The effect of compulsory schooling on health – evidence from biomarkers*

Hendrik Jürges: Schumpeter School of Business and Economics, University of Wuppertal, Rainer-Gruenter-Str. 21 (FN.01), 42119 Wuppertal, Germany.
Email: juerges@uni-wuppertal.de

Eberhard Kruk: Munich Center for the Economics of Aging, Max-Planck-Institute for Social Law and Social Policy, Amalienstr. 33, 80799 Munich, Germany.

Steffen Reinhold: Department of Economics, University of Mannheim, L7 3-5, 68131 Mannheim, Germany.

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Abstract: Using data from the Health Survey for England and the English Longitudinal Study on Ageing, we estimate causal effects of schooling on health. Our study complements earlier studies exploiting two nation-wide increases in British compulsory school leaving age in 1947 and 1973, respectively, by using biological stress markers as measures of health outcomes in addition to self-reported measures. We find a strong positive correlation between education and health, both self-rated and measured by blood fibrinogen and C-reactive protein levels. However, causal effects estimates based on compulsory schooling changes are ambiguous and remain statistically insignificant.

JEL-Classification: I12, I20.
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* Corresponding author: Hendrik Jürges, Schumpeter School of Business and Economics, University of Wuppertal, Rainer-Gruenter-Str. 21 (FN.01), 42119 Wuppertal, Germany; email: juerges@uni-wuppertal.de. We thank two anonymous referees and the editor for helpful comments.

1. Introduction

In this paper, we aim at contributing to the growing literature on the causal link between education and health. Theoretically, the economic literature has identified causal effects of education on health through at least four plausible channels: (a) just as in the labor market, education raises efficiency in health production (raises the marginal productivity of inputs), i.e. it increases an individual's productive efficiency (Grossman 1972); (b) education changes inputs into health production (through information) and thereby increases allocative efficiency (Rosenzweig and Schulz 1981); (c) education itself changes time preference (and thus inputs into health production) because schooling focuses students' attention on the future (Fuchs 1982, Becker & Mulligan 1997); (d) education has an indirect effect mediated through higher income, occupational status, access to better housing, environmental conditions, and many other factors (Lochner 2011).

Numerous studies have indeed documented a strong positive empirical association between education and health (see the surveys by Cutler and Lleras Muney 2008 or Grossman 2006). Interpretation of this correlation as causal is difficult, however, because education is most likely an endogenous variable, for instance because unobserved variables such as time preferences possibly drive both education and health behavior decisions, or because health (at younger ages) affects educational achievement (reverse causation). Recent empirical work addresses causality issues head on using natural experiments such as exogenous changes in compulsory schooling laws for identification.[1] Moreover, identification of causal channels in the education-health relationship is viewed as an important part of the future research agenda. (Grossman 2006; Lochner 2011).

We study the possible causal link between education and health using two nationwide changes in minimum school leaving age in England in 1947 and 1973 as sources of exogenous variation. In those years, minimum school leaving age was raised from from 14 to 15 (affecting birth cohorts born in or after April 1933) and from 15 to 16 years (affecting birth cohorts born in or after September 1957), respectively. Both reforms have already been used in previous studies to study causal effects of education on wages (Oreopoulos 2006; Devereux and Hart 2008), political participation (Milligan et al. 2004), and teenage fertility (Silles 2011). We are also not the first to exploit these reforms for causal analyses of education on

health (see e.g. Oreopoulos 2006; Silles 2009; Clark and Royer 2010; Lindeboom et al. 2009; Powdthavee 2010). For instance, Oreopoulos (2006) reports positive effects of this reform on self-rated health (and a range of labor market outcomes) in the combined UK General Household Surveys from 1983 to 1998, but later re-analysis of this data revealed no effects of education on self-rated health.[2] Critique concerning the external validity of such studies and their value for current policy recommendations could come from the fact that cohorts affected by the reform were born some 75 years ago. Education policy today might have a very different effect. Silles (2009) extends Oreopoulos's analysis and includes cohorts affected by the 1973 reform. Comparing the effects of the two reforms that were 26 years apart might give us some indication whether (causal) education effects on health are stable over time. In fact, Silles (2009) finds positive significant causal effects of education on self-rated health when considering both reforms simultaneously. However, when analyzing the 1973 reform and restricting the sample to younger cohorts she does not find a causal effect. Thus it appears as if the effect has become less relevant. In the most comprehensive analysis of the English compulsory schooling reforms with respect to health outcomes to date, Clark and Royer (2010) use UK vital statistics and data from the Health Survey for England and find very small – not always significant – positive effects of the reforms on mortality, self-rated health, health behaviors, BMI and blood pressure. Overall, the available evidence so far provides only inconsistent support for a causal education effect on health at the compulsory education threshold.

While we analyze the effect of the same reforms as the above authors, partly using the same data, we add to the empirical evidence in a number of ways. The first goal of our paper is to complement the earlier analyses – that have mainly relied on self-reported health measures – by using objective measures of health (biomarkers) as outcomes.[3] An important recent development in survey research is the integration of biomarkers. Biomarkers are often associated with genetic information, i.e., DNA samples. However, the vast majority of biomarkers currently collected and analyzed are non-genetic: anthropometric measurements (height, weight, waist, lung capacity, grip strength, balance), blood pressure, and blood and

[1] See e.g. Adams (2002), Albouy and Lequin (2008), Arendt (2005), Clark and Royer (2010), Lleras-Muney (2005) Oreopoulos (2006), Silles (2009), and Kemptner et al. (2011).
[2] See corrigendum available on http://www.aeaweb.org/articles.php?doi=10.1257/000282806776157641
[3] One recent paper using a objective health measure is by Powdthavee (2010), who uses measured hypertension as, also exploiting the UK education reforms as sources of exogenous variation in health. He finds very large negative effects of one additional year of schooling on the incidence of hypertension among men.

saliva samples. The scientific value of collecting such biomarkers in large surveys is promising (National Research Council 2008).

First, biomarkers improve the measurement of health. Self-reports of health are subject to considerable under-, over-, or misreporting, depending on the circumstances and dimensions at hand (e.g. Jürges 2007, 2008; Bago d'Uva et al. 2008). Objective information can be used to validate respondents' reports and to study the amount and determinants of under-, over-, or misreporting in population surveys. Self-ratings of health may be subject to reporting bias that is correlated with important determinants of health. However, self-reports of health have their own distinct scientific value. For instance, it has been shown that they contain information on health status (e.g. as predictors of mortality) even after conditioning on objective measures of health (Idler and Benyamini 1997). Thus, biomarkers should be seen as complementary measurements rather than substitutes. The value of self-assessments alone as policy outcome measures is less clear, however. It would be hard to evaluate the benefits of a health care reform, say, that improves self-assessed health but leaves more objective measures of health unchanged.

Second, biomarkers provide direct information on pre-disease pathways, in particular by measuring physiological processes that are below the individual's threshold of perception. This could be important for finding causal effects of education on the health at younger ages when diseases have not yet become manifest. Most of the existing studies that exploit the UK education reforms for causal analyses look at contemporaneous health. The biomarkers used in our study as health indicators can be interpreted as markers of *future* health. The medical literature shows they are associated with future adverse health events, particularly with respect to cardiovascular disease (see below). Thus although a comprehensive study such as Clark and Royer (2010) does not find large significant effects of the UK education reforms on contemporaneous health (at the age of about 65 to 70), there might still be effects on future health, and our study could be able to pick up significant education differences in pre-disease indicators.

In our analyses, we concentrate on two biomarkers for inflammatory processes: blood fibrinogen, a blood-clotting factor, and blood C-reactive protein (CRP), a protein released into the bloodstream when there is active inflammation in the body. Inflammation, in turn, is a major feature of atherosclerotic plaques, which causes adverse cardiovascular events such as

angina pectoris, heart attacks, and strokes. Consequently, both fibrinogen and C-reactive protein have gained much interest in the medical literature as predictors of incident cardiovascular disease (for reviews of the literature see e.g. Kamath and Lip 2003 and Hirschfield and Pepys 2003). Elevated levels of fibrinogen and CRP have been shown to be strong, independent predictors of weight gain (Duncan et al. 2000), incident diabetes (Pradhan et al. 2001), or cardiovascular events (Ridker et al. 2002, 2003). Whether these associations are causal could not yet be established (Davey Smith et al. 2004). Still, for the medical practitioner such findings suggests that patients who would benefit most from interventions targeting blood pressure and cholesterol lowering, smoking cessation or exercise promotion, could be identified by (high) blood fibrinogen and CRP levels.

