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Article

Family of origin and educational inequalities in mortality: Results from 1.7 million Swedish siblings

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A B S T R A C T

Circumstances in the family of origin have short- and long-term consequences for people's health. Family background also influences educational achievements – achievements that are clearly linked to various health outcomes. Utilizing population register data, we compared Swedish siblings with different levels of education (1,732,119 individuals within 662,095 sibships) born between 1934 and 1959 and followed their death records until the end of 2012 (167,932 deaths).

The educational gradient in all-cause mortality was lower within sibships than in the population as a whole, an attenuation that was strongest at younger ages (< 50 years of age) and for those with a working class or farmer background. There was substantial variation across different causes of death with clear reductions in educational inequalities in, e.g., lung cancer and diabetes, when introducing shared family factors, which may indicate that part of the association can be ascribed to circumstances that siblings have in common. In contrast, educational inequalities in suicide and, for women, other mental disorders increased when adjusting for factors shared by siblings.

The vast variation in the role of childhood conditions for the education-mortality association may help us to further understand the interplay between family background, education, and mortality. The increase in the education gradient in suicide when siblings are compared may point towards individually oriented explanations ('non-shared environment'), perhaps particularly in mental disorders, while shared family factors primarily seem to play a more important role in diseases in which health behaviors are most significant.

Introduction

The inverse association between educational level and mortality risk has generated a lot of scientific interest over recent decades. In Sweden, the life expectancy gap at age thirty between compulsory and college educated individuals today amounts to slightly more than five years (The Swedish Commission for Equity in Health, 2016). However, precisely why this gap exists is not clear. Education precedes occupation and income - two potentially health-generating resources - but it may also influence health through a greater avoidance of unhealthy behaviors, access to health related information, and increased conceptual thinking (Cutler & Lleras-Muney, 2010). It has been suggested that education is the 'fundamental element' in connecting social positions to health (Mirowsky & Ross, 2005), but the assumption that education has a causal effect on morbidity and mortality has also been questioned (Behrman et al., 2011). Causality from health to education is also conceivable if, for example, ill health in childhood impedes later educational achievements (Case et al., 2005).

Education may also be of particular interest because it signals family background. Socioeconomic conditions during childhood are clearly associated with educational outcomes (Breen & Jonsson, 2005). Family background, in a broad sense, also has important consequences for individuals' health and mortality risks over the life course (Kuh & Shlomo, 2004). The complex interplay between family background, education, and mortality is however not fully understood, and the aim of the present study is to obtain further insights into these relationships. The existing studies have mainly examined the influence of single, fairly well-defined circumstances in the family of origin, such as childhood socioeconomic position (e.g., Galobardes, Lynch & Davey, 2004); however, it is difficult to adequately characterize and obtain information on all of the relevant features of family background that are important to both educational attainments and adult health and longevity. Comparing siblings with different levels of education provides an opportunity to potentially circumvent this problem. Because siblings generally share their family of origin, sibling comparisons will match out any measured or unmeasured characteristics to

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which siblings were equally exposed to when they were growing up. If the family of origin has any influence on the association between education and mortality, one might expect a within-family gradient to be weaker than the association in the population as a whole.

Recent studies from the US and several Nordic countries have shown that an educational gradient in morbidity and mortality exists between siblings (e.g., Krieger, Chen, Coull & Selby, 2005; Madsen, Andersen, Christensen, Andersen & Osler, 2010; Naess, Hoff, Lawlor & Mortensen, 2012; Sondergaard et al., 2012). In addition, some of these studies also show that the relative educational gradient in mortality is less steep in within-sibling comparisons than in the population as a whole, which has been interpreted as evidence of familial confounding of the association between education and mortality. However, the education-mortality association remained unchanged in a Swedish twin study, which suggests that the association is not driven by the environmental and/or genetic factors that are common to the twin pair (Lundborg, Lyttkens & Nystedt, 2016). To examine the genetic input, comparing the health outcomes for dizygotic and monozygotic twins provides a strong research design. A concern with twin studies may however be whether the results are generalizable to the overall population (Strully & Mishra, 2009).

Our contribution

We examine the education-mortality association for Swedish siblings who were born between 1934 and 1959 utilizing population register data. In addition to describing how education and mortality are related for the between and within sibling groups, respectively, we address two gaps in the existing literature. *First*, the previous research that utilizes sibling designs to study the education-mortality association has generally been limited in its ability to examine specific causes of death, particularly for women. It is well-known that the education-mortality gradient varies considerably by cause of death with strong inverse associations for causes such as coronary heart disease, lung cancer and diabetes, but with weak positive or no associations for prostate cancer, breast cancer and (in some studies) external causes for women (Steenland, Henley, & Thun, 2002; Huisman, Kunst, & Bopp et al., 2005). Likewise, measured family background factors such as parents' socioeconomic position show particularly strong inverse associations with the risk of dying from diseases with a considerable behavioral component, even net of later-life social class (Lawlor et al., 2006). Cause-specific variations in the education-mortality pattern may further point towards other possible explanations, for example in suicides for which a psychiatric disease is not only the underlying cause of death, but which may also result in truncated educational attainments, particularly if the onset is early (Kessler, Foster, Saunders & Stang, 1995). This encourages a closer examination of the total influence of family factors on the relationship between education and cause-specific mortality.

