

**Does Education Reduce the Risk of Hypertension? Estimating the Biomarker Effect of  
Compulsory Schooling in England**

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**Abstract**

This paper estimates the exogenous effect of schooling on reduced incidence of hypertension. Using the changes in the minimum school-leaving age law in the United Kingdom from age 14 to 15 in 1947, and from age 15 to 16 in 1973, as sources of exogenous variation in schooling, the regression discontinuity and IV-probit estimates imply that, for the first law change in 1947, completing an extra year of schooling reduces the probability of developing subsequent hypertension by approximately 7 to 10 percentage-points; the result which holds for both genders. No significant effect was found for the introduction of the second law change in 1973. The correct IV-probit estimates of the LATE for schooling indicate the presence of a large and negative bias in the probit estimates of schooling-hypertension relationship.

**Keywords:** hypertension; compulsory schooling; biomarker; regression discontinuity; health

**JEL:** H1, I1, I2

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Ever since Michael Grossman's seminal work on the demand for health in the early 1970s (Grossman, 1972; and subsequently, Grossman, 2000; 2005), researchers have routinely tried to estimate the effects of different socio-economic variables on a variety of health outcomes. So far, years of schooling has stood out as one of the statistically significant determinants of health. This is true whether health levels are measured by self-assessed health status, mortality rates, risky health behaviors, morbidity, physiological measures, and mental well-being (Grossman, 1975; Berger and Leigh, 1989; Kenkel, 1991; Deaton and Paxson, 2001; Oreopoulos, 2007; Blanchflower and Oswald, 2008).

There are, however, two major practical problems associated with the estimation of the schooling effect on health. The first corresponds to the classic estimation bias associated with ordinary least squares (OLS) estimates of the return to schooling. In the economics literature, researchers frequently use the instrumental variables (IV) approach to address the endogeneity of schooling decisions. A valid instrumental variable, which determines whether an individual receives more years of schooling, but does not determine other factors that affect the outcome of interest, can overcome estimation biases that often arise when using the OLS method. Yet, according to Guido Imbens and Joshua D. Angrist (1994) and Philip Oreopoulos (2006), many of the instruments used in the return to schooling literature – e.g., distance from home to college (Card, 1995), restrictive compulsory schooling law (Angrist and Krueger, 1991), and regional spending on education in regions where the individual was still a student (Berger and Leigh, 1989) – only affect a small fraction of the general population. As a result, many of the IV estimates produced in the literature are only approximations of the average treatment effects among a small group of people who happened to be exposed to the instruments (Card, 2001).

The second practical problem concerns the existing measures of health outcomes frequently used in the studies conducted by economists concerning the determinants of health. While there are valid reasons for using self-assessed health, mortality rates, morbidity, and risky health behaviors such as smoking and drinking as proxies for health, these variables can only, at best, be considered by the medical professions as indirect indicators of someone's underlying health. They do not, for example, possess the same clinical properties as such biomarkers as blood pressure, cortisol levels, cholesterol levels, or heart rate, which are normally used by clinicians to measure someone's biologic state. For example, self-reported health, which is the most commonly used measure of health outcomes in economics, is subject to a variety of potential measurement biases and interpersonal comparability problems (for a recent review, see Powdthavee, 2009a). The same holds true for other self-

reported health problems. Mortality rates, morbidity, and disability index may fair better as measures of health, given that each has the required quality of being ‘objective’. Yet both are indicators of the extreme cases of ill-health and physical disablement, which will reflect only a small fraction of the nation’s population. Because nationally representative surveys that contain both biomarker readings and years of schooling are scarce, econometric evidence on the biomarker effect of schooling is virtually non-existent.

Using the unique Health Survey for England data set which combines interviews and physical examinations, this paper estimates the effects of schooling on adult hypertension readings in England. Following Harmon and Walker (1995), Oreopoulos (2006, 2007) and Devereux and Hart (2010), I will rely on exogenous changes in the amount of schooling received by individuals caused by the raising of the minimum school-leaving age in the United Kingdom (which has occurred twice over the age-spread of those over the age of 16 in the English data set). Because the changes of minimum school-leaving age affected a large fraction of people who would have left school at an earlier age before both reforms, IV-probit estimates of the schooling effects on the risk of hypertension is likely to come close to the average treatment effects that apply to the whole population (see Oreopoulos, 2006). In addition to the IV-probit approach, I also follow previous literature on compulsory schooling and adopt a regression discontinuity (RD) approach in order to illustrate the average educational attainment and adult hypertension just before and after the introduction of the two minimum school-leaving age laws.

There are empirically good reasons to use a zero-one indication of hypertension as the main biomarker for the nation’s underlying health and well-being. Hypertension has been shown in the medical literature as the single most important predictor of heart disease and death from heart disease (Hofman, Feinleib, Garrison, and van Laar, 1981; Fraser, 1986; Wilson and colleagues, 1998), which is also the current biggest killer in America and the UK. Hypertension has also been used as a biomarker for stress and general psychological well-being in the work by David Blanchflower and Andrew J. Oswald (2008, 2009).

More generally, the current study aims to make a new contribution to the relatively small literature that has its primary focus on the estimation of the non-market returns to education. According to Gary Becker and Kevin Murphy (2007), comparatively few studies have concentrated on the benefits from education other than earnings, such as health, child rearing, and marriage, despite the evidence that education has been shown to have large effects on such non-market activities. This paper aims to fill that research void by estimating

the causal effect of schooling on an actual health measure as an efficiency parameter in health production.

Section I gives an account of previous empirical evidence on the effect of schooling on health. Data and empirical strategy are discussed in Section II. Section III presents both RD and IV-probit results. Section IV concludes.

### **I. Previous evidence on the effect of schooling on health outcomes**

There are a variety of sources of bias associated with estimation of the schooling effect on health. First, causality may also run in reverse from health to schooling, i.e. healthier students may be more efficient producers of additional human capital via more years of formal schooling, which implies that estimates of the schooling effect on health will be biased upward. Second, there may be omitted third variables such as ability (Angrist and Kruger, 2001; Card, 2001), heritable endowments (Behrman and Rosenzweig, 2002), and time-preference (Fuch, 1982) that influence both schooling and health outcomes. One could imagine, for example, that people who are more future oriented (i.e. those who desire more leisure at older ages) will stay in school for longer, work more at younger ages, as well as have higher levels of health during most stages of the life cycle. Thus, the effect of schooling will be biased upward if one fails to control for time-preference. A third source of potential bias is measurement error, which can bias the estimated schooling effect toward zero (Blackburn and Neumark, 1995).

Previous work on the estimation of the schooling effect on health has mainly dealt with the endogeneity issue using either the IV method or the quasi-experimental approach, and the results have been mixed. Using the state of residence in childhood as instruments for schooling, Leigh and Dhir (1997) find schooling to be negatively related to an index of disability. However, there is little difference in size of the schooling coefficient whether it is treated as exogenous or endogenous. The same IV method is used by Sander (1995) to estimate the causal effect of schooling on smoking in the 1986-1991 National Opinion Research Center's General Social Survey. Applying parents' education, rural residence at age 16, region of residence at age 16, number of siblings as instruments for schooling, Sander finds the schooling effects on the probability of quitting smoking estimated by probit and IV-probit estimators to be virtually the same.

More recently, Lleras-Muney (2005) uses compulsory education laws from 1915 to 1939 as instruments for education in the adult mortality equations. When treating schooling

as exogenous, she finds the IV estimates on the schooling effect on adult mortality to be negative and significantly larger than the ones obtained by OLS: While GLS estimates suggest that an extra year of schooling lowers the probability of dying in the next ten years by 1.3-percentage-points, the IV-estimates suggest that the effect is in fact much larger: at least 3.6-percentage-points. Adams (2002) demonstrates using the same instruments as Lleras-Muney (2005) that the schooling effect on self-assessed health is much larger in the IV equations than in the OLS equations. He also finds the effect to be especially stronger for women relative to men. Similar results are also obtained by Arendt (2005) when two compulsory schooling reforms in Denmark are used to address the endogeneity of schooling in self-assessed health equations. He shows that the estimated effect of schooling on health and health-related behaviors (i.e. self-assessed health, BMI, and smoking) is significantly larger in magnitude when endogeneity is allowed for. Using changes in compulsory schooling in Sweden which implemented randomly and in stages by municipalities in the 1950s to instrument for schooling, Spasojevic (2003) reports positive schooling effects on a constructed index of bad health and an index of body mass index (BMI) in the healthy range, although the effects are only significant when using one-tailed tests. In short, most studies do not find significant differences in the estimated schooling effects on health whether the schooling variable is treated as exogenous or endogenous.

One important issue concerning the use of IV method to estimate the return to investment in human capital is that the only effect we can be sure that this method estimates is the local average treatment effect (LATE), i.e. the average treatment effect (ATE) among those who alter their status because they react to the instrument (Imbens and Angrist, 1994). In many cases, the instruments used in the return to schooling literature only apply to a small fraction of the population (see, e.g., Angrist and Kruger, 1991). What this implies is that many of the IV estimates will only approximate the ATE among a small and peculiar group rather than the general population, whereas OLS estimates, in the absence of omitted third variables and measurement error problems, approximate ATE among everyone (Card, 2001). For example, as in the aforementioned study by Spasojevic (2003), because school reforms in Sweden took place randomly and in stages by municipalities, it is possible that her IV estimates only approximate the average treatment effects among students who happened to be residing in these municipalities when the changes took place.

