**Zeitschrift:** Schweizerische Zeitschrift für Soziologie = Revue suisse de sociologie = Swiss journal of sociology

**Herausgeber:** Schweizerische Gesellschaft für Soziologie

**Band:** 38 (2012)

**Heft:** 2

**Artikel:** Health inequality across time : a growth curve analysis of self assessed health in contemporary Switzerland

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**DOI:** https://doi.org/10.5169/seals-815122

# Health Inequality Across Time: A Growth Curve Analysis of Self Assessed Health in Contemporary Switzerland

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## 1 Introduction

The existence of a socio-economic gradient in health is well documented and different explanations have been proposed to account for it (Blane 1985; Blaxter 1990; Link and Phelan 1995; Macintyre 1997; Wilkinson and Marmot 2003). However, the relationship between health and socio-economic status (henceforth Ses) has mainly been investigated as a static one: Ses is usually assumed to have the same relationship to health across all age groups and in all cohorts, so little attention has been given to how the gradient operates across the life course and between different cohorts (Ross and Wu 1996; Lynch 2003; Willson et al. 2007).

It is well known that health is better conceived as a process rather than as a state: health in later life is the result of multiple social and biological processes, whose effects may interact or cumulate over time (Wadsworth 1997; Willson et al. 2007; Blane 2006). Therefore, the relationship between Ses and health is likely to change with age since the mediators of Ses may act differently in different stages of the life course.

Moreover, the Ses-health relationship is also likely to change across cohorts since cohorts differ in the distribution and patterns of health and mortality, in the distribution and quality of education and in other socio-economic characteristics whose effects may differ across generations (Lynch 2003; Mirowsky and Ross 2008).

Typically two different traditions have studied cohort and age variation in the Ses gradient in health (Lynch 2003). On the one hand, demographers have mainly focused on the relationship between Ses and mortality and have assumed that temporal variations in this relationship are due to cohort and period effects. On the other hand, medical sociologists have focused on the relationship between Ses and a variety of health measures, and have looked for life course variation in the Ses gradient in health. However, these perspectives should be combined, especially

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because an accurate picture of the dynamic of Ses effects on health can only be achieved by considering cohort, period and age interactions with Ses.

Using short-term longitudinal data from the Swiss Household Panel (SHP), this study tries to investigate whether the social gradient in health varies through time, controlling for gender, cohort, father's education, region of residence and nationality.

## 2 Theoretical framework

When discussing the temporal dynamics of the social gradient in health, we should distinguish the variation in the Ses/health relationship over time from the variation across cohorts.

With regard to the first kind of temporal variation, today it is still not clear whether and how the educational-based gap in health varies with age, but answering this question may be particularly useful in our increasingly ageing societies in order to understand whether successful ageing is possible and whether there are people who are able to postpone disease and disability (Ross and Wu 1996).

There are two main hypotheses concerning the variation of the Ses gradient in health across the life course: the *cumulative advantage theory* and the *age-as-leveller* hypothesis

The cumulative advantage theory was initially proposed by Merton (1968) to explain increasing divergence with age in scientific careers, and it was first applied to health by Ross and Wu (1996). However, the concept of cumulative advantage has not been defined in a consistent manner in the health literature, and there are at least two meanings that recur in health studies. In one case, cumulative advantage is synonymous with path dependence, while in the second case it stands for cumulative exposure processes (Blane 2006; DiPrete and Eirich 2006; Willson et al. 2007).

In this study we follow the second meaning and consider cumulative advantage as a process whereby the positive effect of Ses on health increases over time, hence producing greater heterogeneity and inequality in health in older age groups than in younger age groups. Ses effects on health may become stronger as people age because of increasing returns to socio-economic resources or because some mediators of the Ses/education-health relationship require a long exposure in order to produce health consequences. For example smoking is unlikely to have serious health consequences among young people, but over time its effects on health may become more evident (Ross and Wu 1996; Lynch 2003).

The age-as-leveller hypothesis predicts a convergence of health trajectories in old age. The idea is that the effect of Ses on health declines as individuals age, and that age itself becomes more and more important in predicting health. There are

at least three reasons why the Ses-education gap in health might converge with age (Ross and Wu 1996; Willson et al. 2007):

1. Proximity: education might have a greater impact on younger age groups because its effects are more proximate;
2. Social policy: social policies that increase equality among the elderly might produce convergence;
3. Selection: in this case the convergence is only apparent. Since low Ses is associated with higher mortality and poorer health, at some point the most unhealthy people of the lower Ses group are selected out of the sample through death or serious disability. Since only the healthiest survivors of low Ses groups remain in the sample, variance in health and education is reduced and the effect of education appears to weaken

