

# **The causal relationship between education and health-related measures and behaviours: Evidence from England**

## **Abstract**

Two strands of explanations argue for the association between education and health. While the first posits it as spurious, the second suggests a causal relationship. I propose the latter through a natural experiment with the second change in compulsory schooling laws in England. As the laws interacted with the timing of the Ordinary Level examinations to alter the probability of obtaining its qualification, I exploit exogenous variation in the likelihood to attain it for January- and February-born individuals. With data from the Health Survey for England, I use February-born individuals as an instrument for education. I find education has no causal effect on various health-related measures and behaviours.

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## **1. Introduction**

From 2006 to 2007, almost half of the National Health Service's (NHS) costs were attributed to behavioural risk factors: diet-related sickness, sedentary lifestyles, smoking, alcohol and obesity cost £5.8 billion, £0.9 billion, £3.3 billion, £3.3 billion and £5.1 billion respectively (Scarborough et al., 2011). This mammoth sum, deemed an economic burden on public resources, attracted the government's attention. In the recent Budget, the Chancellor introduced a tax on the sugar content of soft drinks from 2018 to tackle childhood obesity aimed at compelling individuals to consider external costs associated with its consumption which they do not bear such as the publicly-funded health costs of treating diet-related diseases. The effectiveness of this or any further government intervention in an attempt to correct this "externality" will influence the way the NHS allocates its limited resources efficiently in promoting public health.

Beyond this political issue runs an underlying discussion of the social determinants of health which have long been studied (Wilkinson and Marmot, 2003; Adams et al., 2003). In particular, the effects of education on health has been of interest since the inception of Grossman's (1972) health model. Two broad explanations have emerged for the correlation between education and health.

The first explains the observed positive correlation as spurious and caused by unobservable variables like family characteristics (Case et al., 2002), non-cognitive abilities (Card, 1999) and time preferences (Cutler and Lleras-Muney, 2006). Reverse causality exists where individuals expected to have better health have greater willingness to invest more in education as they have more time to reap positive returns to education from longer life expectancies.

Although possible mechanisms creating this correlation are still largely unknown, the second argues a possible causal link between education and health for several reasons. Firstly, higher productivity from more education directly translates to a higher level of health production through allocative efficiency (Kenkel, 1991; Rosenzweig, 1995) and productive efficiency (Grossman, 1972) in market (Currie and Moretti, 2003) and non-market outcomes (Michael, 1973). One aspect of the latter is education's impact on health. For example, low literacy is associated with a poor understanding of hospitals' discharge instructions (Spandorfer et al., 1995) while higher educated individuals are more likely to follow medical treatments (Goldman and Smith, 2002). Relatedly, higher educated people spend more time on health-related activities because they are better at allocating inputs (Grossman, 1972). Secondly, higher educated individuals use their higher earnings to purchase healthier lifestyles (Glied and Lleras-Muney, 2003) which entail more expensive medical treatments, healthier food consumption and living in healthier areas.

As education level is an individual choice depending on unobserved characteristics such as family background and time preferences, an endogeneity problem arises when these characteristics also affect health. A common strategy to address this issue is to exploit exogenous variations in education not correlated with other variables influencing health with natural experiments, allowing the chance to test the existence of any causality running from education to health. Recent research which has concentrated on the second strand of arguments have tried to establish a causal relationship between education and health with changes in compulsory schooling laws.

England has had two compulsory schooling law changes. The first, which happened in 1947, kept half of the affected cohorts in school for an additional year. It raised the

minimum age for leaving school from fourteen to fifteen years old following the Butler Education act passed in 1944. The second, which happened in 1973, kept one quarter of the affected cohorts in school for an additional year. It raised the minimum age for leaving school from fifteen to sixteen years old when the Raising of School Leaving Age Order was passed in 1972. Like Braakmann (2011), I focus on the latter treatment effect concerning individuals born between September 1957 and 1970 as my natural experiment.

My dissertation contributes to the literature by incorporating additional years of data from 1991 to 1993 in my sample which were not analysed before. I measure various health-related measures and behaviours including Body Mass Index (BMI) which was not considered by Braakmann (2011). Amongst the different outcomes examined, I lower the threshold of the frequency of unhealthy food consumption from Braakmann's six times a week to twice a week in an attempt to capture a significant effect. I run Ordinary Least Squares (OLS) and two-stage least squares (2SLS) regressions in a sample containing all individuals and a discontinuity sample comprising individuals born only in January and February. My results show education has no causal effect on various health-related measures and behaviours.

The following section presents the existing literature consisting the institutional setting, Grossman's health model and the association between education and health, Section 3 describes the data and statistics, Section 4 introduces the general econometric approach, Section 5 reveals the results for health-related measures and behaviours, Section 6 discusses the results and Section 7 concludes.

## **2. Literature review**

### *2.1 Institutional setting*

In England, children begin school in the academic year once they reach five years old. An academic year starts on 1<sup>st</sup> September and ends on 31<sup>st</sup> August the following year. Each academic year comprises three terms starting in September, January and April respectively.

Children who reach the minimum school leaving age in the United Kingdom (UK) may not leave school immediately unlike the United States (US). From 1962 to 1997, children born between 1<sup>st</sup> September and 31<sup>st</sup> January were allowed to leave school at the end of the Spring term before Easter but those born between 1<sup>st</sup> February and 31<sup>st</sup> August were required to stay in school until the Friday before the last Monday in May (Anderberg and Zhu, 2010).

This created two discontinuities in the mandatory duration of schooling: the first between August- and September-born individuals while the second between January- and February-born individuals. As the August-September discontinuity overlaps between academic cohorts, it is less helpful for comparisons. However, the second discontinuity happens within academic cohorts when January- and February-born children begin school simultaneously with different dates granting permission to leave school. Thus, the second discontinuity controls for differences in curriculum content across academic cohorts and the effects of the age upon entering school while also controlling birth cohort effects. Additionally, there are known differences between August-born children and the others but not between January- and February-born children (Crawford et al., 2007) which may invalidate the analysis for the first discontinuity. For example, August-born children

relative to September-born children are 2 percentage points less likely to attend university at eighteen or nineteen years old and 2.3 percentage points less likely to attend a premier Russell Group institution if they do.

Both discontinuities would change the compulsory duration of education by only one term equivalent to two months. However, birth cohorts until the early 1970s, the school leaving date interacted with the timing of the Ordinary Level (O-level) examinations, the lowest possible school leaving qualification, which were taken at sixteen years old when the summer term ended.

Like (Anderberg and Zhu, 2010; Braakmann, 2011), I focus my analysis on the cohorts which experienced the O-level examinations instead of the later cohorts which took the General Certificate of Secondary Education (GCSE) examinations in place of the O-level examinations from 1988. As the former individuals are considerably older than the latter individuals who are still in their late twenties, they more likely to face any observable health issues. Moreover, combining both cohorts with different education systems may introduce biases and other problems which are difficult to quantify such as the academic rigour of both curriculums.

The interaction with the timing of the examinations is absent for students born before September 1957 who could leave school at fifteen years old and thus missed the O-level examinations if they chose to leave the earliest. However, a natural experiment arises through the interaction of the timing of O-level examinations and compulsory schooling laws for individuals born between September 1957 and the early 1970s. The combination of the timing of O-level examinations and the variation in school leaving dates creates large discontinuities in the probability of attaining any qualification. Across the cohorts

which did the O-level examinations, February-born individuals are two to three percent more likely to leave school with a qualification than January-born individuals (Braakmann, 2011). Thus, the substantial increase in individuals with a O-level qualification from cohorts born from 1957 onwards compared with earlier cohorts is due to the raise in the minimum school leaving age which increased the probability of taking the O-level examinations at sixteen years old. While more academically gifted individuals who continued with higher-level qualifications were unaffected, none of the additional O-level holders proceeded with more advanced qualifications. This observation is consistent with individuals who would have preferred to leave school earlier but complied to take the O-level examinations.