One of the more successful instruments used to estimate the market returns to education in recent times which, when implemented, will produce the IV estimate that is closest to the ATE for the general population as possible, is the changes in compulsory

schooling law in the UK (see Harmon and Walker, 1995; Oreopoulos, 2006). One reason for this is simply because the introduction of the minimum school-leaving age in the UK for the first time in April 1<sup>st</sup>, 1947 affects a large fraction of the population who would have left school at age 14 prior to 1947. The dramatic effect of the introduction of such laws on the amount of schooling received by the general population means that the estimated local treatment effects of education will come close to mirroring population average treatment effects (Oreopoulos, 2006).

The findings on the effect of the compulsory schooling law on health have been mixed. Using the nationally representative Labor Force Survey and General Household Survey for the UK, Philip Oreopoulos (2006, 2007) report some evidence that education improves self-assessed health – although the IV estimates are very similar to the estimates obtained by OLS. However, in a subsequently published corrigendum of his 2006 paper, he provides contradicting evidence to his earlier results by stating that, although the effect of the law on self-assessed health is positive, its effect is in fact not statistically different from zero (Oreopoulos, 2008). This implies that the protective effect of the law on self-assessed health found in his earlier papers may have been caused by coding errors. Using the same GHS data set as Oreopoulos, Mary Sille (2009) shows that the effect of compulsory schooling on multiple measures of health (e.g., self-reported health, an indicator of long-term illness, no activity limiting, and no work-preventing) to be positive, statistically significant, and much larger than standard regression estimates suggest. The positive estimates, however, could have been caused by misspecification of the IV model, i.e. both cohort and higher age polynomials were left out from the regressions, which consequently prevented smooth changes in education and health outcomes over time to be controlled for in her IV estimation (Imbens & Lemieux, 2007).

Although measures of subjective health, mortality outcomes, and health behaviors are reasonably good proxies for health outcomes, they are still far from having the required properties to be representative as a biomarker. By definition, a biomarker is “a characteristic that is objectively measurable and evaluated as an indicator of normal biological processes, pathological processes, or pharmacologic responses to therapeutic intervention.” (Biomarkers Definition Working Groups, 2001, p.91). Unlike self-assessed health, certain biomarkers are clinically accepted as the “true” objective measure of an individual’s underlying health. For example, reduction of elevated arterial blood pressure has been used for decades by clinicians to reflect the reduction in the stress level and in the incidence of stroke and congestive heart failure, whereas serum cholesterol levels are often used as an indicator of the risk of coronary

heart disease (see, for example, Wilson and colleagues, 1998). Biomarkers also do not suffer from certain cognitive biases that are known in the literature to systematically affect the way people rate their health – e.g., the focusing effect and the relative health effect (see, e.g., Powdthavee, 2009a; 2009b).

Despite the clear clinical advantages of biomarkers over subjective health measures, econometric evidence on the relationship between schooling and biomarkers is scarce. This is mainly due to the lack of biomarkers data being available in most household surveys. Of the few studies of biomarkers in economics, Berger and Leigh (1989) use data from the Health and Nutrition Examination Survey to estimate the impact of schooling on reduced blood pressure. Using average real per capita income and expenditures on education in the state in which an individual resided from the year of birth to the age of 6 as their instrumental variables, they found schooling to have a small negative effect on blood pressure: an extra year of schooling reduces both systolic and diastolic blood pressure by approximately -0.6 and -0.2 mmHg, respectively. However, as mentioned previously, their IV estimates obtained by Berger and Leigh are likely to approximate average treatment effects among a subset of population who are responsive to the instruments. Using both the 1998-2007 Health Survey for England and the 2001 Eurobarometer data set, Blanchflower and Oswald (2008, 2009) find schooling to be negatively correlated with blood pressure, although no attempts have been made to treat schooling as exogenous. Like Berg and Leigh, the schooling coefficients obtained in Blanchflower and Oswald, though statistically significant at the 1% level, are very small and of almost no economic importance.

The current paper advances from Oreopoulos (2006, 2007) and study the effect of the compulsory schooling law on ‘actual’ and clinically-accepted – as opposed to proxy – health measures. Evidence on the effect of the law on biomarkers is almost non-existent. However, particular mention should be made of recent work – done independently from this study – by Damon Clark and Heather Royer (2008) and Hendrik Jürges, Eberhard Kruk, and Steffen Reinhold (2009). In both articles, the authors estimate the effect of compulsory schooling on different measures of objective health in the Health Survey for England, including biomarker measures such as blood pressure, hypertension, blood fibrinogen and C-reactive protein levels. Using only the first raising of the minimum school-leaving age in 1947, Clark and Royer conclude that there is no evidence of compulsory schooling effect reducing the risk of adult hypertension for the whole sample, although it should be noted that their hypertension variable does not include those who take medicine to low blood pressure. Jürges, Kruk, and Reinhold also come up with a similar conclusion of no schooling effect when estimated on

blood fibrinogen and C-reactive protein levels. The current study extends beyond these two studies by following Devereux and Hart (2010), who conclude that the effect of compulsory schooling on wages in the UK is only positive for males but not for females, and examines the effect of compulsory schooling on adult hypertension by gender. It also adds to the literature by allowing for the unobserved time shocks, which could affect how both schooling and blood pressure variables are collected in the survey, to be controlled for in the regressions in the form of year fixed effects, something which has not been done previously in either study.

## **II. Data and empirical strategy**

### *A. Data*

The data set used in this paper is the Health Survey for England (HSE). The HSE is an annual survey and is designed to monitor the nation's health. The unit of survey in the HSE is the household. Information is collected through a combination of face-to-face interviews, a self-completed questionnaire, and a series of medical examination (including taking measurements for height and weight, as well as recording of blood and saliva sample for clinical tests) conducted by a trained nurse. Three continuous blood pressure measurements are available in 99% of the case, so I take the average for the second and third measures of systolic and diastolic. By definition, systolic blood pressure measures the rate of contraction of heart chambers while driving blood out of the chambers, whereas diastolic blood pressure measures the time when the heart fills with blood after contraction. Both are measured in millimeter of mercury (mmHg). Here, hypertension takes a value of 1 if the person has a systolic blood pressure of 140 mmHg or over and/or a diastolic blood pressure of 90 mmHg or over, and a value of 0 otherwise. With suggestions from a referee, we also control for the individual's underlying health by including the non-hypertensive cases who reported to take prescribed medicine to help with their high blood pressure in the 'hypertensive' category, i.e. a value of 1. This type of categorization forms our main dependent variable, although other categorizations of hypertension – e.g., 'recorded' hypertension, systolic hypertension, and diastolic hypertension – will also be analyzed in this study.

The schooling variable is recorded as the age an individual finished full-time education. In this paper, I pool data from the 1991 to 2007 HSEs. Using only the cohorts who were born six years prior to and post-changes of the compulsory schooling law – (1<sup>st</sup> change in 1947) those who were born between 1926 and 1939, and (2<sup>nd</sup> change in 1973) those who

were born between 1952 and 1965 – the first set of cohorts consists of 24,574 observations and 34,559 observations in the second cohort<sup>1</sup>. Summary of descriptive statistics are given in Table A1 in the appendix.

The legislation on changing the minimum school-leaving age from 14 to 15 was first introduced in the 1944 Education Act, with the first increase implemented on April 1st 1947. Unfortunately, the month of birth is not available in every wave of the HSE. This makes it not possible for us to test whether the effect of the law varies between those born in 1933, e.g. those born before April 1<sup>st</sup> 1933 (before the law has been implemented) and those born from April 1<sup>st</sup> 1933 (after the law has been implemented). However, according to Devereux and Hart (2008), the exact specification used for the law change – i.e. whether to allow for the differing in the law between those born before April 1<sup>st</sup> 1933 and from that date forward – is not critical. A further increase in the minimum school-leaving age from 15 to 16 subsequently occurred in 1973. Figure 1 plots the fractions of school leavers at age 14 and 15 before and after the first reform in 1947. Figure 2 does the same but for the 2<sup>nd</sup> reform. Consistent with Harmon and Walker (the UK Family Expenditure Survey) and Oreopoulos (the UK General Household Survey), we can see that a very high fraction of individuals in the HSE left school at age 14 (or less) before 1947. There is, however, a significant drop in the fraction of school leavers at age 14 in 1947: the portion of school leavers at aged 14 fell from 54% in 1946 to 8% in 1950. A sharp – albeit relatively smaller – drop in the portion of 15 years-old school leavers occurred in 1973: Over the course of three years between 1972 and 1975, the fraction of school-leavers at age 15 (or less) fell from 22% to 8%.

### *B. RD and IV-probit approach*

This paper focuses on one particular prediction made on the demand for health by Grossman’s human capital model: the causal effect of schooling on subsequent health outcomes. Two econometric strategies are adopted here: regression discontinuity (RD) and IV-probit.

