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A counterfactual decomposition of survival
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The impact of early and adult life conditions on educational health inequality over the life course: A counterfactual decomposition of survival functions and hazard rates

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Summary. The positive relation between education and health has been shown to be remarkably pronounced over the life course. Less is, however, known about the underlying mechanisms. Two groups of explanatory factors have been advanced: Initial life conditions affecting both education and health in early life, and health related educational returns during adulthood. We apply a counterfactual decomposition of hazard rate models to study the relative importance of these two pathways on the transition to poor health. Using data from SHARE (2006/07) and SHARELIFE (2008/09), we find that early socioeconomic conditions contribute most to the educational health differences. This is true especially for the oldest cohort. In the successive cohorts the impact of the early conditions weakens, while determinants during adulthood become also important.

Keywords: Educational health inequality; Hazard rate models; Decomposition; Counterfactual analysis; Weighting

1. Introduction

Education is one of the fundamental causes of diseases. It structures the access to health-related resources and is strongly related to a wide range of health outcomes and mortality (Link and Phelan, 1995; Miech *et al.*, 2011). For, presumably, the first time this relationship was reported by Kitagawa und Hauser (1973) in their book „Differential Mortality in the United States: A Study in Socioeconomic Epidemiology“. These findings spurred intense research in the fields of demography, social epidemiology, economics, and sociology, showing robust negative associations between education and mortality, prevalence and incidence of diseases (Dupre, 2007; Elo und Preston, 1996; Ross und Wu, 1995; Schnittker, 2004).

In order to understand this relationship, different theoretical pathways were proposed (for a review see e.g., Blane, 2003; Cutler and Lleras-Muney, 2008). Although the literature is not conclusive, the main explanations of educational health gradient can be summarized into two major groups, according to whether they take place before or after educational attainment has been completed. First, *early life* experiences of poor health, economic hardships and family instability as well as *early* cognitive abilities and genetic factors produce conditions that have long term effects on individual development, educational attainment, and thus health and mortality in early and later life (Blane, 2003; Crimmins, 2005; Feinstein, 2002; Carvalho, 2012; O’Rand and Hamil-Luker, 2005). Second, in turn, higher education improves financial and psycho-social resources as well as health behaviors which may also accumulate *over the adult periods of life* and thus relate education to health outcomes in later life (House *et al.*, 1994; Ross and Wu, 1995; Ross and Mirowsky, 2010).

These two groups of mediating or moderating factors between education and health over the life course are mainly driven by mechanisms proposed by the cumulative advantage and disadvantage

hypothesis. According to this approach, initial deficits in health-related resources that are attributable to early-life circumstances accumulate over time and lead to additional advantages or disadvantages across the life course (Blane *et al.*, 2007; Dannefer, 1987, 2003; Ferraro and Shippee, 2009; O’Rand, 2001). Most childhood conditions are associated with both education and health. Obviously, cognitive ability in learning and problem-solving skills affects academic performance positively (Rohde and Thompson, 2007). It may, however, also lead to a better knowledge of constitutional behavior and thus improve a person’s health care, health behaviors as well as slow down the progress of diseases (Chandola *et al.*, 2006; Mensah and Hobcraft, 2008). A low parental socio-economic status coupled with economic hardship and poor learning environments in childhood is also strongly related to both poor health during childhood and adulthood and lower educational achievement (e.g., Kuh and Smith, 2004; Moody-Ayers *et al.*, 2007; Case *et al.*, 2002). Further, poor health during childhood was shown to predict lower education and poor self-assessed health in adulthood also independently from parental socio-economic situation (Blackwell *et al.*, 2001; Elo, 1998). Finally, an unstable family situation during early periods in life, especially parental divorce, separation or death as well as such forms of family distress as heavy drinking create unfavorable environments for development during childhood and were also shown to be significantly related to both educational attainment and health during childhood and during later periods (Wickrama *et al.*, 1997).

Although a growing number of studies reveal the importance of early childhood for education and health, most previous literature on the educational health gap over the life course concentrated on the mechanisms which operate after educational attainment has been completed. From this perspective, education is seen as a resource that structures the distribution of health-related advantages and disadvantages as well as the onset and duration of exposure to

environmental and social risks (House *et al.*, 1994; Mirowsky and Ross, 2003). Highly educated individuals accumulate advantages such as higher income, wealth, psycho-social resources, and healthy behaviors during adulthood, whereas the less-educated are subject to chronic stress (resulting, for example, from occupational uncertainty), lack of both financial and emotional support in their social networks, and risky health behaviors (Chandola *et al.*, 2006; Ross and Wu, 1995, 1996). Recent studies also point to such psycho-social aspects of health-related benefits from education as so called “learned effectiveness” which manifests itself in the higher sense of personal control and thus healthier lifestyles as well as to the extent of creativity and autonomy of work (Mirowsky and Ross, 2003; Ross and Mirowsky, 1999). The main pathways by which education was shown to influence health remain, however, the general socio-economic situation during adulthood, sense of personal control, and healthy lifestyles (Chandola *et al.*, 2006; Ross and Wu, 1995; Ross and Mirowsky, 2010).

Early and later determinants of educational health differences, however, are much more complex and much less understood than they may seem at first glance because the health gradient and the its determinants are likely to change over time. First, the educational health gap and the relative importance of its explanatory factors may differ *with age*, as for example financial means, psycho-social resources, and health behaviors are not stable over the life course (Lynch, 2003). Second, they may differ *between birth cohorts*, since the initial chances to achieve a certain education and socio-economic position have changed across birth cohorts (Breen *et al.*, 2009; Goesling, 2007). Recent studies reveal such age- and cohort-specific changes of the relationship between education and health: Although the overall level of health improved (Meara *et al.*, 2008), health inequality between educational groups was not reduced. In fact, the most consistent result

of previous research points to an increasing health gap with age especially among the recent birth cohorts (Goesling, 2007; Liu and Hummer, 2008; Mirowsky and Ross, 2008).

Previous research *on explanations of the* educational health gap largely disregard age- and cohort-varying character of this relationship and of the factors to which it can be traced back. Most studies used cross-sectional or short-term longitudinal data which do not allow for separating age and cohort effects as well as for considering the temporal order of the mediating processes (for a review see Blane, 2003; Leopold and Engelhardt, 2012; Mirowsky and Ross, 2003). A number of longitudinal studies analyzed some pathways between education and health over the life course. They concentrated, however, mostly on *either* early life conditions *or* mediating processes during adulthood (e.g. O’Rand *et al.*, 2009; Avendano *et al.*, 2009; Lynch, 2006; Willson *et al.*, 2007). The relative importance of both periods in life for the educational health gap over the life course and across cohorts remains thus largely unclear.

To our best knowledge, only Chandola *et al.* (2006) considered explanatory factors from both periods in life to account for causal relationship between education and health. Using data on a large sample of children born in 1958, they test several early and later causal pathways between education and health. Their results reveal that parental social class, health in early life and adult life styles as well as adult social class were the main predictors of the health gradient in mid-adulthood.

Two major deficits limit, however, the evidence of this and other studies. First they disregard life-course *development* of the education-health gradient as they mostly use prevalence of diseases or health statuses as measures of health. While informative, such measures do not capture *individual-level changes in health over the life course*. Disease *incidence* or onset of poor health, on the other hand, may better quantify the individual rate of health decline by

incorporating the timing of change in the health status (Dupre, 2007). Furthermore, the onset of major disease or poor health status can be even more interesting and relevant for research and policy on health inequality compared with measures of prevalence at some arbitrary point in the life course. Previous research consists almost exclusively of studies that examine educational differences in disease prevalence. The rather descriptive literature, however, consistently shows that education is negatively associated with incidence of such chronic conditions as hypertension (Diez-Roux *et al.*, 2002), heart disease (He *et al.*, 2001), stroke (Jakovljevic *et al.*, 2001), heart attack (Manrique-Garcia, 2011), and cancer (Menvielle *et al.*, 2009; Geyer, 2008). In addition, Dupre (2007) shows that age-specific educational health trajectories may have different patterns dependent on whether prevalence or incidence of major diseases is studied.

