mechanism might be that better-educated children are better able to help parents navigate the health care system. A Swedish study found that parents with higher educated adult children live longer when comparing adult children who were cousins (Torssander, 2013), thus being able to account for a number of unobserved traits passed from parents to children.

But not all studies yield results as clear-cut as those. A Swedish study of an educational reform suggested that a reduced mortality risk can only be for fathers with daughters affected by the reform (Lundborg and Majlesi, 2018). A Chinese study showed that parents of children affected by an educational reform show improved cognition and lung functioning, but no effects on grip strength, self-rated health, or mental health (Ma, 2019).

Another pathway linking children's education and parental health might be endogenous. Parents who live longer might have passed on traits to their children that are also beneficial to the children's education. An English study of an educational reform, which showed that parents of children affected by the reform did not live longer or report better health (Potente *et al.*, 2019), would be in line with this endogenous pathway.

Data and methods

Data: Health and Retirement Study

The Health and Retirement Study (HRS, Sonnega *et al.*, 2014) is a long-running panel study of the older US population. Between 2006 and 2012, the HRS collected genetic (saliva) samples from approximately 84% of participants undergoing face-to-face interviews. These DNA samples were genotyped for about two million SNPs. Further, the data include information on adult children's education and parental longevity, making it the ideal resource for our research endeavor. Indeed, Friedman and Mare (2014) used HRS data to show that children's education is associated with parental mortality.

We restrict the data to non-Hispanic white respondents who were genotyped and for whom the key variables of interest were observed. White here refers to respondents who self-identify as white and who fall within one standard deviation of all self-identified whites for eigenvectors 1 and 2 in the PCA of all unrelated study subjects. Specifically, we make use of the RAND HRS Longitudinal file (Bugliari *et al.*, 2019), which we merge with the HRS genetic data (Ware *et al.*, 2018).

Our key predictor is the *respondents' schooling* measured in years. We control for *parental education*, respondents' *sex*, *birth cohort*, and respondents' *number of siblings*. Descriptive statistics are reported in Table 1. Our outcome

variable, *parental survival*, is shown in Figure 1.

Table 1: Descriptive statistics

	Prop./Mean	*SD*	Min.	Max.
Father's avg. age at death	72.73	13.65	20	105
Father deceased	0.94		0	1
Mother's avg. age at death	77.72	13.93	21	106
Mother deceased	0.86		0	1
Child's education (in y.)	13.47	2.45	0	17
Father's education:				
Less than high school	0.53		0	1
High school diploma	0.29		0	1
Some college	0.07		0	1
College+	0.12		0	1
Mother's education:				
Less than high school	0.46		0	1
High school diploma	0.37		0	1
Some college	0.09		0	1
College+	0.08		0	1
Female child	0.57		0	1
No. of child's siblings	2.75	2.15	0	18
Child's birth cohort:				
Born < 1924	0.09		0	1
Born 1925-34	0.21		0	1
Born 1935-44	0.30		0	1
Born 1945-54	0.24		0	1
Born 1955+	0.15		0	1
N	10,486			

Source: HRS (Sonnega *et al.*, 2014), own calculations.

Identification strategy: Mendelian Randomization

Intuition A common approach to identify causal relationships—in contrast to associations—using non-experimental designs is an instrumental variable approach (Wooldridge, 2010). An instrumental variable is a variable exogenous to the outcome, but causal to the predictor variable is used to (quasi-)randomize a predictor variable. Traditional instruments are policy changes that occur quasi randomly or the coin flip assigning participants to treatment or control group in a randomized controlled trial. Recently, genes have become very popular instrumental variables in epidemiological research under the name of Mendelian Randomization (MR, Davey Smith and Ebrahim, 2003; Davey Smith and Hemani, 2014; Pickrell, 2015). Genes are essentially a random draw of the genes of our parents, thus might be used as intstruments, and MR is now frequently