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<sup>1</sup> The choice of bandwidth of six years before and after the law seems arbitrary. However, there is an obvious reason why more birth cohorts are not included: the larger the bandwidth, the harder it is to maintain the assumption that no unobserved factors that influence hypertension have changed in parallel to the reform. By contrast, it may be difficult to get precise estimates if too small a bandwidth is used (see Imbens & Lemieux, 2008).

First, the RD approach. Following Imbens and Lemieux (2008) and more specifically, Clark and Royer (2008), a standard procedure for estimating the causal effect of compulsory schooling on a health outcome is to estimate the following pair of equations:

$$S_{ic} = \beta_0 + \beta_1 D_{ic} + X'_{ic} + u_{ic} \quad (1)$$

$$HP_{ic} = \gamma_0 + \gamma_1 D_{ic} + X'_{ic} + v_{ic} \quad (2)$$

Here,  $S_{ic}$  is the number of years formal schooling completed for individual  $i$  in birth cohort  $c$  (calculated from date-of-birth and is defined yearly),  $HP_{ic}$  is a binary variable representing whether the individual has a stage I hypertension (systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg/or if the person takes prescribed medicine to help with high blood pressure),  $D_{ic}$  is a dummy for whether the individual is affected by the change in the minimum school-leaving age (i.e., for the 1947 reform,  $D_{ic}$  will take a value of 1 if the individual was 14 from 1947 onwards, and zero otherwise),  $X'_{ic}$  is a vector of control variables, which include smooth functions of birth cohort and age (low-order polynomials) and year dummies. Both birth cohort and age polynomials are included to capture age and cohort trends in the outcome variables of equations (1) and (2). Year fixed effects are added to control for the unobserved time shocks in both outcome variables which can vary from one year to the next in the survey.

Least squares estimation of (1) and (2) will generate  $\beta_1$  and  $\gamma_1$ , which represent the causal effect of compulsory education on school-leaving age and hypertension respectively. These estimates thus signify the “jump” in both education and health outcome associated with being 14 after the date of the reform, and the ratio of the estimates ( $\gamma_1 / \beta_1$ ) will give the standard IV Wald estimate, which is also a LATE estimate (see Damon and Royer, 2008).

Nonetheless, given that our hypertension variable is a binary outcome variable, estimating equation (2) using least squares estimation may yield inconsistent results. For this reason, I also adopt an IV-probit approach to estimate the LATE for schooling on hypertension (Newey, 1987). In IV-probit estimation, the identification is achieved by the inclusion of a dummy variable that records the exogenous change in the minimum school-leaving age law in the UK in the first-stage regression (see equation (1) above). A probit model on hypertension that incorporates the instrumented schooling as an explanatory variable will then be estimated in the second stage, controlling for birth cohort and age

polynomials (cubed) and year fixed effects. All regressions are clustered by birth cohort, and robust standard errors are reported.

### III. Results

Following Damon and Royer (2008), Figures 2 and 3 adopt the RD approach and provide the graphic illustrations of the compelling effects of the two reforms on educational attainment (see also Oreopoulos, 2006, and Imbens and Lemieux, 2008). Aggregating the data into cell means by birth year in order to create cohort averages, Figure 2 plots the average age cohorts left full-time education by the year they were age 14 between 1940 and 1953 (i.e. cohorts 1926-1939). Figure 3 does the same but for those who were age 15 between 1967 and 1980 (i.e. cohorts 1952-1965). Note that these fitted values are obtained from regressing the means on birth cohort and age polynomials (cubed), year fixed effects, and a dummy for whether or not a cohort faced a minimum school-leaving age – age 15 for cohorts 1926-1939, and 16 for cohorts 1952-1965. The vertical line in Figure 2 represents the change of minimum school-leaving age from 14 to 15 in 1947 and, in Figure 3, from 15 to 16 in 1973. Consistent with Oreopoulos (2006), we can see a clear jump in the average age left full-time education after both reforms. The fit predicts an increase in the schooling level (or  $\beta_1$  in equation (1)) between 1946 and 1947 of 0.35 years, and between 1972 and 1973 of approximately 0.20 years.

Figure 4 and 5 plot the corresponding mean hypertension using cohorts 1926-1939 and 1952-1965 respectively. There appears to be a fall in the mean hypertension between 1946 and 1947. The same cannot be said for the 2<sup>nd</sup> law change in 1973; the effect of the law on hypertension is positive albeit statistically insignificant at conventional levels. Though not shown in the table, the ratio of the estimates ( $\gamma_1 / \beta_1$ ), or the local average treatment effects (LATE), is -0.093 (= -0.033/0.356) for cohorts 1926-1939 (i.e. the 1<sup>st</sup> reform in 1947), and 0.025 (= 0.005/0.199) for cohorts 1952-1975 (i.e. the 2<sup>nd</sup> reform in 1973) respectively. This seems to be in contrast with the findings obtained by Damon and Royer (2008) and Jürges, Kruk, Reinhold (2009), which conclude that there is insignificant compulsory schooling effect on adult blood pressure, hypertension, blood fibrinogen and C-reactive protein levels. Additionally, the statistically insignificant effect of the 2<sup>nd</sup> reform may be due to the fact that the law change in 1973 only led to an increase in the time spent in full-time education by 0.2 years, i.e. one-half of the effect of the 1<sup>st</sup> reform in 1947.

Given that men and women may benefit differently from the reforms (see, e.g., Devereux and Hart, 2010), I re-estimate equations (1) and (2) by gender and report the corresponding results in Figures 6-8. We can see from these figures that the effect of compulsory schooling on hypertension is negative and statistically significant at the 5% level for both men and women. Looking at Figures 6A & 6B, there is a noticeable fall in the mean hypertension between 1946 and 1947 of -0.028-points for men and -0.036-points for women respectively. Regarding the LATE estimates, it appears that an extra year of education leads to a reduction in the risk of developing hypertension by approximately 8-percentage-points (=  $-0.028/0.359$ ) for men and 10-percentage-points (=  $-0.036/0.351$ ) for women, on average. Nonetheless, the effect of the law on both male and female hypertension is positive but not statistically different from zero when it was implemented for the 2<sup>nd</sup> time in 1973.

The RD approach may nevertheless lead to some imprecision, given that hypertension is a binary outcome variable and not a continuous outcome variable. To allow for the fact that the dependent variable is a [0,1] variable, Table 1 estimates for cohorts 1926-1939 the effect of education on adult hypertension using both probit and IV-probit models. Column 1 shows that, without age controls and year fixed effects, the coefficient on educational attainment (i.e. school leaving age) is roughly -0.044, with a statistically well-defined standard error of 0.005. Adding a cubed age control and year dummies leaves this coefficient unchanged.

In the first-stage regression, the minimum school-leaving age variable enters the schooling equation positively and statistically significantly at the 1% level. Independent of birth cohort polynomials, age polynomials, and year fixed effects, individuals who faced the minimum school-leaving age of 15 have approximately 0.36-year more full-time education than those who faced the minimum-schooling age of 14. Note that the 0.36 years jump in the schooling level after 1947 corresponds to the prediction made in Figure 1.

The IV-probit estimates of the schooling effects on hypertension are reported in the last three columns of Table 1. We can see that there is a negative and statistically significant relationship between schooling and hypertension; the coefficient on school-leaving age with a full set of controls is -0.209 and a standard error of 0.074.

Using the same birth cohorts (i.e. 1926-1939), the IV-probit estimates in Table 2 imply that an extra year of schooling helped reduce the probability of men having hypertension by approximately 7-percentage-points compared to the almost 2-percentage-points obtained in the probit model. In addition to this, an extra year of schooling also helped to reduce the probability of hypertension by around 8-percentage-points for women compared to the almost 2-percentage-points obtained in the probit model. These are large effects;

among those who were born between 1926 and 1939, an extra year of education helped reduce the proportions of males and females with hypertension from just above 65% to a level that is below 60%. The sizeable impacts of schooling on hypertension are also consistent with Becker and Murphy (2007) whose work predicts education to have a large effect on non-market consumptions.

At a glance, the differences between the probit and IV-probit marginal effects obtained may seem very large. However, a closer examination reveals that these differences are actually in line with what would have been obtained if OLS and 2SLS were used instead as our main models of choice. For example, running an OLS on males' hypertension equation yields an estimated coefficient of -0.015, with a statistically robust standard error of 0.004, i.e. an extra year of education reduces the probability of men having hypertension by almost 2-percentage-point. On the other hand, a 2SLS model produces an estimated coefficient of -0.068 and a standard error of 0.030, which implies a marginal effect of 7-percentage-point. In other words, it virtually makes no difference whether an OLS or a probit model was used in the estimation of the compulsory schooling effect on hypertension. Moreover, it appears that these differences are also comparable with the differences in the estimates obtained in some of the previous studies on the market returns to education. For example, Harmon and Walker (1995) find the effect of the law to increase the wage-premium from 6-percentage-points to 18-percentage-points, whilst Oreopoulos (2006) finds the differences in the OLS and 2SLS estimates to be an increase of around 7-percentage-points – from 7-percentage-points to 14-percentage-points.