Second, previous studies on the pathways between education and health disregard cohort specifics of this gradient. The results of Chandola *et al.* (2006), for example, are limited to a specific British cohort born in 1958 whose educational attainment and thus the relationship between education and health can be affected by an establishment of comprehensive schools and new universities in the 1960s and 1970s. Although Dupre (2007) and a number of other studies (e.g. Lynch, 2003; Mirowsky and Ross, 2008) show that incidence and prevalence of health conditions vary across birth-cohorts, they barely can test why such cohort specifics are observed (Goesling, 2007).

Our study addresses three main limitations of previous research on the explanatory factors of the relationship between education and health over the life course. First, we examine the relative importance of *early and later periods* of life for the educational health gap. Second, we concentrate on the explanation of the incidence rather than of the prevalence of poor health, which allows for considering *life-course development* of educational health gradient. Finally, we

analyze whether the relative importance of different groups of explanatory factors varies across birth cohorts.

2. Methods

We analyze the educational health gap over the life course by means of survival analysis. The dependent variable is the timing of the first incidence of a period of ill health for more than a year in a respondent's life. In particular, we compare the corresponding survival functions and hazard rates for high-educated and low-educated respondents. That is, the age-related educational health gap is defined in our analysis as the vertical distance between the survival curves of the high educated versus the low educated at a specific age. In addition, we also look at differences in age-specific hazard rates between the two educational groups.

To evaluate the relation between life conditions and the educational health gap we employ a counterfactual decomposition approach. That is, we ask how the survival and hazard curves would look like if the distribution of specific life conditions were the same for the low educated and the high educated. Comparing counterfactual survival functions and hazard rates with their observed counterparts reveals how much the differences in these life conditions between low and highly educated contribute to the health gap at various ages.

Counterfactual decompositions are common in some fields of social science research, but not many attempts seem to have been made to adopt the methodology to survival analysis. Well known is the so-called Blinder-Oaxaca decomposition that is mostly used in labor economics to decompose wage gaps between men and women or between ethnic groups (Blinder, 1973; Oaxaca, 1973; Jann, 2008). Related techniques have also been developed in demography for the standardization and decomposition of rates (see, e.g., Das Gupta, 1993). Over the last decades,

counterfactual decomposition techniques were extended in many directions; see Fortin *et al.* (2011) for a comprehensive overview in economics. Yet, the literature only pays little attention to the application of decompositions in the context of survival analysis.

The only exception we are aware of is the paper by Powers and Yun (2009), in which a decomposition approach for hazard rate models is proposed. In particular, Powers and Yun generalize the Blinder-Oaxaca decomposition to the piecewise constant exponential model. However, subject of decomposition in their approach is the group difference in the observed overall rate, defined as the total number of events divided by the total time at risk. Because it is unclear how the approach could be adapted for the decomposition of differences in survival functions or hazard rates, we follow an entirely different strategy than Powers and Yun (2009) in our analysis.

Instead of directly decomposing results from hazard rate models, we employ a reweighting approach as in DiNardo *et al.* (1996; also see the corresponding sections in Fortin *et al.*, 2011). The approach is closely related to techniques developed in the causal inference literature known as “inverse probability weighting” (section 5.4 in Imbens and Wooldridge, 2009) or “propensity-score reweighting” (Nichols 2008). The basic idea of the reweighting procedure is to weight the observations so as to balance the distributions of covariates between the two groups. As can be shown, the propensity score of group membership plays a central role in determining the appropriate weights. For example, if we want to adjust the sex distribution among the low educated to the distribution observed for the high educated, we can fit a Probit model

$$Y^* = \beta_0 + \beta_1 \cdot SEX + e, \quad e \sim N(0,1)$$

$$Y = \begin{cases} 1 \text{ (high educated)} & \text{if } Y^* > 0 \\ 0 \text{ (low educated)} & \text{if } Y^* \leq 0 \end{cases}$$

and then use the estimated propensity scores

$$\hat{p}_i = \widehat{\Pr}(Y = 1|SEX_i) = \Phi(\hat{\beta}_0 + \hat{\beta}_1 \cdot SEX_i)$$

to define the following weights:

$$w_i^0 = \frac{\hat{p}_i}{1 - \hat{p}_i}$$

Weighting the observations in the low-educated group using weights w_i^0 will result in a sex distribution that exactly matches the observed sex distribution of the high educated. Likewise the sex distribution among the high educated can be adjusted to match the distribution observed for low educated by weighting the observations in the high-educated group using weights

$$w_i^1 = \frac{1 - \hat{p}_i}{\hat{p}_i}$$

Furthermore, the sex distribution in both groups can be adjusted to match the distribution in the pooled sample by weighting the observations using weights

$$w_i^p = \begin{cases} \frac{1}{\hat{p}_i} & \text{if } Y_i = 1 \text{ (high educated)} \\ \frac{1}{1 - \hat{p}_i} & \text{if } Y_i = 0 \text{ (low educated)} \end{cases}$$

The procedure can be extended to adjust the distribution of multiple covariates by adding further variables and, possibly, interaction terms to the Probit equation. While this affects the values of the estimated propensity scores, \hat{p}_i , the basic equations for the definition of the weights stay the same. If the Probit model is saturated, that is, if it contains only categorical variables and includes all possible interaction terms, the distributional adjustments will be exact. In more parsimonious

models or if continuous covariates are included, the adjustments will be approximate. In general, the quality of the approximation will depend on the degree of misspecification of the Probit equation. Therefore, it is essential to find a good model that includes significant interactions and uses reasonable functional forms for continuous covariates.¹

As mentioned above, by comparing observed survival functions and hazard rates with counterfactual functions based on the weights above we can gain insights into how imbalances in health relevant characteristics between low educated and high educated contribute to the educational health gap at various ages. In many cases, however, we are not only interested in the total effect of a set of covariates, but also want to disentangle the total effect into the contributions of single variables or specific subsets of covariates. Unfortunately, there is no best solution for such a detailed decomposition in the reweighting approach. A simple procedure is to introduce the variables in a stepwise fashion to the Probit model and determine separate weights from each step. Using these weights, a series of counterfactual functions can then be computed. An issue with this approach is that the results will depend on the order in which the covariates are introduced to the Probit model, that is, there are path dependencies (unless the covariates are uncorrelated). An alternative would be to start with the full model and then estimate a series of models, each omitting one covariate (or one subset of covariates). This approach identifies the “marginal” or “direct” contribution of each covariate (or subset of covariates). There is no problem with path dependencies in this approach. However, the sum of the marginal contributions does not necessarily add up to the total contribution of all covariates. Yet another approach would be to employ the stepwise procedure repeatedly and average over all possible

¹ Of course, also other approaches such as matching algorithms (e.g. Morgan and Harding, 2006) can be used to find the weights. Results, however, should be fairly similar, at least in large datasets. To evaluate the robustness of our approach we replicated parts of our analysis employing the entropy balancing procedure proposed by Hainmueller (2012), that directly optimizes the balancing of the first three moments of the covariates. The resulting counterfactual survival functions and hazard rates were almost identical.

permutations of the order in which the covariates can be introduced to the Probit model. This approach both eliminates path dependency and ensures that the individual contributions sum up to the total effect. However, it is a rather mechanical solution and the precise interpretation of the individual results appears somewhat unclear.

In our application, we employ the stepwise procedure because we believe that there is a sensible natural order in which to introduce the subsets of variables. In a first stage we control the distribution of countries, birth cohorts and sex between the educational groups. Because these variables are not of substantive interest in our application our only goal is to make the two educational groups comparable and have a good starting point for the subsequent analyses. We therefore adjust the distributions of these variables in the two groups to the pooled distribution using weights as defined by w_i^p . In the second stage, we then introduce subsets of health relevant characteristics using the stepwise procedure where the distribution among the low educated is adjusted to the one of the high educated using weights as defined by w_i^0 . That is, in the second stage we ask how the survival function and hazard rate of low educated would look like if they had the same health relevant characteristics as the high educated. Note that separate Probit models are used for the first stage and the second stage. That is, the Probit model in the second stage does not include country dummies, birth cohorts, or sex, but is weighted by w_i^p from the first stage. The weights used to compute the counterfactual functions in the second stage are then the product of w_i^p from the first stage and the specific w_i^0 from the second stage.