Second, whether family factors affect the education-mortality gradient similarly irrespective of social class background is not known. We hypothesize that both the health-related selection into education and/or the causal effects of education may differ between, for example, working class and service class families. According to some of the social class literature, the association between education and class destination, i.e., how important an individual's own educational achievements are to the subsequent position in the labor market, is weaker for individuals from more advantaged backgrounds compared to those from less advantaged families (Goldthorpe, 2007). However, there is mixed evidence for education having a greater effect on health for individuals from disadvantaged social origins. Some of the research suggests that education is more important to people with low-educated parents, so that they are more dependent on their own educational attainment for good health compared to individuals with highly educated parents (Ross & Mirowsky, 2011). Quite opposite to this assertion, greater health returns to education for individuals from advantaged backgrounds have also been shown (Bauldry, 2014). However, it is unclear if family influences on the education-mortality association differ by social class origin, or, rather, if shared family factors affect the education-mortality association similarly regardless of class background. We believe that describing the variation in the education-mortality gradient between social classes can be one important step towards understanding the processes underlying the association.

In addition to examining cause-specific mortality and the shared family characteristics within distinct social classes, an additional purpose of this study is to shed light on the difficulties in the interpretation of estimates from sibling fixed-effects models. Sibling comparisons come at a cost as only siblings that are discordant on education and age at death can contribute, which reduces precision and induces bias. This has in general received superficial treatment in the literature. Sibling designs cancel out shared family factors, which cause estimates to attenuate, but two mechanisms may counteract and inflate the sibling fixed effects estimates. First, in a Cox regression the inclusion of any variable that is associated with the outcome may cause the estimates of other variables to inflate even in the absence of confounding, which has been referred to as the non-collapsibility of the Hazard Ratio or as a non-linearity effect (Martinussen & Vansteelandt, 2013). Second, the estimates might also inflate due to non-shared confounding from collider stratification induced by design (Frisell, Oberg, Kuja-Halkola & Sjolander, 2012). We review how conditioning on a difference in education between siblings may very likely induce a non-causal association between education and mortality, and we further discuss what this means for the interpretation of our main findings.

Data

The basis for including individuals in this study was the Census of 1990, which covered the entire Swedish population in November of that year. The Multi-generation Register with information on parents was linked to the individuals in the Census to identify full biological siblings. The Multi-generation Register contains parental information for individuals who were born after 1932; however, the parent-child linkage is more complete for individuals who were born in 1934 or later. We therefore include birth cohorts from 1934 and onwards, with the upper limit set to 1959 so that most of the individuals had completed their education before the start of the follow-up. All full siblings were possible to identify if the parents were born in Sweden in the year 1915 or later. The Multi-Generation Register and the validation procedures that weer used have been described in detail elsewhere (Ekbom, 2011).

The data on education were collected from the national Educational Register in 1990. Mortality follow-up was conducted using the Cause of Death Register from January 1991 to December 2012. The study population thus consisted of full biological siblings were born from 1934 through 1959, who were part of a sibling group of two or more siblings who were alive and living in Sweden in January 1991 (N=1,732,119). We also select a subpopulation that was born between 1944 and 1959 (N=1,153,730) for whom we can track information on the social class position during childhood from the Census of 1960.

Variables

Education

Five ordered levels of highest attained education in 1990 were distinguished: Compulsory schooling (8–9 years), short upper-secondary education (mainly vocational, less than 3 years), academic upper-secondary education (3–4 years), some tertiary education (less than 3 years) and tertiary exam (3 years or more, including postgraduate studies). Education was both introduced as a set of dummy variables and as a continuous variable (level 1–5).

Mortality

In addition to all-cause mortality, the following specific causes of death were analyzed separately: Ischemic heart disease (IHD, ICD10: I20-25), lung cancer (C32-34), respiratory diseases (J00-99), diabetes (E10-14), alcohol-related deaths (F10, K70, K73-74), mental diseases excluding alcohol abuse (F00-09; F11-99), suicide (X60-84), and other external causes of death (V01-Y89, excl. X60-84). These particular causes of disease were chosen for two reasons. First, these causes are common ones for which the association with education has been studied previously, and for which an educational gradient has been found. Secondly, the causes were chosen because knowledge about their etiology exists, which is useful in the interpretation of the results.

Social background

Social background was derived from parents' occupation and employment status when the individuals who were born from 1944 to 1959 were between 1 and 16 years old (i.e., in the year 1960). Occupation and employment status were combined to determine social class according to the EGP class schema (Erikson & Goldthorpe, 1992). Thereafter, family social class was assigned in agreement with the dominance principle based on both parents' individual class position (Erikson, 1984). In the analyses, we use the broader classes of manual occupations (47%); non-manual occupations (26%); self-employed (9%); and farmers (12%). For 6% of the individuals who were born between 1944 and 1959, there was no information of the parents' occupation due to missing data or because none of the parents were active in the labor market. It is possible that parents change class position during the individuals' childhood; however, from approximately 30 years of age, the class position is fairly stable (Jonsson, 2001). Typical manual occupations included shop assistants and drivers, and typical non-manual occupations included teachers and engineers. The most common occupation for the self-employed was retailer, but the occupational variation was substantial.