Regarding the variation of the socio-economic gradient in health across cohorts, Mirowsky and Ross (2008) support what is known as the *rising importance hypothesis*, according to which the rate at which health trajectories diverge across levels of education within cohorts as they age is increasing in younger cohorts. This hypothesis arises from research on historical trends in mortality differentials. Notwithstanding improved public health and medical technology, an increase in the Ses differences in mortality was observed in the last third of the twentieth century (Feldman et al. 1989; Pappas et al. 1993; Elo and Preston 1996; Lauderdale 2001; Lynch 2003). In fact, cohorts differ because of a series of factors that may impact on cohort specific Ses-related gaps in health (Lynch 2003). Cohorts differ in composition, size and historical experiences (such as the implementation of specific social programmes and the available medical treatments, but also the prevalence of risk factors affecting health)[1] and are characterized by different prevailing causes of mortality and a distinct timing of mortality. In particular, according to the epidemiologic transition theory, the widening socio-economic gap in health may be due to the fact that since 1960 degenerative diseases have declined and advances in public health and medicine have, once again, been disseminated first among the upper classes (Lynch 2003). Moreover, both the content and the distribution of education have greatly changed across cohorts, and the relationship between education and the mechanisms that mediate its effect on health may have changed too. More precisely, there are at least two changes that may explain the stronger relationship between education and health in younger cohorts (Lynch 2003). First, knowledge of public health, which is likely to be transmitted through schools, is probably greater nowadays than in the past. Second, according to credentialism, education has become increasingly

1 For instance, cohorts differ in terms of significant behavioural risk factors like smoking. If a cohort has lived in a period in which smoking was not associated with the level of education, it would be unlikely to observe a social gradient in smoking in this specific cohort (Willson et al. 2007).

important in assigning individuals to positions in the labour market and hence in creating inequalities in occupation and income.

These are the main hypotheses that have been formulated around the issue of temporal variation in the social gradient in health. However, empirical results in this field are mixed, and sometimes even contradictory, so that we cannot conclusively support any of the hypotheses presented above. Some studies provide evidence of a cumulative advantage process showing that the gap in health across levels of education grows throughout adulthood (Ross and Wu 1996; Lynch 2003; Mirowsky and Ross 2008); conversely other studies show that the social gap in health grows through much of adulthood but eventually converges (House et al. 2005; Herd 2006); finally, there are studies that support the opposite hypothesis of age-as-leveller (Beckett 2000; Cutler and Lleras-Muney 2006).

In fact, the cumulative advantage and the-age-as leveller hypotheses should not necessarily be considered as mutually exclusive and they may be unified in a more comprehensive explanation that can solve some of the ambiguities observed in previous empirical results. Hence, some authors (House et al. 2005; Mirowsky and Ross 2008) suggest making a theoretical distinction between two components of the adult health trajectory. The first, called the erosion component, is essentially a constant annual rate of decline in health and may vary across levels of education. The second factor, called the disintegration component, represents a biological ceiling: it contributes little to the trajectory through much of adulthood but its effect grows at an accelerating rate, becoming substantial in old age. It may be less sensitive to cumulative socio-economic advantages; rather, according to House et al. (2005) it may even be steeper for the more advantaged. If both components exist, the slope of the trajectory at any given age is the sum of the two, and the dominance of the disintegration component in older age could explain the convergence in health at older age.

However, divergences in empirical results are largely attributable to differences and limitations in data and models. In order to correctly understand whether the relationship between Ses and health varies with age and cohort, we should use data and models that allow us to distinguish between the effect of Ses on health trajectories within cohorts from trends in its effect across cohorts. For instance, this problem could cause cross sectional data to produce misleading results. If both the cumulative advantage hypothesis and the rising importance hypothesis are correct, then each phenomenon tends to obscure the other when comparing the health gap across age groups in a particular year or period (Lauderdale 2001). In fact, if older people have had longer time for Ses gap to develop, but have lived in times when those differences grew more slowly, ignoring the age or cohort effect would lead to the conclusion that the Ses gradient in health is timeless.

It would therefore be necessary to work with panel data that allow us to distinguish between intra and inter individual heterogeneity and, more specifically,

with long term longitudinal data that permit an examination of health trajectories covering a long portion of the life course and allow for a clear disentanglement of age and cohort effects (Willson et al. 2007).

Moreover, every analysis that detects a shrinking of the Ses gap in health in older age should try to understand whether this is due to a longevity ceiling or to mortality selection (Beckett 2000; Willson et al. 2007; Mirowsky and Ross 2008).

Many articles concerning health and the cumulative advantage in health focus on the effect of education. This choice, that we share, is supported by a series of good reasons. Education is a very good predictor of health: it is associated with all measures of health (self-rated health and functioning, mortality and morbidity) and shapes a series of resources that contribute to health (economic resources, social psychological resources, behaviours and health related lifestyle) (Ross and Wu 1996; Robert and House 2000).