3. Data

We use data from the Survey of Health, Ageing and Retirement in Europe (SHARE). SHARE is a cross-country longitudinal survey on patterns of ageing in the diverse cultural settings of

Europe (Börsch-Supan *et al.*, 2010). The survey provides micro-data on health, socio-economic status, family characteristics, etc., on nationally representative samples of individuals born before 1955, speaking the official language of the country and not living abroad or in an institution, as well as on their partners irrespective of age.

Three waves of SHARE are currently available. The first two waves (2004-05 and 2006-07) mostly gather current information on the respondents. The third wave, named SHARELIFE and carried out between fall 2008 and summer 2009, collects information on all important areas of the respondents' life, namely events related to partners and children, childhood health and family background characteristics, complete employment transitions from the first job to retirement, information on past residences, health and health care information from childhood to adulthood, income, and assessments of well-being and happiness. SHARELIFE was conducted in 13 countries that represent four important regions of Europe: Nordic countries (Denmark and Sweden), Central Europe (Austria, Belgium, France, Germany, the Netherlands, Switzerland), Eastern Europe (Poland and Czech Republic), and Mediterranean countries (Greece, Italy, Spain). All respondents aged 50+ who participated in at least one of the previous waves and their partners independently of age were interviewed in SHARELIFE (for methodological details see Schröder, 2011). The full sample of SHARELIFE consists of 26,836 individuals, whereby 23,689 participated also in the second wave. After we restricted the initial sample to the individuals born between 1930 and 1954 our data included 18,175 respondents who participated in both the second wave of SHARE and in SHARELIFE.

Most studies on health inequality analyze measures of health at the time of the survey and are thus hardly able to separate age and cohort effects. Contrary to this conventional approach, we draw on retrospective data of SHARELIFE where detailed information on the timing of ill

health is available. As mentioned above, our dependent variable is thus the age when the first period of ill health or disability in adulthood started that lasted for more than a year. Respondents reported specific diseases which were the reasons for such long-term period of poor health. Due to a small number of cases within specific diseases and disability types they could not be considered in our analysis specifically. Individuals who did not experience at least *one year period of poor health* are treated as censored observations.

Education is measured in relative terms. We divided each country-specific sample into three groups, corresponding to the bottom, middle and top thirds of the education distribution.

To test whether childhood health shocks might have affected both education and health at later life, we include a self-reported measure on childhood health (between birth and age 15) ranging from excellent to poor or varying (*excellent or very good childhood health*). This information was collected retrospectively in SHARELIFE. Although skepticism regarding the respondents' ability to remember events taking 50 years ago or longer is understandable, as Smith (2009) discusses, respondents appear to remember important childhood events about themselves, such as illnesses during childhood, quite well. Additionally, we have information whether the respondent suffered from serious diseases conditions including asthma, meningitis, epilepsy, heart trouble, leukemia, cancer etc. during childhood (*serious diseases in childhood*).

Cognitive abilities at age 10 were assessed by respondents' self-evaluation of their math and language skills at school in comparison to their class-mates (*better math skills, better language skills*).

The information on socio-economic status in childhood collected in SHARELIFE includes the occupation of the main breadwinner in the household (*professional, technical or managerial*),

features of the accommodation (*presence of fixed bath, inside toilet, or central heating*), the number of books at home were enough to fill one bookcase (*more than 25 books*), as well as the density at the household defined as the number of people in the household divided by the number of rooms in the household (*persons per room*). To capture additionally early life conditions, we include whether parents drink heavily (*parents drink heavily*) and whether the biological father or mother was absent (*parent absent*). All this information refers to the time when the individual was 10 years old. Although driven by the data we have, the age 10 cut-off is consistent with the child development literature which documents that cognitive and non-cognitive skills developed by age 10 are important determinants of health disparities in adulthood (Conti *et al.* 2010).

To account for materials conditions back in life, we control for *periods of financial hardship* and for *periods of hunger* in childhood (from birth to age 15) and after age 15. Additionally, we consider *periods of happiness* and *periods of stress* compared to the rest of the life. The later variables should measure psycho-social conditions during life. All periods are captured with dummy-variables being one when the period took place from birth to age 15 or after age 15 and before the onset of the period of poor health.

Health care during life is accounted for with a dummy variable measuring whether the respondent always had a usual source of care, that is, a particular person or a place that he or she went to when sick or needing advice about health (*period of no usual healthcare*). Moreover, we have measures for the age interval when no usual health care was available (*no health care age 0-15, age 16-25*).

Table 1 reports for low and highly educated persons summary statistics for the incidence of a period of ill health lasting longer than one year during adulthood. As expected, the statistics

reveal that low educated persons have significantly higher levels of experience of serious diseases during childhood, having lower childhood health, and less often better language skills compared to their class-mates. Moreover, all measured socio-economic conditions of the low educated persons are significantly lower and the family situation is less favorable compared to highly educated persons. Finally, low educated persons experience more often difficult periods in life (financial hardship and hunger) and less often periods of happiness both during childhood and also after age 15. Contrary to our expectation, periods of stress do not differ during childhood for low and highly educated and are even less often for low educated persons after age 15. Furthermore, low educated persons evaluate their math skills better compared to their class-mates and experience less often serious diseases in childhood. The latter could be due to selective (panel) mortality. Concerning the control variables, males are significantly more often among the highly educated persons. Older cohorts are more often low educated compared to younger cohorts.

Table 1: Summary statistics of main variables

Variable	All	Country-specific education: bottom 3 rd	Country-specific education: top 3 rd	Test of mean difference ^{a)}
<i>Health measure</i>				
Period of ill health for more than a year (0/1)	0.211	0.237	0.182	0.000
<i>Childhood health from birth to age 15</i>				
Very good childhood health (0/1)	0.683	0.675	0.708	0.000
Serious diseases in childhood (0/1)	0.263	0.240	0.295	0.000
<i>Cognitive abilities at age 10</i>				
Better math skills (0/1)	0.559	0.684	0.519	0.000
Better language skills (0/1)	0.360	0.249	0.531	0.000
<i>Socio-economic life conditions at age 10</i>				
Main breadwinner: professional, technical or managerial (0/1)	0.122	0.048	0.242	0.000
More than 25 books (0/1)	0.337	0.190	0.559	0.000
Persons per room	1.963	2.255	1.613	0.000
Fixed bath (0/1)	0.299	0.190	0.466	0.000
Inside toilet (0/1)	0.498	0.371	0.669	0.000
Central heating (0/1)	0.166	0.107	0.265	0.000
<i>Family at age 10</i>				
Parent absent (0/1)	0.127	0.140	0.113	0.000
Parents drink heavily (0/1)	0.083	0.102	0.057	0.000
<i>Periods in life from birth to age 15</i>				
No usual health care age 0-15 (0/1)	0.050	0.069	0.032	0.000
Period of financial hardship age 0-15 (0/1)	0.009	0.033	0.009	0.000
Period of stress age 0-15 (0/1)	0.013	0.014	0.013	0.803
Period of hunger age 0-15 (0/1)	0.037	0.076	0.037	0.000
Period of happiness age 0-15 (0/1)	0.041	0.377	0.407	0.001
<i>Periods in life after age 15</i>				
Period of financial hardship age 16+ (0/1)	0.264	0.289	0.264	0.002
Period of stress age 16+ (0/1)	0.522	0.394	0.522	0.000
Period of hunger age 16+ (0/1)	0.012	0.019	0.013	0.002
Period of happiness age 16+ (0/1)	0.383	0.036	0.048	0.001
<i>Controls</i>				
Male (0/1)	0.461	0.419	0.517	0.000
Cohort 1930-39 (0/1)	0.320	0.426	0.228	0.000
Cohort 1940-45 (0/1)	0.249	0.266	0.235	0.000
Cohort 1946-54 (0/1)	0.431	0.309	0.545	0.000