Statistical analyses

To describe the familial aggregation of education, we calculated for each index sibling in a sibling pair the distribution of education among their co-sibling. This metric is known as the probandwise concordance rate, and is the probability of the co-sibling having a certain education, conditional on the index sibling's education. We also calculated the ratio of observed concordance divided by the concordance expected if education was randomly distributed across families, i.e., no familial aggregation. We calculated confidence intervals of the observed and expected proportions and ratios by bootstrapping (sampling from the 1,741,172 pairs with replacement under the observed and expected distributions of education, 2,000 repeats). Because of the vast number of pairs, all of the confidence intervals were very tight with all of the proportions varying less than one percentage point.

In the survival analyses, we used age in months as the time. Individuals were counted as being under the risk of dying from the age that they had in January 1991 until the age of emigration, age of death, or age at the end of follow-up, which was December 2012. All of the analyses were adjusted for sex. The sex-specific estimates of the association between education and mortality were obtained by including an interaction term between sex and education. We conducted analyses for all-cause mortality and cause-specific mortality. We further stratified the analyses by age-group and by social class background. We conducted two sets of Cox proportional hazards regression models: with and without a family fixed effect. We will refer to these as population-based analyses and family-based, respectively. In the analyses without a family fixed effect (i.e., the population-based analyses) the model is of the form $\lambda_{ij}(t)=\lambda_0(t)\ \exp(\beta_1 x_{ij1}+...+\ \beta_m x_{ijm})$, where $\lambda_{ij}(t)$ is the hazard for the *i*th sibling within the *j*th family, $\lambda_0(t)$ is the baseline hazard, β_m is the *m*th element of the vector of regression coefficients, and x_{ijm} is the *m*th element of the vector of covariates for the *i*th sibling in the *j*th family (i.e., dummy variables pertaining to sex and education). In these analyses, we took interdependence of siblings into account by using a robust variance estimator. In the analyses with a family fixed effect, the model is of the form $\lambda_{ij}(t)=\lambda_{0j}(t)\ \exp(\beta_1 x_{ij1}+...+\ \beta_m x_{ijm})$. Note that the model now contains a separate baseline hazard $\lambda_{0j}(t)$ for every *j*th family. The family-specific baseline hazard ensures that comparisons are only made within families. Unlike shared frailty models, this model makes no assumption of independence between the shared component (i.e., the baseline) and covariates. To quantify the importance of the family of origin, we compare the Hazard Ratios between the analyses with and without sibling fixed effects.

Table 1
Descriptives of the Swedish population, born 1934 to 1959, alive and living in Sweden at the end of 1990. No. and % for individuals included and excluded in the study, respectively.

	Included		Excluded, no sibling in the defined population		Excluded, no info. on parents and/or education	
	N	%	N	%	N	%
Level of education						
Compulsory	528731	31	150914	27	238804	39
Short upper secondary (< 3 y)	561673	32	185715	33	179914	29
Long upper secondary (> = 3 y)	191842	11	69389	12	71676	12
Short tertiary (< 3 y)	215007	12	74178	13	60297	10
Long tertiary (> =3 y)	234866	14	80820	14	68021	11
Total	1732119	100	561016	100	618712	100
Siblings						
One	–	–	563830	100	n/a	
Two	816658	47	–	–	n/a	
Three	484119	28	–	–	n/a	
Four	228232	13	–	–	n/a	
Five or more	203110	12	–	–	n/a	
Total	1732119	100	563830	100		
Sex						
Male	884370	51	287409	51	330763	51
Female	847749	49	276421	49	322544	49
Total	1732119	100	563830	100	653307	100
Year of birth						
1934 to 1939	236006	14	101556	18	199144	30
1940 to 1944	342383	20	87462	16	144318	22
1945 to 1949	435020	25	101786	18	130258	20
1950 to 1954	397961	23	95840	17	96799	15
1955 to 1959	320749	19	177186	31	82788	13
Total	1732119	100	563830	100	653307	100
Mortality						
Alive in the end of 2012	1564187	90	504767	90	551986	84
Died 1991–2012	167932	10	59063	10	101321	16
Total	1732119	100	563830	100	653307	100

Results

The study population that is included is fairly similar to the entire Swedish population born 1934 to 1959 (Table 1). Those who were excluded from the analyses were more likely to be born at the beginning of the period and lack information on the variables of

Table 2
Probandwise concordance of education in 1,741,172 sibling pairs.

		Index sibling's education										
		1		2		3		4		5		Expected
	Education	%[a]	Ratio[b]	%[a]	Ratio[b]	%[a]	Ratio[b]	%[a]	Ratio[b]	%[a]	Ratio[b]	%
Co-sibling's education	**1**	50.85	1.67	34.36	1.13	23.19	0.76	19.01	0.62	11.07	0.36	30.53
	2	32.6	1.01	38.96	1.20	31.89	0.98	29.76	0.92	19.33	0.60	32.43
	3	6.68	0.60	9.69	0.88	15.18	1.37	13.24	1.20	12.77	1.15	11.08
	4	6.15	0.50	10.14	0.82	14.85	1.20	18.69	1.51	18.55	1.49	12.41
	5	3.72	0.27	6.85	0.51	14.89	1.10	19.3	1.42	38.29	2.82	13.56

1=compulsory; 2=short sec; 3=long sec; 4=short tertiary; 5=long tertiary

[a] The distribution of co-sibling education for an index sibling with a given education (given in the column above)

[b] Ratio of observed proportion vs. expected proportion under assumption of no familial aggregation of education. Numbers above 1 indicates a higher observed proportion than expected, numbers below 1 indicates lower proportion observed than expected. The expected distribution is given in the last column and corresponds to the marginal distribution of education in the study population.

interest, or to be born at the end of the period and not have any siblings.

