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Human Capital and Longevity. Evidence from 50,000 Twins*

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Abstract

Why do well-educated people live longer? We use unique and high-quality data on about 50,000 monozygotic and same-sex dizygotic twins, born between 1886-1958, to address this question. We demonstrate a positive and statistically significant relation between years of schooling and longevity, which remains when we exploit the twin design. This result is robust to controlling for specific within-twin-pair differences in early life factors, such as birth weight and proxies for ability and health in the form of body height, as well as to restricting the sample to monozygotic twins.

JEL Classification: I12, I11, J14, J12, C41

Keywords: death, longevity, education, endogeneity, stratified partial likelihood, duration analysis; twins

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I Introduction

Well-educated people live longer. But why is it so? Despite hundreds of empirical studies on the topic, the underlying mechanisms behind the correlation between schooling and longevity remain poorly understood. This is unfortunate, since deeper understanding of the association is crucial for proper assessment of policy goals concerning further advancement of life expectancies and reducing socioeconomic disparities in longevity.

In this paper, we consider the role played by unobserved endowments for the relationship between schooling and mortality. Does the relationship merely reflect that well-educated persons are equipped with certain innate endowments that also make them live longer? This does not seem unreasonable, given the evidence suggesting that both schooling and mortality are partly genetically determined (e.g. Plug 2004; Ljungqvist et al. 1998; Hjelmborg et al. 2006). Or does the relationship reflect that growing up under poor environmental conditions leads both to less schooling and a shorter life? This would also seem plausible, given the growing literature that provides convincing evidence on the importance of early life conditions for a range of adult outcomes (see Currie 2009 for a recent overview). Or, could it be that education really affects life expectancy, e.g. via increased ability to apprehend and process health related information and engagement in health related activities? In such a case, the returns to education may extend way beyond the returns that one observes on the labour market.

We test for the influence of unobserved endowments by using a unique data set on a unusually large sample of 50,000 monozygotic and dizygotic twins who were born in Sweden between 1886-1958. A unique feature of the data is that we are able to follow the twins through registers until 2009, which means that we are able to observe complete life-spans *at the individual level* for a large fraction of the sample. We advance the recent literature on the topic by applying stratified partial likelihood duration models to these data, which allow us to cancel out to varying degrees the influence of both shared genetical and early life environmental factors. The large sample size also works to our advantage, as the twin design effectively obtains identification only from twin pairs who differ in schooling.

Our results suggest that schooling do matter for longevity. We find a strong and positive association between education and longevity, both among males and females. When we exploit the twin feature of our data, we obtain similar estimates that are only slightly reduced in magnitude. Our estimates are large in magnitude and imply that being a highly educated person, meaning having at least 12 years of schooling, is associated with 2.5-3 years additional life expectancy at age 60 compared to being a low educated person. This estimate is surprisingly similar across gender and across time. Further, limiting the studied sample to monozygotic twins affects the estimates only very mildly.

Taken together, our results suggest that the relationship between education and mortality is not primarily generated through the influence of shared genetic and/or environmental endowments affecting both education and mortality. They also support the results obtained by Lleras-Muney (2005), using an IV-design, in a US context and imply that the value of schooling may

be much greater than what one would conclude from only considering labour market outcomes.

In our analyses, a number of issues arise. The first one is that even though MZ twins share genes, they may face slightly different environments, even in the womb. This may generate differences in traits such as birth weight, which has been shown to affect later educational attainment (Black et al 2005). If the same trait also affects mortality, our twin-based estimates would be confounded by environmentally induced birth weight differences. Since we observe data on birth weight for a relatively large subsample (3.500 twin pairs), we are able to test for this. Our results confirm earlier findings that birth weight indeed predicts schooling differences within twin pairs. However, our twin fixed effects estimates of the relationship between schooling and mortality are robust to controlling for birth weight differences. Moreover, birth weight does not predict mortality within twin pairs.

Twins may also differ in other traits, such as ability and post-natal health. In order to test if such factors are biasing our results, we use information on a variable known to be strongly correlated with both childhood IQ and early life health; height (see for instance Case & Paxson 2008). Although our results suggest that height is a significant predictor of within-twin-pair differences in education, the inclusion of height in the regressions does not affect our main results of a significant relation between schooling and mortality.

A second concern is that inclusion in the sample is conditional on survival to the years in which the different surveys were conducted. This means that we for some of the older cohorts are likely to observe a sample of unusally healthy twins compared to the more recent twin cohorts, where survival to a particular survey would be more common. One may therefore worry that external validity is threatened for our results based on the older cohorts. Moreover, and *if* education does have a causal effect on survival, a low-educated individual in the older cohorts who still manages to survive to an equally high age as his more highly educated cohort peer, and thus to the point of being included in the sample, likely has better on average unobserved underlying health, partly due to factors such as genes and early life conditions.[1] This would induce a negative correlation between education and certain unobserved traits, such as genes and early life health, leading to downward biased estimates. In the twin design, however, we argue that many such traits are shared by twins and therefore cancels out, which we believe reduces the importance of the selection problem.

Third, the incentive to invest in schooling may be affected by expected longevity, thus inducing a reverse causality problem. Twins, however, share any signalling value that may be reaped from parental or more distant ancestral longevity. It is also comforting that neither birth weight, nor height, being possible early-life markers of expected longevity, predict mortality differences within twin pairs. Nevertheless, accidents and other misfortunes that may affect survival propensities may naturally strike a twin pair asymmetrically. Particularly if they occur in young age, in advance of when final education is determined, and are not manifested by height (which we observe), such

[1] The argument is that the low-educated individual who survived to an old age must have some offsetting traits in order to compensate for his lower education.

asymmetries are bound to affect our results. In order to reduce the risk of our estimates being heavily influenced by the most severe adverse health shocks occurring rather early in life we have limited the sample to those twin pairs where both twins have survived at least to an age of 40.

The paper is organized as follows. In Section 2, we review the literature that is connected to our study, notably the literature on the causal effect of education on health. Section 3 describes our data and the constructed variables, while Section 5 provides some descriptive patterns on education and mortality. Section 6 discusses the econometric methods we use and their underlying assumptions. Section 7 presents our main results and our sensitivity analyses. Section 8 quantifies the results from section 7 in terms of expected survival gains due to education. Section 8 concludes.

II Previous literature

Economists have recently considered the relationship between education and a range of non-monetary outcomes such as health, criminal behaviour, marriage, and political participation.[2] This recent evidence points to the importance of education for a much wider range of outcomes than those observed on the labour market and thus suggests that education may be a crucial component in human development. The evidence thus suggests that the value of educational investments in society may be greater that previously thought.

The idea that human capital may affect health, and thus longevity, was formalized by Michael Grossman already in the 70s (Grossman 1972). Building on Gary Becker's human capital model, Grossman introduced education as a crucial component in individuals' health production function. In the model, education enhances the efficiency of health production, so that well-educated people are able to squeeze out a greater health output from a given health input. A related hypothesis was suggested by Rosenzweig and Schultz (1982), where educated people are instead able to choose a better mix of health inputs in the production of health.

Not everybody was convinced, however, by the Grossman model's treatment of education as exogenously given. Victor Fuchs, for instance, questioned whether there exists any causal relationship between education and health at all (Fuchs 1983). Instead, he argued that education and health may be related only through unobserved "third factors", such as time preferences. People who are future-oriented ought to invest more in both education and health, since the benefits of both types of investments are of long-run character. This would induce a positive correlation between education and longevity, without any causal relation between the two. Besides time preferences, it seems plausible that genes and early life environment also could constitute such "third factors". In fact, since a substantial part of the observed variation in education on the one hand, and mortality on the other, among humans is belived to be of genetic nature, it does not seem farfetched to assume that there could be some genetic "overlap" between the two.

[2] For a recent overview of this literature, see Lochner (2011).