Finally, biomarkers allow studying physiological pathways in the complex relationship between social status and health, providing information on important links that can be used to identify causal relationships. Our paper can be viewed as a step in the direction of finding the causal channels between education and health. In a recent review paper, Lochner (2011) discusses eight different channels through which education may improve health of which several may operate via physiological markers such as fibrinogen and C-reactive protein. The first channel by which education might improve health, prominent in the socio-epidemiological literature, is reduced "environmental stress" or "psychosocial stress". This is due to improvements in social standing (e.g. job autonomy) or socioeconomic well-being (e.g. income). Better educated people have higher status jobs, are more autonomous in their work, and are subject to less external demands. Psychosocial stress, in turn, has been shown to be associated with elevated levels of blood fibrinogen and CRP. Although psychosocial stress can be measured by subjective survey responses, we believe that biomarkers have their advantages and we try to exploit those in our study. First, they naturally measure the cumulative amount of stress; second they are arguably free from the kind of (self-serving) biases that might plague subjective survey responses.[4] Stress-related biomarkers such as fibrinogen and C-reactive protein can thus be used as more "objective" indicators of long-term exposure to environmental stress. The medical and public health literature shows that the biomarkers we analyze in our paper are indeed associated with job stress (Theorell 2002), job control (Steptoe et al. 2003), and daily interpersonal stress (Fuligni et al. 2009). These factors are related to occupational choice, which in turn is dependent on education. Consistent with

[4] Again, this is not to say that subjective measures are useless. We rather stress that we use complementary evidence to study the education-health nexus.

this reasoning, Morefield et al. (2011) and Kelly et al. (2011) find that occupational choice is a relevant determinant of health outcomes.

Another possible channel by which education can improve health – and for which our biomarkers could be informative – is healthier life-style. Better educated individuals smoke less and cigarette smoking is strongly associated with increased fibrinogen and CRP levels. Further, there appears to be a U-shaped association between alcohol consumption and fibrinogen levels, with the lowest fibrinogen levels at moderate alcohol consumption levels.

A number of epidemiological studies have shown associations of fibrinogen and CRP levels with markers of social status. For instance, fibrinogen concentration has been shown to be linked with childhood environmental conditions (measured by adult height and parental socioeconomic status), education level (Brunner et al. 1996), or subjective social status (Demakakos et al. 2008). Higher CRP levels have also been shown to be associated with subjective social status (Demakakos et al. 2008). However, none of these studies has addressed the issue of causal effects in an econometric framework. Our work intends to fill this gap in the literature.

Although we believe that using biomarkers to measure current and future health in economic studies has its virtues, some words of caution are in order. First, biomarkers represent "surrogate" outcomes. As social scientist, we usually do not care about these outcomes as such but in the clinical endpoints that are correlated with them, and a link between a "treatment" and surrogate outcome does not always imply a link between the treatment and the clinical endpoint. Second, the biomarkers we use in our study are linked with a fairly narrow range of health outcomes (cardiovascular) and not health generally. Taken together with the fact that we exploit two specific changes in the number of years of compulsory schooling, we certainly cannot claim to study the effect of education in general on health in general. In other words, external validity is limited both in terms of our identification strategy and in terms of our outcome measure (when using biomarkers).

Our study proceeds as follows. In the next section, we will briefly describe the school reforms analyzed in this paper and their effect on educational attainment. In Section 3, we explain the identification strategy (instrumental variables) which we use to exploit these reforms. The fourth section describes the data and shows some descriptive results on the correlation

between education and self-rated health, blood fibrinogen and blood CRP levels. Section 5 contains the causal estimates and some conventional robustness checks. We discuss our results and give conclusions in Section 6.

2. Institutional background

We now briefly describe the most salient aspects of the changes in schooling laws in Britain that we use for identification. The first change in minimum school leaving age analyzed in our paper was part of the 1944 Education Act and took effect on April 1st, 1947. Individuals who were born before April 1933 and who turned 14 before the law change could leave school at the end of the term in which they turned 14 (the school year was divided into three terms). Individuals who were born in April 1933 or later and who turned 14 after the law change had to stay in school until the end of the term in which they turned 15, i.e. at least until Summer 1948. This law change had a dramatic effect on the average age at which British pupils left school (see below). In 1973, minimum school leaving age was again raised, from 15 to 16, by the Raising of the School Leaving Age (ROSLA) Order of 1972. This reform affected pupils born on or after September 1st, 1957.

Figure 1 illustrates the effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment (these data are from the combined HSE/ELSA samples described below). For both reforms, we show the percentage of pupils who have finished school at age 14, 15 and 16, respectively, for birth cohorts born 5 years before to 5 years after the first cohort that was affected by the reform. Among pre-1947 reform cohorts, roughly 60 percent left school at the age of 14, and 10 percent left school at age 15. The relationship between the two proportions practically reverses after the reform. About 55 percent of each cohort left school at age 15. 7 percent of those immediately affected by the reform still left school at age 14. In principle, nobody born in or after April should report a school leaving age of 14. This is not the case however, which might be due to misreporting, individual non-compliance, or districts failing to provide sufficient school places immediately after the reform – as pointed out by Clark and Royer (2010). Over the years, this proportion decreased to 4 percent for the cohorts born after the first quarter of 1933. It is interesting to note the effect of the reform on the average number of years in school (see Figure 2). Education has been on a secular increase for men and women. The 1947 reform has boosted this increase further but the

increase at the discontinuity was larger for women than men. Average school leaving age has jumped by about 0.4 years for men and 0.6 years for women.

--- about here Figures 1 and 2 ---

The pattern of change we find for the 1973 reform is different (see lower panel of Figure 1). Of the pre-1973 reform cohorts, 32 percent on average left school at the age of 15. A similar proportion, 30 percent, left school at the age of 16. After the reform, the percentage of pupils leaving at age 16 increased to about 52 percent whereas the proportion of those leaving at age 15 became negligible (roughly 7 percent). During the observation period, the average number of years in school (Figure 2) was fairly stable for men, except for the jump of about 0.35 years induced by the 1973 reform. Education of women was still on the increase and the 1973 reform apparently only had a fairly small effect on average years in school.

Based on the described reforms, we aim at identifying the effect of schooling on health by comparing health outcomes of individuals born until March 1933 to those born in or after April 1933 and of those born in or after September 1957 to those born until August 1957. The assumption underlying our empirical approach that allows identifying a causal effect, and which is described in the next section, is that there are no unobserved cohort-level determinants of health that have changed at the time of the reform.

3. Econometric method

The nature of the two reforms analyzed in this paper makes them a candidate for a regression discontinuity design (RD). The idea of the RD approach is that the probability of receiving a particular treatment (here: an additional year of schooling) is a discontinuous function of a continuous treatment-determining variable (here: day of birth). This allows to estimate causal effects of the treatment by comparing outcomes (here: health) for individuals just below and just above the treatment threshold (for an overview of recent econometric developments concerning the RD design see Imbens and Lemieux (2008) and Lee and Lemieux (2009)). As documented in the preceding section, the treatment in our application is not purely assigned on the basis of the birth date (i.e. the treatment is under partial control of the individuals). After both reforms, some individuals left school at younger ages than the legal school leaving age (at least so they said in the HSE and ELSA). They thus did not receive the treatment after

the threshold date, i.e. the probability of treatment did not jump to 1. In such cases, a so-called "fuzzy" RD (FRD) design becomes appropriate. In case of a binary treatment, the FRD design is basically a Wald estimator. To see this, let Y be the health outcome, X be the date of birth as treatment-determining variable, W be the treatment received, and value c be the threshold value of the treatment-determining variable, then the FRD estimator can be written as (Imbens and Lemieux 2008):

$$\tau_{FRD} = \frac{\lim_{x \downarrow c} E(Y \mid X = x) - \lim_{x \uparrow c} E(Y \mid X = x)}{\lim_{x \downarrow c} E(W \mid X = x) - \lim_{x \uparrow c} E(W \mid X = x)} \quad (1)$$

Under certain assumptions (monotonicity or no defiers, i.e. individuals do not leave school earlier *because* of the reform), and by taking limits from above and below the threshold value c, τ_{FRD} identifies the average treatment effect on the treated (averaged across all compliers at the threshold c). Take the 1947 reform as an example. If sample size was no problem, then equation (1) would tell us to just compare the average health of all individuals born on April 1st, 1933 with outcomes of all individuals born on March 31st, 1933 and divide the difference (the numerator) by the difference in average school leaving ages of those two groups of individuals (the denominator). However, sample size at the discontinuity almost always is a problem. For instance, in our pooled sample described below, we have 54 individuals each born in March and April 1933 with valid fibrinogen values. Finding significant health effects of education for such small samples is virtually impossible. The task is thus to appropriately estimate average outcomes and treatments *at the discontinuity* using observations that are *further away from the discontinuity*, for instance using all observations that are born four years before and after the threshold.