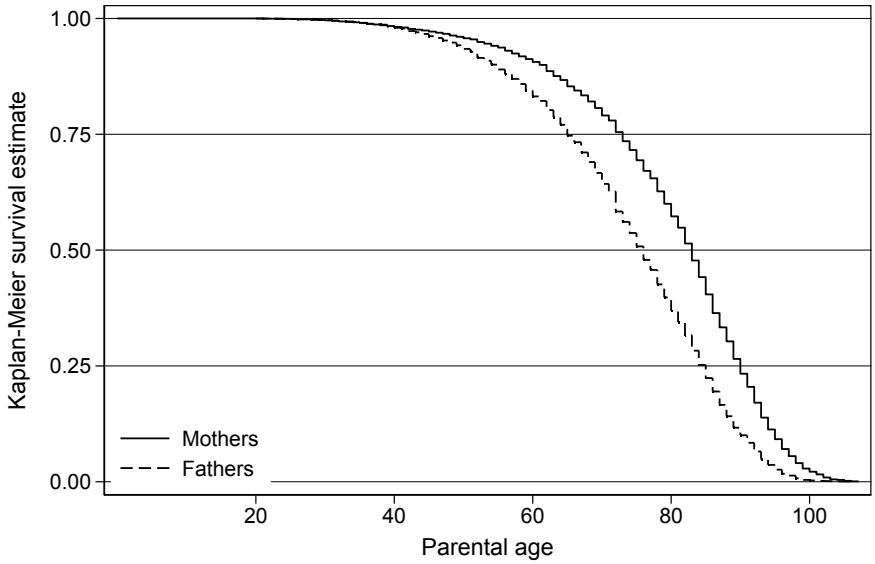

Figure 1: Survival curves for respondents' fathers and mothers, $N = 10{,}355$

Source: HRS (Sonnega *et al.*, 2014), own calculations.

implemented in social science research (DiPrete *et al.*, 2018; Sotoudeh *et al.*, 2019).

Genes as instrumental variables In our study, we combine and extend two approaches to Mendelian Randomization. First, in network studies, it has been highlighted that the genes of ego might be useful predictors of behavior in alter. For instance, genes which predict the smoking behavior of peers also affect ego's smoking behavior, pointing towards social contagion effects in smoking (Sotoudeh *et al.*, 2019). We propose to use the Mendelian Randomization approach for a cross-trait prediction of adult children's education on parents' mortality. However, since parents and children share 50% of their segregating genetic material, we need to take parental education into account and reintroduce a challenge known from the classic MR approach, namely, that the same genes might influence education and mortality (Marioni *et al.*, 2016), also known as biological pleiotropy.

Since individual genetic effects on social outcomes are known to be substantively small, we accumulate known effects across the whole genome in so-called polygenic scores, to avoid potential issues with weak instruments. All polygenic scores were readily available in the HRS data (Ware *et al.*, 2018). Our genetic instrument is the *polygenic score for educational attainment* (EA3, Lee *et al.*, 2018). Further, all models control for the first 10 genetic principal components

to take population stratification, in particular ancestry-based between-family differences, which could genetically or environmentally bias results.

Accounting for biological pleiotropy A recent extension of the classic MR approach (DiPrete *et al.*, 2018) proposes the solution to condition the genetic effects for education on genetic effects for the outcome under study. Thus, we condition our genetic variables also on potential pleiotropic effects due to e.g. heart disease or smoking. Specifically, we further control for the *polygenic score for waist circumference* (Shungin *et al.*, 2015), the *polygenic score for smoking* (number of cigarettes per day, Tobacco and Genetics Consortium, 2010), the *polygenic score for coronary artery disease* (CAD, Schunkert *et al.*, 2011), the *polygenic score for myocardial infarction* (The CARDIoGRAMplusC4D Consortium, 2015), and the *polygenic score for longevity* (Broer *et al.*, 2014).

Instrumental variables in time-to-event models The two-stage least squares approach (2SLS), commonly used in linear models, replaces the endogenous independent variable, here children's education, with the value predicted in the first stage. In the first stage, the endogenous independent variable is regressed on the instrumental variable. As our outcome was risk of parental death, we used a survival model to account for right-censoring of the data (Allison, 2014). For time-to-event models, the two-stage residual inclusion approach (2SRI), which is identical to the 2SLS approach in a linear setting, has been shown to yield consistent estimates (Terza *et al.*, 2008). Unlike the 2SLS, in the second stage of the 2SRI regression, both the first-stage residuals, S_{v_i}, and the endogenous variable, S_i, are included in the model to be fitted. We estimated a Cox proportional hazards model for right-censored data (Cox, 1972):