According to Ross and Mirowsky (1999), the real importance of education lies in the years spent in formal education, not the qualification itself or the prestige of the school attended. These findings suggest that education is not important for health simply because it provides credentials that make it easier to find a good job, but rather that, according to the human capital theory (Becker 1964; Hyman et al. 1976), education provides students with real abilities. More specifically, the idea is that schooling not only provides cognitive abilities but also shapes personality characteristics (such as orientation towards work, self-confidence and a sense of control over one's life) and socializes to values and behaviours that can be useful in the process of status achievement and self fulfilment. Moreover, educated people usually learn to be flexible and see things from different perspectives, abilities that help to build supportive relationships and that have beneficial effects on health (Ross and Mirowsky 1999).

Finally, education may be so important for health also because of its role in disease self-management (Goldman and Smith 2002; Smith 2004). Nowadays, many chronic illnesses can be treated with effective therapies that are clearly beneficial, but that can also be complicated and difficult for patients to fully adhere to. Education improves adherence, probably because of its effects in term of improved cognitive functions and the development of problem-solving ability, decision making and the ability to internalise the future consequences of current decisions.

Various studies suggest that education may be the best Ses predictor of health: it is causally prior to occupation and income, is universal to all adults and is basically constant across time after young adulthood (De Irala-Estévez et al. 2000; Hupkens et al. 2000; Lahelma et al. 2004). However, compared to other Ses measures, such as income or wealth, it may be a less sensitive measure because of its narrower range and variability (Krieger and Fee 1994) and it is worth bearing in mind that the meaning and the relevance of a given level of education (expressed, for instance, in economic returns) vary by gender, race/ethnicity and birth cohort (Willson et al. 2007).

In addition to education, we use also income as a predictor of health. Both income and wealth may be relevant determinants of health since they represent greater resources, better living conditions, and increased access to quality medical care. Income may be a partial mediator of the effects of education on health, but there are mixed results concerning the continued net effect of income on education (Deaton 2002; Willson et al. 2007). We are interested in testing this effect in Switzerland.

## 3 Analytic strategy

Previous studies on the social gradient of health in Switzerland (cf. Budowski and Scherpenzeel 2005; Leu and Schellhorn 2006; Zimmerman et al. 2006) have mainly tried to understand what the best predictor of health is, whereas the principle aim of our study is to investigate the temporal dynamics of the health gradient. More precisely, we focus on the hypothesis of cumulative advantage in health, asking whether the social gradient in health varies over time (that is to say, whether there is a significant interaction between age and Ses) after controlling for cohorts effects. In order to test this hypothesis we adopt a model design very similar to that used on PSID data by Willson et al. (2007). Zimmerman et al. (2006) and Budowski and Scherpenzeel (2005) have previously studied health with Swiss longitudinal data, using, respectively, logistic regression and structural equations, whereas in this study we use growth curves models, a special type of multilevel model for change (Singer and Willett 2003), to model trajectories in individual self-rated health over the period 1999 to 2009. More specifically, we implement a two level model, where the first level is represented by repeated measures (from 1999 to 2009) that are nested into a second level represented by individuals.

We allow individual health trajectories to differ in initial status (that is to say health at the time of the first wave of observation) and annual rate of change. Subsequently, we examine the systematic variation in both initial status and rate of change as a function of education and income, controlling for some socio-demographic variables that are potential confounders.

Before running multilevel models for change, as a preliminary step it is necessary to examine the empirical growth plots in order to decide the appropriate functional form of the relationship between Self-Assessed-Health and time predictor (age) for the level 1 submodel.

The next step is to implement a model with no predictors at either level – named *unconditional means model* (Table 3, model 1) – through which we make a partition of the total outcome variation within and between subjects and calculate the intra-class correlation (rho).

At level 1, the model equation is the following:

$$Y_{ij} = \pi_{0i} + \varepsilon_{ij} \quad \text{where} \quad \varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$$

The model equation at level 2 is:

$$\pi_{0i} = \gamma_0 + \zeta_{0i} \quad \text{where} \quad \zeta_{0i} \sim N(0, \sigma_0^2)$$

The $\pi_{0i}$ parameter represents the mean SAH score of individual *i* across occasions. The $\gamma_{00}$ parameter represents the grand mean across individuals and occasions, while $\zeta_{0i}$ stands for the deviation of mean for person *i* from the grand mean.