## *2.2 Grossman's health model*

Grossman (1972) posits health as a durable capital stock like human capital which is non-transferable and depreciates with age. Unlike human capital which directly increases productivity, health capital indirectly increases productivity by yielding an output of healthy time. Individuals are assumed be endowed with an initial stock of health which depreciates over time but can be increased by investment so tomorrow's health capital equals today's health capital and gross investment minus depreciation:

$$H_{i+1} = H_i + I_i - \delta_i H_i$$

where  $H_i$  is the individual's health stock,  $I_i$  is gross investment and  $\delta_i$  is the depreciation rate during the  $i$ th period.  $\delta_i$  is assumed to be exogenous and may vary with the individual's age.

The demand for health is unlike other goods because individuals allocate resources to consume and produce health. Consumers make gross investments in health and other goods according to the following set of household production functions:

$$I_i = I_i(M_i, TH_i, E_i)$$

where investment in health,  $I_i$ , depends on medical services purchased as a means to achieve a larger health capital stock,  $M_i$ , time spent on maintaining good health such as exercising,  $TH_i$ , and human capital,  $E_i$  and

$$Z_i = Z_i(X_i, T_i, E_i)$$

where production of a good,  $Z_i$ , depends on owning it,  $X_i$ , time spent producing it,  $T_i$ , and human capital,  $E_i$ . The latter production function emphasises healthy time is necessary to produce a good in order to achieve maximum utility. Both production functions demonstrate health is a consumption good which yields direct utility and an investment good which yields indirect utility to consumers through increased productivity, fewer sick days and higher wages. However, investment in health is costly as consumers must trade time and resources devoted to health with other goals.

The optimal amount of investment in health by an individual to produce a desired stock of health capital begins with first-order conditions for gross investment. It states the present value of marginal benefits equal the present value of marginal cost of gross investment in period  $i$ .

Discounted marginal benefits at age  $i$  equals  $G_i \left( \frac{W_i}{(1+r)^i} + \frac{Uh_i}{\lambda} \right)$  where  $G_i$  is the marginal product of health capital,  $\frac{W_i}{(1+r)^i}$  is the monetary value of unit increase in time for market and non-market activities and  $\frac{Uh_i}{\lambda}$  is the monetary value of increase in utility due to



increase in healthy time. Thus,  $\frac{W_i}{(1+r)^i} + \frac{Uh_i}{\lambda}$  is the discounted marginal value to consumers of output produced by health capital.

As the inherited stock of health and the depreciation rate are exogenous and the gross investment production function is homogeneous of degree one, the optimal amount of gross investment yields the optimal amount of health capital.

Since the stock of health capital cannot be sold in the capital market, gross investment must be positive. Thus, the undiscounted value of the marginal product of the optimal stock of health capital must equal the supply price of capital:

$$G_i \left( W_i + \left( \frac{Uh_i}{\lambda} \right) (1+r)^i \right) = \pi_{i-1} (r - \tilde{\pi}_{i-1} + \delta_i)$$

where  $\pi_i$  is the marginal cost of gross investment in period  $i$ ,  $\tilde{\pi}$  is the percentage change. This determines the demand for capital goods exchanged in a competitive market. Households buy one unit of stock in period  $i - 1$  at price  $\pi_{i-1}$  and sell  $(1 - \delta_i)$  units at price  $\pi_i$  at the end of period  $i$ .  $\pi_{i-1} (r - \tilde{\pi}_{i-1} + \delta_i)$  measures the cost of holding one unit of capital for one period. This transaction suggests a set of single-period flow equilibria for durable stocks.

The used cost of capital must equal the value of the marginal product of the stock at equilibrium so exchanges in the stock of health substitute for exchanges in the capital market. This means an individual will rent one unit of capital from himself for one period if he increases his stock of health by one unit in period  $i$  because he must increase gross investment in period  $i - 1$  by one unit. If he simultaneously reduces gross investment in period  $i$  by  $(1 - \delta_i)$  units, then he raises only  $H_i$  by one unit.

The equilibrium quantities of  $H_i$  and  $Z_i$  are obtained by maximising the utility function subjected to the goods budget, time and “full wealth” constraints. The goods budget

constraint equates the present value of expenditure on goods to the present value of income over a lifespan and assets:

$$\sum \frac{P_i M_i + V_i X_i}{(1+r)^i} = \sum \frac{W_i T W_i}{(1+r)^i} + A_0$$

where  $P_i$  and  $V_i$  are the prices of  $M_i$  and  $X_i$  respectively,  $r$  is the interest rate,  $W_i$  is the wage rate,  $T W_i$  is the hours of work and  $A_0$  is the discounted property income. In short, total health expenditure equals total income and total assets.

The time constraint shows the total amount of time in any given period,  $\Omega$ , must equal the amount of time spent earning income,  $T W_i$ , being ill,  $T L_i$ , being healthy,  $T H_i$ , and consuming goods,  $T_i$ :

$$\Omega = T W_i + T L_i + T H_i + T_i$$

The “full wealth” constraint is constructed by substituting the time constraint into the goods budget constraint:

$$\sum \frac{P_i M_i + V_i X_i + W_i (T L_i + T H_i + T_i)}{(1+r)^i} = \sum \frac{W_i \Omega}{(1+r)^i} + A_0$$

which shows “full wealth” equals initial assets and the present value of income if an individual spent all of his time working. The wealth is allocated to goods expenditure and non-market production time while the rest is lost to illness.

Finally, when health stock is specified as a function of prices, wages, depreciation rate and technology parameters, the model of the demand for health in its reduced form is

$$H_{it} = a_w w_{it} + a_e E_i + a_a Age_{it} + a_t D_t + B_{it}$$

where  $w_{it}$  is the wage,  $E_i$  is the education level,  $D_t$  is the time effects capturing prices of health inputs and  $B_{it}$  is an unobserved component.

When the correlation between  $H_{it}$  and  $E_i$  is deemed as spurious,  $B_{it}$  is correlated with  $E_i$  such that standard estimates of  $a_e$  do not necessarily reflect a causal parameter. A possible correlation between  $B_{it}$  and  $E_i$  may be explained by the endowment hypothesis (Card, 1999; Rosenzweig and Schultz, 1983) which posits when those born with higher ability obtain more education and those born higher health stocks become healthier adults, a correlation between ability and health endowments will imply a correlation between education adult health. The endowment hypothesis reveal health may have crucial unobserved persistent components. Empirically, the endowment hypothesis has been tested by using proxies such as family background and test scores as control variables (Behrman and Wolfe, 1989) and shown controlling for unobservable characteristics does not significantly change the effects of education.

Another explanation for a correlation between  $B_{it}$  and  $E_i$  is individuals with higher preferences for the future are more likely to engage in activities with current costs and future benefits such as smoking habits (Farrell and Fuchs, 1982) and schooling.

Grossman's model suggests health can be maintained by health investments, depending on goods and activity consumption, which affect health although health depreciates as individuals age. As better health gives an individual more time to work and enjoy consumption, more educated individuals are expected to demand more health and invest more in their health. This implies more educated individuals are also more efficient health producers. Therefore,  $B_{it}$  and  $E_i$  should not be correlated.

### *2.3 Education and health*

Empirical research on the positive correlation between years of schooling and health status in the US was once considered a 'socioeconomic status' effect and believed to be

significantly influenced by a positive correlation between schooling and income and a positive effect of income on health (Antonovsky, 1967). However, Grossman's (1972) theory of productive efficiency hypothesised additional years of schooling should help an individual become a more efficient producer of his own health through increased knowledge about health effects of behaviour, better understanding of healthcare options and the ability to exercise self-control (Thaler and Shefrin, 1981). Research later revealed a statistically significant relationship between schooling years and health after controlling for income differences (Auster et al., 1969; Taubman and Rosen 1982), suggesting individual actions affected his health more than external factors like additional medical resources (Newhouse and Friedlander, 1980).

Kenkel's (1991) and Rosenzweig's (1995) theory of allocative efficiency models the premise the better educated choose a more productive set of health inputs. More educated individuals acquire more human capital which helps them choose a more efficient combination of inputs in their health production functions. For example, education increases awareness of the harmful effects of smoking (Friedman et al., 1981; Mokdad et al., 2004; US Department of Health and Human Services, 2004) to inform an individual's cost-benefit analysis. Harris (1979) finds a strong cross-sectional relationship between education and smoking where the more educated are much less likely to smoke. A national probability survey in 1975 by the Department of Health, Education and Welfare revealed the proportion of high-school graduates who smoked was more than 50% higher than that of college graduates. This relationship may explain why the more educated are healthier to the extent this correlation is from a causal effect of education.