$h(t) = h_0(t)e^{(\Sigma_k X_k \alpha_k + S_i \rho_1 + S_{v_i} \rho_2 +)}$

where $h_0(t)$ is an unspecified baseline hazard function, Gj is child sex, Bj is child birth order, and Cy is a dummy variable for child cohort y. We controlled for

All coefficients are hazard ratios, and ρ_1 is a consistent estimate for the true effect of child's education on parental mortality. Therefore, $exp(\rho_1)$ is the hazard ratio associated with a one-year increase in child's education. If $exp(\rho_1)$ is smaller than 1 and statistically different from 0, there is a causal negative relationship between child's education and parental mortality. The ρ_2 is the effect of the first-stage residuals on parental mortality; its interpretation is equivalent to that of the Wu–Hausman test in a 2SLS framework, wherein a statistically significant coefficient indicates endogeneity in the relationship between child's education and parental mortality.

Results

Table 2 models parental age at death, for fathers on the left hand side and mothers on the right hand side. The naive Cox estimates for fathers and for mothers show that the hazard of dying is almost four per cent lower with each additional year of children's schooling. Controlling for education of both parents reduces this association somewhat, to almost three per cent for fathers and about three and a half per cent for mothers.

The reduced form models reveal that an increase in the polygenic score for education (PGS EA3) reduces the hazard of dying not for fathers, only for mothers. A one-unit increase in the polygenic score for education reduces the hazard of dying by three per cent, but only for mothers. The F-value of first-stage model reveals that the polygenic score for education is a strong instrument, well beyond the rule of thumb of 10. The second stage model for fathers suggests that there is no causal effect of children's education on father's longevity, for mothers however we find that the hazard of dying is about six per cent lower with each additional year of the child's schooling.

Tentative conclusions and future plans

So far, our Mendelian randomization estimates support a causal effect of adult children's education on mothers' longevity. This finding is a major step forward, as all previous evidence from the US was associational in nature (Friedman and Mare, 2014; Yahirun *et al.*, 2019).

This finding has important policy implications. If improving education in a younger generation has positive effects that spill over to an older generation, education breaks a generational trade-off when policy makers decide what to invest in. Investments in education are not at the expense of the older generation, as beneficial spillover effects to the older generation exist.

One intriguing aspect of our findings is the gendered dimension of the health benefits. While children's education is beneficial for the longevity of mothers, we can't find such an effect for fathers. This contradicts the findings from Lundborg and Majlesi (2018), who were only able to find health benefits for fathers.

Our future plans for this study comprise the following:

- Stratify our models by sex of the child. Previous research has suggested that associations differ by the sex of children (e.g. Lundborg and Majlesi, 2018)—our data allow examining this issue further.
- Stratify the model by variables indicating how far children live away from their parents, how many children parents have, the amount of contact they

Table 2: Cox regression of parental survival on child's schooling, hazard ratios (95 per cent confidence intervals in brackets)