Our analysis shows that the familial aggregation of education was substantial (Table 2). In any sibling pair, an individual with compulsory education was 67% more likely to have a co-sibling with compulsory education and 73% less likely to have a co-sibling with a long tertiary education than expected if paired with a randomly selected individual from the study population. At the other end of the educational distribution, in any sibling pair an individual with a long tertiary education was 182% more likely to have a co-sibling with a long tertiary education.

During the follow-up, 167,932 individuals died. We found a strong and monotone gradient in mortality over the educational categories for both men and women in the population-based analyses (Table 3). When siblings where compared, we found the highest educated sibling to have substantially lower mortality for both men and women; however, the gradient was weaker in the family-based analyses than it was in the population-based analyses with attenuations in the log of the Hazard Ratios (HR) int the estimation of the per one-level increment difference in the association between education and mortality of 15% for men and 28% for women. The importance of the family of origin as measured by the attenuation in the HRs appeared to be greater at younger ages, where the mortality was relatively low and the relative educational differences in mortality high.

When cause-specific mortality was examined (Table 4), we observed substantial attenuation for ischemic heart disease, lung cancer, respiratory diseases, and diabetes-related deaths. For women, the reduction was also clear for alcohol-related mortality. The attenuation was limited for other mental disorders and alcohol-related mortality among men, and deaths from other external causes for both of the sexes. The association between education and suicide was stronger in the family-based analyses than it was in the population-based analyses, and a similar pattern occurred for mental disorders among women. However, the population-based educational gradient in suicide was comparatively moderate among women.

When the all-cause mortality analyses where stratified by childhood social class for the individuals born between 1944 and 1959, the differences between the population- and family-based estimates vary across class origin (Table 5). Within non-manual families, the reduction of the educational gradient was only very marginal when siblings were compared (8% for men, 2% for women). Within manual families, self-employed and farmers, the attenuations of the educational gradients when shared family factors were introduced were much clearer (men 25% and women 29% if they originated from a family in which the parent(s) worked in a manual occupation).

To illustrate the bias that is induced by design in sibling fixed effects, we calculated whether we could induce non-causal associations between the sex of one sibling and mortality in the other by conditioning on education in both siblings (Supplemental material). Our analyses take sex into account, but sex serves as a good test because there is little familial influence on the sexes of siblings (Mortensen, Nielsen, Cnattingius & Andersen, 2011). To explore this bias, we consider sibling pairs, but the argument generalizes to larger sibships. We assume that shared family factors causally affect education and mortality, but not sex as described above, such that the sex of two randomly selected siblings are not statistically associated (see Supplement Fig. 1). If we in this scenario select siblings that have different levels of educational attainment, then the sex of the siblings will be associated as a consequence of the selection. Specifically, if siblings have different educations, there is an increase in probability that they also have a different sex. The underlying logic is further expanded in the Supplemental material, which uses causal Acyclic Directed Graphs to demonstrate theoretically and empirically that some degree of bias is inevitable.

Discussion

In this cohort of Swedish siblings, we observed a substantial, and expected, familial aggregation of education, i.e., education is far from randomly distributed across families, which shows that there is a potential for confounding from shared family factors. We also observed a strong educational gradient in mortality. This was true for men and for women. The gradient was present in the population as a whole, and within families. A sibling with a higher education than their co-siblings will on average have lower mortality than their less educated co-siblings. However, the magnitude of the difference between the population-based and family-based estimates depended on the cause of death. We observed substantially weaker estimates in the family-based than in the population-based analyses for mortality from heart diseases, lung cancer, respiratory diseases, and diabetes. For suicide, the education-mortality association was stronger within families than it was in the population-based analyses, and this was also the case for other mental disorders among women. For these causes of death, we believe that the stronger within- than between-family association can be attributed to the influence of psychiatric morbidity on educational outcomes. This is to be expected because severe psychiatric conditions that eventually lead to suicide or a death from other mental disorders are likely to have affected educational outcomes. In the context of the family-based analyses, psychiatric conditions can thus be described as a cause of discordance of education and a cause of discordance in suicide and other mental disorders. This is an example of the type of non-shared confounding that can be amplified by family-based designs.

The degree of the influence from shared family factors on the education-mortality association further varied across social classes; there were only very marginal changes between the population and family-based educational mortality gradient for individuals from non-

Table 3
All-cause mortality. Hazard Ratios (HR) and 95% Confidence Intervals (95% CI)* for levels of education (and for education treated as a continuous variable) for men and women born 1934–1959. All ages pooled and three age groups separately.