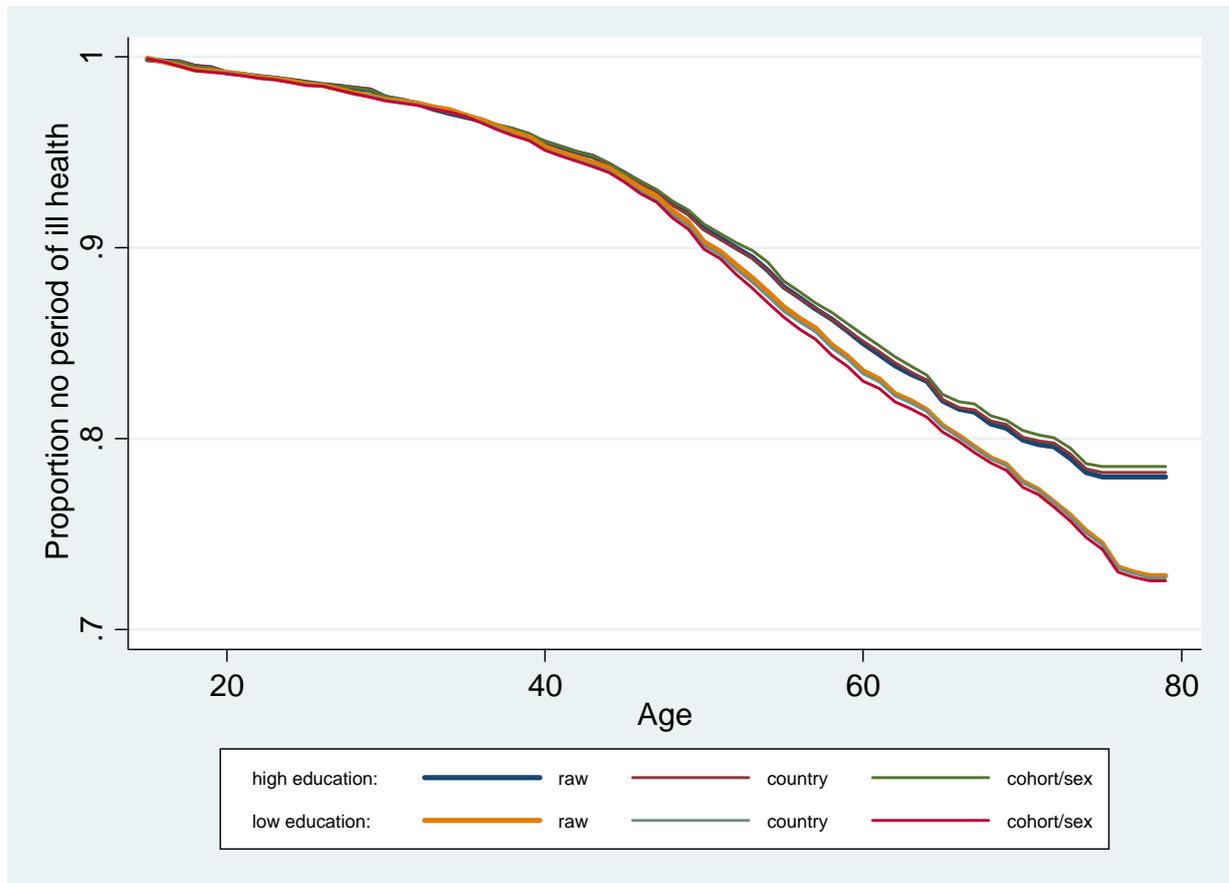
Note: Calculations are based on the full sample, including persons with a mediocre education. a) Test on mean difference between bottom and top 3rd of country-specific education, H1: diff \neq 0, p-values

4. Empirical Evidence

4.1 Counterfactual analysis by education

We start our empirical analysis by estimating the Kaplan-Meier survival rates of not yet having experienced a period of ill health for more than a year separately for low and highly educated persons (Figure 1). The results clearly reveal an increasing education-health gradient with age: Low educated persons do not only experience worse health during the life cycle compared to the highly educated, but also a steeper decline in health with increasing age. Since the distribution of survival times differ significantly by country, cohort, and gender (Table 1), we weight the sample of the low (highly) educated by the inverse of the propensity of being low (highly) educated, conditional on country, cohort and gender. As Figure 1 shows, the adjusted survival rates for the low educated are even slightly lower and the survival rates for the highly educated are slightly higher under control of the country- as well as cohort- and gender-specific composition of the highly and low educated persons.

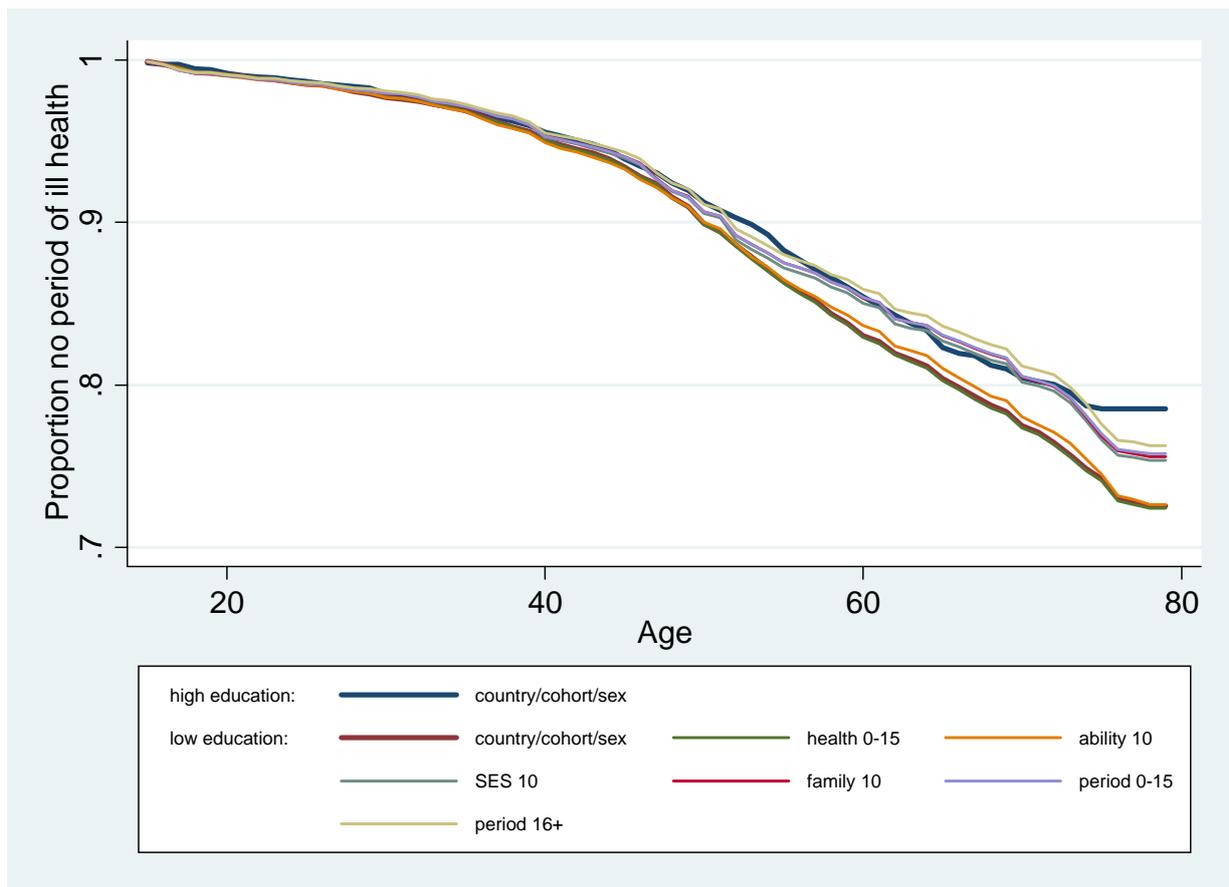
Figure 1: Kaplan-Meier survival rates in good health by education and survival rates weighted by the inverse of the propensity of being low or highly educated, conditional on country, cohort, and gender