Recent evidence also points to the importance of early life conditions, including childhood health, for the production of education capital (Heckman 2007). It is straightforward to conceptualise an education production function were poor health early in life interfers with learning and is also associated with health later in life. Some evidence suggests, for instance, that low birth weight is associated with less schooling (Behrman & Rosenzweig 2004; Black et al. 2007; Oreopoulos et al. 2008; Royer 2009).[3] Health during childhood has also been found to be of importance for later life outcomes (Currie et al. 2010).

Other mechanisms may be at work as well. Expected longevity may affect the incentives to invest in education, since lower mortality means more years to reap the benefits of schooling. Such a mechanism is suggested in the study by Jayachandra and Lleras-Muney (2009), where the large declindes in maternal mortality in Sri Lanka was associated with increased schooling among women.

Few studies have tried to distinguish between the different mechanisms that may induce a positive correlation between education and mortality. The first paper to address the causal effect of education on mortality appears to be Lleras-Muney (2005), who exploited changes in compulsory schooling policies and child labor laws at the state-level across the United States during the first half of the 20th century. The results obtained in the article were dramatic; an extra year of schooling was estimated to reduce the 10-year mortality rate by more than 30 percent, thus supporting the predictions obtained from the Grossman model.

In a French context, Albouy and Lequien (2009) used a regression discontinuity approch to estimate the causal effect of education on mortality. They exploited two compulory schooling reforms and used year-of-birth differences across the tresholds of the reforms to estimate the effect. In contrast to Lleras-Muney, they find no effect of education on mortality. The difference may reflect differences in the institutional context, where France in contrast to the US has universal health coverage.

There are also a few working papers on the topic. Cipollone and Rosolia (2006) exploits a natural experiments in Italy where an earthquake made some people exempted from military service in order to identify the causal effect of education on mortality. The results did not show any significant effect. Using a reform in compulsory schooling laws in the Netherlands, van Kippersluis et al (2009), however, found a sigificant effect of education on mortality. Clark and Royer (2010), exploiting British schooling reforms, found no effect. Meghir et al. (2011) exploited the Swedish schooling reform during the 50s and 60s but found no effect of education on mortality.

The only twin study to study the relationship between education and mortality that we are aware is the article by Behrman et al. (2011), examining the effect of schooling on hospitalizations and mortality among Danish twins born 1921-1950. Their mortality outcome variable is whether or not a twin died before the year 2003. They find that the negative association between education, on the one hand, and hospitalizations and mortality, on the other hand, disappears when exploiting within-twin-pair variation among monozygotic twins inclining them to suggest that the education has no or little causal

[3] Other twin studies, however, find no significant association (Bonjour et al. 2003; Miller et al. 2005; Petersen et al 2006).

effect on health and mortality.

III Data

Our data originates from the Swedish twin registry, held by the Karolinska Insitutet in Stockholm. The registry is the world's largest twin registry and covers information on about 170,000 twins. We use data on the twins born between 1886 and 1958, who have surpassed 40 years of age, which gives us access to about 50,000 same-sex twins, of which 18,000 are monozygotic twins. The twin registry assigns zygosity based on questions about intrapair similarities in childhood. Several validations with DNA have shown this method as having 98% or higher accuracy (Lichtenstein et al., 2002).

Our sample consists of two different cohorts, each of which differs in the extent of data collected and the type of data. The first cohort is labeled the "old cohort" and includes twins born 1886-1925. In order to assemble data for these cohorts, the twin registry contacted all parishes concerning multiple births during the period 1886-1925. All twin births were then followed up in 1959, where the status of the twin pair was recorded. During 1960-61 the first questionnaire was then send out to all same-sexed twin pairs that were still alive and were living in Sweden. Additional questionnaires were then send out in 1963, 1967, and 1970.

The second cohort, labeled the "middle cohort", was compiled in 1970 and included twins born between 1926 and 1958. A questionnaire, similar to those send to the older cohorts, was send out in 1972-73. During the years 1998-2002, a new survey, the SALT survey, was conducted, that followed up on all twins born between 1886-1958.

The method used to collect the twin data gives rise to some selection issues. These are most important for the older cohorts, since the group of complete twin pairs that survived until the first survey in 1961 was probably healthier than the average twin during that period. Note that both twins needed to have survived to 1961 and agreed to answer the survey, in order to be included in the data. In the Methods section, we discuss the potential bias that including these "survivors" may generate. Under reasonable assumptions, we will argue that our twin design to a large extent deals with this selection problem and that any remaining selection bias gives rise to downward biased estimates.

Selection issues are likely to be less serious for the middle cohort, i.e. those born between 1926-1958, since average life expectancy had increased and most twins would have survived until the first survey in 1972-73.

Since the data contains personal identifiers, we have been able to link the data to various administrative registers. Key among these is the causes-of-death register, held by the National Board of Welfare. This register records the date and cause of death for all individuals that die in Sweden and is considered to be very accurate. Furthermore, the register is updated frequently and we have access to deaths that occur as late as 2009. Since we information from the causes-of-death register from 1961-2009 on death dates, we are able to observe all deaths that took place during this period in our twin sample, since all twins were alive in 1961.

Our data on education originates from several sources. For twins alive in 1990, we have access to register-based data on education from 1990 and 2007. The information in these registers is reported from the various educational institutions directly to statistics Sweden. This also means that measurement errors should be small. Besides these register data, several of the surveys contains self-reported education information. Such data is for instance included in the first survey that was conducted in 1961, which means that we do have access to self-reported education information for the oldest cohort. In order to get even more detailed data on education for the older cohorts, we have also linked the data to census information from 1960 and 1970. These are self-reported as well, but contains a higher level of detail compared to the survey in 1961. Our main education measure will be years of schooling but we will consider educational degrees as well.

Birth weight information was collected from the SALT-survey. It is self-reported, since there is no register information on birth weight for cohorts born prior to 1972. This also means that birth weight is only available for the younger cohorts that was still alive between 1998-2002. Information on height, however, which we use as a proxy for cognitive ability, is available for the larger part of the sample. Questions about height were asked at multiple waves of the twin surveys.

Table 1 shows some descriptive statistics on some of the key variables in our analyses. The mean number of years of schooling for male MZ and DZ twins is 10.4, whereas the corresponding number of females is 10.2. Numbers are slightly higher in the sample of MZ compared with DZ twins. The table also shows that we observe a relatively large number of deaths in our data. Among male MZ and DZ twins, 41 percent is observed to die and 38 percent among females. For MZ twins, the figures are slightly lower; 40 percent among males and 35 among females. This is a unique feature of the data that comes from having access to old cohorts. The mean death age for those with observed spells is 73.8 among MZ and DZ males and 77.5 among females. Among the sample of MZ twins, the mean death age is just slightly higher.

Since the twin design effectively exploits variation in schooling within twin pairs, it is crucial to have sufficient variation within twin pairs. Here, our large sample comes in handy. Table 2 shows the distribution of differences in schooling within MZ and DZ twin pairs. The figures has been rounded to the nearest integer so if, for instance, the difference is less than half a year, it is presented as 0. In the pooled sample of MZ and DZ twins, 50 percent of the male twin pairs show no variation in schooling. Thus, almost half of the twin pairs show a difference in schooling, commonly between one and four years. Among female MZ and DZ twins, 48 percent of the pairs differ in schooling, again guaranteeing sufficient variation. Although the fraction of pairs that differ in schooling in the MZ sample is somewhat lower, there is still a substantial fraction of pairs who differ. Among males, 43 percent of the pairs differ in the number of years of schooling, whereas the corresponding figure for MZ females is 44 percent. The lower fraction of MZ twins who differ is as expected, since MZ twins share genes and thus are more similarly endowed with characteristics influencing schooling decisions and success.[4]

[4]Our figures on the fraction of pairs who differ in schooling corresponds relatively well

IV Empirical model

Since the dependent variable measures the duration until death and there is prominent censoring among the younger cohorts, we will use duration models in the empirical analysis.[5] We will focus on partial likelihood models, where we allow for each twin-pair to have a separate baseline hazard. Such models are labelled stratified partial likelihood models and allow us to eliminate the influence of omitted variables at the twin-pair level.[6] To see how, consider first the twin-pair specific baseline hazard:

$$\theta_{0p}(t, \eta_m), \tag{1}$$

where p denotes the p^{th} twin pair and η_j denotes unobserved heterogeneity at the twin-pair level. Assuming proportional hazards, we could then write the hazard rate with twin-pair specific effects as:

$$\theta_p(t; x, \eta_p), \beta) = \theta_0(t, \eta_p) \cdot \theta_1(x; \beta), \tag{2}$$

where x includes explanatory variables, such as years of schooling, and t denotes time. While the baseline hazard θ_0 is an arbitrary function of unobserved individual-specific heterogeneity and duration dependence, θ_1 is a function of observed characteristics x. This approach thus applies the traditional partial likelihood approach to strata, representing units, such as firms, schools, familes, or, as in our case, twin pairs. In our case, the risk sets are replaced by twin–pair-specific risk sets. Since all unobserved twin-pair specific effects are irrelevant in a comparison of individuals belonging to the same twin pair, they therefore cancel out from the expression for the likelihood. We therefore effectively rely on within twin-pair variation in the covariates to identify the regression coeffcients.