Equation (1) shows the fuzzy regression discontinuity estimator written as a Wald or IV estimator. FRD and IV differ in their assumptions about what is happening in the neighborhood of the threshold (cf. Lee and Lemieux 2009). In order for RD to be valid, the distribution of observable *covariates* should not change discontinuously at the threshold, just as the distribution of observable covariates should not differ across treatment and control group in a randomized experiment. Covariates that show jumps at the threshold indicate a non-random allocation of individuals left and right of the threshold. We found at least one important pre-treatment covariate (adult height) that shows some discontinuity at the threshold. Thus our estimator – although numerically equivalent to a FRD estimator– recovers

the causal effect of education under slightly different assumptions, namely that being born after the threshold is a valid instrument for years of education *conditional on observed covariates* as in regular instrumental variables approach. In contrast to a valid regression discontinuity design, the choice of covariates can matter for the results and the plausibility of the identifying assumption.

In our regression analyses shown below, we will use information on season of birth (spring, summer, fall, winter) and adult height to control for both the economic and disease environment in utero and early childhood. Both have been shown to have long-lasting effects on adult health. For instance, season of birth is related to mortality. Doblhammer and Vaupel (2001) show that in the Northern Hemisphere people born in fall (October–December) live longer than those born in spring (April–June). In the Southern Hemisphere, the pattern reversed. Adult height reflects the accumulated nutritional experience during childhood including the fetal period, and is shown to have considerable predictive power both for morbidity and mortality (see Fogel 1997, Deaton 2007) and also educational outcomes (Magnusson et al. 2006). Controlling for height hence serves two purposes. First, in the descriptive (OLS) regressions, inclusion of height captures the effect of a potentially important third factor (childhood conditions) driving both adult health and educational outcomes. We should again stress at this point that adult height is practically determined before schooling decisions are made, either by its genetic component or by early childhood environment. Second, in the instrumental variables regressions, inclusion of height also helps controlling for unobserved cohort effects that cannot readily be captured by (local) polynomial cohort trends. Note that the first cohort affected by the first reform was born in 1933, i.e. in the immediate aftermath of the great depression, and it is a priori unclear if and how the depression has effected childhood environment (and thus adult health and education) of the cohorts in our analytical sample. For instance, we find some indication in our data that, also after controlling for cohort trends, children born after March 1933 are slightly taller than older cohorts.

Despite the fact that our identification is not strictly RD, we follow some of the recommendations for conventional robustness checks in RD designs, such as modelling the long-run relationship between the treatment-determining variable and the outcomes by local linear regression, i.e. linear regressions of Y on x separately for individuals below and above the threshold (within some bandwidth h) and to predict Y at the threshold value of the

treatment-determining variable. Alternatively, we choose some parametric form, such as a fourth-order polynomials. Alternative specifications should lend credibility not only to a RD but also to our IV approach. One practical issue is to choose the appropriate bandwidth for the local or global regression. When we show our results we arbitrarily choose one bandwidth (4 years) and estimate local linear regressions. We present results using alternative bandwidths and alternative parametric specifications in Section 5 as part of our robustness checks. Finally, as suggested in Lee and Card (2008), all standard errors are cluster-corrected, where clusters are given by each value of the treatment-determining variable (month of birth).

4. Data and descriptive results

We use data from the Health Surveys for England (HSE) 1993, 1994, 1998 to 2000, and 2003 to 2006 and the English Longitudinal Study on Ageing (ELSA) 2006. The Health Survey for England is an annual health interview survey of around 15,000 to 20,000 respondents in England conducted by the National Centre for Social Research (separate surveys are available for Scotland and Wales). The English Longitudinal Study on Ageing is an ongoing multi-disciplinary panel survey of the older population covering around 12,000 respondents in England. It was started in 2004 based on a sample that was derived from three waves of the Health Surveys for England 1998, 1999 and 2001. Part of our ELSA sample consists of respondents already present in the HSE 1998 and 1999, i.e. some individuals are represented twice in our data. We are, however, not able to identify these respondents present in both data sources. In fact, the data use contract explicitly forbids re-identification of such respondents. The data are distributed by the Economic and Social Data Service (ESDS). We restrict our analyses to the survey years listed above because only data from these years contain information on blood fibrinogen and CRP levels. Biomarkers are collected during nurse visits after the actual health interview and include not only blood samples but also anthropometric measurements, blood pressure measurements, and saliva samples.

We further restrict our analytical samples in two ways. First, for most of our analyses we use only birth cohorts that are born at most 4 years before and after the two relevant thresholds April 1933 and September 1957 (we lift this sample restriction when we try different bandwidth in our regression approach). Second, we eliminate from our sample all respondents who were not born in England, Wales, or Scotland, i.e. respondents for whom it is unclear if they have been in the British school system at the time of the reform.

We use two main health outcome measures: blood fibrinogen levels and blood C-reactive protein levels: For comparison with earlier studies we also analyze effects of education on self-rated general health (dichotomized to good/poor health). The blood fibrinogen level is measured in grams per liter and the blood C-reactive protein level is measured in mg per liter. One difficulty with combining biomarkers spanning more than 10 years of data collection is that measurements are not necessarily comparable across years due to changes in collection methods, assays, and laboratories. Indeed, the HSE user guide explicitly warns against comparing biomarker levels over time. In order to make our data compatible for use in a pooled data set, we have standardized all measurements to have the means and standard deviations of the 1998 measurement. Moreover, all analyzes conducted include survey year fixed effects to account for differences in data collection methods over time. By controlling for survey year fixed effects, we effectively use only the within-survey year variation of biomarker measures.

As discussed in the introduction, higher levels of fibrinogen and CRP indicate the presence of inflammatory processes and have been shown to be associated with higher risks of obesity, diabetes and cardiovascular disease. In accordance with other studies analyzing the relationship of socio-economic status and CRP levels, we exclude cases with a CRP level of over10 mg/L from further analysis. In cases of acute inflammation CRP values can increase by as much as 10,000-fold. High CRP values might thus relate to acute inflammation and not be informative of chronic pathogenic processes (Pearson et al., 2003). Including these cases in the data potentially biases our results.

--- about here Table 1 ---

Table 1 briefly describes the analytical samples, separately for the 1947 reform cohorts (born between 1929 and 1937) and the 1973 reform cohorts (born between 1953 and 1961). Columns (1) to (4) show descriptive statistics for the full samples. The average age at the date of the survey in older cohorts is 66 years for men and 67 years for women. In the younger sample it is 41 years for both sexes. The average age at which respondents left school has increased substantially from 15.4 years for the older cohorts to 16.7 years for the younger cohorts. The proportion of respondents who reported to be in poor health is 36 percent (men) and 34 percent (women) among the 1947 reform cohorts and 17 percent (men) and 19 percent

(women) among the 1973 reform cohorts. Log fibrinogen and log CRP levels are slightly higher among women and lower in the younger cohorts. Table 1 also shows that both men and women in the 1947 reform cohorts are on average 4 cm shorter than men and women in the 1973 reform cohorts.

The number of observations with valid information on fibrinogen and CRP levels is substantially lower than the full samples. Not all HSE respondents have given consent to be visited by a nurse or to have blood samples taken. Sometimes, respondents are not eligible for blood testing because of medical or other reasons. Further, it is sometimes not possible to identify blood values from samples taken from respondents and finally, some results are invalid for analysis because respondents take medication that affects blood fibrinogen or blood CRP levels. Especially non-compliance on the part of the respondents or medical ineligibility might be a cause of worry due to selection effects. Rather than dealing with this issue formally at this stage, we simply look at differences in average sample characteristics between those with valid fibrinogen/CRP levels and the full samples. As it turns out, the full sample and the sample with valid blood test data are very similar as far as observable characteristics are concerned (a more detailed analysis of participation in the nurse visit is presented in the Appendix). In addition, we checked whether the reform itself has changed the probability of having a valid fibrinogen/CRP value recorded in the data. We found fairly precisely effected zero effects. Still, to get some information on the possible effect of differences between the full sample and the nurse visit sample on our regression results, we also estimated all regressions using self-rated health as outcome but restricting the sample to those also participating in the nurse visit. We find only small changes in our results, so that we believe that sample selectivity should not be a cause of concern.