The analysis carried out by Becker and Murphy (2007) also predicts that the returns to education should be even larger for non-market activities relative to market activities. Their conclusion is based on the assumptions that individuals typically perform a relatively small range of tasks in the market and are paid based on their marginal productivity, but perform a wider range of tasks in the household and collect the full product of their household production. Given that wages are not observed in the HSE, it is not possible for us to do a comparative study on the market versus non-market returns to education. However, the sizeable rates of returns from schooling on biomarkers of the order of 7-10% appear to be comparable with the 7-15% rates of market returns typically found in the literature.

Could the results be sensitive to the way hypertension is defined? So far in this paper, a person is said to have hypertension if his or her measured systolic BP  $\geq$  140 mmHg and/or diastolic BP  $\geq$  90 mmHg/or the person taking prescribed medicine to help with high blood pressure, and 0 otherwise. What this means is that everyone who takes prescribed medicine to

help with blood pressure will be included in the hypertensive category regardless of whether or not a high blood pressure was recorded by the visiting nurse in the year of the interview. Since more years of schooling may increase individual's tendency to start taking medicine more regularly (i.e. highly educated individuals may procrastinate less than those with less years of education), it might be worth analyzing the effect of education on the probability of 'recorded' hypertension (i.e. having systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg, but excluding those non-hypertensive cases who take prescribed medicine to help with high blood pressure), systolic hypertension, and diastolic hypertension separately. The estimation of different dependent variables is carried out for both genders in Table 3.

The IV-probit yields the estimated schooling effect that is negative and significant at least at the 5% level in all hypertensive equations for men but not for women. The effect of male education is negative, sizeable and statistically robust even when a dummy variable which takes a value of 1 if the individual takes prescribed medicine to help with high blood pressure has been controlled for in the estimation (see Column 2). For both genders, the coefficient on taking an HBP medicine is positive and statistically significant at the 1% level, suggesting that people who take medicine to help with blood pressure are also likely to fall within the hypertensive rather than the non-hypertensive category.

Why is the causal effect of schooling on women's hypertension statistically insignificant when the dependent variable is the 'recorded' hypertension variable and not the original hypertension variable that includes the non-hypertensive cases of those who take prescribed medicine? One plausible explanation for this may be that education encourages both men and women to take prescribed medicine more regularly, thus leading to an increase in the proportion of non-hypertensive individuals falling within the original hypertensive category, i.e., systolic blood pressure  $\geq 140$  mmHg and/or diastolic blood pressure  $\geq 90$  mmHg/or those who take prescribed medicine to help with high blood pressure. This is also reflected in the the positive and statistically significant schooling effect on the likelihood of taking prescribed medicine, which can be found in Table 2A in the appendix. However, what Table 3's results are implying is that there may be other additional reduced form effects of schooling on men's health and not women's, which extend beyond its impacts on individual's drug-taking behavior. For example, there is evidence in health economics of a positive association between education and healthy lifestyles, e.g., exercising, reducing alcohol consumption, and quitting smoking (see, e.g., Contoyannis & Jones, 2004; Balia & Jones, 2008; Jones, Rice & Rosa Dias, 2010). And since women generally lead a significantly

healthier lifestyle than men (Contoyannis & Jones, 2004), it is possible that an exogenous increase in the level of schooling will benefit men's health more than women's. Further to this, there is also slightly more variability in males' schooling and hypertension variables relative to females – i.e., the average levels (and standard deviations) of males' and females' schooling are 15.48 (1.60) and 15.41 (1.50) years – which would partly explain why the effect of the law is more pronounced for man relative to women. Finally, the results are consistent with Devereux and Hart (2010) who find a zero rate of return to education for women. Yet in order to shade further light on this issue, future research will have to come back and explore the potential gender differences in the effect of the law on other health-related behaviors, e.g., smoking and healthy eating.

Table 4 moves on to examine by gender the effect of the 2<sup>nd</sup> reform in 1973 on adult hypertension. Using the second group of cohorts (i.e. those who were born between 1952 and 1965) and the original hypertension variable as the dependent variable, we can see that there is a negative and statistically well-determined relationship between school leaving age and hypertension. However, an IV-probit estimation produces a schooling coefficient that is positive albeit statistically insignificant for both men and women, which is consistent with what was obtained earlier in our RD analysis of the effect of the 2<sup>nd</sup> reform (i.e. Figures 7 & 9).

What explains why the 2<sup>nd</sup> school reform produces weaker effects than the 1<sup>st</sup>? One explanation for this is that the 2<sup>nd</sup> reform impacted only a very small proportion of people who would have left school at 15 in 1973, i.e. 22% of the population sample left school at 15 one year before the introduction of the 2<sup>nd</sup> reform in 1973. And because of this, the IV estimates for the 2<sup>nd</sup> reform will only approximate the ATE among this small fraction of population rather than the general population. This explains why the 2<sup>nd</sup> reform produces generally weaker results compared to the 1<sup>st</sup> reform in which more than 50% of the population sample was affected by the raising of the school-leaving age from 14 to 15.

#### **IV. Conclusions**

This paper contributes to the literature of human capital by estimating the biomarker effect of compulsory schooling. Its main value-added is in the use of an 'objective' and clinically-accepted indicator of an individual's underlying health rather than the commonly-used self-assessed health variable in the estimation of the causal effect of education on health. Using two education reforms in the UK as instruments in the hypertension equations, the RD and

IV-probit estimates imply that completing an additional year of schooling helps reduce the probability of both men and women developing subsequent hypertension by approximately 7 to 10 percentage-points. In the IV-probit regressions, the correct estimates of the LATE for schooling indicate the presence of a large and negative bias in the probit estimates of schooling-health relationship. There is also some evidence to suggest that education may be more beneficial for men's health than for women's.

The current study also has important policy implications. First, it provides partial evidence in favor of Grossman (1975) who has suggested that an increase in expenditure on education rather than on health itself is perhaps the most cost effective way to improve the nation's health. Secondly, according to the Office for National Statistics in the UK, death by heart disease explains around 20% of total death in 2005, which is considerably higher than the second-place killer: cerebrovascular diseases (8%), and the third-place killer: lung cancer (7%). If an additional year of schooling can help reduce the incidence of hypertension by up to 10-percentage-points – as is the case for the 1947 law, then the implications of a nationwide change in the minimum school-leaving age of two years rather than one, i.e. from age 16 to 18, which is scheduled to take place in 2013, on the nation's well-being may have been underestimated if one was to simply look at the market returns to education. Finally, the potentially different results by gender (more beneficial for men than for women) are of some interest in themselves. Further studies should be conducted to uncover the underlying mechanisms that education plays in improving men's health more than women's.

The results of this paper also call for further inquiry into the estimation of compulsory schooling on other biomarkers, including, among others, cholesterol and cortisol levels, in order for academics and policy makers to obtain a more complete picture of the relationship between schooling and the true health of a nation.

The current study, however, is not without limitations. One shortcoming concerns the literature critical of the linear regression equations typically used to estimate Grossman's demand for health model. According to the life-cycle model of the demand for longevity under conditions of certainty (see Ehrlich & Chuma, 1990), the production function of gross investment in health needs to exhibit decreasing and not constant return to scale as originally implied by Grossman. Because of this, the demand for health cannot be linear in education, the wage rate, or age, as their impact varies by initial health and wealth and one's life cycle position. This is even more apparent in the demand for longevity model derived under uncertainty of life's end, as the (conditional) explicit solution for optimal investment and value of life saving imply (Ehrlich, 2000). In addition to this, the nonlinear assumption in

education in the estimation of the demand for health has also been supported by the recent empirical evidence in the human capital literature. For example, Jason Fletcher and David Frisvold (2009) reported lower estimates on the effects of schooling versus college attendance on preventive health care use. According to the authors, one plausible explanation for this is that individuals who attended college, who differ in both education and accumulated wealth, have a greater and nonlinear incentive to pursue self-protective measures than do individuals with less than college attainments<sup>2</sup>. Given the potentially nonlinear effects of education on the demand for health at different stages of the life-cycle, a linear regression model – one that assumes a constant effect of education on health – may not be the most appropriate strategy to estimate the overall effects of education on health across an individual’s lifespan. Consequently, this limits the kind of inferences which could be made based on the current study’s findings, i.e. the effect of the law on hypertension may be age-specific. However, to pursue an analysis of a non-linear model empirically would require significantly more stringent work that extends beyond the scope of the paper. In addition to this, the current data set does not contain essential variables – such as the individual’s initial health status, for example – which are required for a non-linear model to be estimated. For these reasons, future research should return to examine the causal effect of different educational attainments on health outcomes at various points in the life-cycle using a more appropriate data set.

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<sup>2</sup> For the complete numerical solutions which illustrate the non-linearity in the effects of education over the life-cycle, and its dependence on the wealth accumulation path, see Ehrlich and Yin (2005).

