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An empirical investigation of the health and health inequality impacts of education and drinking age laws

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Health, inequality and the impact of public policy

An empirical investigation of the health and health inequality impacts of education and drinking age laws

Gawain Heckley

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Abstract

This thesis consists of an introductory chapter and four independent research papers. The introductory chapter introduces the relevant background to all four papers, the methods and results and then discusses what the results tell us about the impact of education and drinking laws on health and its inequality together as a whole.

Paper I, A general method for decomposing the causes of socioeconomic inequality in health, introduces a new method for determining the causes of socioeconomic related inequality in health that requires few identifying assumptions to yield valid estimates. Using the Swedish Twin Registry and a within twin pair fixed effects identification strategy, no evidence of a causal effect of education on income-related health inequality is found.

Paper II, The long-term impact of education on mortality and health: Evidence from Sweden, estimates the impact of education on health using two school reforms in Sweden. Both Regression Discontinuity and Difference in Differences are applied to two Swedish school reforms that are different in design but were implemented across overlapping cohorts born between 1938 and 1954. The observation period is up until 2013 (aged up to 75). The results find no support for a positive causal effect of additional years of education on health.

Paper III, Could easier access to university improve health and reduce health inequalities?, estimates the impact of university education on medical care use and its income related inequality. Exploiting an arbitrary university eligibility rule in Sweden combined with Regression Discontinuity design a clear jump in university attendance is observed due to university eligibility. This jump coincides with an increase in women's contraceptive use without increasing its socioeconomic related inequality. At the same time, the results highlight that universities may need to take greater care of the mental health of their least able students.

Paper IV, Too young to die: Regression Discontinuity of a two-part minimum legal drinking age policy and the causal effect of alcohol on health, examines the impact of Sweden's unique two-part Minimum Legal Drinking Age (MLDA) policy on alcohol consumption and health using a Regression Discontinuity design. In Sweden, on-licence purchasing of alcohol is legalised at 18 and off-licence purchasing is legalised later at 20 years of age. A jump in alcohol consumption is observed at age 18 but no discernible increases in mortality at age 18 or 20 are found. Hospital visits due to external causes do see an increase at both 18 and 20 years. Compared to previous findings for single MLDAs the alcohol consumption impacts found are smaller and the health impacts less severe.

Overall, no evidence has been found that increasing levels of education leads to improvements in health or changes in income related health inequality. Public health policies aimed specifically at health behaviours are potentially likely to be more effective. An example of this is the combination of Sweden's two-part MLDA policy, restrictive access to alcohol through the state run alcohol monopoly off-licence and stringent limits on alcohol levels in the blood for driving which altogether have eliminated the negative health consequences of increased consumption of alcohol among young adults observed elsewhere.

Key words: Inequality measurement, Concentration index, Decomposition methods, Recentered influence function. Socioeconomic Related Health Inequality, Regression Based Decomposition, Regression Discontinuity, Difference in Differences, Twins Study,

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MADE IN SWEDEN

To my family.

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Acknowledgements

The idea of chance and probability theory underpins a lot of thinking in economics. Illustrating this nicely, a few chance encounters have influenced the creation of this thesis. The first such encounter was meeting Bob Smith, my A-level economics teacher. His love for economics was infectious and he inspired me to study economics at university. The idea was to get rich in the finance sector in London then move out to the Home Counties and teach economics to a new generation of students in great luxury, just like him. That plan didn't work out, but instead I landed a job I really enjoyed in the Civil Service. This led me to study for a Masters in economics at UCL. Chance struck again and it was when working in the Civil Service that I met my future wife, Lotta, also an economist. Lotta is Swedish and we moved to Sweden in 2011 so I could experience the Swedish way of life, immerse myself in the language and it is here that I started my PhD.

My first thanks go to Ulf Gerdtham, my supervisor. Chance brought us together after noticing he was involved in the commission for a socially sustainable Malmö. Ulf offered to take me on as a researcher and then offered me the opportunity to do a PhD. Ulf has been very supportive from the beginning, opening doors, sharing his network of contacts, always being positive and believing that it will all turn out great in the end.