Ross and Wu (1995) analyse cross-section samples from a national probability sample of US households in which 2031 respondents between eighteen and ninety years old in 1990 and another set of 3025 respondents between twenty and sixty-four years old in 1979 and again in 1980 to measure two health outcomes: self-reported health (SRH) and physical functioning. They find a positive association between education and health and offer three explanations. Firstly, more educated individuals are more likely to work full-time, enjoy fulfilling jobs and higher incomes which significantly improve health. Secondly, higher educated individuals report a greater sense of control over their lives and have greater social support. Thirdly, the well-educated are less likely to smoke, more likely to exercise and drink moderately. Thus, favourable work conditions, social-psychological resources and healthy lifestyles and good health resulting from education reflect a positive association between education and health.

Adams (2002) provides evidence of an association between education and health among older adults from a US sample drawn from the Health and Retirement Study (HRS) after controlling for observable individual and family background characteristics. His OLS results show higher levels of educational attainment result in healthier outcomes especially among women. He further investigates the extent to which this relationship represents an independent effect of education on health by using the birth quarter and a set of parental and sibling characteristics as instrumental variables (IV) for education to remove biases from error terms correlated with education. His 2SLS results show education's effect on health is independent of omitted variables. Most of his estimates of education on health are positive and significant at least at the ten-percent level. Thus, he

finds education's effect on health remains positive and significant for most reliable health measures.

However, a competing hypothesis suggests education does not play a causal role in explaining health behaviours but attributes the relationship to unobserved characteristics correlated with both variables instead. A potential variable is an individual's preference for the future as these preferences affect the probability of engaging in activities with current costs and benefits which are realised only in the future. Cutler and Lleras-Muney (2006) suggest time preferences lead to different decision-making patterns. Individuals with lower discount rates are more likely to invest in education and health in the long run. For example, they choose not to smoke although the harmful effects from smoking are not realised immediately but only years later.

This "third variable", time preference, can create a positive correlation between education and health even in the absence of any causal relationship between the two variables. Farrell and Fuchs (1982) test whether schooling causes differences in cigarette-smoking habits by examining the smoking behaviour of various cohorts of men and women before and after their formal schooling years. They find the strong negative relationship between schooling and smoking observed at twenty-four years old for individuals with twelve to eighteen schooling years is explained by differences in smoking behaviour at seventeen years old when they were in the same standard. The duration of formal schooling an individual would eventually complete predicted his smoking behaviour before he actually completed the entire duration and finishing the additional schooling years had no marginal effect on their smoking habits. Instead, the robust negative correlation between schooling and smoking appeared only after they were warned of its health consequences. Thus, the

hypothesis of a causal relationship between schooling and smoking differences is rejected in favour of underlying “third variables”.

Tenn et al. (2010) estimate the effect of education on smoking differently by matching individuals who are a year apart in their life cycles to address the potential endogeneity of education. Individuals at a given age, education and student status in the current and previous year are compared with others of the same respective characteristics in the current and following year. The main identification assumption, both groups have similar unobservable characteristics, is reasonable because they are born only a year apart and make the same education decisions at identical life stages. This empirical approach cancels the impact of unobserved characteristics correlated with education to isolate its causal effect. They find one extra schooling year does not have a causal effect on smoking. Instead, unobserved characteristics correlated with education wholly explain their cross-sectional relationship.

Therefore, the omitted variables problem biases estimates measuring the effect of education on health. Due to unobserved variables like health endowments and time preferences, more educated individuals may enjoy better health and vice versa even when schooling does not play a causal role in health outcomes. This underlying “third variable” potentially present in the association between education and health has prompted recent research to use compulsory schooling law changes as natural experiments to establish a causal relationship between education and health. Arendt (2005), Albouy and Lequein (2009), Clark and Royer (2010) and Braakmann (2011) do not find such a causal relationship unlike Glied and Lleras-Muney (2003), Lleras-Muney, (2005), Oreopoulos (2006) and Spasojevic (2010).

Arendt (2005) tests whether the relationship between education and health can be interpreted causally while acknowledging both may be determined by common unobserved factors such as individual time preferences. He employs institutional changes, Danish school reforms, to identify education effects on health by instrumenting education effects on SRH and BMI. Using a panel data set of Danish workers which controls for unobserved heterogeneity over time, he finds expected associations between education and SRH and BMI: longer education is associated with better SRH for men and women. When endogeneity is accounted for, this relationship increases in magnitude but the standard errors also increase when education is instrumented. Therefore, it cannot be rejected education is exogenous to SRH so the null hypothesis of no effect of education cannot be rejected. When school reforms are used as instruments for education, similar results are obtained for BMI as a health outcome despite the jump of educational attainment to a higher level in the years following the 1958 reform.

However, education laws are a fully valid instrument only when used on a small number of consecutive birth cohorts because IV regressions must consider a possible age effect on health to obtain unbiased estimates of the effect of education. Moreover, the aggregate health status of a population improves over time as medicine and hygiene advance: individuals who are fifty years old today are healthier than their counterparts a few decades ago. Although these improvements in health are difficult to measure, this generation effect cannot be ignored when a sample contains cohorts born many years apart. Otherwise, its effect on health will be captured by the birth year, violating the exclusivity condition because being born before or after the reform has a direct impact on



health and possibly an indirect one through education. Nonetheless, Arendt's results show effect of education on health remains open.

Albouy and Lequein (2009) use a French longitudinal dataset to focus on the effect of school leaving age on mortality at older ages. They study two treatment effects in large samples of approximately 40,000 individuals, the Zay and Berthoin reforms, which raised the minimum school leaving age by one year to fourteen years old in 1923 and by two years to sixteen years old in 1953 respectively. Compared with the traditional approach of estimating return to education in an instrumental variable framework like Arendt's (2005), they use a regression discontinuity design to compare cohorts born immediately before or after the reforms which is more appropriate to the nature of such legal changes and a parametric two-stage approach with information from a larger part of the sample. This is because assumptions required for identification are weaker in regression discontinuity design than in the IV approach and provides a nonparametric estimate of return to education. Unlike most empirical work on cross-sectional datasets where individuals in their samples are of different ages when their health is measured, health status measured by Albouy and Lequein concerns individuals of the same age. This ensures health differences are not due to any misspecification in the health dependence in age when studying survival rates at a constant age. By restricting the sample to only six birth cohorts, they limit the impact of any generation effect on the estimates. Both techniques fail to reveal a significant causal relationship between education and health. Despite the increased education levels from these reforms, subsequent declines in mortality observed appears to be insignificant. The results show living until fifty and eighty years old does not seem to be affected by attending school between thirteen and sixteen

years old. The choice of school leaving age to measure an individual's education level best explains the absence of this relationship.

However, the result does not eliminate the existence of returns to education between thirteen and sixteen years old on other unmeasured health aspects. It may be possible schooling years during early childhood or above sixteen years old has a causal impact on mortality. Formal education may improve human capital in terms of knowledge and skills to affect health status. Specific knowledge from medicine and biology acquired in school may be useful for health production but knowledge acquired between thirteen and sixteen years old is unlikely to directly increase the ability to preserve one's health capital. Rather, only two subjects, physical education and biology, in that age range can impart specific knowledge relevant to health production. Physical education may raise awareness amongst students that physical activities may reduce health risks like obesity while biology topics focussing on human metabolic processes may emphasise the importance of eating balanced diets. Additionally, education may develop cognitive skills to enable better selection and consolidation of health-related information. Critical thinking, literacy and numeracy skills which enhance human capital are required to understand health-related issues, therefore, leading to a more efficient allocation of inputs for health production (Grossman, 1972).

As the impact of formal education on health is a cumulative process, each additional schooling year improves health if it increases the amount of knowledge and skills for health production. However, returns to education may depend on a student's motivation during schooling. Individuals obliged to remain in school may benefit less from an extra year of schooling than those who continue voluntarily. The Zay and Berthoin reforms

forced individuals to study longer than they wanted, implying this extra attainment did not significantly contribute to those compliers' intellectual growth. Returns to education on health for the compliers will be smaller than for those who study further even without the Berthoin reform. Thus, both reforms may be weak instruments because they are not significantly correlated with the effect of education on health.