	Fathers				Mothers			
	Cox	Cox	Reduced form	2SRI	Cox	Cox	Reduced form	2SRI
Child's years of schooling	0.964***	0.971***		0.979	0.960***	0.966***		0.936**
	[0.956,0.972]	[0.962,0.980]		[0.940,1.021]	[0.952,0.969]	[0.957,0.975]		[0.896,0.978]
Mothers's education (*ref.* college+)								
Less than high school		0.990	1.037	1.009		1.113*	1.180***	1.080
		[0.901,1.088]	[0.944,1.139]	[0.901,1.129]		[1.009,1.228]	[1.069,1.301]	[0.960,1.215]
High school diploma		0.988	1.013	0.996		1.096	1.131**	1.075
		[0.905,1.078]	[0.928,1.105]	[0.906,1.095]		[0.999,1.202]	[1.031,1.240]	[0.972,1.188]
Some college		0.999	1.003	1.004		1.060	1.063	1.067
		[0.902,1.106]	[0.906,1.111]	[0.907,1.112]		[0.953,1.180]	[0.955,1.183]	[0.958,1.188]
Father's education (*ref.* college+)								
Less than high school		1.202***	1.254***	1.217***		1.082	1.116*	1.029
		[1.107,1.305]	[1.156,1.360]	[1.101,1.346]		[0.992,1.179]	[1.025,1.216]	[0.926,1.143]
High school diploma		1.204***	1.240***	1.216***		1.033	1.057	0.997
		[1.112,1.303]	[1.146,1.342]	[1.112,1.330]		[0.950,1.124]	[0.972,1.150]	[0.906,1.096]
Some college		1.056	1.077	1.065		1.075	1.083	1.067
		[0.956,1.166]	[0.975,1.189]	[0.964,1.178]		[0.969,1.193]	[0.976,1.202]	[0.961,1.185]
PGS Education (EA3)			0.990				0.967**	
			[0.969,1.011]				[0.945,0.989]	
Daughter (*ref.* son)	1.040	1.039	1.051*	1.044*	1.044*	1.041	1.051*	1.032
	[0.999,1.083]	[0.997,1.082]	[1.009,1.094]	[1.000,1.089]	[1.001,1.089]	[0.998,1.086]	[1.008,1.097]	[0.987,1.079]
Child birth cohort (*ref.* born 1935-44)								
Born < 1924	1.002	0.984	0.992	0.990	1.055	1.024	1.039	1.030
	[0.932,1.078]	[0.911,1.062]	[0.918,1.071]	[0.917,1.069]	[0.981,1.135]	[0.948,1.106]	[0.962,1.122]	[0.953,1.112]
Born 1925-34	1.030	1.028	1.035	1.030	1.005	0.998	1.008	0.995
	[0.975,1.088]	[0.973,1.086]	[0.979,1.094]	[0.974,1.088]	[0.951,1.062]	[0.943,1.055]	[0.953,1.066]	[0.941,1.053]
Born 1945-54	0.909***	0.919**	0.908***	0.917**	0.965	0.978	0.965	0.997
	[0.861,0.960]	[0.869,0.971]	[0.859,0.960]	[0.865,0.972]	[0.911,1.023]	[0.923,1.038]	[0.910,1.023]	[0.937,1.060]
Born 1955+	0.770***	0.790***	0.782***	0.788***	0.800***	0.823***	0.816***	0.830***
	[0.719,0.824]	[0.737,0.848]	[0.729,0.839]	[0.734,0.846]	[0.739,0.866]	[0.759,0.892]	[0.752,0.886]	[0.765,0.901]
No. of child's siblings	0.998	0.997	1.001	0.997	0.997	0.996	1.002	0.989
	[0.988,1.007]	[0.987,1.006]	[0.992,1.011]	[0.985,1.010]	[0.987,1.008]	[0.986,1.006]	[0.992,1.012]	[0.977,1.002]
PGS control variables								
Waist circumference			1.017	1.017			1.020	1.021
			[0.993,1.041]	[0.994,1.041]			[0.996,1.046]	[0.996,1.046]
Smoking (cigarettes per day)			0.995	0.995			1.024*	1.022
			[0.974,1.016]	[0.974,1.016]			[1.001,1.047]	[0.999,1.046]
Coronary artery disease			0.985	0.986			0.990	0.991
			[0.963,1.007]	[0.964,1.008]			[0.967,1.013]	[0.968,1.014]
Myocardial infarction			1.052***	1.050***			1.040***	1.036**
			[1.029,1.076]	[1.026,1.074]			[1.016,1.065]	[1.011,1.061]
Longevity			0.980	0.980			0.993	0.992
			[0.956,1.005]	[0.956,1.004]			[0.968,1.019]	[0.967,1.018]
First-stage residuals				0.992				1.035
				[0.951,1.035]				[0.990,1.082]
First ten principal components	No	No	Yes	Yes	No	No	Yes	Yes
Observations	10,355	10,355	10,355	10,355	10,355	10,355	10,355	10,355
First-stage F				132.2				132.2

* p < 0.05, ** p < 0.01, *** p < 0.001.

have with their parents, and whether they provide their parents with help to investigate the mechanisms underlying the causal effect.

- Explore the effects of conditioning on more or different polygenic scores to rule out further potential pleiotropic effects.

- As further tests of the effect, we will 1) analyze respondent's longevity as a function of their children's education and instrument the childrens' education with the polygenic scores of their parents and 2) instrument children's education with only the genes which have not been transmitted from the parent under study.

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