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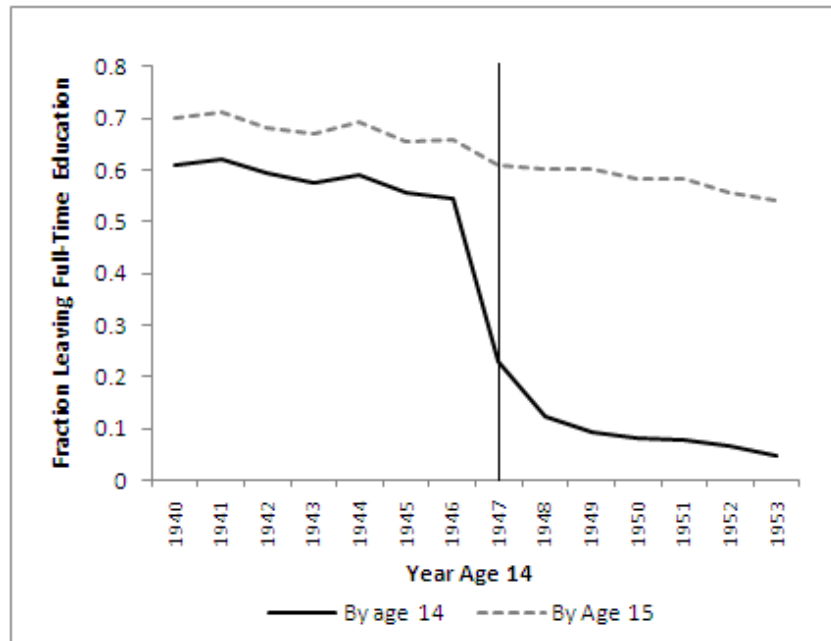
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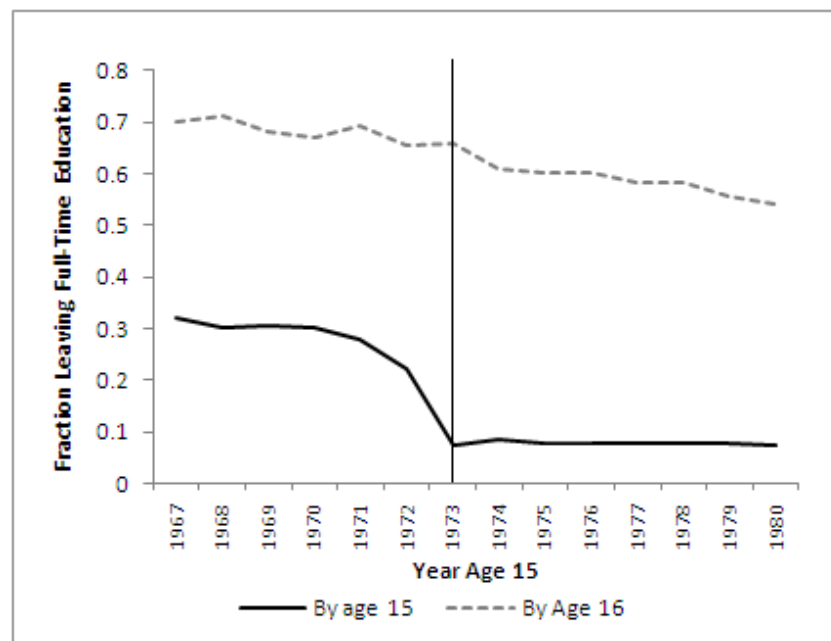
**Figures 1A & 1B: Fraction of Students Left Full-Time Education By Age 14 and 15,**

**HSE 1991-2007**

**Fig.1A**

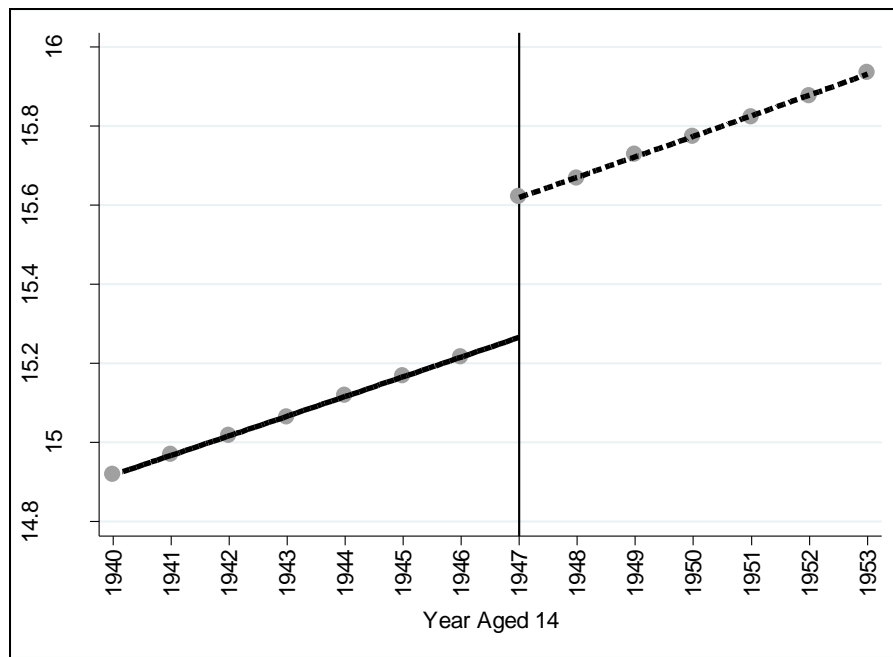


**Fig.1B**



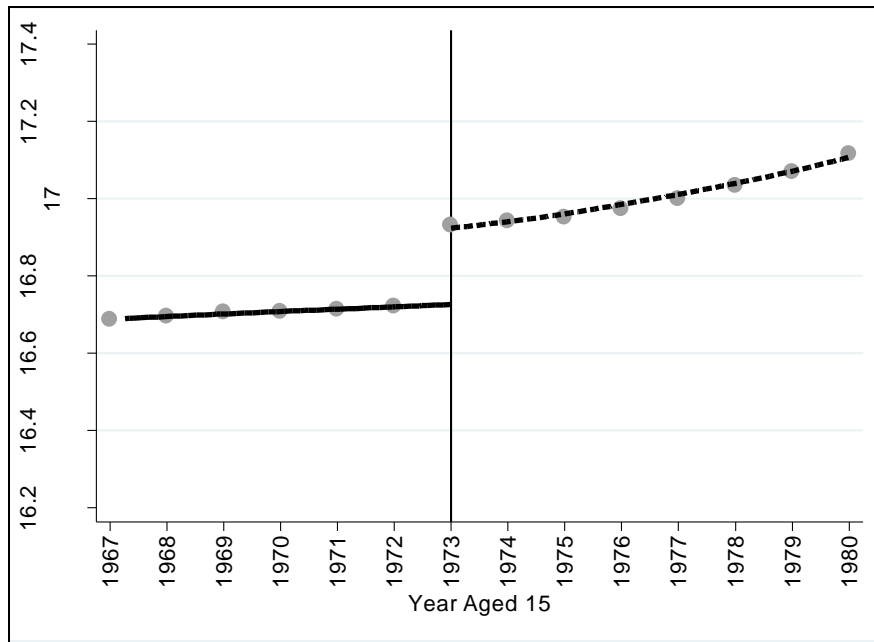
**Note:** the bottom line in Fig.1A represents the proportion of adults in the HSE who left school at or before the age of 14 between 1940 and 1953, and the top is the same but for age 15. The bottom line in Fig.1B represents the proportion of adults in the HSE who left school at or before the age of 15 between 1967 and 1980, and the top is the same but for age 16. Note that the first introduction of minimum school-leaving age law in the United Kingdom took place in 1947 (from age 14 to 15) and then again in 1973 (from age 15 to 16), as indicated by the vertical lines.

**Figure 2: Local Averages and Parametric Fit of Average Schooling Age  
By Year Aged 14 (Age-Adjusted and Time-Detrended)**



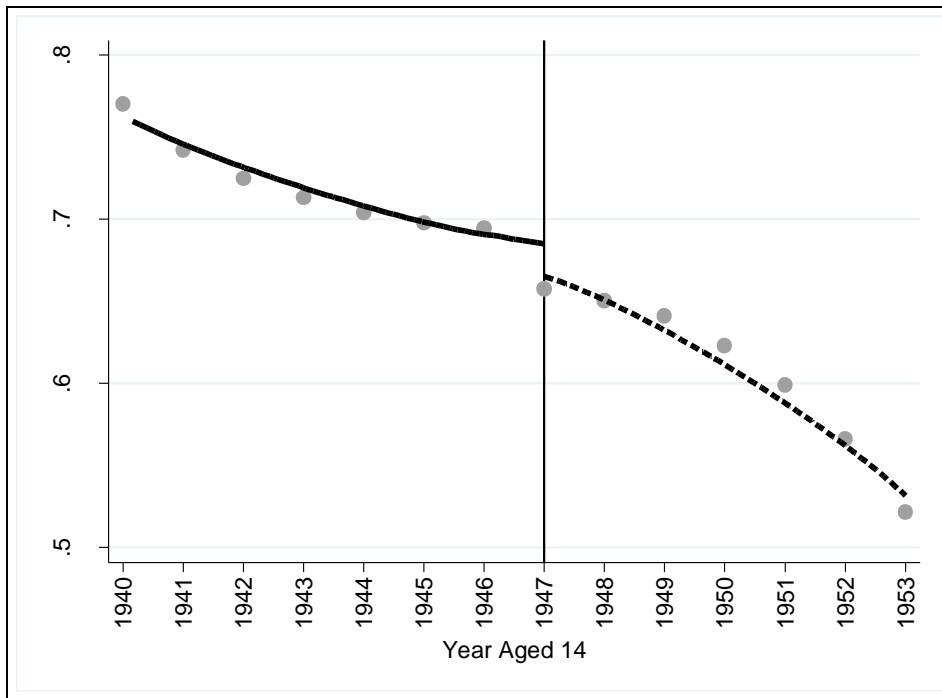
**Note:** Local averages are plotted for English individuals who were aged 14 between 1940 and 1953. The line represents the predicted fit by regressing mean age finished full-time education on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 14. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 14 to 15 in 1947, as indicated by the vertical line.