Secondly, we implement an *unconditional growth model* (Table 3, model 2), with age as the only level-1 predictor and no time constant covariates at level 2. This model quantifies at level-1 the proportion of outcome variation "explained" by the process of ageing. In this specification, the SAH score $Y_{ij}$ is expressed as a *linear* function of time predictor. At the second-level this model expresses how the individual growth parameters (i. e. initial status and annual rate of change) differ across subjects. By inspecting the variance components and comparing this model with the previous one, we can assess the share of within-person variance explained by the linear temporal predictor.[2] The level-1 equation is specified as follows:

$$Y_{ij} = \pi_{0i} + \pi_{1i} age_{ij} + \varepsilon_{ij} \quad \text{where} \quad \varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$$

where AGE is a time varying covariate, it is measured in years at the time of each wave.

The level-2 equation is specified as follows:

$$\begin{aligned} \pi_{0i} &= \gamma_{00} + \zeta_{0i} \\ \pi_{1i} &= \gamma_{10} + \zeta_{1i} \end{aligned} \quad \text{where} \quad \begin{bmatrix} \zeta_{0i} \\ \zeta_{1i} \end{bmatrix} \sim N\left( \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_0^2 & \sigma_{01} \\ \sigma_{10} & \sigma_1^2 \end{bmatrix} \right)$$

In model 3 we predict health trajectories adding only one time-constant covariate, that is cohort, in second level equations.

The equation at level 1 is the following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} age_{ij} + \varepsilon_{ij} \quad \text{where} \quad \varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$$

At level 2 the equations are:

$$\begin{aligned} \pi_{0i} &= \gamma_{00} + \gamma_{01} cohort_i + \zeta_{0i} \\ \pi_{1i} &= \gamma_{10} + \gamma_{11} cohort_i + \zeta_{1i} \end{aligned}$$

2 We have not expressed SAH as a quadratic function of age because the effect of the squared age (a parameter through which we could capture a possible curvature) was found not statistically significant.

Finally, we run two conditional growth models (Table 3, model 4 and 5), in which we add education and then income as covariates of interest and we control for some potential confounders as gender, father's education, region of residence and nationality.

In these models we allow individual health trajectories to differ across levels of education or income, respectively. More precisely, individual growth parameters – intercepts and slopes – become level-2 outcomes, each of which can be related separately to the predictors specified in the equation.

For model 4, in which we focus on the effect of education alone, the equation at level 1 is the following:

$$Y_{ij} = \pi_{01} + \pi_{1i} age_{ij} + \pi_{2i} region_{ij} + \pi_{3i} nationality_{ij} + \varepsilon_{ij} \quad \text{where} \quad \varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$$

where region of residence and nationality are estimated as fixed effects and specified as control variables at first level because they may vary across waves.

At level 2 the equations are:

$$\pi_{0i} = \gamma_{00} + \gamma_{01} cohort_i + \gamma_{02} isced2_i + \gamma_{03} isced3_i + \gamma_{04} cohort * isced2_i + \gamma_{05} cohort * isced3_i + \gamma_{0q} control_i + \zeta_{0i}$$

$$\pi_{1i} = \gamma_{10} + \gamma_{11} cohort_i + \gamma_{12} isced2_i + \gamma_{13} isced3_i + \gamma_{14} cohort * isced2_i + \gamma_{15} cohort * isced3_i + \zeta_{1i}$$

$$\pi_{2i} = \gamma_{20}$$

$$\pi_{3i} = \gamma_{30}$$

$$\text{where} \quad \begin{bmatrix} \zeta_{0i} \\ \zeta_{1i} \end{bmatrix} \sim N\left( \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_0^2 & \sigma_{01} \\ \sigma_{10} & \sigma_1^2 \end{bmatrix} \right)$$

For model 5 in which we focus on the effect of income the equation at level 1 is the following:

$$Y_{ij} = \pi_{0i} + \pi_{1i} age_{ij} + \pi_{2i} region_{ij} + \pi_{3i} nationality_{ij} + \varepsilon_{ij} \quad \text{where} \quad \varepsilon_{ij} \sim N(0, \sigma_\varepsilon^2)$$

At level 2 the equations are:

$$\pi_{0i} = \gamma_{00} + \gamma_{01} cohort_i + \gamma_{02} income_i + \gamma_{03} cohort * income_i + \gamma_{0q} control_i + \zeta_{0i}$$

$$\pi_{1i} = \gamma_{10} + \gamma_{11} cohort_i + \gamma_{12} income_i + \gamma_{13} cohort * income_i + \zeta_{1i}$$

$$\pi_{2i} = \gamma_{20}$$

$$\pi_{3i} = \gamma_{30}$$

$$\text{where} \quad \begin{bmatrix} \zeta_{0i} \\ \zeta_{1i} \end{bmatrix} \sim N\left( \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \sigma_0^2 & \sigma_{01} \\ \sigma_{10} & \sigma_1^2 \end{bmatrix} \right)$$

## 4 Data and measurements

Data for this study come from the 1999–2009 waves of the Swiss Household Panel (SHP). More specifically, we work on an unbalanced sub-sample including all people aged between 30 and 80 years old at the time of the first observation. We keep people over 30 because, since we are interested in the effect of education on health, we prefer to focus on people who are supposed to be out of educational training. We include people up to 80 years of age because previous studies have shown that in Switzerland most of the decline in health occurs in older age (Höpflinger and Hugentobler 2005).