Associations between self-rated health and biomarkers

To illustrate the correlation of traditional health measures such as self-rated health and the biomarkers used in the present study, Table 2 shows average levels of (log) blood fibrinogen and (log) blood CRP for different levels of self-rated health, separately for the two analytical cohorts. Within each cohort and for both measures, we find a clear gradient with higher levels of fibrinogen and CRP for respondents who self-report worse health (fibrinogen and CRP levels are also correlated with each other, $r = 0.50$). The younger cohorts generally have lower values than the older cohorts even when reporting the same level of self-rated health,

reflecting lower risk of cardiovascular disease. Members of the younger cohort who report to be in poor health have higher CRP levels than members of the older cohorts reporting to be in good health. Table 2 also documents the correlation between adult height and health measured by biomarkers. Individuals in the top half of the cohort-sex-specific height distribution generally have lower blood fibrinogen and CRP levels than individuals in the lower half.

--- about here Table 2 ---

Associations between education and health

Table 3 shows the associations between education (measured as the age when the respondent left school) and the three health outcomes: self-rated health (again dichotomized to good versus poor), log fibrinogen level, and log CRP level. In each regression, we control for cohort (year and month of birth), season of birth, height and sex. We also control for survey year to account for possible unobserved differences across surveys.

--- about here Table 3 ---

For each of our measures, the results shown in Table 3 provide evidence for a significant association between education and health. Leaving school one year later is associated with about a 5 to 6 percentage point decrease in the probability of reporting poor general health in the older cohorts and a 3 to 4 percentage point decrease in the younger cohorts. When the sample is restricted to respondents participating in the nurse visit, these associations become somewhat smaller. Also, controlling for cohort, season of birth, height, and survey year reduces the strength of the association. Still, the slope of the education-self assessed health gradient is fairly large. Health differences related to one additional year of education correspond to health differences related to being more than ten years younger. Our findings for subjective health are corroborated by the more "objective" biomarkers. Each year of education is associated with a reduction in the blood fibrinogen level by 1.5 (women) to 1.9 percent (men) in the older cohorts and by between 1.8 percent (men) and 2.1 percent (women) in the younger cohorts. Controlling for covariates reduces this association but it remains highly significant. The effect size corresponds to about two to three years of chronological age for men and women, respectively, i.e. the effect size is smaller than for self-rated health. For log CRP levels, effect sizes are in the range of about three years of chronological age.

While one cannot interpret these results causally they indicate that elevated levels of fibrinogen and CRP blood levels, indicating e.g. job-related psychosocial stress, are a potential pathway from education to health outcomes.

5. Instrumental variables results

The findings described in the preceding section reveal significant and sizeable associations between education and various measures of health. In this section we study whether this association is causal. As described above, we make use of two general increases in the minimum school leaving age in 1947 and 1973 that affected all cohorts born in or after April 1933 and September 1957, respectively. A graphical analysis of health outcomes by birth cohort can be found in Figures 3 and 4. Graphically, one finds some effect of the 1947 reform for self-rated health and CRP levels for women but only small effects for men, but no effects of the 1973 reform are visible for women, whereas there seems to be a jump in the levels of self-rated health for men indicating that the reform has improved health for men of those birth cohorts.

--- about here Figures 3 and 4 ---

Results of instrumental variables regression are shown in Table 4. The first stage parameter shows the effect of the treatment dummy on the average school leaving age within the estimating samples. Here we find considerable differences between men and women and reform cohorts. In line with our graphical analysis in section 2, we find that the 1947 reform had a stronger effect on the education of women than on the education of men. In 1973, the effect was slightly stronger for men. First stage F-statistics (for the instrument) are larger or close to 10 for all regressions except one, indicating that our results do not suffer from a weak instrument problem.

--- about here Table 4 ---

Turning to the IV parameters, we find that education has a mixed effect on health self-ratings. For men in the 1947 reform cohort, the point estimate is *plus* 2 percentage points, indicating that education might actually harm health. However, the standard error is 15 times as large as the one we get for the OLS estimate in Table 3. Statistically, the 2 percentage points are

neither different from zero nor different from the OLS estimate of minus 5 percentage points. For men in the 1973 reform cohort, our IV point estimate is negative and somewhat larger than the OLS coefficient, but again, it is neither different from zero nor different from the OLS coefficient. For women in the 1947 cohort, we obtain an IV estimate of minus 7 percentage points, i.e. a larger effect than OLS, that is statistically different from zero. In contrast, in the 1973 cohort, we find an implausibly large positive effect of education on the probability of reporting poor health. This finding could be indicative of a large heterogeneity in the effect of education on health, either because the group of women affected by the reforms is different or because the reforms affected schooling decisions at different margins. Overall, although most of these results are not inconsistent with a positive causal effect of education on health, it does also not lend much credibility to such an assertion.

Similar to health self-assessments, we do not find convincing evidence for a significant causal effect of education on biomarker levels. Estimates for log fibrinogen levels have mixed signs and are never significantly different from zero. The coefficients for log CRP levels are negative throughout, indicating a positive effect of education on health. Effect sizes are in the vicinity of the OLS estimates – but not significantly different from zero – for men in both reform cohorts and for women in the younger reform cohort. For women in the 1947 reform cohorts point estimates are much larger than OLS estimates. Again, given the large standard errors of our estimates, a Hausman test would not reject the assumption of exogeneity of education. These results point to one potential problem when complementing survey data with information on biomarkers. Because of the large variance in measurements the results using those variables will become rather imprecise.

Robustness checks

We now attempt to evaluate the robustness of our results presented in the preceding section. The results shown and discussed in the last sections were based on few observations. In this section we inspect whether we find more precise results when changing the bandwidth and the specification of the time trend. Following the recommendations in Lee and Lemieux (2009) for regression discontinuity designs, we primarily test the robustness of our results with respect to the bandwidth around the discontinuity and the functional form of the relationship between the outcome and the treatment-determining variable. Another check is to restrict our sample to respondents with either 14 or 15 years of education in case of the 1947 reform and

15 or 16 years of education in case of the 1973 reform. Among these respondents, the reforms had the largest impact on years in school, so that restricting the sample will increase the strength of our instrument.

Our first robustness check is to estimate the IV parameters using local linear regression and varying the bandwidth from one year to eight years (in half year steps). The results are shown in Table 5. With the exception of very small bandwidths which lead to imprecise estimates due a substantial loss of information, the results appear to be qualitatively robust to changing the bandwidth (beginning at about $h = 2.5$ years). However, effect sizes appear to become smaller in absolute value when the bandwidth is increased but this does not necessarily affect statistical significance because estimates also get more precise.

Changes in the size of the estimates suggest that results might be sensitive to how one models non-linearities in cohort effects. We have thus also experimented with alternative specifications using polynomial cohort trends of varying degrees on samples of varying bandwidths (see Table 6). This exercise essentially confirms our findings based on local linear regressions.

--- about here Tables 5 and 6 ---

Regression results based on the restricted samples are shown in Table 7. Notably, whereas the association between years in school an health usually gets larger (as indicated by the OLS regression parameters also reported for comparison purposes), it partly loses significance. This might not only be due to smaller sample sizes, but also due to less variation in the education variable.

6. Summary and discussion

We use data from several rounds of the Health Survey for England and the English Longitudinal Study on Ageing to estimate the causal effect of education on health. Our identification comes from two increases in mandatory school leaving age in 1947 and 1973. We are not the first to exploit these reforms for causal analyses in an instrumental variables approach. However, the specific contribution of our paper is the use of biomarkers (blood fibrinogen and C-reactive protein) in addition to health self-reports as health outcome

measures. These measurements are important markers of psychosocial stress caused for instance by low occupational and social status. Using these markers therefore allows studying important pathways by which education affects health outcomes.

We do not argue that biomarkers represent "better" or "more precise" measures of health. Rather, we use them as indicators of health that are complementary to subjective measures such as self-rated health. We analyze blood fibrinogen and blood C-reactive protein because high levels in each are known risk factors for cardiovascular disease. Thus our analyses allow identifying whether education has a causal effect not only on manifest conditions but also on the risk of developing a disease.