**Figure 3: Local Averages and Parametric Fit of Average Schooling Age  
By Year Aged 15 (Age-Adjusted and Time-Detrended)**



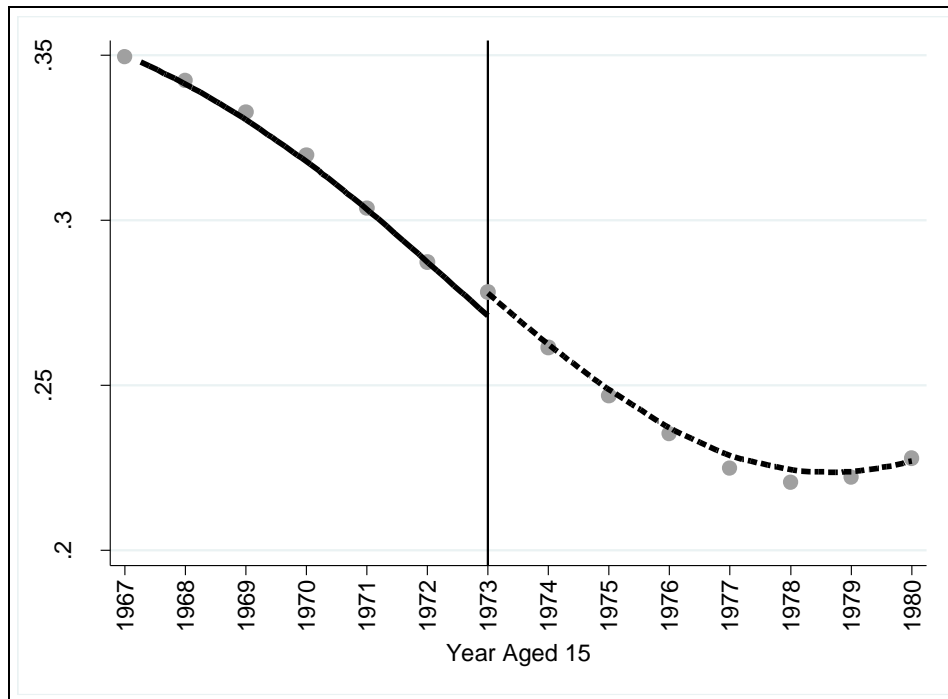
**Note:** Local averages are plotted for English individuals who were aged 15 between 1965 and 1990. The line represents the predicted fit by regressing mean age finished full-time education on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 15. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 15 to 16 in 1973, as indicated by the vertical line.

**Figure 4: Local Averages and Parametric Fit of Average Hypertension  
By Year Aged 14 (Age-Adjusted and Time-Detrended)**



**Note:** Local averages are plotted for English individuals who were aged 14 between 1940 and 1953. The line represents the predicted fit by regressing mean hypertension (systolic BP $\geq$ 140 and/or diastolic BP $\geq$ 90 or taking medicine prescribed for high blood pressure) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 14. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 14 to 15 in 1947, as indicated by the vertical line.

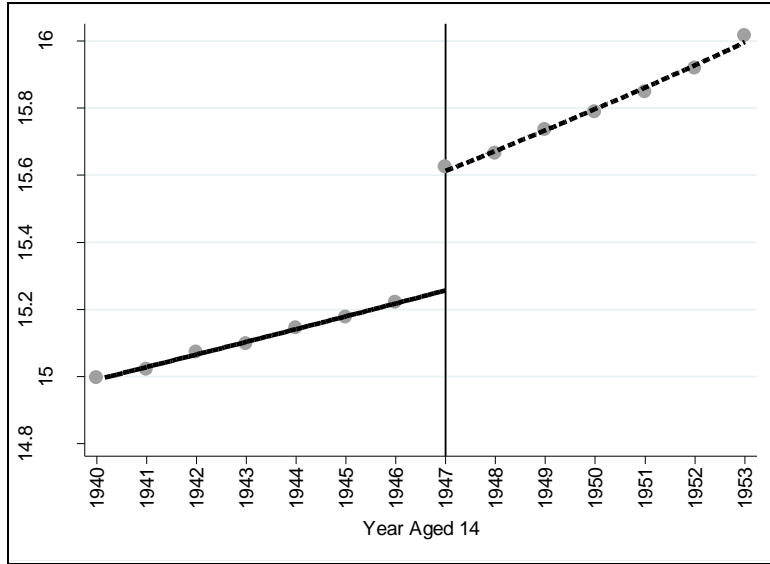
**Figure 5: Local Averages and Parametric Fit of Average Hypertension  
By Year Aged 15 (Age-Adjusted and Time-Detrended)**



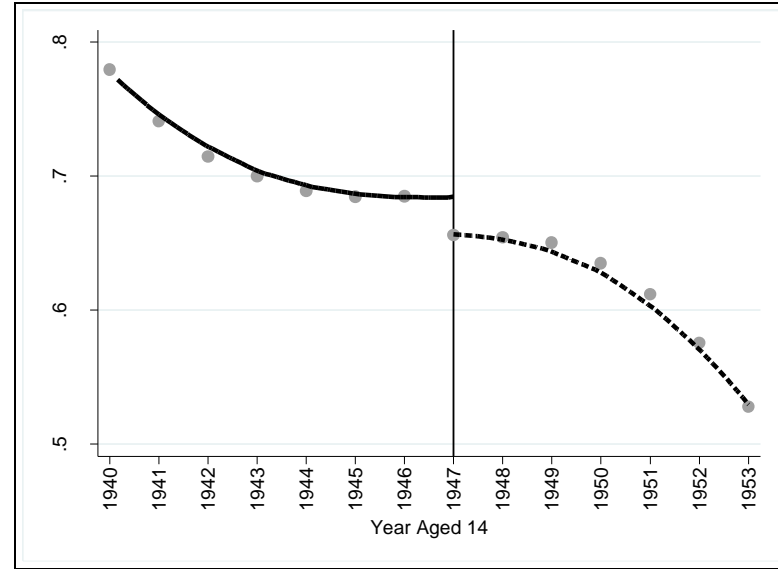
**Note:** Local averages are plotted for English individuals who were aged 15 between 1967 and 1980. The line represents the predicted fit by regressing mean hypertension (systolic BP $\geq$ 140 and/or diastolic BP $\geq$ 90 or taking medicine prescribed for high blood pressure) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 15. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 15 to 16 in 1973, as indicated by the vertical line.

**Figures 6A & 6B: Local Averages and Parametric Fit of Average Schooling Age and Average Hypertension**  
**By Year Aged 14 (Male Subsample, Age-Adjusted and Time-Detrended)**