In the next step we investigate how the survival rate of the low educated persons would change if they had the same chances of receiving higher education. First we stepwise compute the odds of being highly educated using the information on childhood conditions and on benefits of higher education during adulthood and weight stepwise the sample of low educated persons with the odds of being highly educated. Starting with a model controlling for childhood health and healthcare, we successively add cognitive abilities at age 10, socio-economic life-conditions at age 10, familial situation at age 10, as well as periods in life during childhood and adulthood (Figure 2). As the counterfactual survival rates show, the decline in health would not change

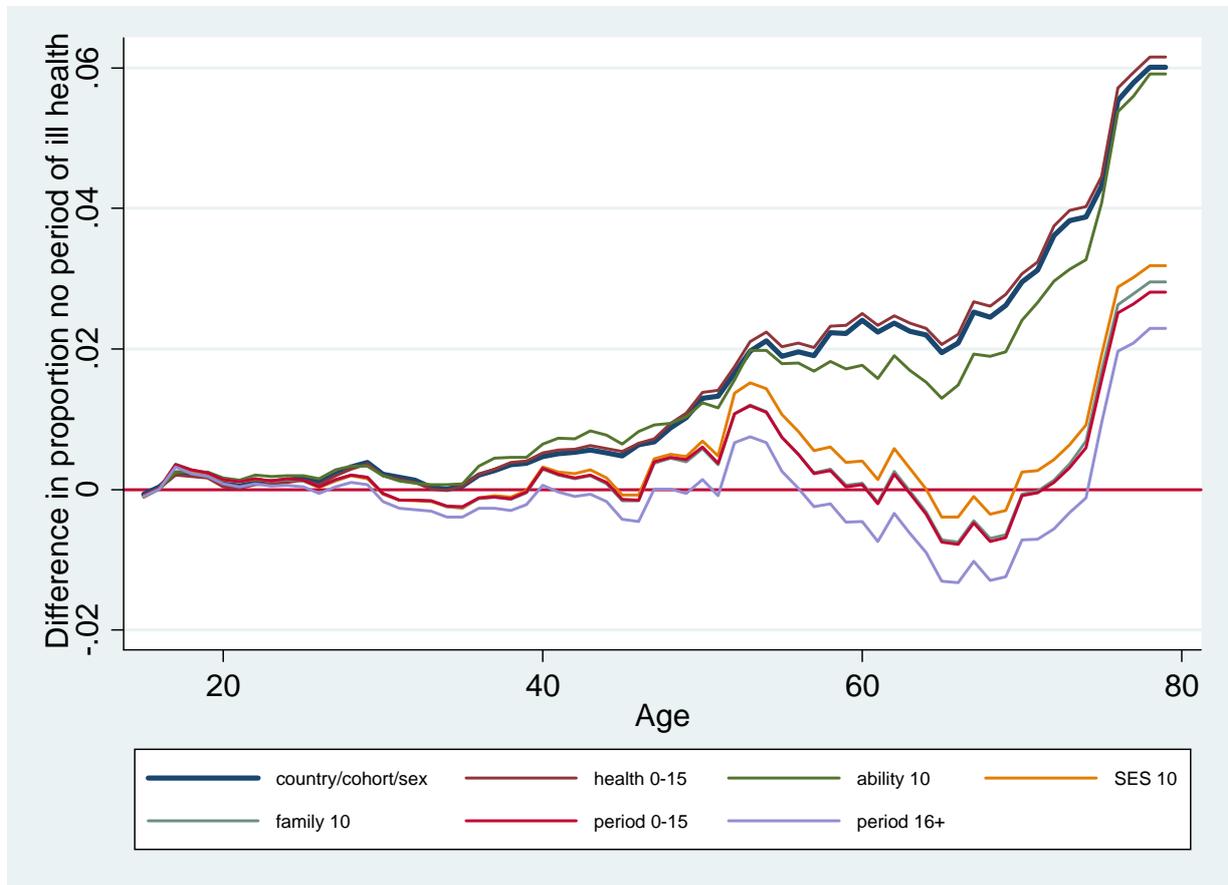
much given the better preconditions of highly educated persons with regard to health during childhood and cognitive abilities. However, taken additionally into account the better socio-economic life conditions at age 10, the decline in health would be almost the same as for the highly educated persons. While the familial situation at age 10 and the different periods in life during childhood do not add much to improving health, the more favorable periods in life during adulthood would contribute to further improving the health of the low educated.

Figure 2: Kaplan-Meier survival rates in good health by education and counterfactual survival rates for the low educated weighted by the odds of being highly educated; control variables are childhood health, cognitive abilities, socio-economic life-conditions, family, as well as periods in life during childhood and adulthood



This result is even more visible when taking the differences in the survival rates for the highly and low educated persons. Weighting these differences with the odds of being highly educated under control of health and cognitive abilities during childhood as well as the specific socio-economic circumstances during childhood and in adulthood (Figure 3) show that cognitive abilities in childhood would pay off after age 40. Considerably more important are socio-economic life conditions at age 10. With the endowments of the highly educated persons, the gap in the survival rates would almost vanish. Additionally, the more favorable familial situation at age 10 of the highly educated contributes to closing the health gap while special periods in life during childhood of the highly educated help in explaining the health gradient (the counterfactual hazard rate is almost identical when additionally considering periods in childhood next to the familial situation at age 10). Accounting additionally for more favorable endowments regarding special periods in life after age 15 of the highly educated, the low educated persons would further improve health during life.

Figure 3: Difference in survival rates in good health for highly and low educated persons and counterfactual differences in survival rates weighted by distributions of highly educated persons