We find that education is clearly correlated with lower levels of fibrinogen and C-reactive protein (indicating worse health and higher risk of cardiovascular disease for respondents with a smaller number of years in school). This points to the potentially important role of psychosocial stress as pathway by which education affects health outcomes. Analyses based on biomarkers, therefore, are an important additional source of information in addition to analyses based on subjective health assessments or physician-diagnoses illnesses. However, our results contain no evidence for a significant *causal* effect of education on the examined biomarkers. This lack of significance is largely due to the imprecision of the estimates. Contemporary identification strategies generally take their toll in terms of statistical power by deriving effect estimates from the presumably exogenous part of the variation in the treatment variable. The lack of statistical power is a nuisance. But we believe that our paper also conveys a message for future research. Collection of biomarkers in general population surveys is currently high on the agenda (National Research Council 2008). However, econometric identification of causal links in observational data requires large amounts of data. If 3,000 observations (as used in our study) are too few to get precise enough results, the collection of biomarkers as "objective" markers of intermediate health outcomes may thus – at least for economists – not prove to be the magic bullet that solves issues of endogeneity and survey response bias in the SES-health literature.

Still, while our causal results are inconclusive, the descriptive OLS results support the notion that cumulative psychosocial stress as measured by biomarkers is a correlate of both education and health and thus is a potential causal pathway from education to health. Biomarkers thus contain valuable information for researchers in addition to more subjective

health markers. We believe it would be worthwhile to investigate this question further in larger samples. In contrast to biomarker results, we find some evidence for an effect of education on self-rated health, which we also analyzed for comparison purposes, among women. This difference might arise from the fact that self-rated health is a more comprehensive (but possibly biased) measure of overall health. Overall, our results are mostly in line with earlier studies analyzing the effect of the 1947 and 1973 UK education reforms on health outcomes. For instance, regarding "objective" health measurements (log fibrinogen and CRP blood levels), our finding of no significant education effect confirms findings in Clark and Royer (2010) who look at BMI and blood pressure.

Although a causal effect of education on health (both direct and indirect) is theoretically plausible, we believe that there are a couple of reasons why the effect may actually be hard to identify in observational studies. As other authors, we use changes in compulsory schooling for identification. It is well known that IV and RD estimators have good internal validity but that external validity is a problem. For instance, using our instrument, we are (only) able to identify local treatment effects. The main effect of the reforms studied in the present paper on education was to keep those who wanted to quit as early as possible in school for one more year. One important question is what has been learned in this one year? There is some evidence that this year had some positive effect on the wages of the affected cohorts (Oreopoulos 2006, Devereux and Hart 2010), i.e. something has been learned and people have become more productive in the labor market, but the higher income apparently had no sizeable impact on health. One explanation for the absence of a causal effect on objective measures of health is that what needs to be learned to make individuals more productive producers of their own health is different from skills that are valued at the labor market. It is likely that the emerging literature on health literacy sheds more light on this issue (Nutbeam 2008).

Another reason why we may not find strong evidence for a causal effect of education on health measured by biomarkers is that our samples are limited to individuals born shortly before and after the relevant cut-off dates for being affected by the reforms. The reason not to choose too wide intervals is obvious. The more birth cohorts are included, the harder it becomes to maintain the assumption that no unobserved factors that influence health have changed in parallel to the reform. One example for such unobserved factors affecting the validity of the instrument is medical progress. Put differently, the instrument loses validity

when the sample is extended too far because the exclusion restriction does no longer hold. Of course, by including cohort trends and adult height we hope to account for such unmeasured factors, but the risk that the cohort trend is incorrectly specified rises with the number of cohorts included in the regression. The downside of "staying close" to the discontinuity is that the number of observations may quickly become too small to get precise estimates. This may also be one explanation for our non-findings with respect to biomarkers. However, we do find some significant effects of education on self-rated health in samples of similar size. The contradiction between results for self-rated health and biomarkers could thus also be due to differential reporting styles of respondents of different education levels. This issue has raised some attention in the recent literature (e.g. Bago d'Uva et al. 2008, Jürges 2008), but cannot be solved in the present paper. Further, the biomarkers we use in our study are surrogate outcomes representing a narrow range of health outcomes. Thus to find no effect on those biomarkers but positive effects on a more comprehensive measure of health is logically possible (although we believe that the theoretical arguments for an effect of education on cardiovascular health are fairly convincing).

Finally, it must also be noted that our parameters only identify the effect of education for compliers to the two specific reforms of raising *mandatory* school leaving age. Interventions at other stages of the life-cycle might have more systematic causal effects on health. For instance, a recent strand of the human capital literature has stressed the importance of early childhood education for the development of cognitive and non-cognitive skills (Cunha et al. 2006). If early childhood education changes the whole lifetime path of human capital accumulation, early interventions might substantially improve health, while later life interventions like increasing the number of school years remain largely ineffective.

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Table 1: Sample description

	Full HSE/ELSA sample				With valid blood sample			
	1947 reform cohorts		1973 reform cohorts		1947 reform cohorts		1973 reform cohorts	
	Men	Women	Men	Women	Men	Women	Men	Women
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age at survey	66	67	41	41	66	67	41	42
Age left school	15.4	15.4	16.7	16.7	15.4	15.4	16.7	16.7
Poor health (%)	36	34	17	19	37	35	18	19
Height (cm)	172	159	176	163	172	159	176	163
Ln(fibrinogen)					1.00	1.04	0.86	0.93
Ln(CRP)					0.63	0.66	0.12	0.14
N	4787	5280	5925	7252	2135	2240	3074	3409

Table 2: Relation between self-rated health, height and biomarker levels

	1947 reform cohorts				1973 reform cohorts			
	Men		Women		Men		Women	
	Fib	CRP	Fib	CRP	Fib	CRP	Fib	CRP
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Self-rated health								
Poor	1.05	0.81	1.08	0.80	0.91	0.37	1.01	0.47
Good	0.98	0.55	1.02	0.61	0.85	0.06	0.92	0.06
abs. t-value	6.4	5.6	5.6	4.2	5.6	5.0	8.4	6.3
Height (relative to cohort and sex specific median)								
Below	1.01	0.64	1.06	0.70	0.87	0.16	0.94	0.21
Above	0.99	0.60	1.02	0.61	0.84	0.07	0.92	0.04
abs. t-value	1.8	1.1	4.5	2.1	3.1	1.8	3.3	3.5

Notes: CRP values available in 1998 and 1999 only.

Table 3: Descriptive (OLS) regressions of health measures on education

	Poor health	Poor health	Poor health[a)]	Poor health[a)]	log(fibrin.)	log(fibrin.)	log(CRP)	log(CRP)
	1947 reform cohorts (1)	1973 reform cohorts (2)	1947 reform cohorts (3)	1973 reform cohorts (4)	1947 reform cohorts (5)	1973 reform cohorts (6)	1947 reform cohorts (7)	1973 reform cohorts (8)
				Men – no controls				
Age left school	-0.054***	-0.044***	-0.053***	-0.032***	-0.019***	-0.018***	-0.102***	-0.074***
	(0.004)	(0.003)	(0.006)	(0.004)	(0.003)	(0.003)	(0.018)	(0.020)
	Men – controlling for birth cohort (year and month), season of birth, height, and survey year							
Age left school	-0.051***	-0.042***	-0.048***	-0.030***	-0.015***	-0.016***	-0.102***	-0.073***
	(0.004)	(0.003)	(0.006)	(0.004)	(0.003)	(0.003)	(0.019)	(0.020)
N	5577	5925	2735	3074	2737	3074	1611	1630
				Women – no controls				
Age left school	-0.063***	-0.039***	-0.058***	-0.033***	-0.015***	-0.021***	-0.075***	-0.110***
	(0.004)	(0.003)	(0.005)	(0.004)	(0.003)	(0.003)	(0.017)	(0.018)
	Women – controlling for birth cohort (year and month), season of birth, height, and survey year							
Age left school	-0.059***	-0.038***	-0.054***	-0.033***	-0.011***	-0.020***	-0.070***	-0.106***
	(0.004)	(0.003)	(0.005)	(0.004)	(0.003)	(0.003)	(0.017)	(0.018)
N	6199	7252	2909	3409	2911	3410	1749	1848

Notes: Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%;
[a)] Restricted to observations with valid fibrinogen values.