Note that all observable covariates that do not vary within twin pairs, such as age and gender, also cancels out in the SPL model. In our main specification, we will only include years of schooling as an explanatory variable. The reasons for not including variables such as marital status, income, or occupation are twofold. First, we want the allow the effect of education on mortality to run through exactly such mechanisms. Controlling for such factors would thus disguise part of the effect. Second, controlling for income, for instance, leads to difficulties in interpretation. The reason is that one is then comparing the effect of education for people with the same income. This could induce a selection problem, since the lower-educated person who is able to reach

with figures from previous studies using a twin design to study the returns to education (see for instance Ashenfelter and Rouse 1998 and Holmlund et al. 2010).

[5] An alternative would have been to run fixed effects regressions on the probability of surviving until certain ages or to study 5 or 10 year survival. We prefer duration models in our main specification, since they better exploit the variation in the data. For 10-year survival, for instance, two related twins who die within the same 10-year period, but at different dates, would not contribute to the identification of the estimates in a fixed-effects model. In our preferred duration model, however, any differences in the death dates would contribute to the identification of the estimates. In order to shed some more light on the magnitude of our estimates we will also exploit fixed-effects models, however.

[6] See Ridder & Tunali (1999) for a thorough exposition of the stratified partial likelihood model.

the same income as a higher educated person likely has higher-than-average ability. In such as case, the estimated effect would be downward biased, since a negative relationship between unobserved ability and schooling would be induced.

Before discussing the assumptions made in the twin-design, we discuss a number of technical issues that arise when applying the stratified partial likelihood estimator. One is due to the fact that we re-group spells from asynchronous to synchronous, using the termiology of Ridder & Tunali (1999). Asynchronous mean spells starting at different points in time, but when applying the stratified partial likelihood estimator one instead order the spells of each cluster from the shortest to the longest and assume them to start at the same point in time. This is not an issue in our case, since the spells of two twins start at the same point in time. Another assumption needed is that there is no causal effect of the death of one twin on the death hazard on the other twin, given x_i and η_j. This a more debatable in our case, since the death of a sibling may affect the health of the other siblings.[7] This would most likely, however, give rise to a downward bias in the estimated effect of education on mortality, since it would induce smaller differences in mortality across twins with different educations.

The SPL estimator controls for the influence of unobserved variables at the twin-pair level, much like the standard fixed effects estimator. While the SPL-estimator removes twin-pair specific unobserved heterogeneity, there may still exist environmentally induced unobserved differences within twin pairs that may relate to differences in both human capital and longevity. One threat to our particular application of the twin design would be the existence of unobserved health differences within twin pairs early in life. As discussed in the Background section, a number of twin studies report a significant effect of birth weight on education, although the evidence is mixed. A significant association between birth weight and education would be a problem for us if there is also a significant association between birth weight and mortality. Here, the evidence is very limited, since such studies put great demands on the data, following people from the "cradle to the grave". We will attempt to deal with this potential threat by running regressions on the subsample on which we have access to measures of birth weight and examine if inclusion of birth weight affect the relationship between education and mortality. As a "by-product" we will also obtain twin estimates of the relationship between birth weight and mortality.

Another potential threat to our twin design is unobserved ability differences within twin pairs. If such differences are related to both schooling and mortality, our estimates would most likely be upward biased. There is however very little evidence on the extent to which twins differ in ability and to what extent such differences predict schooling differences. In a recent study by Sandewall et al. (2010), however, IQ differences within twin pairs are shown to predict schooling differences.[8]

[7] Such patterns have been observed for spouses, see for instance van den Berg et al. (2011).

[8] Note, however, that one can not rule out that such differences in IQ between twins are generated through earlier differences in schooling inputs, such as teachers.

We are not aware, however, of any twin studies on the effect of IQ on mortality, although a large number of epidemiological studies have reported statistical correlations between early life IQ and later life health and mortality. A recent Swedish study report results that may be informative for our purposes. The study was a long-term follow up study of 1,500 children, where each additional year of schooling reduced the mortality hazard by between 9 to 12 percent (Lager et al. 2009). When adding a measure of childhood IQ, however, the relationship between education and mortality was not affected.

Although we cannot observe cognitive ability, or IQ, we can at least control for variables that are known to be strongly correlated with IQ. One such variable is height, which has been shown in Case & Paxson (2008), for instance, to be strongly related to childhood IQ. Admittedly, height is often taken as an indicator of early life health and it can thus be expected to reflect variation in both height and IQ. If the results changes drastically when controlling for this proxy, however, we would be concerned that unobserved differences in cognitive ability are being picked up. We fully acknowledge that this by no means is a perfect solution. Since height is also related to early life health, any change in the results when accounting for height could obviously be interpreted as picking up health differences. However, an *absence* of change in the result could provide some indicative evidence that differences in cognitive abilities play no great role, although we then need to assume that the variation in IQ that is relevant for mortality is picked up by our height measure.

Another issue that aries when interpreting twin-based estimates, is that parents may reinforce or compensate for differences in endowments between twins. In the former case, the importance of such differences would be magnified. Most studies on data from Western countries seem to support the hypothesis that parents try to compensate for ability differences between children (see Almond and Currie (2010) for a recent overview of this literature). To the extent that ability is associated with underlying health – or if parents also compensate by investing more in the education of their less healthy offspring – our estimates of the impact of education on mortality would be downward biased. Without data on parental behaviour, it is obviously impossible to address these concerns, besides trying to control for observable differences that may induce or reflect parental responses, such as birth weight and height.[9]

As mentioned in the data section, the sampling of the twins may introduce some selection bias. Twins born between 1886-1925 have to be alive in 1961 to be included in the sample. For the older cohorts, we are thus likely to observe a sample of unusally healthy individuals. Moreover, it is plausible that that a low-educated individual who is still able to survive to 1961 has better unobserved underlying health on average compared to a high-educated individual. In such a case, a negative correlation between education and certain unobserved traits, such as genes and early life health, would arise, leading to downward bias in the estimate of schooling. Here, the twin design gives us

[9]In Lundborg (2011), the relation between parental investments early in life and the twin's later educational attainment was analyzed. The twin estimates showed a negative relationship between parental time investments and years of schooling. This is consistent with compensating behaviour by parents, where parents invest more in the less able twin in order to reduce differences between their twins.

an advantage, since many such unobserved traits are shared by twins in a given twin pair and therefore cancels out, which reduces the importance of the selection problem.

Certain selection issues may remain, however. For instance, there may still remain some selection within twin pairs, such that the lower educated twin is likely to have better underlying health than his higher educated co-twin in pairs where both survive until a high age, which would push the estimates downwards. Moreover, since survivors can be expected to healthier on average, the effect of education may be smaller for this group, if there is decreasing marginal productivity in the production of longevity with respect to education. Note, however, that these selection issues are much less important for the younger cohorts. We will therefore in the sensitivity analysis analyze to what extent the results change by cohort and by age.