In our unbalanced sample we choose people who had been followed at least for three waves and at most eleven waves. We actually analyse 50926 observations corresponding to 7360 individuals (the number of average observations for individual is 6.9). Table 1 shows the frequency of participation patterns; 23.4% of the subjects have been followed for eleven waves.

This study therefore uses a short term longitudinal data set and as such it has some limits. This kind of data does not make it possible to perfectly disentangle age, period and cohort effects and forces us to ignore the possible selective survival across cohorts up to the age at which individuals entered the study. Consequently, this study examines how health trajectories develop in a very limited temporal window, where the effect of time may appear weak simply because of the nature of our data.

Our dependent variable is self assessed health[3] ranging from 1 (very well) to 5 (not well at all).

Subjective health measures have been proved to be surprisingly accurate and reliable (McDowell and Newell 1996) and self-rated health is highly correlated with mortality, morbidity and with objective measures such as functional limitations and health problems (Idler and Benyamini 1997; Ferraro and Farmer 1999; McDonough and Amick 2001; Jylha 2009). In addition to this, as a relative measure, self-rated health can effectively depict someone's health status regardless of age (Willson et al. 2007).

Our temporal predictor, the age of the subject, is measured in years at the time of each wave. We control for cohort, which is determined by a respondent's year of birth. Age is rescaled at age 50, whereas cohort is rescaled in the year 1949 in order to give the intercept a substantial meaning.

We focused on education and household income as crucial indicators of socioeconomic status.[4] The level of education is expressed in terms of International

3 The question is "We are now going to talk about various aspects of your health. How do you feel right now?"

4 As we have already explained in the theoretical framework, both income and education may be relevant for health and they should be considered separately because they may act on health through partly different pathways (cf. Ross and Mirowsky 1999; Lynch 2003; Smith 2004).

Table 1 Number of participation patterns

| Frequency | Percent | Cumulative percent | Pattern |
|---|---|---|---|
| 1720 | 23.4 | 23.4 | 1 1 1 1 1 1 1 1 1 1 1 |
| 1089 | 14.8 | 38.2 | • • • • • 1 1 1 1 1 1 |
| 360 | 4.9 | 43.1 | 1 1 1 1 1 • • • • • • |
| 324 | 4.4 | 47.5 | 1 1 1 • • • • • • • • |
| 201 | 2.7 | 50.2 | 1 1 1 1 1 1 • • • • • |
| 164 | 2.2 | 52.4 | 1 1 1 1 • • • • • • • |
| 162 | 2.2 | 54.6 | • • • • • 1 1 1 • • • |
| 122 | 1.7 | 56.3 | • • • • • 1 1 1 1 • • |
| 122 | 1.7 | 57.9 | 1 1 1 1 1 1 1 • • • • |
| 3096 | 42.1 | 100.0 | (other patterns) |
| 7360 | 100.0 | | X X X X X X X X X X X |

Source: SHP.

Standard Classification of Education (ISCED) and it has been coded in the following three categories:

1. lower secondary level (ISCED 0–2) combining: incomplete compulsory school, compulsory school, elementary vocational;
2. secondary level (ISCED 3) including: domestic science course, 1 year school, general training school, apprenticeship, full-time vocational school, bachelor and maturity;
3. third level (ISCED 4–6) combining: vocational high school with master certificate, technical or vocational school, vocational high school, university, academic high school.

Household income is coded as the mean across wave of the logarithm of the household equivalent net income. We specify a logarithmic relationship between health and income because there is a non-linear relationship according to which income

Income is supposed to mediate the relationship between education and health by facilitating access to medical care, by enabling one to purchase healthy food, vitamins, exercise equipment, a healthy house in a neighbourhood with a high quality of life and so on. However, education may also affect health through socio-psychological and behavioural pathways. Persons with different levels of education have different social psychological resources such as coping resources, social support, a sense of personal control, problem-solving skills and cognitive abilities. Moreover, highly educated people more often show healthy behaviours (exercise, better nutrition, avoidance of smoking, etc.).

changes at the lower end of the income scale have a greater effect on health than income changes at the upper part of the distribution.

In the first conditional growth model we focus on the effect of education controlling for gender[5], father's education (coded as ISCED classification), region of residence[6] (Lake Geneva, Middleland, North-West, Zurich, East-Switzerland, Central Switzerland, Ticino) and nationality (swiss and foreign nationality).