Moreover, the specific aspects of human capital relevant to health production may react to changes in education level but not to changes in school-leaving age. As education improves one's relative position in society, Ross and Mirowsky (1999) suggest more educated individuals have a higher self-esteem and hence better health. Thus, relative education level amongst those who leave school during the same year will affect health. Incomes in the French labour market depended more on ranking in education level hierarchy than on the actual school-leaving age (Grenet, 2003). As the Zay and Berthoin reforms did not change this education position, market opportunities of a given decile of school leaving ages distribution would not have changed even if their average school leaving age increased. Thus, the absence of a significant causal impact may be due to the choice of using school leaving age as a proxy for education level. Nonetheless, it is unclear if school leaving age changes captured human capital changes relevant to health production necessary to support the indirect causal mechanism linking education and health through an income effect.

In the UK, Clark and Royer (2010) exploits both changes to British compulsory schooling laws which generated sharp differences in educational attainment amongst individuals born months apart. They verify the cohorts just affected by those changes completed significantly more education than slightly older cohorts under the old laws. As these law

changes induced such sharp changes in educational attainment, regression discontinuity design detected the effects of education on health, assuming individuals' proximate in birth date would have had similar health outcomes absent the treatment. However, they find little evidence the additional education improved health outcomes or behaviours: smoking, drinking, diet and exercise.

They argue it is difficult to attribute the results to the content of the additional education for similar reasons explained by Albouy and Lequein (2009) or the wider circumstances the affected cohorts faced such as distinct socioeconomic differences in access to care and quality of care (Acheson et al., 1998). For example, while the NHS provides universal health insurance, it can be inequitable when more educated parents manipulate the system such as persuading their general practitioners (GP) for specialist treatment (Lyall, 2008). Despite this, even if the NHS had weakened health returns, higher educated individuals would have engaged in less risky behaviours and more efficient self-management. Evidence suggests highly-educated parents in the UK are more responsive to changes in medical knowledge regarding the perceived safety of childhood vaccines (Anderberg et al., 2008).

Another explanation is these compulsory school changes are likely to have generated only weak peer effects. As these changes affected many students, the affected students are likely to have left school with the same types of peers as the unaffected students. Gaviria and Raphael (2001) and Powell et al. (2005) suggest teenage peers affect health outcomes and behaviours like the propensity to smoke. Thus, the absence of teenage peer effects may explain the absence of a causal relationship in this setting.

However, these explanations assume health effects of education are large and attribute the null results to unique features of the British setting. It may be possible the health effects of education are rather small. Compared with Albouy and Lequien's (2009) who also find small effects on mortality, their estimates are less precise partly because the French compulsory schooling changes had smaller effects on educational attainment and they used smaller samples. Nonetheless, health returns to this extra education are, at best, small.

Braakmann (2011) exploits exogenous variation in the probability to achieve any educational qualification between January- and February-born individuals for thirteen academic cohorts in England with only England's second compulsory schooling law change. The law interacted with the timing of the Certificate of Secondary Education (CSE) examinations to change the probability of obtaining a qualification by around two to three percentage points for these cohorts. With data on individuals born in January and February from the British Labour Force Survey (LFS) and the Health Survey for England (HSE), he investigates the effects of education on health by instrumenting education by being February-born. The OLS results in the samples containing all individuals and in the discontinuity samples containing only January- and February- born individuals are always very similar, suggesting the latter are not that different from other individuals with regards to the relationship between education and health. The OLS results reveal individuals with any qualification are always much less likely to have a health problem but the 2SLS results show a random pattern of insignificant positive and negative point estimates of education on various health-related measures and behaviours: smoking, drinking and diet

in all samples. This insignificant effect of education on health further supports the “third variables” explanation.

As an extension to health based on Nelson and Phelps’ (1966) theory that the return to education is greater the faster the advance of theoretical technology level, Glied and Lleras-Muney (2003) hypothesise health inequalities across socioeconomic groups in the US are vast and increasing because more educated people are better able to take advantage of technological advances in medicine than the less educated. By focussing on overall mortality and cancer mortality through examining the incidence of cancer and survival conditional on illness incidence, they test their hypothesis by relating education gradients in mortality with measures of medical innovation.

Unlike Albouy and Lequien (2009), Glied and Lleras-Muney’s (2003) sample contained individuals of different ages. Thus, they use a flexible specification of the generation effect with birth cohort dummies and a quadratic function of age to control for bias from health differences due to age and the generation effect as assuming a linear dependence for the age or generation effects may have been too restrictive. They find evidence supporting steeper education gradients for diseases with more innovation, suggesting education may enable people to use technological progress more effectively in reducing mortality or in surviving cancer.

In their model, an individual’s health,  $H$ , is a function of the technology level,  $A$ , which the individual has access to and other inputs,  $C$ :

$$H = H(A, C)$$

The frontier technology level,  $T(t)$  is given as

$$T(t) = T_0 e^{\lambda t}$$

where  $T(t)$  is instantaneously diffused and  $\lambda$  is the exogenous rate of technological progress.

They assume the technology level available to any individual depends on the ease individuals “adopt” new technologies and the lag between innovation and adoption is a decreasing function of education such that

$$A(t) = T(t - w(e)) = T_0 e^{\lambda(t-w(e))}$$

where  $w'(e) < 0$ . This assumption implies the more educated adopt new technologies at a faster rate because of greater access to and use of information and larger capacity to find better health providers and treatments (Rosenzweig, 1995).

Thus, an individual’s health production function can be expressed as

$$H = H(T_0 e^{\lambda(t-w(e))}, C)$$

“The education gradient” in health, the first derivative of the health production function with respect to education, is the marginal gain in health from an additional unit of schooling. Since  $w'(e) < 0$ , the model predicts health is an increasing function of education and the rate of return of education is larger the higher the rate of technological change like Grossman’s (1972).

They test their model’s prediction in the data by estimating the disease-specific education gradient and then relating the gradient size to innovation measures which proxy for the parameter  $\lambda$ . Technology includes all innovations which change the way and rate individuals transform inputs into health, implying new knowledge is innovation.

With data from the National Health Interview Survey (NHIS) and Surveillance Epidemiology and End Results (SEER), they find average family income has an independent effect on cancer survival but the relationship between the education gradient

in survival and progress persists even when controlling for family income. They compare the effects of education for those diagnosed before and after Medicare eligibility at sixty-five years old. The correlations between the gradient and progress appear greater for the population with Medicare than for the those below sixty-five years old. Moreover, the compulsory schooling measure shows a causal effect of education, particularly for the effects of survival after cancer diagnosis. These findings do not explain the mechanisms behind each process but suggest two observations: the relationship between the gradient and technological progress cannot be explained away by family income and education has an effect on cancer survival rather than through other characteristics which affect an individual's decision to become educated.

Lleras-Muney (2005) uses US compulsory education laws from 1915 to 1939 as instruments for education to identify the effects of education on a census-based measure of mortality. These laws increased education by five percent a year from an additional year of compulsory schooling. Her model includes birth cohort dummies to account for the generation effect and leverages a unique feature in her data concerning all American states: changes in education laws did not happen simultaneously in all American states. She examines the direct effect of changes in compulsory schooling on the mortality rates of the cohorts immediately before and after the change in legislation in a regression discontinuity design. The results suggest compulsory laws had an effect on adult mortality and support the hypothesis education affects health. Her estimates suggest an additional year of education has a relatively large effect on mortality, reducing ten-year mortality rates by three percentage points off a ten-percent base mortality rate. Thus, education has a causal impact on mortality.



To obtain comparable estimates to estimate the impact of the school leaving changes on health outcomes, Clark and Royer (2010) construct a ten-year mortality rate using Census data for 1991 and 2001. For each birth month cohort, they measure mortality between 1991 and 2001 as one minus the 2001 population count divided by the 1991 population count, yielding estimates much smaller than Lleras-Muney's (2005). For example, men born after April 1933 reduce the 1991-2001 mortality rate by 0.2 percentage points and 1.5 percent of the mean mortality rate for those born before April 1933. Even when this estimate is scaled up by two, it is still much smaller than Lleras-Muney's (2005) General Least Squares (GLS) and IV estimates.