Finally, one need to assume that investments in education do not depend on the future moment of death of the main person, given x_i and η_j. This assumption would fail, for instance, if the main respondent early on in life has some idea about his expected lifelength. If this expectation is low, he or she may then invest less in education and what looks like an effect of education on mortality may then just reflect reverse causality. We expect such anticipation effects to be rather weak, however, since any signalling value that parental health or longevity may have for the twins own expected longevity is shared by the twins. Differences in early life health could lead to differences in lifelength expectations, however. Here, we expect the regressions including controls for height and birth weight to be informative, since variation in these factors could generate differences in expectations about life length. In order to preclude the influence of more severe negative early life health shocks the sample is limited to those twin pairs where both have survived at least to an age of 40.

A Results

We start our analysis by analyzing the relationship between years of schooling and mortality. Later, we will refine the analysis, in order to investigate potential heterogeneity in the impact of education on mortality across genders. Table 3 show the Cox regression results for pooled MZ and DZ twins and MZ twins only, with and without allowing for twin-pair specific baseline hazards. In the models without twin-pair specific baseline hazards, we adjust for gender and birth year.

As shown in the first column of Table 3, the hazard ratio of 0.944 is significant and implies that one additional year of schooling is associated with about a 6 percent lower mortality hazard in our pooled sample of MZ and DZ twins. This is a substantial, although somewhat lower, estimate compared to the estimates obtained in some previous studies using Swedish data, such as Lager et al. (2009).

In column II, we then show the results when allowing for twin-pair specific baseline hazards in our sample of MZ and DZ twins. Here, any unobserved factors that may affect education and mortality that are shared by the twins within a twin pair are cancelled out. Interestingly, the results do not change much and the hazard ratio only slightly decreases to 0.935, implying a some-

what greater effect. It thus appears that unobserved common early life environment and shared genes do not importantly bias the estimates obtained in column I. Admittedly, DZ twins, who constitute the major part of the pooled sample, only share 50 percent of their genes, but the fact that the estimates hardly change when moving to the SPL model talks against any strong influence of genetics in the relationship between education and mortality. This conclusion is supported when we restrict the sample to MZ twins only, as shown in columns III and IV. Here, the results are indeed very similar to the estimates obtained for the sample of MZ and DZ twins.

What else could explain the small difference in results when accounting for all possible twin-pair specific factors? Perhaps the difference is not that small after all since one may argue that the Cox estimates and the SPL estimates are not strictly comparable. The reason is that in the latter case, the estimates are based only on twin pairs who differ in schooling, whereas in the former case both variation between and within twin pairs is used. Clearly, if the twin pairs that differ in schooling are systematically different compared to other twin pairs, the estimates that are based on them may not be generalizable to the full twin population and they have a LATE (Local Average Treatment Effect) flavour. We checked this by also running Cox regressions only on twin pairs that differed in schooling. This generated a hazard ratio of 0.953, which is somewhat larger than the estimate of 0.944 that we obtained for the Cox model on the full population. This also implies that the difference between the Cox and SPL estimates are somewhat larger than what our first results implied, since the estimate shrinks from 0.953 to 0.935.[10] We get a similar pattern when focusing on MZ twins only; here the Cox estimates based on only the twins that differ in schooling generates a hazard ratio of 0.959. Still, the difference in magnitude of the estimates between the Cox and SPL model appers rather small and the main message of the analysis does not change; there remains a statistically significant relationship between schooling and mortality also when accounting for shared genes and environment.

How do our estimates compare with those obtained in some previous studies? Lleras-Muney (2005) also found that her estimates of the effect of schooling on mortality did not change much when accounting for the influence of unobserved characteristics, in her case through the use of instrumental variables. We find it interesting that we get the same result with an alternative research design and one conclusion may be, as also discussed by Lleras-Muney, that education is in fact rather exogenous to future mortality. Our results differ from Behrman et al. (2011), however, where no significant relationship between education and mortality was obtained, although using the twin design and although the analysis was based on data from a neighboring country to Sweden; Denmark. We are not sure what explains the difference between our results but we note that the dependent variable in their case was a dummy for

[10] We also checked whether the twin pairs that differed in schooling also differed from other twin pairs in terms of observable characteristics. The results suggested that differences within twin pairs have become more common over time, as the average birthyear of twin pairs that differed in schooling was 1937 compared to 1927 for twin pairs who did not differ. This difference was statistically significant. We return to the issue of changes in the estimates over cohorts/ages in subsequent sections.

survival until 2003, that their data concerned a narrower time period (1921-1950), and that our sample size is substantially greater.[11] One possibility is that the type of twin pair that differs in schooling, and thus contributes to the identification of the schooling effect, is different between Sweden and Denmark, meaning that the estimates are not strictly comparable.

Before proceeding with gender-specific results, we also want to make sure that our results are not driven by some outliers in the data. As shown by Amin (2011), twin fixed effects estimates are especially sensitive to the influence of a few outliers in the explanatory variable. We therefore re-ran our results first excluding all twin pairs where the twins differed by more than 4 years in terms of schooling. Doing so actually *decreased* the hazard ratio somewhat, to 0.928. Further restricting the sample to pairs with at most 3 years of a difference resultet in a hazard ratio of 0.932. Even when restricting the sample to those with at most a 2 year difference, the estimate did not change to any important extent and remained significant (0.923). These results are of interest also for another reason; it appers reasonable to us that twin pairs who differ less in terms of schooling also differs less in unobserved factors that could potentially bias our results. If these unobserved factors were of great importance, however, we would expect the estimates based on twin pairs close in terms of schooling to be of lower magnitude. This is not what we find, however.[12]

B Results by gender

We next consider the possibility that the estimates differ between males and females and run separate regressions by gender. Given the different roles of males and females in our society, it would not be surprising if the nature of the relationship between mortality and education differs. For example, for males, having a university degree might mean not ending up in manual and accident-prone occupations. For women, the effect during our study period will likely work through the propensity to belong to the labour force. Secondly, for those who would have belonged to the labour force in any case, a higher education might mean that monotonous job with a high risk job injuries are avoided.

In the mid panels of Table 3, we repeat the exercise of the previous section but this time only for males. Allowing for twin-pair specific baseline hazards in the sample of male twins, we now obtain a hazard ratio of 0.942, whereas the corresponding Cox estimate is 0.946. When we restrict the sample to MZ twins only, the SPL estimate only slightly decreases (0.936).

For females, the results are not radically different. In Table 3, the SPL hazard ratio of 0.925 in the pooled sample of female twins is rather close to that of the male twins. Restricting the sample to MZ twins only, and applying the SPL estimator, the hazard ratio increases somewhat to 0.953. The somewhat greater effect of education obtained for males in the SPL model is in line with previous evidence of a somewhat stronger gradient among males, although our

[11] When we restrict our sample to the period 1921-1950, and run our SPL model on MZ and DZ twins, our estimates do not change much, however, and remain significant (0.93).

[12] We also ran regressions excluding people who survived the age of 100 and 90, respectively. We lose about 3,000 individuals by imposing this restriction but the results did hardly change at all.

results suggest that the differences are quite small. The results for females also suggests that unobserved heterogeneity may affect their estimates more, as the estimated effect of education on mortality shrinks more when applying the SPL estimator in the sample of female MZ twins.