In the second conditional growth model we focus on household income controlling for the same confounders of the previous model to which we add the level of education of the subject; controlling for this important antecedent variable, we can measure the net effect of income. All these confounders are specified as time constant covariates included at level 2 of the hierarchical linear models.

Before commenting on the results of our model, it can be useful to look at the bivariate relationship between self-assessed health and all the variables that we are going to use (cf. Table 2).

As expected, we can see that health is worse for older people, for females, and for people with lower levels of education and income. Moreover, health is worse for people whose fathers have a lower level of education, for people living in the regions of Lake Geneva and Ticino and for foreign people.

## 5 Results

The unconditional means model (Table 3) shows that the grand mean of the latent factor expressing the dissatisfaction with health is equal to 2.116. In this model we assume that the individual trajectories of change are completely flat and that they may only vary in elevation around the grand mean.

The intraclass correlation is equal 0.54. This means that in Switzerland more than half of the total variation in health status lies between persons.

The next step is to develop a growth curves model including only age. The process of ageing synthetises the effect of biological and social time-varying covariates that we are not able to specify in the equation. Looking at column 2 of Table 3, we can see that with a one unit increase in age there is a worsening in self-assessed health equal to 0.027 (this is the average true rate of change).

5 Gender is a crucial control since women have a longer life expectancy but they generally report lower levels of self-rated health and suffer more from chronic illness and disability (Cockerham 2007; Bird and Rieker 2008; Jylha 2009).

6 Different studies have demonstrated that the socio-economic status of the area of residence has its own – albeit small – effect on health, independent of the effect of individual Ses ( Robert and House 2000). It is also important to consider neighbourhood and region because they often differ in terms of social policies (and hence in the provided services and infrastructures); these differences are likely to affect health.

Table 2 Self rated health by socio-demographic characteristics at time of first observation

| Characteristics | Mean | Standard deviation |
|---|---|---|
| Age | | |
| 30–40 | 1.6 | 1.7 |
| 41–50 | 1.9 | 2.0 |
| 51–60 | 2.1 | 2.00 |
| 61–70 | 2.2 | 1.9 |
| 71–80 | 2.5 | 2.0 |
| Gender | | |
| male | 1.9 | 1.8 |
| female | 1.9 | 2.0 |
| Education | | |
| ISCED1 | 2.5 | 2.4 |
| ISCED2 | 1.9 | 1.9 |
| ISCED3 | 1.8 | 1.8 |
| Household income quintile* | | |
| 1 quintile | 2.1 | 2.2 |
| 2 quintile | 2.0 | 1.9 |
| 3 quintile | 1.8 | 1.8 |
| 4 quintile | 1.9 | 1.8 |
| 5 quintile | 1.7 | 1.7 |
| Father's education | | |
| ISCED1 | 2.1 | 2.0 |
| ISCED2 | 1.8 | 1.8 |
| ISCED3 | 1.9 | 1.8 |
| Region of residence | | |
| Lake Geneva | 2.1 | 2.1 |
| Middleland | 1.9 | 1.9 |
| North-West | 1.9 | 1.9 |
| Zurich | 1.9 | 1.8 |
| East-Switzerland | 1.8 | 1.8 |
| Central Switzerland | 1.8 | 1.8 |
| Ticino | 2.0 | 2.1 |
| Nationality | | |
| Foreign | 2.2 | 2.1 |
| Swiss | 1.9 | 1.9 |

* Yearly household income equalised, oecd, net.

Source: SHP.

Comparing this model to the first one, we can also see a proportional reduction in the first level variance component equal to 0.02, meaning that 2% of the individual's variation in health is associated with linear time.

In the third model we add only cohort as a second level predictor in order to describe age trajectories of health controlling for differences by cohorts. Controlling for age, younger cohorts display worse health (b = +0.030). The decline in health associated with the ageing process is not uniform across cohorts: in younger cohorts health worsens at a faster rate over time (0.048 + 0.0002).

In the first conditional growth model (Table 3, model 4), we focus on the effect of education allowing people belonging to different groups formed on the basis of the heterogeneity factors specified in the equation to have different trajectories of health. The results of this model are shown here below.

As already known in literature, we can see that in contemporary Switzerland having a second and third level education is a protective factor for health (b = –0.413 and b = –0.526). Whereas a male aged 50, with a primary education, and with a father also having a primary education, living in the region of Geneva and with Swiss nationality, has an initial health score of 2.695 (the intercept); the same subject with a second level education has a score equal to 2.282 (2.695–0.413); with a third level education this same subject has a score of 2.169 (2.695–0.526). In particular both the effects of secondary and tertiary education on health initial status appear to be statistically significant.