Table 4: Instrumental variables estimates for the effect of education on health

	Poor health	Poor health	Poor health[a)]	Poor health[a)]	log(fibrin.)	log(fibrin.)	log(CRP)	log(CRP)
	1947 reform cohorts (1)	1973 reform cohorts (2)	1947 reform cohorts (3)	1973 reform cohorts (4)	1947 reform cohorts (5)	1973 reform cohorts (6)	1947 reform cohorts (7)	1973 reform cohorts (8)
Men								
First stage parameter	0.38***	0.33***	0.32***	0.32***	0.33***	0.32***	0.40***	0.39***
	(0.08)	(0.07)	(0.09)	(0.10)	(0.09)	(0.10)	(0.14)	(0.13)
First-stage F statistic	23.49	22.47	14.23	10.55	14.67	10.55	8.03	9.43
IV parameter	0.02	-0.08	0.05	-0.08	-0.03	0.01	-0.12	-0.18
	(0.06)	(0.06)	(0.09)	(0.10)	(0.06)	(0.05)	(0.23)	(0.25)
Observations	5577	5925	2735	3074	2737	3074	1611	1630
Women								
First stage parameter	0.52***	0.31***	0.41***	0.31***	0.41***	0.31***	0.49***	0.20
	(0.08)	(0.07)	(0.11)	(0.09)	(0.11)	(0.09)	(0.15)	(0.14)
First-stage F statistic	37.39	17.92	14.59	10.95	14.53	11.09	10.24	1.95
IV parameter	-0.07*	0.15**	-0.12	0.11	0.04	-0.01	-0.37	-0.09
	(0.04)	(0.06)	(0.08)	(0.08)	(0.04)	(0.05)	(0.24)	(0.49)
Observations	6199	7252	2909	3409	2911	3410	1749	1848

Cluster corrected standard errors in parentheses, * significant at 10%; ** significant at 5%; *** significant at 1%
Controlling for year and month of birth, survey year, sex, and height
[a)] Restricted to observations with valid fibrinogen values

Table 5: Instrumental variables estimates for the effect of education on health, various bandwidths

Bandwidth (years)	Poor health 1947 reform cohorts (1)	Poor health 1973 reform cohorts (2)	Poor health [a] 1947 reform cohorts (3)	Poor health [a] 1973 reform cohorts (4)	log (fibrin.) 1947 reform cohorts (5)	log(fibrin.) 1973 reform cohorts (6)	log(CRP) 1947 reform cohorts (7)	log(CRP) 1973 reform cohorts (8)
Men								
1	-0.10	0.09	0.69	6.67	1.43	0.71	-2.79	16.05
1.5	0.02	-0.09	-0.13	-0.01	-0.10	-0.05	-0.67*	-0.33
2	0.04	-0.10	-0.13	-0.03	-0.15*	-0.01	-0.67**	-0.29
2.5	0.06	-0.07	-0.03	0.04	-0.09	0.03	-0.40	-0.30
3	0.10	-0.06	0.08	0.02	-0.08	0.05	-0.39	-0.26
3.5	0.07	-0.05	0.11	-0.00	-0.06	0.02	-0.29	-0.25
4	0.02	-0.08	0.05	-0.08	-0.03	0.01	-0.12	-0.18
4.5	0.02	-0.06	0.09	-0.05	-0.01	-0.02	0.05	-0.20
5	0.03	-0.07	0.10	-0.06	-0.02	-0.01	0.01	-0.07
5.5	0.02	-0.08	0.11	-0.08	-0.00	-0.03	-0.02	-0.17
6	0.01	-0.09	0.08	-0.09	-0.01	-0.02	-0.03	-0.17
6.5	-0.00	-0.07	0.09	-0.05	-0.01	-0.00	0.03	-0.09
7	-0.03	-0.08	0.05	-0.05	-0.02	-0.02	0.02	-0.07
7.5	-0.03	-0.07	0.05	-0.04	-0.02	-0.03	-0.02	-0.20
8	-0.04	-0.06	0.04	-0.01	-0.03	-0.03	-0.05	-0.21
Women								
1	0.79	0.06	0.10	0.34	0.03	-0.19	1.78	-0.82
1.5	0.00	0.07	-0.14	0.03	0.02	-0.01	-0.23	-0.73
2	0.00	0.10	-0.16	0.10	0.06	-0.04	-0.39	-1.24
2.5	-0.05	0.13**	-0.14	0.20	0.00	0.06	-0.18	-0.70
3	-0.04	0.09*	-0.11	0.06	0.00	0.03	-0.21	-0.30
3.5	-0.05	0.16**	-0.11	0.11	0.04	0.01	-0.26	-0.27
4	-0.07*	0.15**	-0.12	0.11	0.04	-0.01	-0.37	-0.09
4.5	-0.08**	0.10**	-0.15**	0.06	0.03	0.02	-0.23	0.02
5	-0.08**	0.06	-0.15**	0.03	0.02	0.03	-0.17	0.13
5.5	-0.08**	0.09**	-0.14**	0.05	0.01	-0.01	-0.23	-0.00
6	-0.07**	0.08*	-0.11*	0.02	0.00	-0.01	-0.21	0.10
6.5	-0.06**	0.09*	-0.10*	0.02	-0.01	-0.00	-0.20	0.06
7	-0.06**	0.09*	-0.10**	0.04	-0.02	0.01	-0.20	0.07
7.5	-0.07***	0.08	-0.11**	0.04	-0.02	0.01	-0.19	0.19
8	-0.07***	0.08	-0.09**	0.06	-0.02	0.02	-0.22	0.44

Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%;
Controlling for month of birth cohort, survey year, season of birth, and height
[a] Restricted to observations with valid fibrinogen values

Table 6: Instrumental variables estimates for the effect of education for the effect of education on health, various polynomial trends (k) and bandwidths (h)

	Poor health	Poor health	Poor health [a)]	Poor health [a)]	log (fibrin.)	log(fibrin.)	log(CRP)	log(CRP)
Polynomial, Bandwidth	1947 reform cohorts (1)	1973 reform cohorts (2)	1947 reform cohorts (3)	1973 reform cohorts (4)	1947 reform cohorts (5)	1973 reform cohorts (6)	1947 reform cohorts (7)	1973 reform cohorts (8)
Men								
k=1, h=4	0.02	-0.08	0.05	-0.08	-0.03	0.01	-0.12	-0.19
k=2, h=4	0.02	-0.08	0.05	-0.08	-0.03	0.01	-0.12	-0.18
k=3, h=4	0.12	-0.06	0.03	0.10	-0.11	0.03	-0.64	-0.39
k=4, h=4	0.12	-0.06	0.04	0.10	-0.11	0.03	-0.64	-0.39
k=1, h=8	-0.04	-0.07	0.04	-0.01	-0.03	-0.03	-0.05	-0.23
k=2, h=8	-0.04	-0.06	0.04	-0.01	-0.03	-0.03	-0.06	-0.22
k=3, h=8	0.04	-0.09*	0.11	-0.08	0.01	-0.02	0.02	-0.14
k=4, h=8	0.04	-0.09*	0.11	-0.08	0.01	-0.02	0.02	-0.14
Women								
k=1, h=4	-0.07*	0.15**	-0.12	0.11	0.04	-0.01	-0.37	-0.10
k=2, h=4	-0.07*	0.15**	-0.12	0.11	0.04	-0.01	-0.36	-0.09
k=3, h=4	-0.02	0.12	-0.13	0.12	0.02	0.03	-0.15	-0.66
k=4, h=4	-0.02	0.12	-0.13	0.12	0.02	0.03	-0.16	-0.64
k=1, h=8	-0.07***	0.08	-0.09**	0.06	-0.02	0.02	-0.22	0.39
k=2, h=8	-0.07***	0.08	-0.09**	0.06	-0.02	0.02	-0.22	0.44
k=3, h=8	-0.06*	0.10**	-0.16**	0.03	0.01	-0.01	-0.20	-0.16
k=4, h=8	-0.06*	0.10**	-0.16**	0.03	0.01	-0.01	-0.20	-0.16

Cluster corrected standard errors in parentheses; * p<10%; ** p<5%; *** p<1%;
Controlling for survey year and height, season of birth
[a)] Restricted to observations with valid fibrinogen values

Table 7: Instrumental variables estimates for the effect of education on health (restricted samples)