C Birth weight, education, and mortality

Having established a set of baseline results, we next turn to the potential threats to our estimates. We start by looking at the role of birth weight differences among twins, where the results could also be of interest in their own right. Since birth weight information is only collected for those who survived to the SALT survey, conducted 1998-2002, the sample size is now reduced from about 50,000 to about 7,000. In order to increase power, we will focus on the pooled sample of MZ and DZ twins, as was also done in several previous studies, such as Black et al. (2006) and Royer (2009).[13]

Since our measure of birth weight is self-reported, and measurement errors therefore a concern, we first check whether there is any evidence that birth weight differences are associated with differences in schooling in our sample. This analysis also serves as a comparison with previous studies and helps to evaluate the credibility of our estimates. As shown in Table 4, we find a significant and positive relationship between birth weight and years of schooling. In a linear OLS model, as shown in column 1, a 100 grams increase in birth weight is associated with 0.014 more years of schooling.[14] When imposing twin fixed effects, the estimate is practically unchanged amounting to 0.013. This estimate corresponds rather closely to the estimate obtained by Royer (2009) for US data, where a 100 gram increase in birth weight is associated with 0.02 additional years of schooling. In order to compare the results with those of Black et al. (2005), we also estimated the effect of log birth weight on high school completion. The estimates showed that a 10 percent increase in birth weight is associated with a 0.7 percentage points increase in the probability of high school completion. This is very similar to the 0.9 percentage points increase obtained in Black et al. (2005), who, unlike us, used register data on birth weight. The similarity of our findings with those obtained in previous studies suggests that the self-reported data on birth weight used here are valid.[15]

The fact that birth weight differences predict schooling differences raises some concerns about our main results reported above. If birth weight is also positively related to longevity, it would mean that our estimates would most

[13] It turns out that the point estimates are very similar when we restrict the sample to MZ twins, although they become less precisely estimated.

[14] We have also tried alternative specifications of the birth weight variable, such as an indicator of low birth weight (birth weight $<$ 2500 grams). The indicator of low birth weight is significant, but when including both this indicator and the birth weight variable, the latter always dominates and the former becomes insignificant.

[15] One reason for a limited role of measurement errors is that there may be twin-pair specific component to the measurement error. In other words, if twins tend to misreport in similar ways, this source of measurement error will be taken out in the SPL model. A similar argument was made by Smith (2009), who used sibling data on self-reported retrospective health during adolesence.

likely be upward biased. Next, we therefore re-estimate our main models, while adding birth weight to the regression. One concern in these analyses is, however, that our sample with birth weight information is younger and that education may have little or no relationship with mortality in this younger sample. The mean birth year in the sample with birth weight information is 1946, which means that the average person was between 52 and 57 years of age when the birth weight data was collected. We therefore first establish if there is a significant association between education and mortality also in this restricted sample with information on birth weight. As shown in the first and third columns of Table 5, this is indeed the case, where the estimated hazard ratios in both the standard Cox and the SPL models are rather similar to the corresponding estimates obtained for the full sample.

How does then the inclusion of birth weight affect the relationship between education and mortality? As shown in columns two and four of Table 5, the magnitude of the hazard ratio of education is almost unchanged when accounting for birth weight differences. Moreover, the results show that birth weight is not significantly related to mortality, with a hazard ratio of 1.008 in the Cox model and 0.993 in the SPL allowing for twin-pair specific baseline hazards.[16] We believe this is an interesting finding in its own right and we are not aware of any previous twin studies on the relationship between birth weight and mortality. We also again tried other indicators of birth weight, such as an indicator of low birth weight (below 2,500 grams) and log birth weight but the results did not change. Our results thus suggest that although birth weight predicts schooling, the omission of birth weight in our main regressions do not bias the estimate of schooling, since birth weight is not related to mortality. At least, this is true in the still quite substantial sample with information on birth weight.

D Proxies for ability differences

We next turn to the question of potential ability differences between twins, using our proxy measure height. As discussed in the Methods section, it is well established that height is related to childhood and adolescent IQ, as well as with early life health and nutritional inputs. We therefore believe that an *absence of a change* in results when controlling for a variable known to be strongly correlated with childhood IQ could be informative.

First, we want to establish that there is a significant relation between height and education in our sample. As shown in Table 6, this is indeed the case, where a ten centimeter increase in height is associated with an about 0.5 increase in the number of years of schooling. Note also that since height information is present for the lions share of the full sample, we are no longer restricted to the smaller sample size used for assessing the role of birth weight. When we impose twin-pair specific baseline hazards, as shown in column II, the estimate decreases to about one third (to 0.15) but remains significant at the 1 percent level. Similar, and significant, estimates are obtained if we

[16] Among the 10,100 observations with birth weight information, 543 deaths occured. There is also substantial birth weight variation, since all twins differ in birth weight grams, the average difference being 45 grams.

restrict the sample to MZ twins (not shown).

Does our main result on the relationship between education and mortality change when we control for height in the regression? To answer this, we first estimate the relation between education and mortality in the sample where we have information on height. As seen in columns 1 and 3 of Table 7, the resulting estimates in both the standard Cox model and the SPL model are very similar to the ones obtained for the full sample. When we also include height in the regression, the results do not change to any considerable extent. Moreover, the results show no significant relationship between height and mortality in either model.

Since height is strongly correlated with cognitive ability, an alternative sensitivity check would be to examine if the results change when only including twin pairs who are very similar in terms of height and who, thus, should also be more similar in terms of cognitive ability and early life health. We therefore reran our regressions, this time only including twin pairs that differed at most two centimeters in height, resulting in a sample size of about 20,000 twins. Also, the estimated hazard ratio is still significant and of similar magnitude as for the full sample (0.94). Even when we restrict the sample to include only twins who differed at most one centimeter our estimates remain rather stable and significant (0.95).

E Learning from DZ twins

We have shown that birth weight differences and differences in height, which arguable picks up some variation also in cognitive ability, do not affect our results to any great extent. There of course remains the possibility that there are differences in other unobserved factors between twins that will bias our estimates. We may shed some light on this by exploiting the information on zygosity in the data. It seems plausible that MZ twins are more alike than DZ twins not only in terms of education but also in terms of preferences and personality traits. Evidence suggesting so is for instance implied in variance decomposition studies that tries to single out the genetic component in such traits, where contrasts between MZ and DZ twins are used.[17] In our case, it means, for instance, that certain preferences that affects both schooling and longevity, should be more alike among MZ twins than among DZ twins. If this is the case, we would also expect the MZ estimates to come out as smaller than the DZ estimates, since a larger part of the influences of such preferences will be differenced out among MZ twins.[18] On the other hand, if the results do not change much, it would suggest that differences in unobserved factors,

[17] Jang et al. (1996), for instance, used data on MZ and DZ twins and showed a strong genetic influence on the five personality dimensions of Neuroticism, Extraversion, Openness, Agreeableness. Cesarini et al. (2009) found evidence of genetic contribution in preferences for giving and risk-taking.

[18] This would be true as long as at least some part of the variation in such preference traits is due to genetics. This is also what the studies cited above suggest. A difference in results between MZ and DZ twins could also be explained by the greater role that measurement errors play among MZ twins than DZ twins. Since data on education is taken from registers for a large part of our sample, we do not believe that measurement errors play a great role, however.

such as preferences, play less of an important role.

The results in Table 3, based on pooled MZ and DZ twins and MZ twins already suggest that we should not expect any great differences in the estimates for MZ and DZ twins. When we explicitly make the comparison between DZ and MZ, this is indeed also what we find. When moving from DZ to MZ twins, and pooling males and females, the hazard ratio changes only very mildly, from 0.932 to 943. While this may suggest that some more unobserved heterogeneity is taken out in the MZ estimates the difference is small and we cannot reject that the estimates are in fact the same. The same conclusions are reached when running separate regressions by gender, again suggesting that the role of unobserved heterogeneity between the twins perhaps should not be overemphasized.

F Non-linear returns

So far, the analyses have been restricted to linear returns to schooling. Next, we instead consider the possibility that there are important non-linearities in the relationship between schooling and mortality. For this purpose, we defined three groups; those with less than 10 years of schooling, those with at least 10 but less than 13 years of schooling, and those with at least 13 years of schooling. The latter category includes people with a university exam.