Three reasons may explain the difference between Clark and Royer's (2010) and Lleras-Muney's (2005) findings. Firstly, the compulsory school laws are different across the two countries. The US compulsory school laws are used in with child labour restrictions and compulsory attendance laws but the UK laws refer only to school attendance. If child labour laws changes affect whether children work in potentially dangerous conditions, the US laws are expected to have larger effects on health. Secondly, the US laws are less binding as only five percent of the relevant US cohorts drop out at the earliest possible age before the law changes Lleras-Muney (2005) in contrast to the larger proportions in the UK. Thirdly, the methods used in both studies differ. In particular, since the British school leaving laws are binding at the national level and cause sharp changes in educational attainment, regression discontinuity methods can capture flexible cohort trends in health outcomes. In contrast, Lleras-Muney's (2005) difference-in-difference approach to exploit cohort and state variation in compulsory schooling laws assumes these laws are uncorrelated with state-specific trends in the outcomes.

Oreopoulos (2006) studies only the first change to British compulsory schooling laws to estimate local average treatment effects (LATE) of high school and returns to education using a regression discontinuity design with data from combining fifteen UK General Household Surveys (GHS) from 1983 to 1998 with fourteen Northern Ireland ones from 1985 to 1998. He compares LATE estimates of the US and Canada where very few students were affected by compulsory school laws with the UK's to test whether IV returns to schooling often exceed OLS' because gains were high only for small groups in the UK's population. As changes in compulsory education were implemented country-wide which was the larger geographic area covered by usual datasets, it is impossible to separate the before and after treatment dummy from the birth cohort dummies in an IV specification. Thus, Oreopoulos (2006) used polynomial controls for birth cohort and age instead of dummy variables, similar to Albouy and Lequien's (2009) parametric estimation. He finds the returns to compulsory schooling are substantial, between ten and fourteen percent, regardless of whether the laws impacted a majority or minority of those treated. Since many students in the UK left school at the earliest opportunity, these changes affected a larger portion of the population in comparison to the US where its compulsory school law changes affected only five percent of the cohort (Lleras-Muney, 2002). Thus, estimates based on UK law changes are more likely to be closer to the average treatment effect. The larger IV returns to schooling estimates than OLS' suggest a considerable dropout pattern. The similarity of IV results across countries may imply students from the UK face greater financial constraints from staying than students from the US and Canada. However, removing fees in 1944 did not affect attainment beyond fifteen years old

although half of secondary students in the UK paid some school fees. Moreover, many early school leavers did not work (Oreopoulos, 2006).

Three other explanations may account for dropout behaviour. Firstly, dropouts may feel compelled to leave school even at the expense of forgoing large monetary gains (Lee and Burkam, 2003) due to poor student performances and negative attitudes from their peers and teachers. Secondly, the uncertainty of additional earnings from increased schooling for risk-averse dropouts encourages them to leave school early (Levhari and Yoram, 1974). Thirdly, dropouts may severely discount future consequences of their current decisions (O'Donoghue and Rabin, 1999) and incorrectly calculate present values of future returns.

Spasojevic (2010) uses the 1950 Swedish comprehensive school reform which was implemented by municipal areas in stages and finds extra schooling caused by Sweden's compulsory school reform improves adult health. The cohort most affected by the compulsory schooling reform consisted individuals born between 1945 and 1955. Most individuals born in 1945 were still part of the old system when the new system started unless they lived in one of the municipalities which adopted the reform promptly. However, most individuals born in 1955 attended the new post-reform schools. Thus, individuals experienced two different school systems as one of them required at least one more schooling year than the other. Despite this, the characteristics of municipalities which adopted reform were representative of Sweden (Meghir and Palme, 2001) such that the reform should not be correlated with unobserved characteristics in health outcomes.

With data from the 1981 and 1991 Swedish Level of Living Survey, she uses an IV technique to estimate formal schooling's causal effect on adult health in Sweden by

instrumenting exposure to the compulsory reform based on an individual's municipality for education. After controlling for cohort and county effects, family background and individual income, she obtains a schooling coefficient of -0.210 on poor health which is significant at least at the ten-percent level in her 2SLS model. Additional schooling significantly reduces the poor health index and considerably improves BMI when educated is treated as endogenous. The IV schooling coefficients are larger than the OLS estimates, similar to Currie and Moretti (2003), Arendt (2005) and Lleras-Muney (2005). Thus, the 2SLS regression with schooling reform status as the instrument shows a consistent causal estimate of education's effect on health.

### **3. Data and statistics**

I use data from the HSE to study the English population. The HSE is an annual survey conducted since 1991 by the joint Health Surveys Unit of the National Centre for Social Research and the Department of Epidemiology and Public Health, Royal Free and University College Medical School, London for the Department of Health. Its aim is to inform policies in health services and raise health awareness publicly through information provision on the nation's health. The yearly sample sizes vary between 12000 and 20000 individuals. The survey consists a questionnaire enquiring a specific topic which changes annually and a nurse visit to the participant's residence for medical tests.

I consolidate statistics on obesity, physical activity and diet in England from a diversity of sources: Active People Survey 2013/14, HSE 2013 and HSE 2014.

Figure 1

Obesity prevalence of adults (16+) in England from 1993 to 2013.



Figure 1 depicts a stark increase in the share who were obese from 1993 to 2013: 13.2 percent to 26 percent for men and 16.4 percent to 23.8 percent for women.

<sup>1</sup> Based on author's calculations.

Figure 2

Adult participation in sport in England, 2013/14.

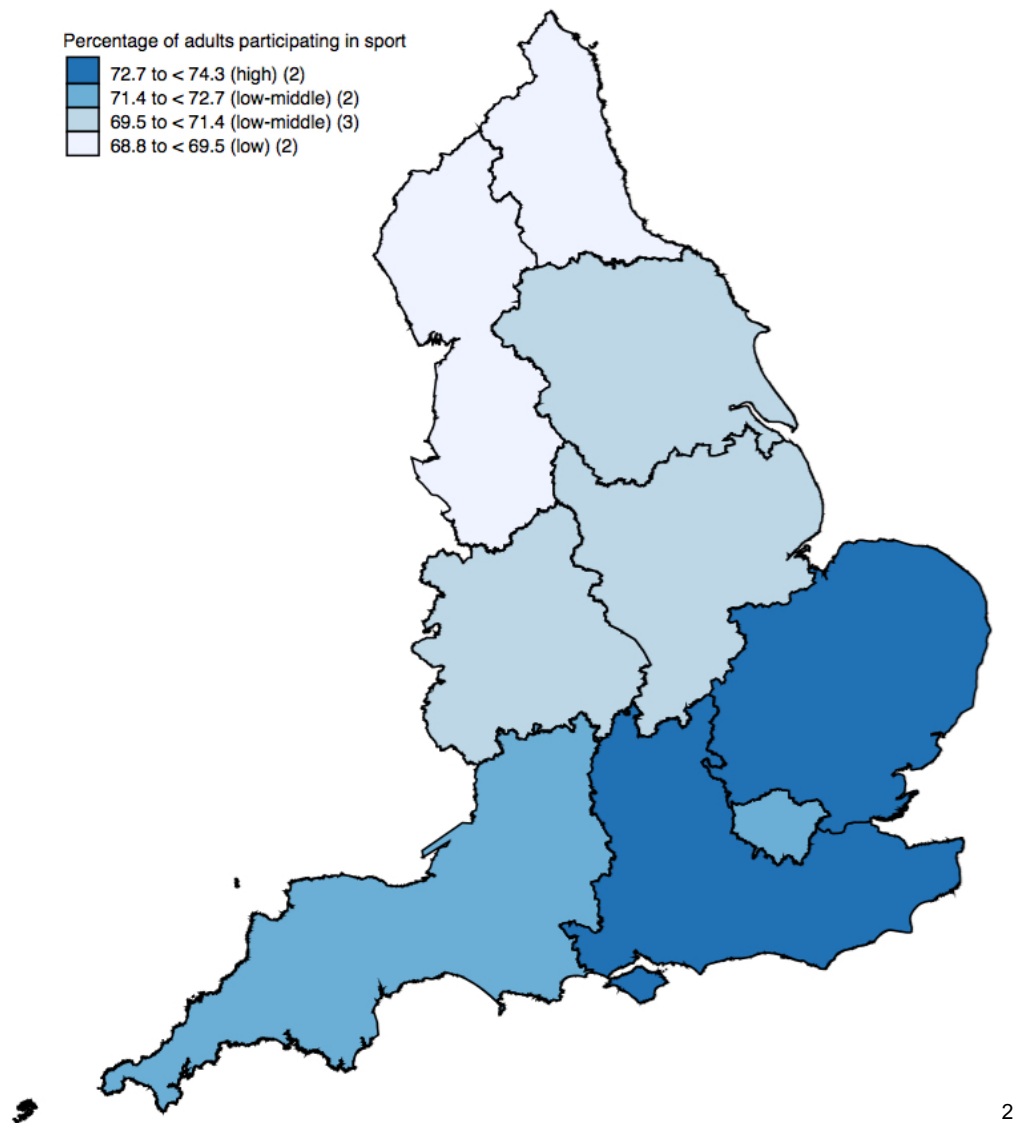


Figure 2 shows the share of adults in each region who participated in moderate intensity sport for thirty minutes at least once a week, the minimum requirement to be considered physically active in the HSE data according to physical activity guidelines, from October 2013 to October 2014.