		Men				Women			
		Population-based		Family-based		Population-based		Family-based	
	Education	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
All ages	Basic	2.10	2.05,2.15	1.91	1.84,1.99	1.92	1.86,1.98	1.63	1.56,1.70
	Upper sec < 3y	1.83	1.79,1.88	1.70	1.64,1.78	1.50	1.45,1.54	1.37	1.31,1.43
	Upper sec ≥3y	1.37	1.34,1.41	1.34	1.28,1.40	1.30	1.25,1.36	1.30	1.22,1.38
	Tertiary < 3y	1.18	1.15,1.22	1.20	1.14,1.26	1.07	1.03,1.11	1.08	1.03,1.14
	Tertiary ≥3y	1 (ref)		1 (ref)		1 (ref)		1 (ref)	
	Cont. education	1.21	1.21,1.22	1.18	1.17,1.19	1.19	1.18,1.20	1.13	1.12,1.14
	% red in ln(HR) cont. education			*15%*				*28%*	
Age group:									
31–49	Basic	2.76	2.56,2.98	2.29	2.02,2.60	2.13	1.95,2.33	1.65	1.44,1.90
	Upper sec < 3y	2.27	2.10,2.45	1.99	1.76,2.25	1.44	1.32,1.57	1.24	1.08,1.41
	Upper sec ≥3y	1.52	1.39,1.67	1.45	1.26,1.67	1.21	1.07,1.37	1.30	1.10,1.54
	Tertiary < 3y	1.14	1.03,1.27	1.12	0.96,1.30	0.99	0.89,1.10	0.97	0.84,1.13
	Tertiary ≥3y	1 (ref)		1 (ref)		1 (ref)		1 (ref)	
	Cont. education	1.31	1.29,1.33	1.25	1.21,1.28	1.23	1.21,1.26	1.14	1.11,1.18
	% red in ln(HR) cont. education			*19%*				*35%*	
50–64	Basic	2.20	2.13,2.27	1.97	1.88,2.07	1.94	1.87,2.01	1.65	1.56,1.74
	Upper sec < 3y	1.89	1.83,1.95	1.75	1.66,1.84	1.50	1.44,1.56	1.39	1.32,1.47
	Upper sec ≥3y	1.40	1.35,1.45	1.35	1.28,1.43	1.32	1.25,1.39	1.27	1.18,1.37
	Tertiary < 3y	1.17	1.12,1.22	1.22	1.15,1.30	1.07	1.03,1.13	1.11	1.04,1.18
	Tertiary ≥3y	1 (ref)		1 (ref)		1 (ref)		1 (ref)	
	Cont. education	1.23	1.22,1.24	1.19	1.17,1.20	1.19	1.18,1.20	1.13	1.12,1.15
	% red in ln(HR) cont. education			*17%*				*28%*	
65–78	Basic	1.79	1.72,1.86	1.62	1.49,1.76	1.87	1.78,1.97	1.59	1.44,1.74
	Upper sec < 3y	1.62	1.55,1.69	1.48	1.35,1.61	1.52	1.45,1.61	1.38	1.25,1.51
	Upper sec ≥3y	1.28	1.22,1.34	1.23	1.13,1.35	1.30	1.20,1.41	1.36	1.18,1.56
	Tertiary < 3y	1.25	1.19,1.32	1.20	1.08,1.33	1.09	1.02,1.17	1.05	0.94,1.18
	Tertiary ≥3y	1 (ref)		1 (ref)		1 (ref)		1 (ref)	
	Cont. education	1.15	1.14,1.16	1.13	1.11,1.15	1.18	1.17,1.19	1.13	1.11,1.15
	% red in ln(HR) cont. education			*17%*				*27%*	

* based on standard errors adjusted for family clustering.

manual social classes, but there were clear reductions in the other social classes. The attenuation in the education-mortality association when controlling for shared family factors was more substantial for women, which may suggest that the family influence was generally greater for women than for men. However, it is difficult to say whether this is because the early family environment has more lasting implications for women's educational attainment and health in general. This study examines the relative gradients in mortality. Women have lower mortality than men, and similar changes in absolute risk attributable to confounding in men and women would attenuate the relative gradient more strongly among women. We also note that the education gradient in suicide increased more for women than for men in the family-based analyses, which may point towards the comparatively high impact of psychiatric disease on women's educational attainments. However, our analyses could not disentangle these various explanations.

Studies from other Nordic countries have similarly reported a weakening of the educational gradient in all-cause mortality when adjusting for conditions shared between siblings (Elo, Martikainen & Myrskylä, 2014; Naess et al., 2012, Sondergaard et al., 2012). Our all-cause mortality results for Sweden are in line with these other Nordic studies. Interestingly, however, a recent Swedish twin study suggests that family environment and genetic factors have a very limited impact on the education-mortality link (Lundborg, Lyttkens & Nystedt, 2016), while our sibling design revealed a much greater attenuation of the association. An important strength of this twin study is the inclusion of child health and cognitive ability proxies, which we missed.

The unobserved and observed influence on the educational gradient in cause-specific mortality has been less studied, but clear reductions have previously been observed for lung cancer accidents, and alcohol-related deaths (Elo, Martikainen & Myrskylä, 2014). To our knowledge, the causes of death where we found an increase in the educational gradient within siblings (such as suicide), have not been studied before. The association between birth order and suicide might potentially influence our results, as it has been shown that younger siblings are more likely to commit suicide than older siblings (Rostila, Saarela & Kawachi, 2014). However, adjustment for birth order did not alter the education-suicide association within siblings.