**Fig.6A**

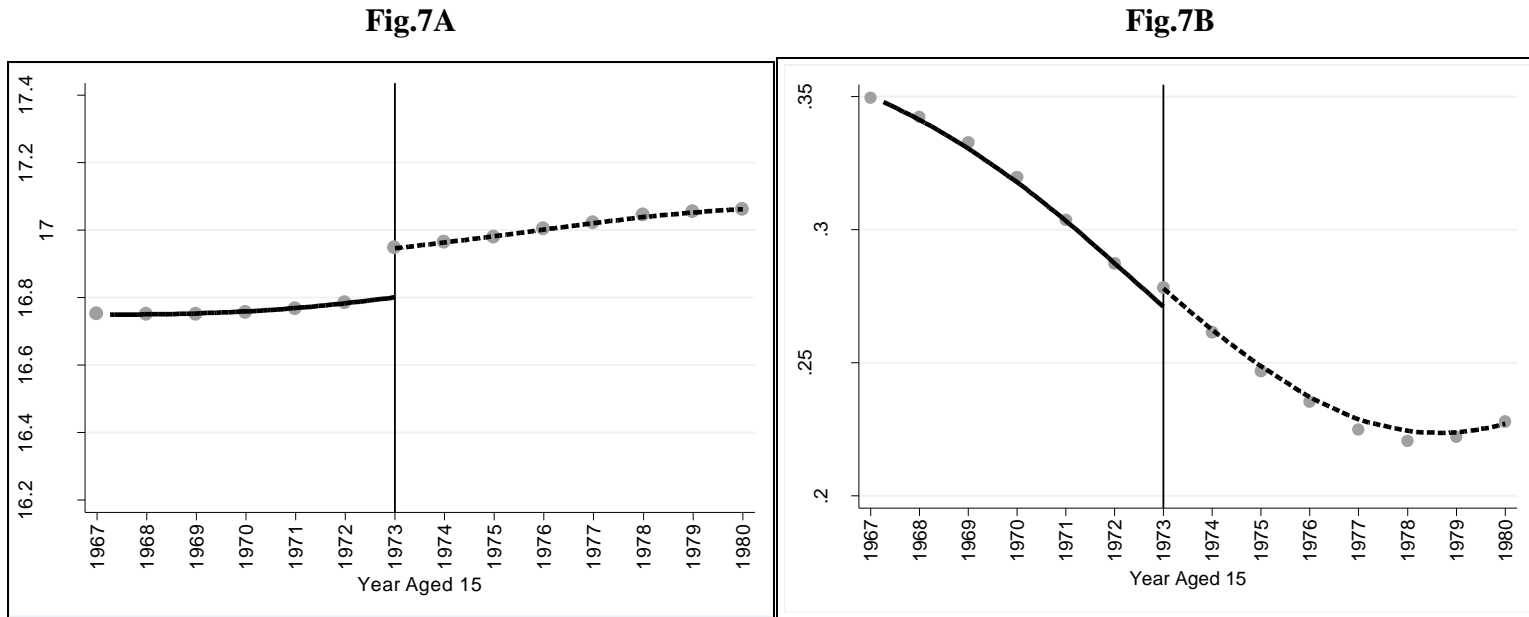


**Fig.6B**



**Note:** Local averages are plotted for English men who were aged 14 between 1940 and 1953. The line represents the predicted fit by regressing mean age finished full-time education in the first diagram (and average hypertension in the second diagram) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 14. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 14 to 15 in 1947, as indicated by the vertical line.

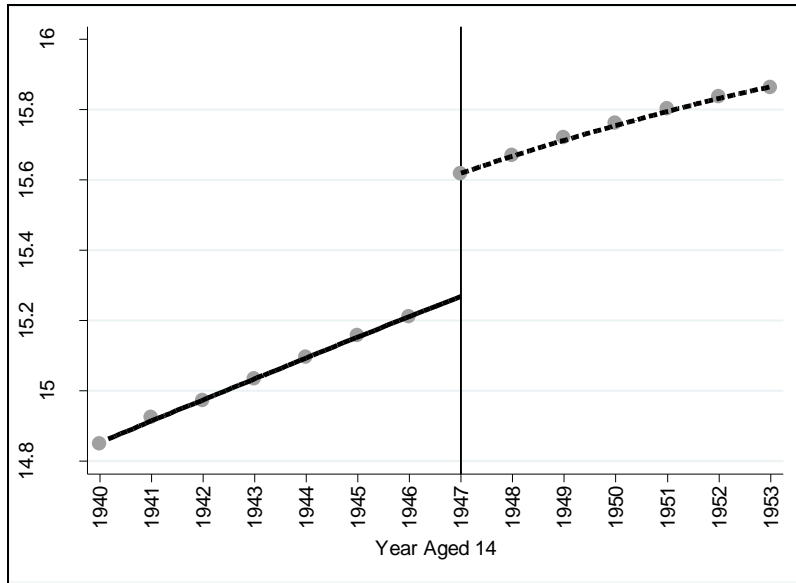
**Figures 7A & 7B: Local Averages and Parametric Fit of Average Schooling Age and Average Hypertension**  
**By Year Aged 15 (Male Subsample, Age-Adjusted and Time-Detrended)**



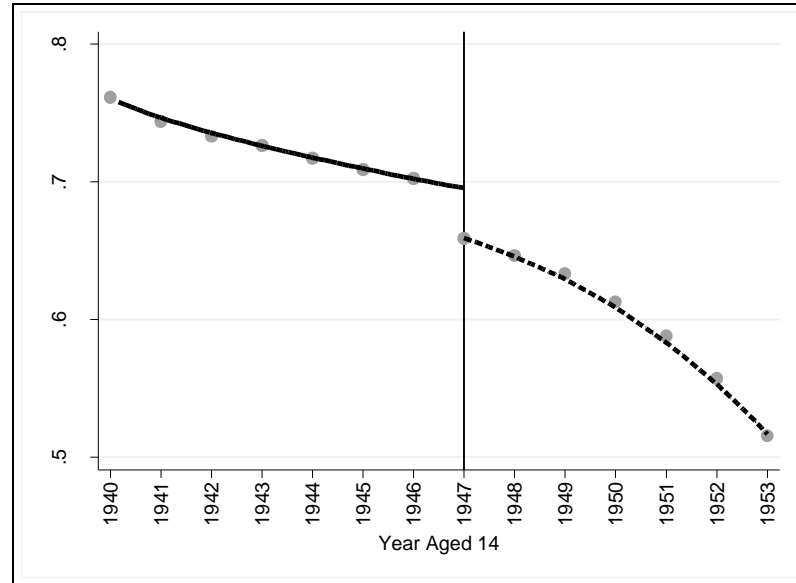
**Note:** Local averages are plotted for English men who were aged 15 between 1967 and 1980. The line represents the predicted fit by regressing mean age finished full-time education in the first diagram (and average hypertension in the second diagram) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 15. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 15 to 16 in 1973, as indicated by the vertical line.

**Figures 8A & 8B: Local Averages and Parametric Fit of Average Schooling Age and Average Hypertension**  
**By Year Aged 14 (Female Subsample, Age-Adjusted and Time-Detrended)**

**Fig.8A**



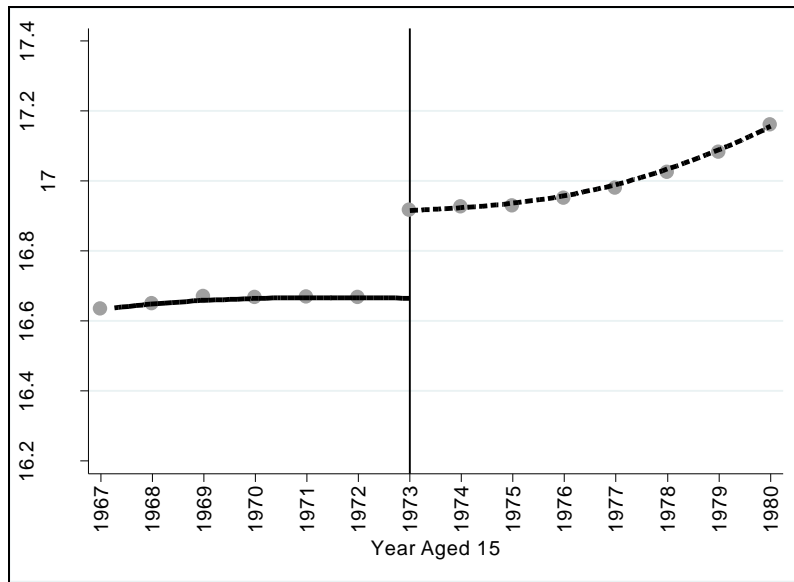
**Fig.8B**



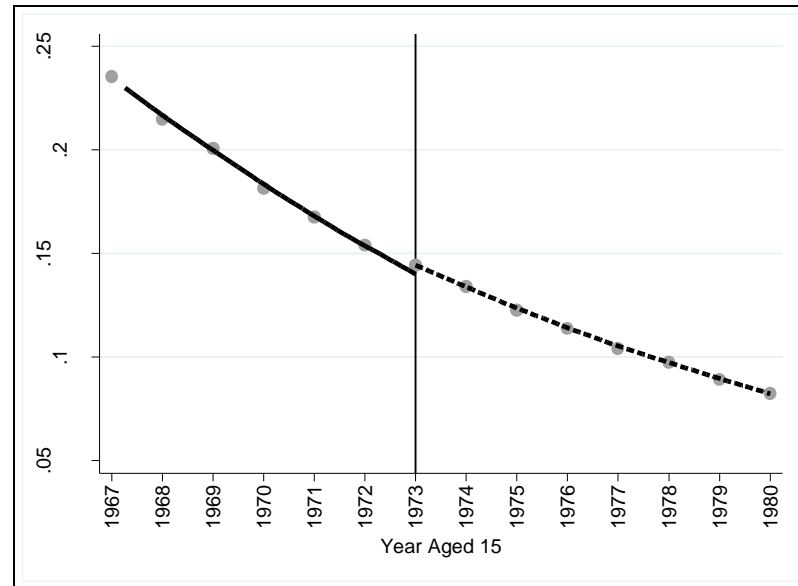
**Note:** Local averages are plotted for English women who were aged 14 between 1940 and 1953. The line represents the predicted fit by regressing mean age finished full-time education in the first diagram (and average hypertension in the second diagram) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 14. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 14 to 15 in 1947, as indicated by the vertical line.