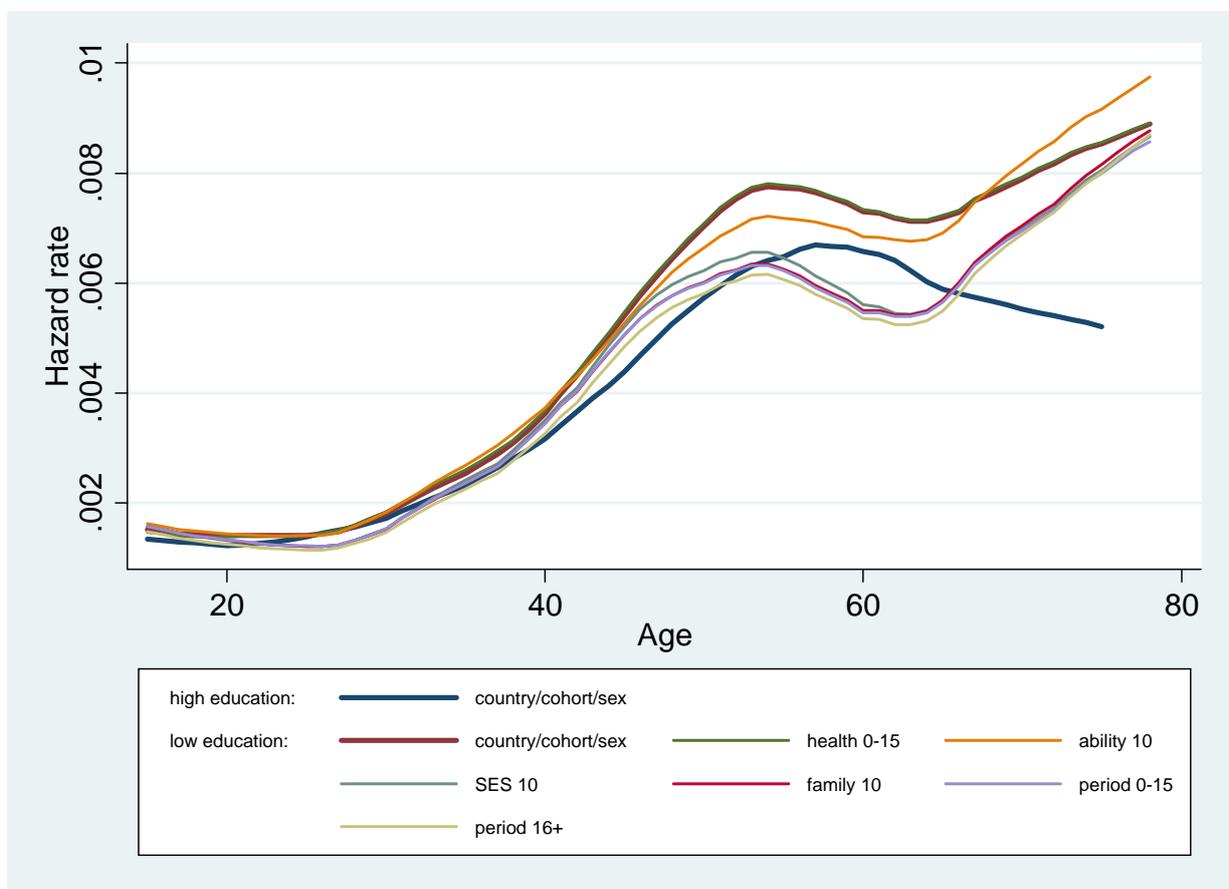


Finally, Figure 4 shows the actual hazard rates for the highly educated persons under control of country-, cohort- and gender-specific heterogeneity as well as the counterfactual hazard rates for the low educated weighted with the odds of being highly educated.² The results clearly reveal that highly educated persons experience the transition to ill health not only at a later age but also to a lower extent at every age compared to low educated persons even when the low educated are weighted with the odds of being highly educated under control of the cohort- and gender-specific composition of this reference group. Under control of all factors referring to early childhood

² Displayed are smoothed hazard rates using an Epanechnikov kernel with a bandwidth of 10 years and renormalization boundary correction as implemented by Jann (2005).

circumstances and conditions during adulthood of the highly educated, the transition to bad health of the low educated still remains at an earlier point of time, though with a decreasing maximum.

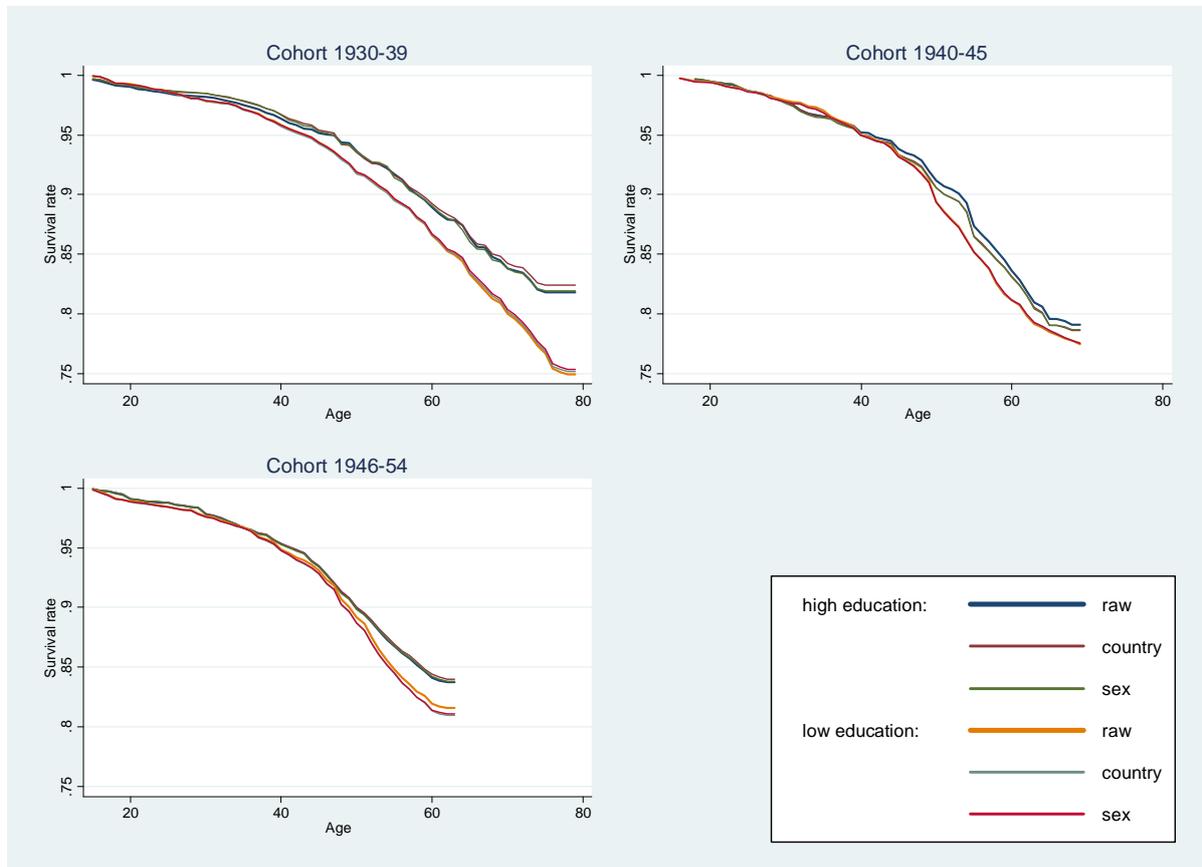
Figure 4: Hazard rates by education and counterfactual hazard rates for the low educated weighted by the odds of being highly educated; control variables are childhood health, cognitive abilities, socio-economic life-conditions, family, as well as periods in life during childhood and adulthood



5.1 Cohort-specific counterfactual analysis

Now we turn to the question whether the impact of early and adult life conditions on educational health inequality has changed over cohorts. Figure 5 shows the cohort-specific Kaplan-Meier survival rates of not yet having experienced a period of ill health for more than a year separately for low and highly educated persons. The results reveal for all cohorts an increasing education-health gradient with age, which does not seem to change much across cohorts. What is changing is the timing of the transitions: In the younger cohorts transitions to ill health occur both earlier and to a higher extent compared to older cohorts. Since the distribution of survival times differ significantly by country and gender, we weight the sample of the low educated (or highly educated respectively) by the inverse of the propensity of being low educated (or highly educated respectively), conditional on country and gender. As Figure 5 shows, the adjusted survival rates both for the low educated and for the highly educated are very similar to the raw survival rates in all cohorts.

Figure 5: Cohort-specific Kaplan-Meier survival rates in good health by education and survival rates weighted by the inverse of the propensity of being low or highly educated, conditional on country, cohort, and gender



To investigate the cohort-specific mechanisms of educational health inequality, we weight the sample of the low educated persons with the odds of being highly educated under control of childhood conditions and circumstances during adulthood (Figure 6). As the counterfactual survival rates show, the decline in health would not change much given the better preconditions of highly educated persons with regard to health during childhood and cognitive abilities. However, taken additionally into account the better socio-economic life conditions at age 10, the familial situation at age 10 as well as distinct periods in life during childhood and adulthood, the decline in health would be almost the same as for the highly educated persons in the oldest

cohort. For the younger cohorts, the effect of this group of variables is also evident, although less strong compared to the oldest cohort. This result is also supported by Figure 7, which shows the cohort-specific differences in survival rates in good health for highly and low educated persons and the counterfactual differences in survival rates weighted by distributions of highly educated persons.