	Poor health	Poor health	Poor health[a)]	Poor health[a)]	log(fibrin.)	log(fibrin.)	log(CRP)	log(CRP)
	1947 reform cohorts (1)	1973 reform cohorts (2)	1947 reform cohorts (3)	1973 reform cohorts (4)	1947 reform cohorts (5)	1973 reform cohorts (6)	1947 reform cohorts (7)	1973 reform cohorts (8)
Men								
OLS parameter	-0.067***	-0.086***	-0.050*	-0.080***	-0.023	-0.031**	-0.194***	-0.169**
	(0.021)	(0.019)	(0.030)	(0.022)	(0.015)	(0.015)	(0.073)	(0.070)
IV First stage parameter	0.63***	0.33***	0.67***	0.30***	0.67***	0.30***	0.74***	0.34***
	(0.03)	(0.03)	(0.03)	(0.04)	(0.03)	(0.04)	(0.04)	(0.06)
First-stage F statistic	499.92	111.48	417.41	49.26	422.46	49.26	365.18	30.42
FRD parameter	-0.02	-0.10	-0.03	-0.07	-0.03	0.06	-0.12	0.32
	(0.05)	(0.09)	(0.06)	(0.14)	(0.04)	(0.08)	(0.19)	(0.41)
Observations	3690	3496	1790	1800	1792	1800	1077	965
Women								
OLS parameter	-0.086***	-0.083***	-0.079**	-0.110***	-0.013	-0.015	-0.123	-0.213**
	(0.021)	(0.017)	(0.031)	(0.022)	(0.015)	(0.014)	(0.079)	(0.092)
IV First stage parameter	0.63***	0.41***	0.59***	0.43***	0.59***	0.43***	0.62***	0.41***
	(0.02)	(0.03)	(0.04)	(0.03)	(0.04)	(0.03)	(0.05)	(0.04)
First-stage F statistic	741.47	190.28	269.84	161.49	270.34	161.49	181.49	91.69
FRD parameter	-0.03	0.15***	-0.04	0.08	0.03	-0.06	-0.30	-0.27
	(0.06)	(0.05)	(0.09)	(0.08)	(0.03)	(0.05)	(0.19)	(0.33)
Observations	4009	4064	1815	1892	1817	1892	1059	1009

Cluster corrected standard errors in parentheses, * significant at 10%; ** significant at 5%; *** significant at 1%
Controlling for year and month of birth, survey year, sex, and height
[a)] Restricted to observations with valid fibrinogen values

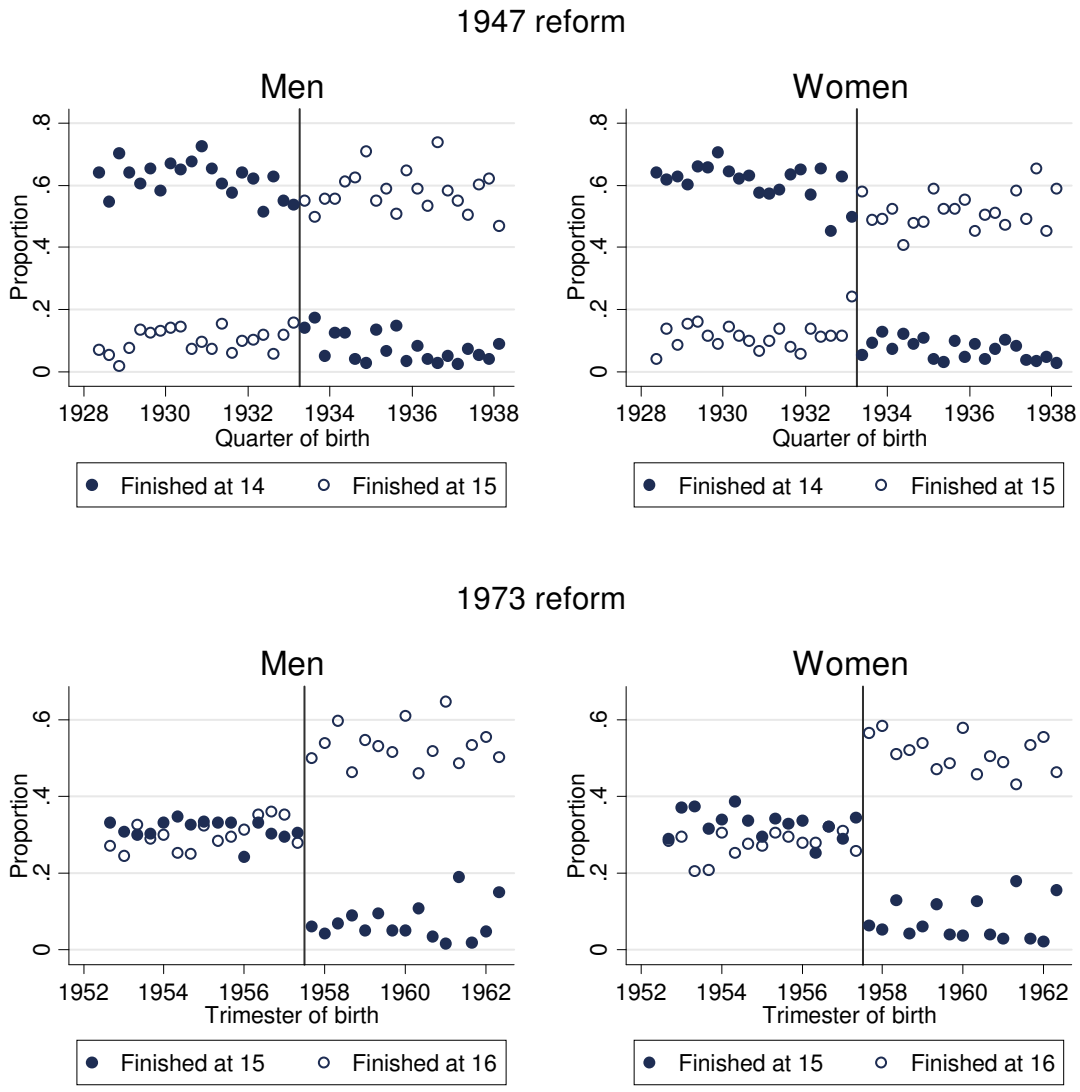

Figure 1: Effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment, measured by the proportion of respondents who left school at ages 14, 15 or 16, respectively.

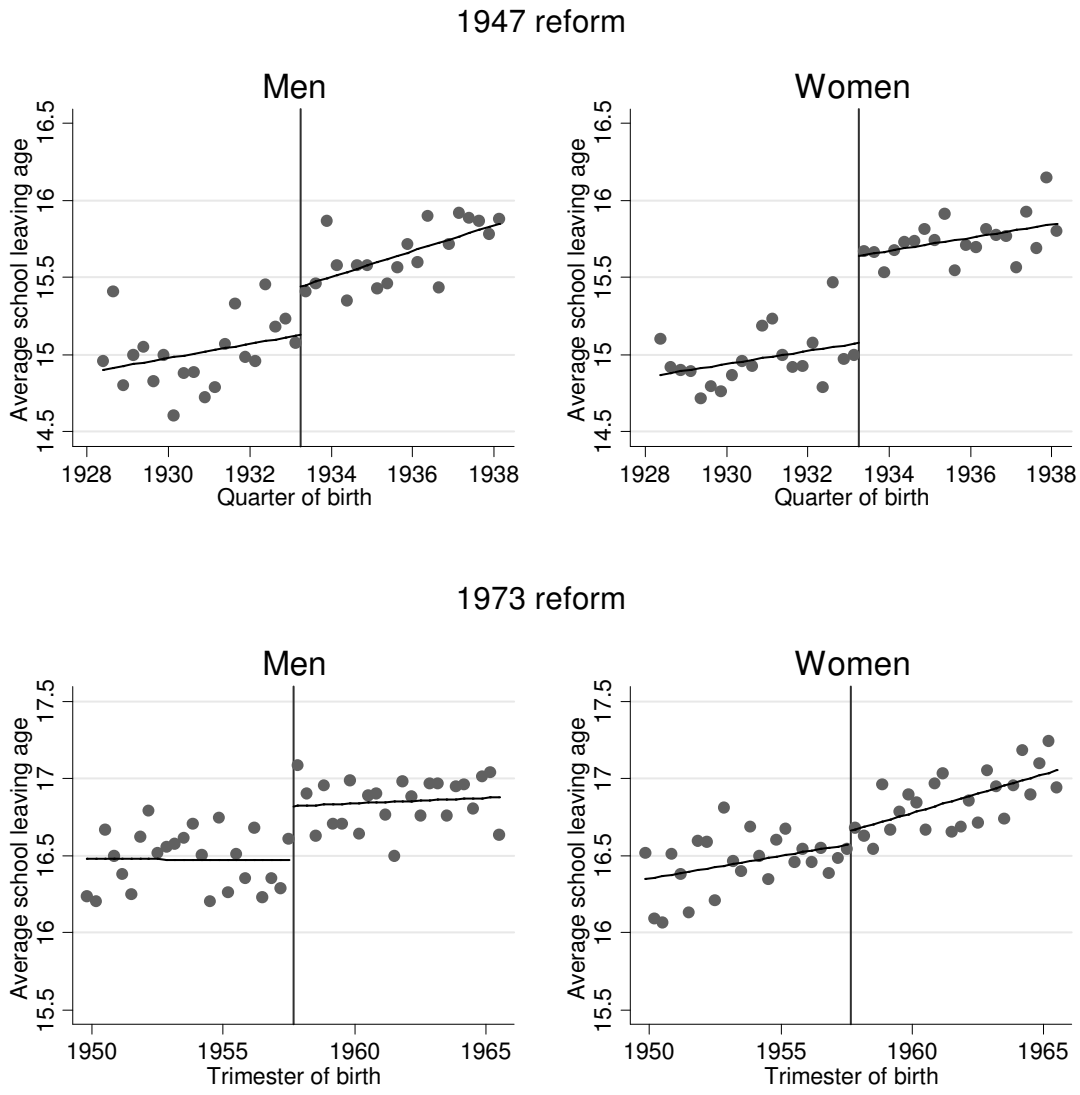

Figure 2: Effect of the 1947 and 1973 changes in compulsory school leaving age on educational attainment, measured by the average school leaving age.