The cause-specific patterns of attenuation might be explained by the differences in the intermediary causal pathways that were involved in the different causes of death. Perhaps the clearest example is that of lung cancer, for which the causal intermediary is well known. The pattern of attenuation is consistent with the explanation that smoking (initiation and/or cessation) is affected by family of origin above and beyond the social influences on these processes that comes with education. The role of smoking in explaining educational differences in mortality illustrates the complexities of causal explanation from observational studies: Smoking exposure is often taken to be a mediator between education and mortality (Mackenbach, 2011), but it is also a part of a confounding pathway that runs from the family of origin to mortality (Gilman et al., 2008). In fact, family influences on smoking are likely to be a contributor to a number of the causes for which we find a weakened association between siblings.

Childhood social class is associated with mortality with low risks for people with non-manual and farmer's backgrounds, and it is higher for manual and self-employed (not in Tables). However, this association is greatly weakened when individual education is controlled for. In

Table 4
Cause-specific mortality. Hazard Ratios (HRs) of mortality for levels of education and % reduction in ln(HR) for continuous education between the population-based and the family-based models. 95% confidence interval in parentheses for continuous education (omitted for education levels due to space restrictions).

		MEN							WOMEN						
		Basic	Upper sec < 3y	Upper sec ≥3y	Tertiary < 3y	Tertiary ≥3y	Continuous education	*% reduction*	Basic	Upper sec < 3y	Upper sec ≥3y	Tertiary < 3y	Tertiary ≥3y	Continuous education	*% reduction*
All causes (167,932 deaths)	Population-based	2.10	1.83	1.37	1.18	ref	1.21 (1.21–1.22)		1.92	1.50	1.30	1.07	ref	1.19 (1.18–1.20)	
	Family-based	1.91	1.70	1.34	1.20	ref	1.18 (1.17–1.19)	*15*	1.63	1.37	1.30	1.08	ref	1.13 (1.12–1.14)	*28*
IHD (24,744 deaths)	Population-based	2.76	2.31	1.62	1.28	ref	1.29 (1.28–1.31)		4.57	3.05	2.03	1.33	ref	1.49 (1.45–1.52)	
	Family-based	2.14	1.88	1.41	1.28	ref	1.21 (1.18–1.23)	*27*	3.34	2.44	1.86	1.16	ref	1.38 (1.33–1.43)	*19*
Lung cancer (13,831 deaths)	Population-based	2.66	2.20	1.58	1.22	ref	1.28 (1.26–1.31)		2.71	2.24	1.78	1.27	ref	1.28 (1.25–1.31)	
	Family-based	2.13	1.80	1.42	1.11	ref	1.22 (1.18–1.26)	*21*	1.79	1.79	1.47	1.21	ref	1.14 (1.11–1.18)	*46*
Respiratory (6,998 deaths)	Population-based	3.42	2.66	1.68	1.26	ref	1.38 (1.34–1.42)		4.85	2.92	1.92	1.34	ref	1.53 (1.47–1.58)	
	Family-based	2.54	2.17	1.40	1.05	ref	1.30 (1.23–1.36)	*19*	3.36	2.16	1.67	1.01	ref	1.42 (1.35–1.51)	*16*
Diabetes (3,343 deaths)	Population-based	3.19	2.69	1.66	1.27	ref	1.35 (1.30–1.40)		5.15	2.94	1.85	1.09	ref	1.60 (1.50–1.71)	
	Family-based	2.35	2.10	1.36	0.89	Ref	1.29 (1.21–1.38)	*15*	3.00	2.05	1.69	1.13	ref	1.35 (1.24–1.47)	*36*
Alcohol-related (6,423 deaths)	Population-based	3.95	3.57	2.01	1.41	ref	1.40 (1.37–1.43)		3.62	2.45	2.40	1.21	ref	1.39 (1.32–1.45)	
	Family-based	3.48	3.16	1.77	1.48	ref	1.35 (1.29–1.42)	*10*	3.24	2.62	2.50	1.57	ref	1.28 (1.20–1.37)	*24*
Other mental (1,306 deaths)	Population-based	3.56	3.16	1.58	1.48	ref	1.38 (1.30–1.46)		2.47	1.59	1.01	0.77	ref	1.35 (1.24–1.47)	
	Family-based	3.78	4.01	2.16	1.84	ref	1.34 (1.20–1.49)	*9*	3.77	2.43	1.29	1.15	ref	1.43 (1.24–1.65)	*-19*
Suicide (6,933 deaths)	Population-based	1.73	1.59	1.09	1.13	ref	1.16 (1.14–1.19)		1.16	1.13	1.06	1.05	ref	1.04 (1.00–1.07)	
	Family-based	2.03	1.71	1.25	1.36	ref	1.18 (1.14–1.23)	*-12*	1.39	1.38	1.45	1.30	ref	1.06 (1.01–1.12)	*-60*
Other external (9,917 deaths)	Population-based	2.52	2.38	1.48	1.25	ref	1.27 (1.25–1.29)		1.73	1.29	1.08	0.99	ref	1.17 (1.13–1.20)	
	Family-based	2.41	2.37	1.53	1.33	ref	1.24 (1.20–1.28)	*10*	1.66	1.28	0.99	1.03	ref	1.15 (1.09–1.20)	*12*

Table 5
All-cause mortality. Hazard Ratios (HR) and 95% Confidence Intervals (95% CI)* for levels of education for men and women born *1944–1959*. Separate models by childhood social class (manual, non-manual, self-employed, farmers, no information/parents' not working).