Regarding the interactions between education and the temporal predictor, we can see that they are not statistically significant for both tertiary and secondary education (Table 3, model 4). This means that the effect of education on health is stable over time (at least in our 11 years temporal window of observation).[7] However we should remember that with this kind of short-term data we are not able to definitively reject the cumulative advantage theory.

In the next model (Table 3, model 5) we examine the effect of household income on health net of education and controlling for other confounders. There is a strong evidence of a protective effect of income on self-assessed health: the parameter b (–0.453) expresses the variation of self-assessed health for each unit increase in the logarithm of the mean household equivalent net income for the reference group[8].

7 All the tests illustrated in table 3 involve only one restriction; however, if we want to test a hypothesis involving multiple restrictions on the coefficient vector we need to do a joint test. In this case we would like to know if the following interaction terms are jointly zero: a) interaction between ISCED2 and age; b) interaction between ISCED3 and age; c) interaction between ISCED2 and cohort; d) interaction between ISCED3 and cohort; e) interaction between ISCED2, age and cohort; f) interaction between ISCED3, age and cohort. The joint Chi2 statistic with 6 degrees of freedom (as many as the restrictions on the coefficient vector) has a value of 4.17, and a probability of 0.6542; so we cannot reject the null hypothesis at the 5% level that all the coefficients above are jointly equal to zero.

8 We make a joint test involving restrictions on the following parameters: a) income, b) interaction between income and age, c) interaction between income and cohort; d) interaction between income, age and cohort. We obtain a Chi2 equal to 81,5 with 4 degrees of freedom. Since

Table 3 Multilevel models for change: estimates of the parameters b expressing the net effects of covariates and their standard errors (in brackets)

| | Model 1 uncond. mean | Model 2 uncond. growth | Model 3 only cohort | Model 4 education | Model 5 income |
|---|---|---|---|---|---|
| Intercept | 2.116*** | 2.072*** | 1.965*** | 2.695*** | 2.550*** |
| | (0.017) | (0.017) | (0.025) | (0.094) | (0.083) |
| Age | | 0.027*** | 0.048*** | 0.043*** | 0.049*** |
| | | (0.001) | (0.002) | (0.007) | (0.002) |
| Cohort | | | 0.030*** | 0.022*** | 0.031*** |
| | | | (0.002) | (0.008) | (0.003) |
| age*cohort | | | 0.0002* | 0.0005 | 0.0003*** |
| | | | (0.0001) | (0.0003) | (0.0001) |
| Female | | | | 0.098** | 0.084* |
| | | | | (0.036) | (0.036) |
| ISCED1 (ref.) | | | | | |
| ISCED2 | | | | –0.413*** | –0.302*** |
| | | | | (0.081) | (0.063) |
| ISCED3 | | | | –0.526*** | –0.321*** |
| | | | | (0.090) | (0.070) |
| ISCED2*age | | | | 0.006 | |
| | | | | (0.007) | |
| ISCED3*age | | | | 0.009 | |
| | | | | (0.008) | |
| ISCED2*cohort | | | | 0.009 | |
| | | | | (0.008) | |
| ISCED3*cohort | | | | 0.014 | |
| | | | | (0.009) | |
| ISCED2*age*cohort | | | | –0.0002 | |
| | | | | (0.0003) | |
| ISCED3*age*cohort | | | | –0.0004 | |
| | | | | (0.0003) | |
| log_av_income[a] | | | | | –0.453*** |
| | | | | | (0.056) |
| log_av_income*age | | | | | 0.016** |
| | | | | | (0.005) |
| log_av_income*cohort | | | | | 0.025*** |
| | | | | | (0.006) |
| log_av_ income*age*cohort | | | | | –0.0003 |
| | | | | | (0.0002) |

*Continuation of Table 3 on the following page.*

the probability to observe this value under the null hypothesis is 0.000, we can reject the null hypothesis that the above specified parameters are jointly zero.