<sup>2</sup> Based on author's calculations.

Figure 3

Adult daily fruit consumption in England, 2013/14.

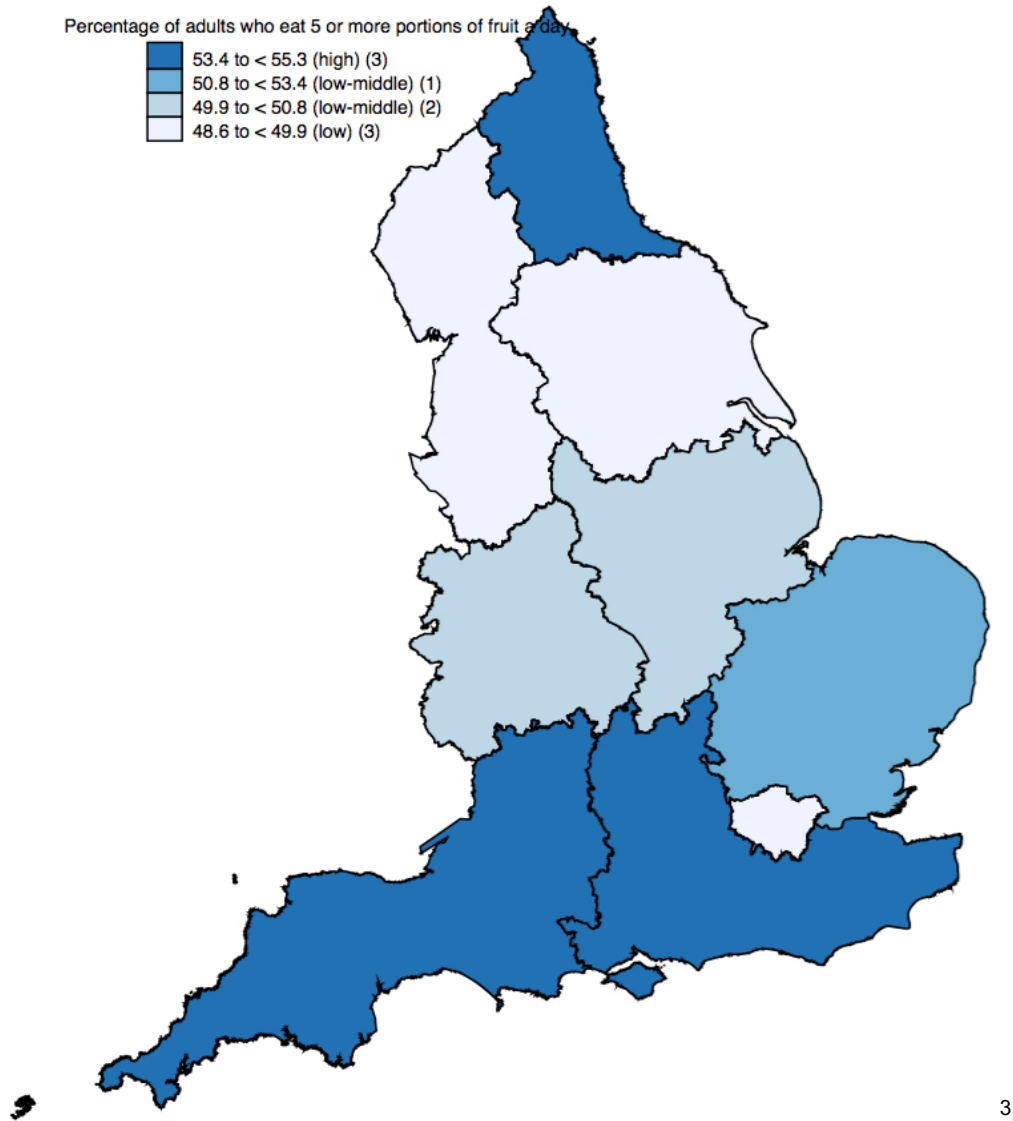


Figure 3 illustrates the share of adults in each region who ate at least 5 portions of fruit daily which exceeds the NHS' recommended daily fibre intake, assuming they would also eat at least a daily portion of vegetables, from October 2013 to October 2014.

<sup>3</sup> Based on author's calculations.

Among gender differences, fewer men than women consumed the recommended five or more portions of fruit and vegetables on the previous day: 25 percent and 28 percent respectively. Higher consumption was also associated with higher income: 30 percent of men and 35 percent of women in the highest income quintile had consumed the recommended portions on the previous day compared with 19 percent of men and 23 percent of women in the lowest quintile.

The HSE datasets I consider for my econometric analysis are from 1991 to 1993 and from the first quarter of 1998 to the last quarter of 2002. Month of birth, which is essential to construct the instrument in my analysis, is available only during these two periods.

Tables 1 and 2 detail the descriptive statistics on all HSE variables which I use. Although the average age of the samples is relatively young, there are individuals who already suffer from long-term health issues.



Table 1

Descriptive statistics, estimation sample of all individuals.

Variable	Observations	Mean	Standard deviation
Health Survey for England			
Has at least an O-level qualification (1=yes)	15921	0.7505	0.4328
Born February-August (1=yes, vs. September-January)	15921	0.5919	0.4915
Born in February (1=yes, vs. born in January)	2626	0.0755	0.2642
Age (years)	15921	40.68	7.16
BMI	15921	26.48	4.81
Smoker (1=yes)	15921	0.3030	0.4596
Drinks over weekly limit	15921	0.3204	0.4666
Eats sweets, biscuits, chocolates or crisps more than 2 times a week	15921	0.2700	0.4440
Eats fruits and vegetables more than or equal to 5 times a week	15921	0.3331	0.4713
Male (1=yes)	15921	1.5318	0.4990

Table 2

Descriptive statistics, estimation sample of individuals born in January and February.

Variable	Observations	Mean	Standard deviation
Health Survey for England			
Has at least an O-level qualification (1=yes)	2626	0.7464	0.4352
Born February-August (1=yes, vs. September-January)	2626	0.4577	0.4983
Born in February (1=yes, vs. born in January)	2626	0.4577	0.4983
Age (years)	2626	40.36	7.17
BMI	2626	26.35	4.79
Smoker (1=yes)	2626	0.3180	0.4658
Drinks over weekly limit	2626	0.3191	0.4662
Eats sweets, biscuits, chocolates or crisps more than 2 times a week	2626	0.2784	0.4482
Eats fruits and vegetables more than or equal to 5 times a week	2626	0.3332	0.4714

#### 4. General approach

Consider the basic relationship between health and education by OLS:

$$BMI_{it} = c_0 + c_1 gcsequali_{it} + u_{it}$$

where  $BMI_{it}$  is an individual's BMI,  $gcsequali_{it}$  indicates whether the individual has at least an O-level qualification and  $u_{it}$  is the error term.  $E(u_{it}|gcsequali_{it})$  is required to be zero for valid inferences. However, it does not hold when there are errors-in-variables bias, simultaneity causality bias and omitted variable bias.