		Men				Women			
		Population-based		Family-based		Population-based		Family-based	
	Education	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
All born 1944-59	Basic	2.43	2.34,2.51	2.12	1.98,2.27	2.15	2.06,2.25	1.79	1.66,1.93
(N=1153730)	Upper sec < 3y	2.04	1.96,2.11	1.87	1.74,1.99	1.54	1.48,1.61	1.41	1.31,1.52
	Upper sec ≥3y	1.46	1.40,1.53	1.44	1.34,1.55	1.28	1.21,1.35	1.27	1.16,1.39
	Tertiary < 3y	1.17	1.12,1.23	1.21	1.12,1.32	1.08	1.03,1.14	1.12	1.04,1.22
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.26	1.25,1.27	1.21	1.19,1.23	1.22	1.21,1.24	1.16	1.14,1.17
	% red in ln(HR) cont. education			*19%*				*29%*	
Manual family background									
(N=544566)	Basic	2.38	2.22,2.54	1.94	1.72,2.18	2.10	1.94,2.27	1.81	1.58,2.06
	Upper sec < 3y	2.02	1.89,2.16	1.77	1.57,1.99	1.52	1.40,1.65	1.41	1.24,1.61
	Upper sec ≥3y	1.40	1.30,1.51	1.29	1.13,1.46	1.23	1.11,1.36	1.22	1.04,1.43
	Tertiary < 3y	1.16	1.06,1.26	1.15	1.00,1.32	1.06	0.97,1.17	1.16	1.00,1.34
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.26	1.25,1.28	1.19	1.16,1.22	1.24	1.22,1.26	1.16	1.14,1.19
	% red in ln(HR) cont. education			*25%*				*29%*	
Non-manual family background	Basic	2.82	2.65,2.99	2.66	2.35,3.01	2.17	2.00,2.35	2.16	1.87,2.50
(N=296928)	Upper sec < 3y	2.05	1.93,2.17	2.03	1.81,2.27	1.57	1.46,1.68	1.60	1.41,1.81
	Upper sec ≥3y	1.55	1.46,1.66	1.69	1.51,1.91	1.37	1.25,1.50	1.54	1.32,1.79
	Tertiary < 3y	1.20	1.12,1.30	1.34	1.18,1.52	1.08	1.00,1.17	1.15	1.01,1.31
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.30	1.28,1.31	1.27	1.23,1.31	1.20	1.18,1.23	1.20	1.16,1.24
	% red in ln(HR) cont. education			*8%*				*2%*	
Self-employed family background	Basic	1.98	1.76,2.22	1.68	1.34,2.11	2.16	1.87,2.49	1.73	1.36,2.20
(N=106130)	Upper sec < 3y	1.75	1.55,1.97	1.51	1.21,1.89	1.48	1.29,1.70	1.33	1.05,1.67
	Upper sec ≥3y	1.35	1.19,1.55	1.32	1.03,1.68	1.26	1.05,1.51	1.14	0.85,1.52
	Tertiary < 3y	1.04	0.89,1.21	0.96	0.74,1.25	1.02	0.87,1.20	1.03	0.80,1.34
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.20	1.18,1.23	1.16	1.10,1.22	1.23	1.19,1.27	1.15	1.09,1.22
	% red in ln(HR) cont. education			*22%*				*30%*	
Farmer family background	Basic	1.87	1.64,2.13	1.60	1.26,2.04	1.87	1.62,2.16	1.44	1.14,1.82
(N=132762)	Upper sec < 3y	1.51	1.32,1.73	1.33	1.05,1.69	1.38	1.21,1.59	1.25	1.01,1.55
	Upper sec ≥3y	1.20	1.02,1.40	1.09	0.83,1.42	1.13	0.93,1.38	0.97	0.73,1.30
	Tertiary < 3y	1.07	0.90,1.26	1.04	0.78,1.38	1.22	1.04,1.44	1.16	0.91,1.48
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.19	1.16,1.23	1.15	1.09,1.20	1.15	1.12,1.19	1.08	1.03,1.14
	% red in ln(HR) cont. education			*23%*				*44%*	
No information on parents' occupation	Basic	2.73	2.34,3.19	1.88	1.38,2.57	2.24	1.86,2.69	1.30	0.90,1.88
(N=73344)	Upper sec < 3y	2.38	2.04,2.79	1.67	1.22,2.27	1.64	1.36,1.97	1.02	0.71,1.46
	Upper sec ≥3y	1.54	1.29,1.85	1.34	0.94,1.89	1.20	0.93,1.54	0.95	0.60,1.51
	Tertiary < 3y	1.15	0.94,1.42	1.09	0.75,1.60	0.98	0.78,1.24	0.66	0.43,1.01
	Tertiary ≥3y	1 (ref)		1		1		1	
	Cont. education	1.30	1.26,1.34	1.18	1.11,1.26	1.27	1.22,1.32	1.15	1.07,1.24
	% red in ln(HR) cont. education			*37%*				*40%*	