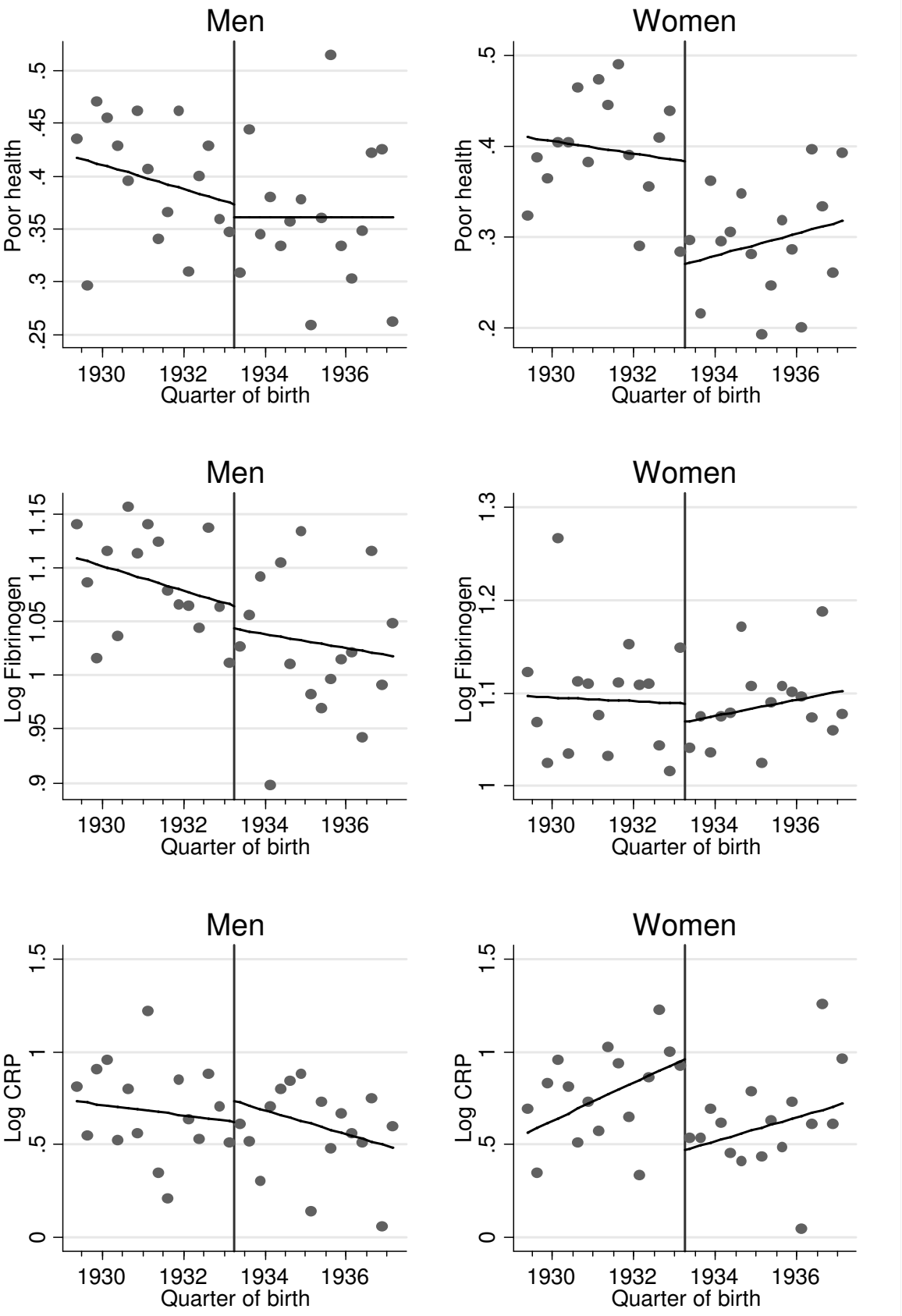

Figure 3: Effect of the 1947 changes in compulsory school leaving age on health measures.

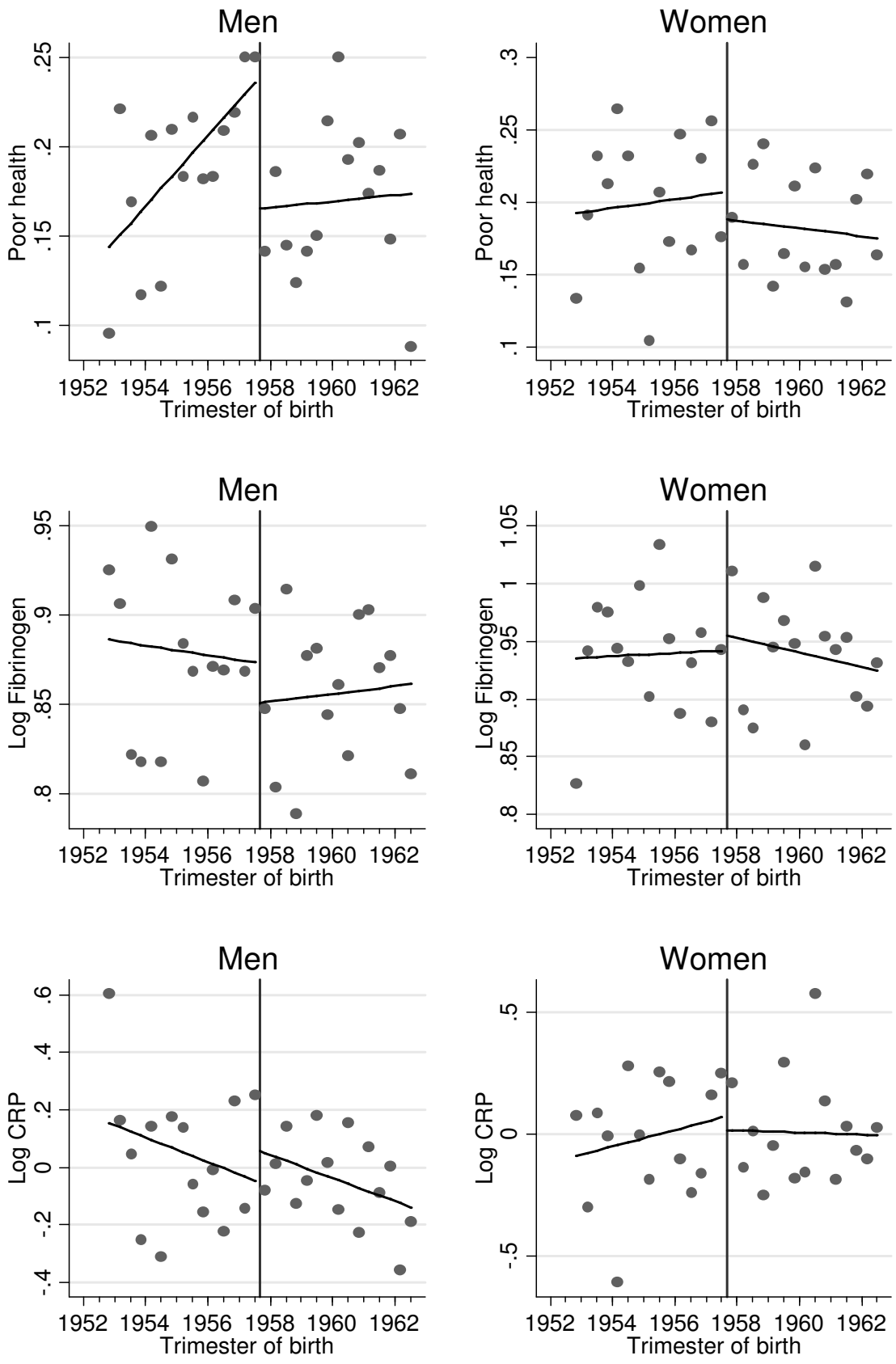

Figure 4: Effect of the 1973 changes in compulsory school leaving age on health measures.