Errors-in-variables bias occurs when  $gcsequali_{it}$  is measured wrongly:

$$gcsequal_{it} = gcsequali_{it} + \varepsilon_{it}$$

where  $\varepsilon_{it}$  such as when the survey participant reports his highest educational qualification incorrectly. Thus, the estimated relationship becomes:

$$BMI_{it} = c_0 + c_1 gcsequal_{it} + [u_{it} - c_1 \varepsilon_{it}]$$

which biases  $\hat{C}_1$  because  $E(u_{it} - c_1 \varepsilon_{it}|gcsequali_{it} + \varepsilon_{it})$  will not be zero.

Simultaneity causality bias occurs when a dependent variable,  $X_{it}$ , like  $BMI_{it}$  affects  $gcsequali_{it}$  such that:

$$gcsequali_{it} = \gamma_0 + \gamma_1 BMI_{it} + \varepsilon_{it}$$

so increases in  $u_{it}$  lead to increases in  $BMI_{it}$  which lead to increases in  $gcsequali_{it}$ . For example, having at least an O-level qualification leads to a lower individual BMI as he is expected to have the knowledge and means to live healthily but a lower individual BMI indicating a healthier person will also provide him a greater quantity of healthy days to obtain an educational qualification (Grossman, 1972), creating a feedback effect. Therefore,  $u_{it}$  and  $BMI_{it}$  will not be independent, biasing  $\hat{C}_1$ .

Omitted variable bias occurs when there is an important determinant of  $BMI_{it}$  which is excluded from my regression and is correlated with  $gcsequali_{it}$  such as the parents' income of the individual. The effects of parental background on their children's health have been shown to be long-lasting (Case et al., 2002, Currie, 2004; Currie and Hyson,

1999): poor health in childhood is associated with lower educational attainment, inferior labour market outcomes and poor health in adulthood. Thus, I expect a positive correlation between parental income and an individual's education level because parents with more resources are more likely to invest in their children's education and household's diet which should lead to lower and healthier individual BMIs. Case et al. (2002) analyse the relationship between family income and child health using the US National Health Interview Survey (NHIS), a cross-section dataset like the HSE. They find the existence of a significant and positive effect of income: children in poorer families have significantly worse health than children from wealthier families. The income gradient in child health increases with child age in the US as the protective effect of income accumulates over childhood. However, unlike the US where there has been only private health insurance available until the Affordable Care Act enacted in 2010, the UK has had the NHS since 1948 which offers free healthcare at the point of delivery (Culyer and Wagstaff, 1993). Thus, the lack of evidence for parental income effect on children's health increasing with children's age (Currie et al., 2004) may be because the NHS has been successful in insuring children's health of low income households. Nonetheless, parental income will be part of my regression error if it is correlated with  $gcsequali_{it}$  and a determinant of  $BMI_{it}$  in my dataset, resulting in a downward bias of  $\hat{C}_1$ .

Therefore, I use an IV approach in 2SLS regressions to overcome the problems highlighted above and obtain consistent estimates in my natural experiment. The instrument I use is  $febborn_{it}$ , February-born individuals, As February-born individuals has a two to three percent higher probability of obtaining a qualification (Braakmann, 2011), my instrument is relevant,  $cov(febborn_i, X_i) \neq 0$ . While families can plan for the

season when they desire to give birth to some extent which may lead to parental characteristics differences for individuals born at different times of the year (Buckles and Hungerman, 2008), this is far less possible for the exact birth month. Thus, my instrument is also exogenous,  $cov(febs_{it}, u_{it}) = 0$ . I control for age, sex and the exogenous increase in the dependent variable not explained by other variables with a time trend. Thus, the regressions in my main analysis follows this form:

$$1^{\text{st}} \text{ stage: } gcsequal_{it} = a_0 + a_1 febs_{it} + a_2 t + sex_{it} + age_{it} + v_{it}$$

$$2^{\text{nd}} \text{ stage: } BMI_{it} = b_0 + b_1 \widehat{gcsequal}_{it} + b_2 t + sex_{it} + age_{it} + e_{it}$$

Since  $\widehat{gcsequal}_{it}$  is estimated from the exogenous variables, it is not correlated with  $u_{it}$  unlike  $e_{it}$ , so  $\beta_1$  is consistent. For categorical variables such as whether the individual is a smoker, I use probit and ivprobit regressions which works like OLS and 2SLS respectively in obtaining consistent estimates. I analyse additional years of data from 1991 to 1993 and measure various health-related measures and behaviours, in particular, BMI, which was not examined by Braakmann (2011). Amongst the various outcomes examined, I lower the threshold of the frequency of unhealthy food consumption from Braakmann's six times a week to twice a week in an attempt to capture a significant effect. I run the regressions for two samples: one containing all individuals and another containing only January- and February-born individuals. I report my estimates from the OLS, 2SLS, probit and ivprobit regressions in the following section.

## 5. Results

Table 3 outlines the first-stage regression results for the relationship between month of birth and education in the HSE for the whole sample.

Table 3

First-stage regression results, Health Survey for England, dependent variable: has at least an O-level qualification (1=yes).

	Men and women	
	All individuals	January- and February-born individuals
Individuals born from February to August (1=yes)	0.0153* (0.0070)	
Individuals born in February (1=yes)		0.0427** (0.0169)
Observations	15921	2626
R <sup>2</sup>	0.0003	0.0024
F-statistic	149.30	40.15
Cragg-Donald Wald F-statistic	1.687	5.159
Kleibergen-Paap Wald rk F-statistic	1.782	5.191

Coefficients, robust standard errors in parentheses, \*/\*\*/\*\* Statistical significance on the 10%, 5% and 1% level. All estimations control for age, sex and a time trend.

The first-stage F-statistic value is smaller and slightly problematic in the discontinuity sample because of the smaller sample size. The results suggest my instrument is not weak according to Staiger and Stock's (1997) rule of thumb which rejects the null hypothesis of weak instruments if the F-statistic is more than or equal to 10. However, Stock and Yogo (2005) disagree because the first-stage F-statistics assumes conditional homoskedasticity. Thus, I also use the Kleibergen-Paap rk Wald-statistic to test for weak instruments when exercising robust options and the Cragg-Donald test to account for independently and identically distributed errors. They reveal a potential weak instrument problem which I consider when interpreting the results from the main analysis.

Table 4

The impact of education on BMI, Health Survey for England, OLS and IV results.

	BMI
<hr/>	
OLS estimates: all individuals	
At least an O-level qualification (1=yes)	-0.8647*** (0.0939)
Observations	15921
IV results: all individuals	
At least an O-level qualification (1=yes)	-8.2408 (10.1466)
Observations	15921
<hr/>	
OLS estimates: January- and February-born individuals	
At least an O-level qualification (1=yes)	-0.9500*** (0.2271)
Observations	2626
IV results: January- and February-born individuals	
At least an O-level qualification (1=yes)	-0.3091 (4.7552)
Observations	2626

Each cell is from a different regression. Coefficients, robust standard errors in parentheses, \*/\*\*/\*\* Statistical significance on the 10%, 5% and 1% level. All estimations control for age, sex and a time trend.

Table 5

The impact of education on lifestyle habits, Health Survey for England, Probit and IVProbit results.

	Smoker	Drinks over weekly limit
Probit estimates: all individuals		
At least an O-level qualification (1=yes)	-0.1918*** (0.0089)	0.0460*** (0.0086)
Observations	15921	15921
IVProbit results: all individuals		
At least an O-level qualification (1=yes)	-0.1783 (0.4213)	0.0439 (0.4557)
Observations	15921	15921
Probit estimates: January- and February-born individuals		
At least an O-level qualification (1=yes)	-0.1644*** (0.0219)	0.0401** (0.0210)
Observations	2626	2626
IVProbit results: January- and February-born individuals		
At least an O-level qualification (1=yes)	-0.1559 (0.4410)	0.0369 (0.2734)
Observations	2626	2626

Each cell is from a different regression. Coefficients, robust standard errors in parentheses, \*/\*\*/\*\* Statistical significance on the 10%, 5% and 1% level. All estimations control for age, sex and a time trend.

Table 6

The impact of education on food consumption habits, Health Survey for England, Probit and IVProbit results.