* based on standard errors adjusted for family clustering

contrast, the educational gradient in mortality is rather unaffected by controlling for childhood socioeconomic position (a similar result can be found in Erikson (2001)). In this paper, we observed that for non-manual social class families, the difference between the population- and family-based estimates were small. Thus, factors shared between siblings in non-manual families did not explain the education-mortality association to any considerable extent. There might be several explanations for this, although it is far from clear why the family environment would be more important and/or limited in one social class but not another. However, the selective elements of a low (high) education may differ by social origin, and various individual traits may be more decisive for educational attainments for children with a working-class background for whom higher education is less likely. Given school performance, children from advantaged origins more often continue to higher educational levels (Erikson & Rudolphi, 2010). In addition, even when their cognitive ability is relatively low, individuals from advantaged backgrounds more often tend to continue to higher levels of education in comparison with individuals from the least advantaged parental backgrounds (Bukodi, Erikson & Goldthorpe, 2014). It is thus possible that there is a comparatively strong negative selection into lower educational levels within these advantaged families where higher education of the offspring is both more likely and expected. Such a selection would increase the family-based estimates and thus work in the opposite direction of familial confounding.

Historical and social contexts are important to the education-mortality association (Hayward, Hummer & Sasson, 2015), and the family influence on this relationship may also be context dependent. As a consequence, the interplay between family, education, and health outcomes may change over time. However, the present study averages

across a wide range of birth cohorts, age groups, and time periods. During the twentieth century, the educational system became more comprehensive, and upper-secondary and tertiary education expanded substantially (Jonsson and Mills, 1993). Thus, all else equal, a younger sibling is more likely to attain a higher educational level than an older sibling. Assuming that a causal effect of education on health is present, changes in the educational system may generate a difference in siblings' life expectancy. However, if the education-mortality association is mainly confounded by other factors, e.g., familial circumstances, health and mortality differences between siblings would not be affected by educational expansion. Further, there is a negative effect of birth order on educational attainment but the educational expansion may counteract this effect (cf. Barclay, 2015), particularly in large families in which the age gap between the first and last child is substantive. As a robustness check, we tested whether our results were substantially changed when we restricted our all-cause mortality analyses to sibling groups with a maximum age gap of four years. The results of these restricted analyses are nearly identical to the estimates based on all sibling groups, which suggests that a large age gap between some sibling groups is not driving the results.

We have briefly outlined the statistical problems with interpreting the attenuation in education-mortality association between the population-based and family-based estimates. Because of the problem of the non-collapsibility of the HR, the associations within families may inflate even in the absence of confounding. The most attractive solution to this problem is Aalen's additive survival model where the subgroup and population-averaged associations are the same (Martinussen & Vansteelandt, 2013), but work is needed to implement a solution in software that works with covariates with very high degrees of freedom such as family of origin in the present study.

We want to stress that the educational gradient in mortality between siblings must be interpreted with caution. Despite earlier enthusiasm (McGue, Osler & Christensen, 2010; Rutter, 2007), it is clear that sibling comparisons do not mimic experiments that are characterized by exogenous variation in exposure. Sibling comparisons are a form of imperfect matching, and they come at the dual loss of precision (which may be less important in the context of this study) and bias away from the causal effect of education. One source of bias might arise from the fact that families in which all siblings have the same education are informative for the population as a whole, but uninformative for the between sibling analyses. For example, it is an open (but unanswerable) question if the unobservable educational gradient in siblings in families who are 'doomed' to low education can be inferred from the educational gradient in families in which the siblings actually have different educations. Using the association between the sexes of siblings as an example, we have also demonstrated empirically that restriction to discordant pairs will induce non-causal association that will bias the estimates away from the causal effect of education. This should be taken into account when interpreting the findings for causes of death, where the underlying conditions are likely to affect educational success and mortality, for example schizophrenia in the analyses of education and risk of suicide (Agerbo, Byrne, Eaton & Mortensen, 2004). Because the familial clustering of education is very strong, the potential for bias is considerable: Having an education that is different from one's siblings is likely to have a reason. It may be possible to obtain an idea of the directionality of the bias. In general, we would expect the bias induced by design to work to increase the educational gradient in mortality because we believe that the majority of factors that limit educational success are likely to be detrimental to health if they have any effect on health at all. In theory one might imagine other scenarios: Devoting one's time to sport activities might impede the chances of obtaining a long education while having a positive effect on health, or, for example, a sibling may have been encouraged to obtain a long education because it was felt that he or she was too frail for a manual job. However, we believe that the net result across families is likely to result in bias away from the null, but this assumption is contingent on the context-specific distribution of all contributory factors according to education and familial background, which is obviously not something that is easily documented.

In conclusion, our findings reaffirm that family of origin explains a part of the educational gradient in mortality. For some causes of disease, it seems likely that a causal effect of education may be an important contributor to the observed association. Strong educational gradients exist within families particularly for causes of death where the sibling design itself introduces confounding. Given the limitation of the design, the findings should be interpreted with caution, particularly as it is difficult to know how to balance the competing explanations of confounding and causation.

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Appendix A. Supplementary material

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