The results in Table 8 show the potential importance of degrees. Compared to twins with less than 10 years of schooling, twins having between 10 to 12 years of schooling has about 13 percent lower mortality hazard. This estimate is only mildly affected when allowing for twin-pair specific baseline hazards. For twins with a university exam, or some university studies, the estimates are more dramatic; the mortality hazard is now more than 40 percent lower in the SPL model. For MZ twins, the estimates are only slightly less pronounced amounting to about 35 percent regardless of whether we employ the SPL or not.[19]

Table 8 shows estimates separately for male and female MZ twins. For the combined sample of MZ and DZ twins, the estimates for both men and women are very similar to the ones obtained for the pooled sample. Turning to MZ twins it seems as if the effect of 10-12 years of education compared with less education is somewhat stronger among women.[20]

G Selection and changes in the estimates over time

As argued in the data and methods section, the sampling used in the twin registry may induce some selection problems. We pointed out that this most likely leads to conservative estimates using the Cox model. The reason is that inclusion in the registry is conditional on survival to certain points in time and since a low-educated person who still manages to survive long ought to

[19] We also tried specifications including both degrees and years of schooling. The latter variable was never significant in any of these specifications, underscoring the importance of degrees.

[20] We also repeated the sensitivity tests of the previous sections, controlling for birth weight and height, but the results were again robust.

have better than average unobserved underlying health this may generate a negative correlation between the latter and education. We also argued that the SPL estimator may partly solve this, since twins share many unobserved characteristics, such as underlying health.

If selection is an important issue, we would expect that the downward bias in the estimates is larger for the older cohorts, since they had to survive longer in order to be included in the sample. A person born 1890, for instance, had to survive to the age of 71 to be included. We can therefore check the sensitivity of our results to the inclusion of the older cohorts, where more selection has likely taken place. Note, however, that two things happen when we exclude older cohorts. Besides excluding cohorts were more selection has taken place, we are also less likely to observe completed lifespans in the remaining cohorts and we will thus only be able to evaluate the effect of education at younger ages. We will therefore need to assume, for the moment, that the proportional hazards assumptions holds, i.e. that the effect of education remains constant over time (age). Further down, we will investigate this assumption as well.

In Table 9 we show both Cox and SPL estimates with different restrictions on what cohorts to include. In the first row, we replicate the main results for ease of comparison. Excluding the oldest cohort, born between 1886-1895, does not bring any radical change in the results, as shown in the second row. No dramatic changes occur when also excluding the cohorts born between 1896-1905 and between 1906-1915. If selection bias would have been important, one would have expected that the Cox estimates would grow in magnitude as the older cohorts were excluded. Moreover, we would expect the SPL estimates to differ more from the Cox estimates in the older cohorts, if the SPL model deals with some of the selection problem. This is not what we find, however.

Note that one cannot rule out that a strong downward bias from selection is offset by a strong upward bias from unobserved ability in the older cohorts. In later cohorts, a weaker selection bias would then also be offset by a weaker ability bias. It appears likely to us that improved access to education over time leads to less rationing of education according to skills. Moreover, when credit constraints are less binding, parents could easier afford to send both their twins to higher education, whereas more binding constraints may have led parents to invest more in the more able twin. We can shed some light on this possibility by examining if the effect of height, which partly picks up cognitive ability, on education decreases over time. This is not the case, however, and if anything, the effect is greater for the post 1940 birth cohorts.

We next turn to the results for the later born cohorts, where the results suggest that there have been some changes over time in the estimates. Including only cohorts born between 1936-1958 gives a stronger impact of education, with a hazard ratio of 0.89 in the SPL model. This pattern becomes even more pronounced when restricting the sample to the even younger cohorts born between 1946-1958, where the hazard ratio is now 0.82. These results are consistent with the pattern observed in many previous studies that the socioeconomic gradient in mortality has grown stronger over time. This has been explained by, for instance, that well-educated people have been more able to take up new medical interventions (Glied and Lleras-Muney 2008).

There are also other ways to interpret the changes in estimates in the recent

cohorts, however. As mentioned above, the age composition of the sample by automation changes when imposing different cohort restrictions. For the younger cohorts, many more deaths are censored simply because fewer people have reached high ages. Hence, it is possible that the cohort effect found in Table 9 rather reflects that the impact of education varies by age. We can shed some light on how plausible this explanation is by comparing the estimates for similar age groups who belong to different cohorts, i.e moving the age window across time.

In order to investigate how the impact of education on mortality varies over age and/or birth cohorts, we focus on specific cohort subdivisions of the main sample for which we are able to make similar age cutouts. If the changing age composition of the sample explains the changes in the results above, we would expect to see no great differences when comparing the results for the same age groups across cohorts. On the other hand, if the results change also across the same age groups across cohorts, this suggests that real changes in the relationship between education and mortality have taken place. This, again, assumes that our SPL estimator takes care of selection issues and that any remaining ability bias remains more or less constant over time.

Table 10 presents the SPL results from this exercise. The first two columns give the mortality hazard ratios during the age span 50-60 for the cohorts born 1922-34 and 1935-47 respectively, while the subsequent columns give the corresponding hazards for later age spans and comparable cohorts. Clearly, the relationship between education and mortality *within a particular age group* is rather similar across cohorts. In the age group 50-60, for instance, the hazard ratio for the cohorts 1922-34 and 1935-47 is 0.91 and 0.89, respectively. Thus, the estimates suggest that the relationship between education and mortality looks rather similar over time but that it varies by age. The difference in results when putting restrictions on which cohorts to include, shown in Table 9, therefore reflected that the relationship between education and health becomes weaker by age rather than changes in the relationship across cohorts. The reduced importance of education by age could be explained by a mechanism where the underlying health of those surviving to higher ages is greater and where education matters less for health production at higher levels of underlying health.

H How much life expectancy is really gained by higher education?

The Cox and SPL survival models analysed above are both semi-parametric in the sense that whereas the impact of explanatory factors (e.g. schooling) is estimated, the parameters of the underlying baseline hazard parameters are not. These models make use of the ordering of relevant events (deaths) but do not use information on how far apart or exactly when on the time scale different events occur. Hence, these models yield estimates of the mortality hazard ratios but given the absence of a specified baseline, such hazards are not directly translatable in terms of more detailed longevity prospects. To capture such prospects via survival analysis, the baseline has to be specified and its parameters estimated as well.

Unfortunately, it is infeasible to apply a twin design alá SPL when estimating a parametric proportional hazards model. The reason is obvious; there is simply not enough information (or degrees of freedom) from two observations to allow for specification of one specific baseline for each and every stratum or twin pair. Nevertheless, the results presented so far strongly suggest that unobserved twin characteristics operating at the twin level do not influence the estimated impact of education on mortality in any important way. In order to illustrate how the effect of education on mortality hazard ratios may translate into survival propensities, we therefore present estimates of mean remaining life expectancy at age 60, based on the Gompertz distribution.[21]

The Gompertz distribution has been shown to describe old age mortality rather well. This model is a parametric proportional hazard model in which the baseline hazard takes the form:

$$\theta_0(t, a, b) = ae^{t/b}, \tag{3}$$

where a and e are shape and scale parameters respectively, of the baseline and t is, just as in the Cox and SPL models, the randomly distributed death time. Hence, the model can be expressed in terms similar to the Cox used above with the addition that the baseline is specified according to a Gomperz distribution with specified shape and scale parameters.

The sample included in this analysis was restricted to include only twin pairs who were both alive at the age of 60. Hereafter we estimated Gompertz survival models for different subpopulations according to gender, educational group (as defined above) and birth cohort (1900-1919, 1920-29), only including birth year as a continuous explanatory variable. On a computational level, the studied time/age profile was rescaled so that entrance in the relevant risk set occurred at time point (t) 0 (at the age of 60) in order to more accurately capture old age mortality. The resulting estimates of the shape and scale parameters were then used to calculate life expectancies by integrating the random variable t with respect to its probability measure given by the estimated Gomperts density, over its support (0, inf), that is, from age 60 (t=0) onwards.