**Figures 9A & 9B: Local Averages and Parametric Fit of Average Schooling Age and Average Hypertension**  
**By Year Aged 15 (Female Subsample, Age-Adjusted and Time-Detrended)**

**Fig.9A**



**Fig.9B**



**Note:** Local averages are plotted for English men who were aged 15 between 1967 and 1980. The line represents the predicted fit by regressing mean age finished full-time education in the first diagram (and average hypertension in the second diagram) on a birth cohort cubed polynomial, age cubed polynomial, surveyed year fixed effects, and an indicator for the school leaving age faced at 15. The regression is weighted by cell size and clustering by birth year. The circles represent local averages. The minimum school-leaving age was raised from age 15 to 16 in 1973, as indicated by the vertical line.

**Table 1: Probit, First Stage School Leaving Age Regression, and IV-probit Hypertension Equations,**

**Health Survey for England 1991-2007, Cohorts 1926-1939**

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
<b>Cohort 1916-1951</b>	<b>probit</b>			<b>(First stage)</b>			<b>IV-probit</b>		
	<b>Dependent variable: Hypertension</b>			<b>Dependent variable: School leaving age</b>			<b>Dependent variable: Hypertension</b>		
School leaving age	-0.044 [0.005]**	-0.043 [0.006]**	-0.044 [0.006]**				-0.204 [0.073]**	-0.204 [0.073]**	-0.209 [0.074]**
Minimum school leaving age = 15				0.355 [0.069]**	0.354 [0.068]**	0.356 [0.069]**			
Birth cohort polynomial controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Age polynomial controls	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes
Year fixed effects	No	No	Yes	No	No	Yes	No	No	Yes

**Note:** +<10%, \*<5%, \*\*<1%. N = 24,574 in all columns. Dependent variable: Hypertension (BP>=140 and/or BP>=90 = 1 or take medication prescribed for blood pressure, and 0 otherwise). Robust standard errors are in parentheses, and are clustered by birth cohort. The raising of the minimum school leaving age from 14 to 15 took place in 1947.

**Table 2: Probit, First Stage School Leaving Age Regression, and IV-probit Hypertension Equations by Gender,**  
**Health Survey for England 1991-2007, Cohorts 1926-1939**

	(1)	(2)	(3)	(4)
<b>1) Men</b>				<b>Percentage of people with hypertension</b>
<b>Cohort 1926-1939</b>	<b>probit</b>	<b>(First Stage)</b>	<b>IV-probit</b>	
School leaving age	-0.041 [0.011]*		-0.189 [0.078]*	
Minimum school leaving age = 15		0.389 [0.052]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-1.51		-6.96	65.91%
	<b>(5)</b>	<b>(6)</b>	<b>(7)</b>	<b>(8)</b>
<b>2) Women</b>				<b>Percentage of people with hypertension</b>
<b>Cohort 1926-1939</b>	<b>probit</b>	<b>(First Stage)</b>	<b>IV-probit</b>	
School leaving age	-0.046 [0.007]**		-0.229 [0.107]*	
Minimum school leaving age = 15		0.351 [0.091]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-1.68		-8.43	65.76%

**Note:** \*<5%, \*\*<1%. N = 11,669 (male subsample) and 12,905 (female subsample). Robust standard errors are in parentheses, and are clustered by birth cohort. Same control variables (i.e. birth cohort cubed polynomial, age cubed polynomial, and year fixed effects) as in Table 1.

**Table 3: IV-probit Hypertension Equations with Different Dependent Variables, Health Survey for England 1991-2007,**

**Cohorts 1926-1939**

	(1)	(2)	(3)	(4)
<b>1) Men</b>				
	<b>‘Recorded’ hypertension (not including those taking medicine)</b>	<b>‘Recorded’ hypertension (controlling for those taking medicine)</b>	<b>Systolic hypertension</b>	<b>Diastolic hypertension</b>
<b>Cohort 1916-1951</b>				
School leaving age	-0.254 [0.086]**	-0.268 [0.085]**	-0.242 [0.086]**	-0.187 [0.084]*
Taking prescribed medicine for HBP		0.213 [0.044]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-10.03	-5.47	-9.61	-5.02
Fraction of people with specified hypertension	56.11%	56.11%	53.87%	18.73%
	(5)	(6)	(7)	(8)
<b>2) Women</b>				
	<b>‘Recorded’ hypertension (not including those taking medicine)</b>	<b>‘Recorded’ hypertension (controlling for those taking medicine)</b>	<b>Systolic hypertension</b>	<b>Diastolic hypertension</b>
<b>Cohort 1916-1951</b>				
School leaving age	-0.114 [0.142]	-0.138 [0.147]	-0.047 [0.109]	0.137 [0.254]

Taking prescribed medicine for HBP		0.254		
		[0.029]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-4.52	-5.47	-1.87	2.66
Fraction of people with specified hypertension	55.34%	55.34%	53.70%	11.50%

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**Note:** \* $<5\%$ , \*\* $<1\%$ . N = 11,669 (male subsample) and 12,905 (female subsample). ‘Recorded’ hypertension (not conditioning on taking medicine for blood pressure) = 1 if BP $\geq$ 140 and/or BP $\geq$ 90 = 1, and 0 otherwise. Systolic hypertension = 1 if systolic BP  $\geq$  140 mmHg, and 0 otherwise. Diastolic hypertension = 1 if diastolic BP  $\geq$  90 mmHg, and 0 otherwise. Robust standard errors are in parentheses, and are clustered by birth cohort. Same control variables (i.e. birth cohort cubed polynomial, age cubed polynomial, and year fixed effects) as in Table 1.

**Table 4: Probit, First Stage School Leaving Age Regression, and IV-probit  
Hypertension Equations by Gender, Health Survey for England 1991-2007,**

**Cohorts 1952-1965**

	(1)	(2)	(3)	(4)
<b>1) Men</b>				<b>Percentage of people with hypertension</b>
<b>Cohort 1952-1965</b>	<b>probit</b>	<b>(First Stage)</b>	<b>IV-probit</b>	
School leaving age	-0.025 [0.004]**		0.222 [0.283]	
Minimum school leaving age = 16		0.136 [0.031]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-0.83		7.52	28.31%
	(5)	(6)	(7)	(8)
<b>2) Women</b>				<b>Percentage of people with hypertension</b>
<b>Cohort 1952-1965</b>	<b>probit</b>	<b>(First Stage)</b>	<b>IV-probit</b>	
School leaving age	-0.042 [0.009]**		0.063 [0.143]	
Minimum school leaving age = 16		0.251 [0.039]**		
Marginal effect of one extra year of full-time education (in percentage-point)	-0.92		1.39	13.79%

**Note:** \*\*<1%. N = 15,867 (male subsample) and 18,692 (female subsample). Robust standard errors are in parentheses, and are clustered by birth cohort. The raising of the minimum school leaving age from 15 to 16 took place in 1973. Same control variables (i.e. birth cohort cubed polynomial, age cubed polynomial, and year fixed effects) as in Table 1.

**Table 1A: Summary Statistics (Cohorts 1926-39 and 1957-1965)**

	All		Left school age 14		Left school age 15		Left school age 16	
	M	SD	M	SD	M	SD	M	SD
Hypertension*	0.421	0.493	0.723	0.447	0.542	0.498	0.173	0.378
Systolic hypertension (systolic $\geq$ 140mmHG)*	0.315	0.464	0.595	0.490	0.327	0.469	0.136	0.343
Diastolic hypertension (diastolic $\geq$ 90mmHG)*	0.110	0.313	0.156	0.363	0.123	0.329	0.054	0.226
Take prescribed medicine*	0.278	0.448	0.313	0.464	0.248	0.432		
Age finished full-time education	16.268	1.626	15.071	1.606	16.236	1.569	16.975	1.373
Minimum school-leaving age = 15*	0.622	0.484						
Minimum school-leaving age = 16*	0.248	0.431						
Age	50.512	12.167	68.728	4.820	52.305	8.530	36.501	4.914
Female*	0.531	0.499	0.524	0.499	0.530	0.499	0.538	0.498

**Note:** \*dummy variable. Hypertension =1 if systolic BP $\geq$ 140 and/or diastolic BP $\geq$ 90, and 0 otherwise. The ‘hypertension’ variable is constructed from the averages of the systolic BP and diastolic BP, which are the mean of the second and third readings in each case, conditioning on these readings being valid. The hypertension dummy also takes the value of 1 if individuals also take medicine to help with their blood pressure. To have any BP reading, HSE respondents had to agree to the nurse visit and agree to have their blood pressure read. For the nurse to classify the reading as valid, the nurse had to be satisfied that, among other things, the respondent did not smoke, drink alcohol or exercise in the 30 minutes preceding the reading.

**Table 2A: IV-probit Take Prescribed Medicine Equations by Gender,**  
**Health Survey for England 1991-2007, Cohorts 1926-1939**

	<b>(1)</b>	<b>(2)</b>
	<b>Men</b>	<b>Women</b>
School leaving age	0.181 [0.074]*	0.263 [0.077]**
Marginal effect of one extra year of full-time education (in percentage-point)	5.99	9.27
Fraction of people taking prescribed medicine	27.12%	30.98%

**Note:** \* $<5\%$ , \*\* $<1\%$ . N = 11,368 (male sample); N = 12,581. Robust standard errors are in parentheses, and are clustered by birth cohort. Same control variables (i.e. birth cohort cubed polynomial, age cubed polynomial, and year fixed effects) as in Table 1.