Figure 6: Cohort-specific Kaplan-Meier survival rates in good health by education and counterfactual survival rates for the low educated weighted by the odds of being highly educated; control variables are childhood health, cognitive abilities, socio-economic life-conditions, family, as well as periods in life during childhood and adulthood

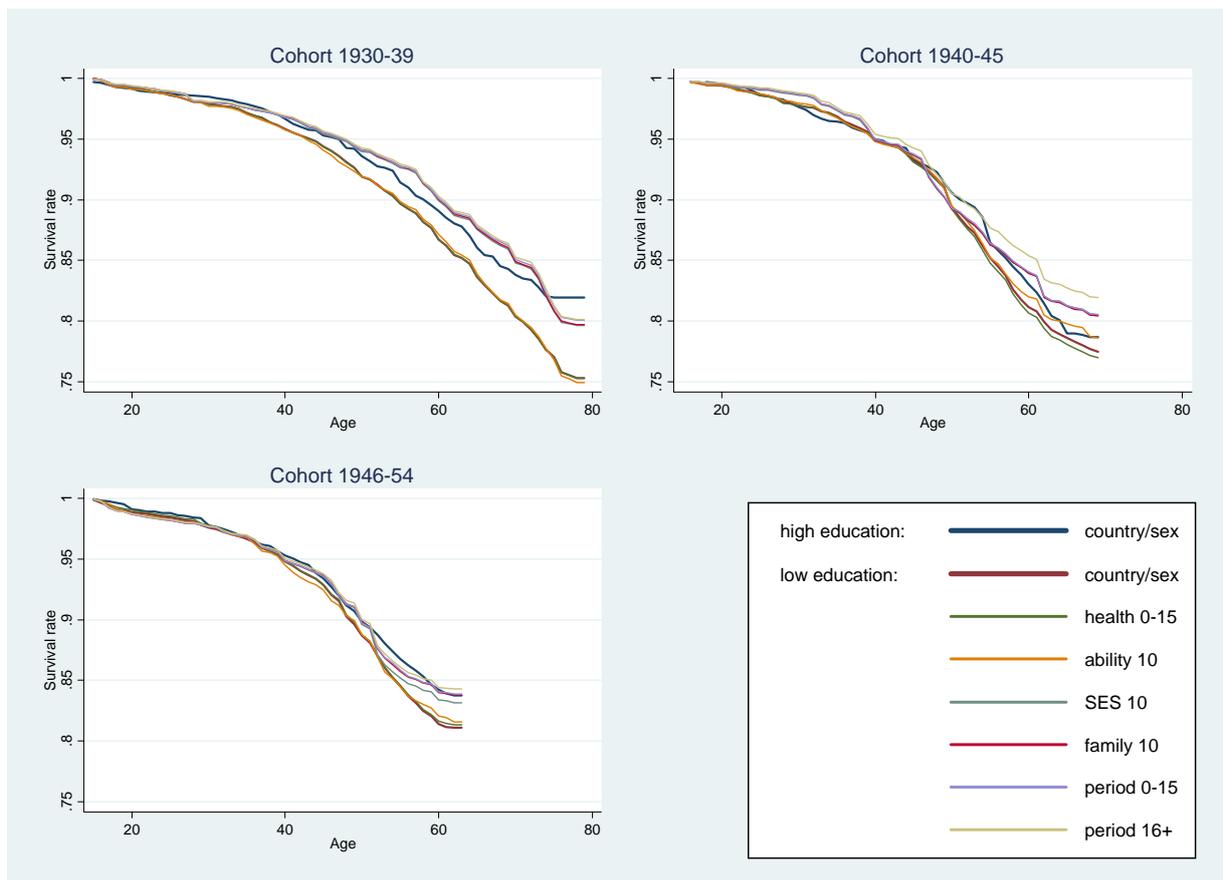
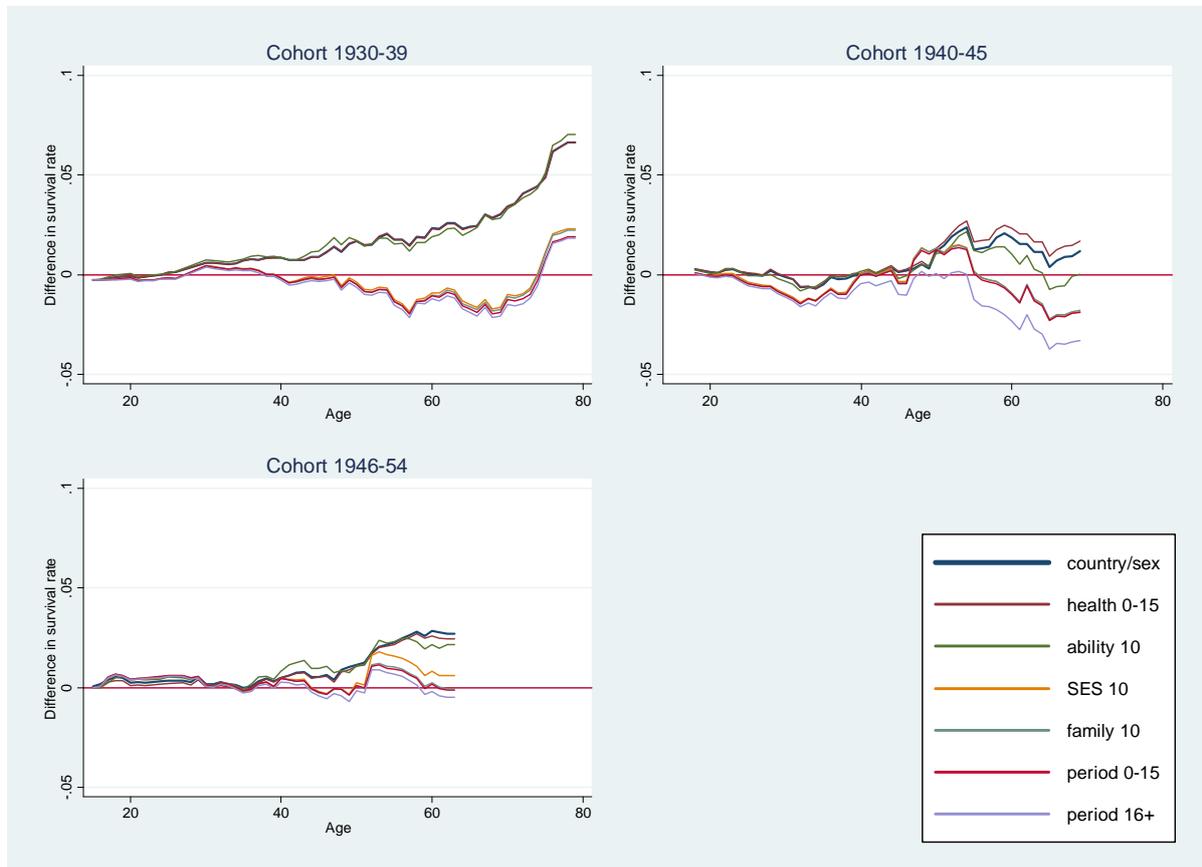


Figure 7: Cohort-specific differences in survival rates in good health for highly and low educated persons and counterfactual differences in survival rates weighted by distributions of highly educated persons