Table 12 presents the results in terms of mean expected remaining life expectancies at age 60 together with the estimated scale and shape parameters (a,b) in parenthesis. Low educated men in the old cohort could expect to live 19.1 years on average when they were 60, whereas high educated men could expect to live for an additional 2.8 years. The corresponding figures for females are 23.1 and 2.5 respectively. For the younger cohorts the educational impact on expected survival is similar. Low educated 60-year old men expecting to live for 20.7 years and highly educated men for additional 2.8 years. Low educated females for 25.3 years and highly educated for an extra 3 years. Hence, overall it seems as if higher education is associated with 2.5-3 years additional life expectancy at age 60 compared to low education.

An alternative way to illustrate the impact of education on survival propen-

[21] It should be emphasized that the main purpose of this exercise is not to exactly capture specific remaining life expectancies for different subgroups but rather to illustrate the differences in these expectancies, i.e. what the impact of education on mortality may translate into in terms of longevity.

sities is to study how the probability of reaching different ages is related to education. Contrary, to the survival analysis based on the Gompertz distribution presented above, this allows for correction for unobserved within-twin-pair common characteristics. Table 12 present twin fixed effects estimates of linear probability models of survival up to age 70 and 80. The identification for these models emanates solely from twin pairs of which one twin has reached the considered age and the other one has not. The first rows present results for the sets of MZ and DZ twins combined. Note that in order to get some crude reference point to the relative impact of education on survival, the figures in italics (Survivors) gives the fraction of survivors up to the considered age in the lowest educational group, as obtained by simple OLS of survival on a constant.

The results from the first rows and first column of table 12 imply that 84 percent of the low educated sample lived to see their 70th birthday, whereas high education (at least 13 years of) education is associated with about 6 percentage greater chance of experiencing this day, the figures being slightly greater for females and smaller for males. Now, an increase of an survival expectancy from 84 to 90 percentage points may not seem that dramatic, but it is also equivalent to a decrease of the mortality risk from 16 to 10 percentage point (or by almost 40 percent). The gender discrepancy is reversed when it comes to the propensities of becoming 80 years old, for which the chance is about 10 percentage points higher for males and 6 (insignificant) percent higher for females if they belong to the highest educational category. Again, together with the figures on the fraction of survivors among the lowly educated, this implies that the death risks are reduced by almost 20 percent for highly educated men. Turning to monozygotic twins the precision of the estimates is lower due to less sample sizes but, with the exception for highly educated men (whose estimates are about halved), for men and women combined as well as for women alone, the magnitude of the estimations rather closely mirrors the ones obtained in the pooled MZ/DZ sample discussed above. Altogether, our estimates in this section suggest that the effect of education on mortality is rather large.

I Summary and conclusion

It is well known that well-educated people live longer. Less is known about why this is so. In this paper, we provided evidence on the topic, by analyzing a large data set on Swedish twins, born between 1886-1958. A unique feature of these data is that we are able to observe complete lifespans for a larger number of individuals. By applying a twin design, we were able to account for many often unmeasured confounders, such as genes and family background. Our results show that there is a strong and significant relationship between education and mortality, even when accounting for such potential confounders. Moreover, our estimates suggest that the effect of education may be large; being a highly educated person, having at least 12 years of schooling, is associated with 2.5-3 years additional life expectancy at age 60 compared to being a low educated person. We also found the results to be robust to a number of controls for within-twin-pair differences in early life factors, such as birth weight

and proxies for ability and relatively stable over the time period considered.

Our analyses used data going to the end of the 19th century, when levels of schooling were low. Between the start and the end of our study period, average years of schooling increased from about 9 to 12 years of schooling. This also means that we do not know if our results generalize to a situation with much higher levels of average schooling, as in Sweden today. Clearly, however, the levels of schooling in our study period are similar to the ones found in many less developed countries today and our results therefore suggests that the longevity gains from extending levels of schooling can be substantial in these countries.

A further implication of our results is that the returns to schooling are much larger than what is seen by narrowly focusing on productivity gains.[22] The extent to which this fact is of relevance for policy depends on whether or not people internalize these additional gains when taking their educational decisions. If people already take this into account in the decisions, there would perhaps be less room for policies aimed at reducing the costs of education. On the other hand, if there are substantial externalities involved, the scope for policy may be much greater.

Further studies should aim at improving our understanding the underlying mechanisms for the relationship between education and longevity. Some additional clues may be given by analyzing specific causes of death. Moreover, analyzing the relation between education and various health inputs, such as smoking, may yield important insights. Such analyses may also shed some light on why the schooling-mortality gradient has changed over time.

[22] Some of the health benefits of education may already be captured in the higher earnings that follow from education, however.

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Table 1: Descriptive statistics. Data from the Swedish twin registry.

	MZ and DZ twins		MZ twins	
	Males	Females	Males	Females
Years of schooling	10.39	10.15	10.51	10.36
	(0.02)	(0.02)	(0.03)	(0.02)
Mean death age (non-censored)	73.78	77.52	74.02	77.59
	(0.12)	(0.12)	(0.20)	(0.20)
Mean censoring age	65.21	67.41	65.08	66.68
	(0.08)	(0.09)	(0.13)	(0.13)
Fraction of observed deaths	0.41	0.38	0.40	0.35
Observations	23154	26446	8608	10088
Birth weight (grams)	2544	2428	2468	2365
	(16.76)	(8.43)	(22.59)	(11.98)
Observations	1882	5164	922	2440
Height (cm)	175.62	163.31	175.47	163.19
	(0.05)	(0.04)	(0.08)	(0.06)
Observations	19612	23450	7466	9034

Notes: This table reports descriptive statistics. Standard errors of mean within parantheses.

1.

Table 2: Descriptive statistics. Frequency of differences in education within twin pairs. Data from the Swedish twin registry.

	MZ and DZ twins		MZ twins	
Difference in years of schooling	Males	Females	Males	Females
Zero	50.5	52.4	57.1	56.1
One	12.3	15.1	12.5	14.9
Two	9.1	10.5	8.2	10.2
Three	16.2	14.3	14.2	12.9
Four	6.5	3.3	4.9	2.7
Five	2.1	2.2	1.4	1.8
Six or more	3.2	2.2	1.7	1.5
Number of twin pairs	11577	13233	4304	5044

Notes: This table reports descriptive statistics. Standard errors of mean within parantheses.

Table 3: Cox regression on mortality and education. Data from the Swedish twin registry.

	MZ and DZ twins		MZ twins	
	Cox	SPL	Cox	SPL
		Pooled sample		
Years of schooling	0.944***	0.935***	0.944***	0.943***
	(0.937 - 0.952)	(0.916 - 0.954)	(0.931 - 0.956)	(0.908 - 0.980)
Observations				
	49600	49600	18696	18696
		Males		
Years of schooling	0.946***	0.942***	0.951***	0.936**
	(0.936 - 0.955)	(0.918 - 0.966)	(0.935 - 0.968)	(0.890 - 0.985)
Observations				
	23154	23154	8608	8608
		Females		
Years of schooling	0.942***	0.925***	0.933***	0.953*
	(0.931 - 0.954)	(0.896 - 0.955)	(0.913 - 0.952)	(0.900 - 1.008)
Observations				
	26446	26446	10088	10088

Confidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1
Notes: This table reports estimates from Cox regression models. Models 1 and 3 are standard Cox models. Models 2 and 4 are Cox stratified partial likelihood models. Models in columns 1-3 include controls for gender and birth year (dummies).

Table 4: Regression on birthweight and education. Data from the Swedish twin registry. Data from the Swedish twin registry.

	MZ and DZ twins	MZ and DZ twins (FE)
Birthweight	0.0140***	0.0132*
(100 grams)	(0.00472)	(0.00680)
Observations	7046	7046

Confidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1 Notes: This table report estimates from linear OLS and FE regressions

Table 5: Regression on mortality and years of schooling, controlling for birth weight. Data from the Swedish twin registry.