	Eats sweets, biscuits, chocolates or crisps more than 2 times a week	Eats fruits and vegetables more than or equal to 5 times a week
<hr/>		
Probit estimates: all individuals		
At least an O-level qualification (1=yes)	0.0068 (0.0080)	0.0611*** (0.0085)
Observations	15921	15921
IVProbit results: all individuals		
At least an O-level qualification (1=yes)	0.0060 (0.2731)	0.0538 (0.7143)
Observations	15921	15921
<hr/>		
Probit estimates: January- and February-born individuals		
At least an O-level qualification (1=yes)	0.0117 (0.0198)	0.0598*** (0.0207)
Observations	2626	2626
IVProbit results: January- and February-born individuals		
At least an O-level qualification (1=yes)	0.0089 (0.2326)	0.0520 (0.3422)
Observations	2626	2626

Each cell is from a different regression. Coefficients, robust standard errors in parentheses, \*/\*\*/\*\* Statistical significance on the 10%, 5% and 1% level. All estimations control for age, sex and a time trend.

Tables 4, 5 and 6 display the expected positive correlation between education and health-related behaviour in the OLS estimates. From Table 4, individuals with at least an O-level qualification have a lower BMI by 8.2 on average. For lifestyle habits, the marginal effects in table 5 imply individuals with at least an O-level qualification are 19 percent less likely to be a smoker but 4.6 percent more likely to drink over the weekly limit than the rest. For food consumption habits, the marginal effects in table 6 imply individuals with at least a O-level qualification are 6.1 percent more likely to eat fruits and vegetables more than or



equal to five times a week than the rest. However, the IV estimates display a mix of positive and negative results and are always insignificant which do not support a causal relationship between education and health.

The OLS and IV results are different because these two techniques estimate different effects. In particular, the IV estimates are LATE estimates for those individuals whose educational status are affected by the instrument which meant any changes in health resulted from being considered as completing a first qualification because they were born in February. The IV estimates are smaller than the OLS estimates because the 2SLS regressions removed the feedback effect caused by simultaneity causality bias present in the simplified OLS regressions. However, the IV results do not support a causal relationship between education and health.

In summary, while there are large discontinuities in education between January- and February-born individuals highlighted in the literature, such discontinuities in various health measures are absent. Thus, there is no causal relationship between education and health. All observed health differences between individuals with different education levels are because of “third variables” like natural health endowments or family background.

Additionally, the causal relationship between education and health is not based solely on the no or low qualification margin as the relationship between education and health becomes stronger towards the higher end of the educational distribution (Cutler and Lleras-Muney, 2010). This explanation is plausible given the instrument I use informs changes only at the lower end of the educational distribution. Braakmann (2011) tests this with a restricted sample containing only individuals with at most an O-level qualification and finds the OLS results generally indicate considerable health advantages for

individuals with O-levels relative to those without any qualification. However, the IV estimates indicate no significant relationship between having an O-level qualification and health outcomes.

## **6. Discussion**

Grossman's health model hypothesises a positive relationship between education and health: higher educated individuals demand a greater quantity of healthy days to enjoy consumption and so invest more in maintaining good health. In the following, I will offer some insight to explain this lack of effect on health my particular quasi-experiment reveals. Although the natural experiment considerably changed the probability of attaining a qualification, February-born individuals gained only one additional term in education, a relatively little amount of time. While research in the literature has examined increases in compulsory schooling which changed the duration of education, this experiment allows February-born individuals to acquire a signal which may be valued in the labour market. Hence, the main causal channel for a potential effect on health could have been through income differences rather than through productivity-based explanations.

The significant OLS estimates show substantial returns to having an O-level qualification relative to having none but the insignificant IV estimates suggest the individuals I consider who sit and pass their O-level examinations are very likely marginal passes. Regression estimates using employment and two measures of wages as outcomes from LFS are qualitatively identical when limiting the sample to individuals with at most an O-level qualification (Braakmann, 2011). Thus, employers may view them as more like individuals without a qualification than individuals who passed with higher marks. This implies marks are likely to be very observable by employers as the OLS estimates do not

distinguish between these two groups unlike the IV estimates which reveal the effects for marginal cases. As the compliers from the IV estimates represent the individuals who just managed to attain O-level qualifications do not gain a labour market advantage over individuals without such qualifications, it seems plausible education has no effect on health.

Furthermore, time inconsistent preferences instead of education may explain lifestyle habits. Quasi-hyperbolic discounting (Phelps and Pollak, 1968; Laibson, 1997) such as this form:

$$\mu_0 + \frac{\alpha}{1+1}\mu_1 + \frac{\alpha}{1+2}\mu_2 + \dots + \frac{\alpha}{1+n}\mu_n$$

induces dynamically inconsistent preferences contrary to geometric discounting in this form:

$$\mu_0 + \beta\mu_1 + \beta^2\mu_2 + \beta^3\mu_3 + \dots + \beta^n\mu_n$$

The following payoff matrices model a hypothetical situation where an individual fails to quit smoking due to quasi-hyperbolic discounting:

	$t_n$	$t_{n+1}$
Quit	4	10
Do not quit	7	6

Under geometric discounting where  $\alpha \approx 1$  and  $\beta \approx 0.8$ ,

	Today	A week later
Quit	12	2.52
Do not quit	11.8	2.47

he makes time consistent choices regardless of when benefits to those choices are delayed. Since he gets more utility from quitting in both periods, he quits immediately.

However, under Quasi-hyperbolic discounting where  $\alpha \approx 1$  and  $\beta \approx 0.8$ ,

	Today	A week later
Quit	9	1.75
Do not quit	10	1.54

he changes his choices based on his distance in the future. Unlike geometric discounting, he gets more utility from quitting only in future and not at present and hence do not quit. The empirical evidence from Gruber and Köszegi's (2001) addictive behaviour model which incorporates time-inconsistent preferences to the standard "rational addiction" model (Becker et al., 1994) suggests smokers exhibit forward-looking behaviour with time inconsistent preferences concerning smoking. Thus, individuals start smoking often as adolescents when they are most present biased (Hammond, 2005) and do not anticipate the difficulty of quitting. Conversely, individuals who quit smoking successfully may have used commitment devices (Ashraf et al., 2006; Kaur et al., 2010; Beshears et al., 2011) like quitting with friends to constrain their own future choices by deciding ahead of time to make future deviations costly. Thus, lifestyle habits may not be correlated with education.

Moreover, BMI can be explained by a whole host of variables of health such as physical activity which may have changed the results concerning the relationship between education and health in the OLS and IV regressions. The absence of exercise data from the HSE collected between 1991 and 1993 makes this analysis impossible. While BMI has been widely linked with health-related life quality (Yan et al., 2004; Patterson et al., 2004), other research has cast doubts on the suitability of using BMI to measure an individual's health (Ashwell et. al, 2012; Janssen et al., 2004). Although the OLS point estimates suggest the health benefits of increased education, the imprecision of the IV

estimates makes it impossible to reject the null hypothesis there is no association between education and health.

## **7. Conclusion**

In my dissertation, I use a natural experiment in England to render an exogenous variation in the probability of attaining any qualification between January- and February-born individuals. Compulsory schooling laws interacted with the timing of the O-level examinations to alter the probability of obtaining a qualification by about 3 percentage points and within-cohort differences in education did not lead to differences in health measures and health-related behaviour (Braakmann, 2011). While OLS estimates show the expected correlation between holding a qualification and health outcomes in samples containing all individuals and in a discontinuity sample consisting only January- and February-born individuals, this relationship does not appear in both samples with IV estimates. Hence, the results do not support a causal link between education and health for the individuals affected by this particular treatment I considered. However, the results do not eliminate a causal link between higher forms of education and health because the treatment had affected individuals only at the margin of achieving a first qualification.

A possible extension for further research is to consider individuals with higher educational qualification since there is a stronger relationship between educational attainment and health at the right tail of the distribution (Cutler and Lleras-Muney, 2010). As different parts of the outcome distributions may have been affected differently, quantile regression may be used to reveal how far right of the distribution education has had its most significant effect on health.

Nevertheless, my results echo some of the previous evidence using changes in compulsory schooling laws (Arendt, 2005; Albouy and Lequein, 2009; Clark and Royer, 2010; Braakmann, 2011) but contradict others based on the same technique (Glied and Lleras-Muney, 2003; Lleras-Muney, 2005; Oreopoulos, 2006; Spasojevic, 2010). In retrospect, the causal link between education and health remains inconclusive.

## 8. References

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