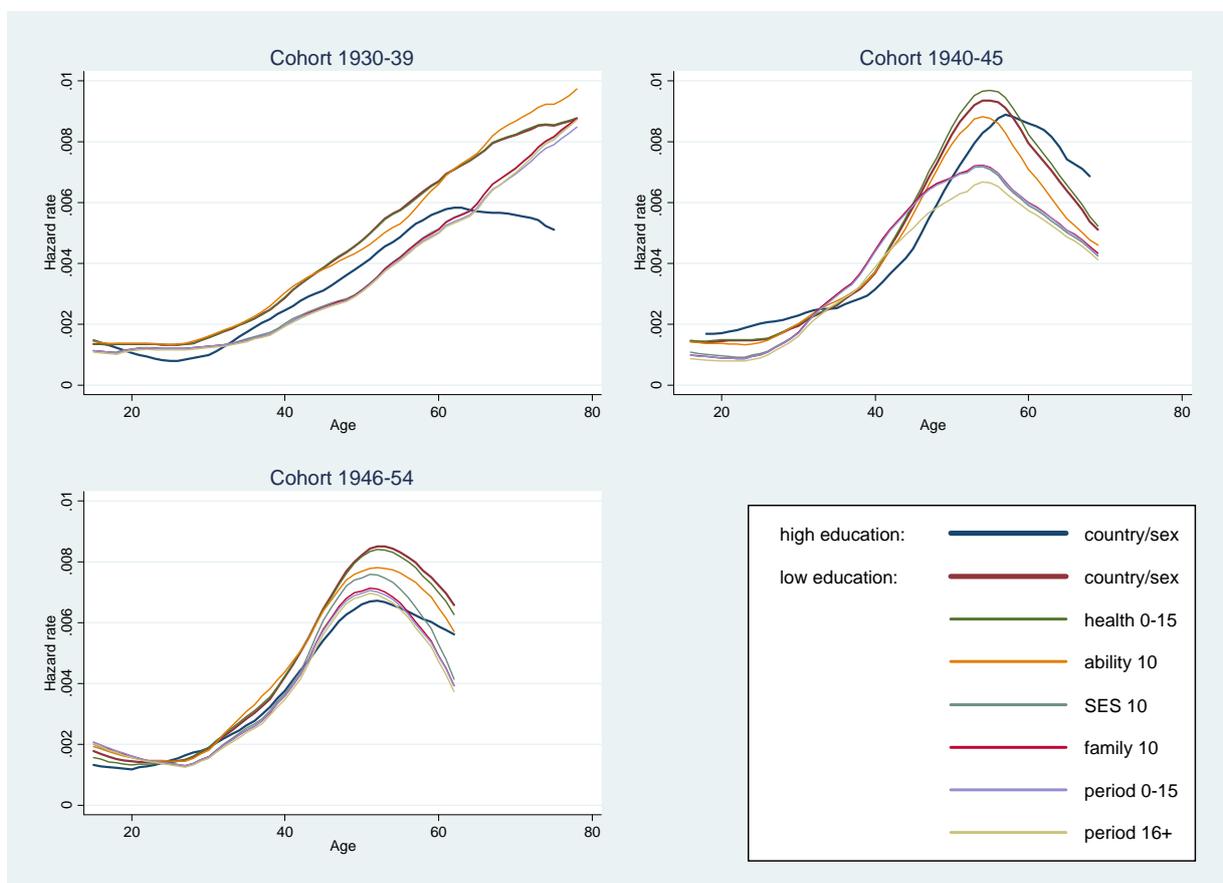


Finally, Figure 8 shows the cohort-specific actual hazard rates for the highly educated persons under control of country-, cohort- and gender-specific heterogeneity as well as the cohort-specific counterfactual hazard rates for the low educated weighted with the odds of being highly educated. The results clearly reveal that highly educated persons experience the transition to ill health in all cohorts only in the cohorts 1930-39 and 1940-45 both at a later age and also to a lower extent at every age compared to low educated persons even when the low educated are weighted with the odds of being highly educated under control of the cohort- and gender-specific composition of

this reference group. In the youngest cohort, the timing for low and highly educated persons are almost the same, although the level differs even under control of all factors referring to early childhood circumstances and situations during adulthood of the highly educated.

Moreover, in the oldest cohort, the hazard rate for the low educated persons does not show the inverted u-shaped pattern of the hazard rate as in other cohorts. Since heterogeneity in populations can result in inverted u-shaped hazard rate curves (Vaupel and Yashin 1985), this result points to selection processes resulting in a less heterogeneous group of low educated persons.

Figure 8: Cohort-specific hazard rates by education and counterfactual hazard rates for the low educated weighted by the odds of being highly educated; control variables are childhood health, cognitive abilities, socio-economic life-conditions, family, as well as periods in life during childhood and adulthood



5. Summary and Discussion

In this paper we studied the differences in individual transitions to long-term ill health between highly and low educated people in Europe. Following the life-course approach of cumulative advantage/disadvantage, we divided the determinants of the educational health gradient into factors during the early life periods before education has been completed and later periods in life. We invented counterfactual weighting procedures to decompose hazard rate models between highly and low educated persons. Unlike previously suggested hazard-rate decomposition techniques, our model allows for an explicit appreciation of the duration dependence of the terms of decomposition. Thus, this method provides a possibility to study the age-dependent impact of different decomposition components. In our analysis we weighted the health trajectories of low educated with odds for being highly educated. The propensity of high education was computed successively according to the temporal order of its determinants over the life course, beginning with childhood health, ability, socio-economic conditions and family situation, followed by periods of financial hardship, stress and happiness up to age 15, and finishing with such periods in life after age 16.

All in all we find that the relative impacts of the early and later determinants of the educational gap in the onset of ill health vary over the life course and across cohorts. Our results reveal that socioeconomic conditions during childhood contribute most to the educational differences in the onset of long-term ill health. This applies especially to the oldest cohort. In the successive cohorts the impact of the early conditions weakens, while periods of financial hardship, hunger, stress and happiness during adulthood become also important.

In more detail, our results first indicate that health conditions and ability during childhood do not contribute much to the explanation of the educational health gradient over the life course. These

results are largely consistent with the results of Chandola et al. (2006). In their study they also found that controlling for these both characteristics only marginally reduces the effect of education on adult health.

Further, we found childhood socio-economic conditions such as parental occupational status, living and learning environments to be the most important determinant of the educational health gap over the life course. Weighting low educated with the chances of being highly educated based on such early socio-economic conditions almost diminished the educational gap in the onset of ill health. The familial situation at age 10 further reduced the gradient. Finally, periods of financial hardship, happiness and stress later in life additionally contributed to the explanation of the educational health gap. The impact of these periods was, however, much less pronounced compared to the impact of socio-economic conditions during childhood. Although we are not aware of other studies simultaneously analyzing the relative impact of early and later determinants of educational health gradient over the life course, the results seem plausible. First, we study educational gap among a rather old cohort. As social stratification research reveals, in the most European countries parental socio-economic background was much more important for educational attainment in these cohorts compared with the younger cohorts (Breen et al., 2009). Our cohort-specific results reveal a similar tendency for educational health inequality. We show that while for the oldest cohort socio-economic conditions during childhood almost completely explain the gap, their contribution weakens in successive cohorts. In the middle and in the youngest cohort periods of financial hardship, hunger, stress and happiness after the age 16 additionally reduce the gap. The youngest respondents in our study were born between 1945 and 1954. For the “next” cohort, born 1958, Chandola et al. (2006) have also shown that early socio-economic conditions do not contribute much for the educational health gap in the adulthood.

Thus, our results may have identified a cohort-specific tendency of declining importance of early socio-economic conditions for reducing the educational health gap over the life course.

Finally, the explanatory power of the determinants of the educational health gradient seems to vary with age. It was especially strong approximately up to the age 70. After this age both early and later life predictors of the educational health gradient do not explain much of the educational gap in the onset of ill health. This result may identify a positive selectivity of those individuals who did not experience a long-term illness up to age 70. As recent research shows, such positive selection into exceptional health among low educated may result from a combination of family and religious factors, health behaviors, psychological makeup, and biological attributes (Dupre and George, 2011). Unfortunately our data did not include this information.

The contribution of this study has to be evaluated in view of its limitations. First of all we could only partly measure the mechanisms behind the education health gradient. This was particularly the case for the explanatory mechanisms after age 16. First we could only very broadly quantify the socio-economic and material resources, since our data included only the information on whether or not respondents experienced financial hardship or periods of hunger. This indicator does not deliver direct information on income, social class and occupational status of the respondents over the life courses, which were previously found to explain to a great extent the effect of education on health during adulthood and which have also changed considerably across cohorts (Ross and Wu, 1995; Cutler and Lleras-Muney, 2008). Second our data did not provide a sufficient measure of health behaviors which were shown to be strongly associated with both early socio-economic conditions and health in adulthood as well as to vary across cohorts (Chandola et al., 2006; Goesling, 2007). Third, we could not directly account for such psychosocial pathways from education to health, as sense of personal control, to which however

educational health differences during adulthood and old age could also be traced back to a great extent (Ross and Mirowsky, 1999; Groffen et al., 2012). Thus, more detailed data is required in order to gain a better understanding of the relative importance of early and later determinants of the education health gradient over the life course and its changes across cohorts.

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