	MZ and DZ twins		MZ and DZ twins (SPL)	
Years of schooling	0.937***	0.937***	0.923*	0.924
	(0.898 - 0.978)	(0.898 - 0.978)	(0.840 - 1.014)	(0.841 - 1.016)
Birthweight		1.008		0.995
(100 grams)		(0.992 - 1.023)		(0.962 - 1.029)
Observations	7046	7046	7046	7046

Notes: This table reports estimates from Cox regression models. Models 1 and 2 are standard Cox models. Models 3 and 4 are Cox stratified partial likelihood models Model 1 and 2 control for gender and birthyear
Confidence intervals in parentheses. *** p<0.01
** p<0.05, * p<0.1 Notes: This table report estimates
from the Cox model

Table 6: Regression on height and education. Data from the Swedish twin registry. Data from the Swedish twin registry.

	MZ and DZ twins	MZ and DZ twins (FE)
Height	0.0468***	0.0149***
(centimeter	(0.00175)	(0.00261)
Observations	43062	43062

Confidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1 Notes: This table report estimates from linear OLS and FE regressions

Table 7: Regression on mortality and years of schooling, controlling for height, Data from the Swedish twin registry. Data from the Swedish twin registry.

	MZ and DZ twins		MZ and DZ twins (SPL)	
Years of schooling	0.948***	0.947***	0.938***	0.939***
	(0.940 - 0.956)	(0.939 - 0.955)	(0.919 - 0.959)	(0.919 - 0.959)
Height		1.002		0.995
(centimeters)		(0.999 - 1.004)		(0.989 - 1.002)
Observations	43062	43062	43062	43062

Confidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1 Notes: This table report estimates from Cox regressions

Table 8: Cox regression on mortality and education. Non-linear returns. Data from the Swedish twin registry.

	MZ and DZ twins		MZ twins	
	Pooled	SPL	Pooled	SPL
10-12 years of	0.874***	0.843***	0.850***	0.818***
schooling	(0.845 - 0.905)	(0.781 - 0.910)	(0.802 - 0.900)	(0.712 - 0.940)
13 or more years	0.647***	0.587***	0.675***	0.659***
of schooling	(0.606 - 0.692)	(0.505 - 0.682)	(0.608 - 0.750)	(0.501 - 0.867)
Observations	49600	49600	18696	18696
		Males		
10-12 years of	0.885***	0.854***	0.867***	0.832*
schooling	(0.843 - 0.928)	(0.768 - 0.949)	(0.800 - 0.940)	(0.684 - 1.012)
13 or more years	0.657***	0.591***	0.714***	0.606***
of schooling	(0.603 - 0.716)	(0.486 - 0.717)	(0.621 - 0.819)	(0.419 - 0.877)
Observations	23154	23154	8608	8608
		Females		
10-12 years of	0.854***	0.832***	0.815***	0.802**
schooling	(0.812 - 0.898)	(0.745 - 0.929)	(0.749 - 0.886)	(0.658 - 0.976)
13 or more years	0.635***	0.583***	0.650***	0.743
of schooling	(0.573 - 0.705)	(0.461 - 0.738)	(0.551 - 0.765)	(0.491 - 1.123)
Observations	26446	26446	10088	10088

Condidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Notes: This table reports estimates from Cox regression models. Models (1) and (3) control for sex and birth cohort. Models (3) and (4) are Cox stratified partial likelihood models.

Table 9: Cox and SPL regressions on mortality and with different sample restrictions. Data from the Swedish twin registry.

Year of schooling	Pooled		Males		Females	
Sample restriction	Cox	SPL	Cox	SPL	Cox	SPL
(1886-1958)	0.944***	0.935***	0.948***	0.942***	0.937***	0.925***
n	49600	49600	23154	23154	26446	26446
(1896-1958)	0.944***	0.937***	0.948***	0.945***	0.937***	0.924***
n	48374	48374	22658	22658	25716	25716
(1906-1958)	0.943***	0.934***	0.946***	0.941***	0.936***	0.922***
n	45434	45434	21378	21378	24056	24056
(1916-1958)	0.939***	0.923***	0.944***	0.929***	0.930***	0.914***
n	39600	39600	18720	18720	20880	20880
(1926-1958)	0.930***	0.919***	0.937***	0.935***	0.916***	0.893***
n	31714	31714	15202	15202	16512	16512
(1936-1958)	0.910***	0.894***	0.910***	0.918***	0.910***	0.854***
n	24384	24384	11822	11822	12562	12562
(1946-1958)	0.867***	0.821***	0.850***	0.814***	0.892***	0.830***
n	14308	14308	7042	7042	7266	7266

Confidence intervals in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 10: Cox SPL regressions on mortality and education. Regressions for MZ and DZ twins by age groups. Data from the Swedish twin registry.

	Ages 50-60		Ages 60-70		Ages 70-80	
	Born 1922-1934	Born 1935-1947	Born 1901-1920	Born 1921-1938	Born 1890-1910	Born 1911-1929
Years of schooling	0.908**	0.885***	0.948	0.941**	0.930	0.936***
	(0.826 - 0.998)	(0.814 - 0.963)	(0.878 - 1.024)	(0.892 - 0.994)	(0.840 - 1.030)	(0.894 - 0.980)
Observations	9532	14428	10683	12943	5365	12022

Condidence intervals in parentheses. *** p<0.01, ** p<0.05, * p<0.1. Notes: This table reports estimates from Cox SPL regression models. Separate estimates by age groups and cohorts. Regressions include both MZ and DZ twins.

Table 11: Remaining life expectancy at the age of 60 for different educational and age groups based on Gompertz estimations of the survival function. Data from the Swedish twin registry.

	1900-1919		1920-1929	
Education	Males	Females	Males	Females
Low: < 10	19.104	23.124	20.741	25.303
	(0.0120, 10.380)	(0.0058, 9.088)	(0.0105, 10.842)	(0.0050, 9.604)
Middle: 10-12	20.188	24.723	21.757	25.810
	(0.0103, 10.238)	(0.0051, 9.402)	(0.0090, 10.636)	(0.0025, 7.396)
High: >=13	21.930	25.613	23.575	28.302
	(0.0094, 11.062)	(0.0032, 8.046)	(0.0061, 9.587)	(0.0024, 8.247)

Estimated scale and shape parameters (a,b) in parentheses. Estimates are based on Gompertz survival models for different subpopulations according to gender, educational group, and birth cohort, only including birth year as an explanatory variable.

Table 12: Twin fixed effects estimates of the probability of survival ages 70 and 80. Data from the Swedish twin registry.

	60 (birth year < 1950)			70 (birth year < 1940)			80 (birth year < 1930)		
	Pooled sample								
Education	M+F	Males	Females	M+F	Males	Females	M+F	Males	Females
Middle: 10-12	0.0142***	0.0139**	0.0145***	0.0209**	0.0230*	0.0189*	0.0201	0.0308	0.0103
	(0.00417)	(0.00666)	(0.00521)	(0.00824)	(0.0132)	(0.0103)	(0.0136)	(0.0203)	(0.0183)
High: >=13	0.0386***	0.0427***	0.0342***	0.0606***	0.0510**	0.0725***	0.0834***	0.108***	0.0562
	(0.00675)	(0.0104)	(0.00876)	(0.0151)	(0.0227)	(0.0201)	(0.0280)	(0.0396)	(0.0399)
Observations	40196	18434	21762	28490	12872	15618	20810	9298	11512
					MZ twins				
Middle: 10-12	0.0219***	0.0365***	0.0106	0.0275**	0.0376*	0.0188	0.0238	0.0458	0.00467
	(0.00693)	(0.0114)	(0.00843)	(0.0140)	(0.0228)	(0.0172)	(0.0233)	(0.0353)	(0.0310)
High: >=13	0.0381***	0.0682***	0.0108	0.0507*	0.0399	0.0644*	0.0819*	0.0781	0.0893
	(0.0114)	(0.0181)	(0.0144)	(0.0260)	(0.0406)	(0.0335)	(0.0473)	(0.0696)	(0.0647)
Observations	14930	6830	8100	10258	4642	5616	7392	3336	4056

Notes: Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1. This table report linear fixed effects estimates for different groups.