*Continuation of Table 3.*

| | Model 1 uncond. mean | Model 2 uncond. growth | Model 3 only cohort | Model 4 education | Model 5 income |
|---|---|---|---|---|---|
| ISCED1_father (ref) | | | | | |
| ISCED2_father | | | | –0.027 | –0.001 |
| | | | | (0.042) | (0.043) |
| ISCED3_father | | | | 0.014 | 0.063 |
| | | | | (0.053) | (0.054) |
| Lake Geneva (ref.) | | | | | |
| Middleland | | | | –0.042 | –0.063 |
| | | | | (0.051) | (0.052) |
| North-West | | | | –0.061 | –0.059 |
| | | | | (0.059) | (0.060) |
| Zurich | | | | –0.095 | –0.068 |
| | | | | (0.056) | (0.057) |
| East-Switzerland | | | | –0.178** | –0.206** |
| | | | | (0.059) | (0.060) |
| Central Switzerland | | | | –0.127 | –0.139* |
| | | | | (0.065) | (0.066) |
| Ticino | | | | –0.046 | –0.080 |
| | | | | (0.092) | (0.094) |
| Swiss_nationality | | | | –0.352*** | –0.325*** |
| | | | | (0.052) | (0.053) |
| Var(age) | | 0.001*** | 0.001*** | 0.001*** | 0.0009*** |
| | | (0.000) | (0.000) | (0.000) | (0.0002) |
| var(intercept) | 1.824*** | 1.679*** | 1.670*** | 1.596*** | 1.567*** |
| | (0.035) | (0.036) | (0.036) | (0.036) | (0.037) |
| cov(age,_intercept) | | 0.006*** | 0.006*** | 0.006*** | 0.007*** |
| | | (0.001) | (0.001) | (0.002) | (0.002) |
| var(l level res.) | 1.576*** | 1.542*** | 1.539*** | 1.516*** | 1.493*** |
| | (0.011) | (0.011) | (0.011) | (0.011) | (0.012) |
| rho | .536 | | | | |
| Log-likelihood | –91 675.3 | –91 350.2 | –91 276.0 | –84 277.1 | –75 013.5 |
| Wald $chi^2(21)$ | | 532.55 | 711.41 | 829.9 | 853.8 |
| Prob > $chi^2$ | | 0.0000 | 0.0000 | 0.0000 | 0.0000 |
| n.observations | 50 926 | 50 926 | 50 926 | 47 244 | 42 086 |
| n.of groups | 7 360 | 7 360 | 7 360 | 6 778 | 6 645 |
| Average obs. per group | 6.9 | 6.9 | 6.9 | 7.0 | 6.3 |

* P < .05, ** P < .01, *** P < .001.
a log_av_income: logarithm of the average household equivalent net income.
Source: SHP.

However, this protective effect appears to weaken in younger cohorts (b = 0.025). As concerning the interaction between income and age, we can see that people with higher household income experience a faster health decline in contrast to those with a lower household income (b = 0.016).[9] This result seem to confirm the age as leveller hypothesis, however we need to be cautious in our conclusion because we work with short term longitudinal data.

## 6 Conclusions

We propose a multilevel analysis for longitudinal data with the main aim of assessing whether individual health trajectories vary significantly on the basis of two crucial indicators of socio-economic status (education and household income) and whether the possible effects of these indicators vary over the course of a lifetime, according to the hypothesis of cumulative advantage.

While different American studies (cf. Lynch 2003, Willson et al. 2007; Mirowsky and Ross 2008) have found support for this hypothesis, we cannot entirely support it on the basis of our data and models.

As for the effect of education on health, our predictions show a fanning out in the older cohorts, meaning that health trajectories tend to become more heterogeneous as people age. However in Switzerland the interactions terms expressing the temporal variation of the gradient are not statistically significant, suggesting that the effect of education on health may be stable over time. Hence, we cannot conclusively support the cumulative advantage theory, at least with our limited temporal window that may be too short to observe a significant change in the protective effect of education.

As regards the effect of household income on health, our data confirm the existence of an economic gradient in health and our predictions show that this advantage decreases over time, at least in the younger cohorts. This result prompts us to drop the cumulative advantage hypothesis in favour of the age as leveller hypothesis. Again, we should stress that, because of our data limitation, our conclusions may need to be revised when long term panel data (which permit the observation of different birth cohorts at the same ages) will be available for Switzerland and better analyses will be possible.

Further development in the study of the social gradient in health concerns the ability to control for genetic factors in order to measure in the most accurate way the effect of environmental variables (such as Ses) on health, a phenotypic trait with a strong

9 We make another joint test involving restrictions on the interaction terms only: a) interaction between income and age, b) interaction between income and cohort; c) interaction between income, age and cohort. In this case we obtain a Chi2 equal to 20.45 with 3 degrees of freedom and a probability to observe this value under the null hypothesis of 0.0001. Hence we can reject the null hypothesis that the above specified parameters are jointly zero.

biological base. Social surveys are increasingly including bio and genetic markers that may be used for this purpose in the near future (for further information, see *Biosocial Survey* [Committee on Advances in Collecting and Utilizing Biological Indicators and Genetic Information in Social Science Surveys et al. 2008]). Considering genetic effect as a black box, behavioural genetics models already make it possible to distinguish between genetic and environmental components that account for the variability observed among individuals for a certain trait. However, these models focus on variation rather than means and much work is needed in order to understand how to model mean values of a trait and control for genetic heterogeneity without the use of biomarkers.

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