Like any thesis, the contents of this book are fairly specialised and therefore the majority reading this acknowledgement are unlikely to make it beyond the next page. I would therefore like to extend my enormous gratitude to everyone who has or will. This includes Ulf, obviously, and also my co-supervisors Johan Jarl and Gustav Kjellsson. Johan has been very supportive from the beginning and an expert guide on all things alcohol. Gustav pushed me to raise my algebraic game, introduced me to Lisa and Karin whilst we were on parental leave with our two sons and also introduced me to a number of other co-authors: Martin Fischer, Martin Karlsson and Therese Nilsson. Martin Fischer has been great fun to work with and is a huge fountain of statistical knowledge. He is also, oddly for a German, possibly the leading expert on education reforms in Sweden and Swedish administrative data. Therese has been very inclusive and a great support and her motivation for all things academic is very contagious. Martin Karlsson has also been very inclusive and is full of innovative ideas and enthusiasm. I am also indebted to Martin Nordin, for co-authoring one of the papers. Heroically, both Lotta and my mum have also read the thesis and helped improve the drafting.

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not be blamed for any remaining mistakes found in the thesis, however, as that responsibility is all mine.

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My final thanks go to my family. Thank you for all your support, your love and your understanding during what has been both a challenging and rewarding time.

Lund, May 2018.

Gawain Heckley

List of papers

This thesis is formed of four original papers referred to by their Roman numerals:

I. Heckley, G., Gerdtham, Ulf-G., & Kjellsson, G. "A general method for decomposing the causes of socioeconomic inequality in health." Journal of health economics 48 (2016): 89-106.

II. Heckley, G., Fischer, M., Gerdtham, Ulf-G., Karlsson, M., Kjellsson, G., & Therese Nilsson. "The long-term impact of education on mortality and health: Evidence from Sweden." Manuscript

III. Heckley, G., Gerdtham, Ulf-G., & Nordin., M. "Could easier access to university improve health and reduce health inequalities?" Manuscript

IV. Heckley, G., Gerdtham, Ulf-G., & Jarl, J. "Too young to die: regression discontinuity of a two-part minimum legal drinking age policy and the causal impact of alcohol on health." Manuscript

 Paper I is accompanied by an erratum correcting some typos.

1. Introduction

This thesis combines three large areas of economic research: the measurement of socioeconomic related health inequality; the decomposition of socioeconomic related inequality into its explanatory factors; and the treatment effects and policy evaluation literature. The thesis is comprised of four papers that stand as individual contributions but together they have a common theme: they assess the impact of public policy (education and drinking age laws) on health and health inequality.

Background

Health inequality

On the 4th of August 1997 Jeanne Calment died aged 122 years. According to the Guinness Book of World Records she is the oldest verified person to have lived. Not everyone lives to 122, nor do people expect to either. Indeed, average life expectancy is a lot less than 122 years. Life expectancy for someone born in Sweden today is 82 years (Statistics Sweden, 2015). There is a lot of variation in length of life and this is because, amongst other things, we are not born genetically equal. In health we appear to accept that fortune has an inevitable role to play. The health inequality literature has therefore taken the view that it is not differences in health themselves that are of interest. Instead it is differences in health that also mirror differences observed elsewhere, especially differences in socioeconomic status (Wagstaff et al., 1991). Socioeconomic status is a descriptive term for an individual's position in society often thought of as some combination of income, education and occupation but not necessarily limited to these criteria.

Another reason that socioeconomic related health is of interest stems from the fact that many countries have introduced public health care provision providing free access to healthcare at the point of need. The expectation has been that access to medical care, a key determining factor of health differences by socioeconomic status, was then in principle equalised for all members of society. These countries

should therefore observe much smaller differences in health that are related to socioeconomic status.

Despite the existence of comprehensive public health systems across countries it is now well documented that socioeconomic inequalities in health exist for many different measures of health including mortality, various morbidities and even health related behaviours (Deaton, 2003, 2013; Mackenbach et al., 2008, 2015). This observation has led to the formation of a number of health inequality commissions, including the World Health Organisation commission on the social determinants of health (Marmot et al., 2007) in Britain (Marmot et al., 2010), Malmö, Sweden and also Europe (Marmot et al., 2012).

Decomposition methods

The natural question that follows from the discovery of extensive socioeconomic inequalities in health is: what could explain this? To this end decomposition methods have been developed for a range of inequality measures including measures of socioeconomic related health inequality (Wagstaff et al., 2003).

Decomposition methods in general, including the method of Wagstaff et al. (2003), do not seek to recover the economic mechanisms underpinning a measure of inequality. Instead they have the aim of highlighting which potential explanatory factors are quantitatively important. For example, if we have found a strong relationship between health and socioeconomic status, decomposition methods can indicate if differences in education are an important explanation behind this. Indeed, results using the method of Wagstaff et al. (2003) tell us, for a fixed level of inequalities (the mean of health and the socioeconomic ranking of individuals is fixed), which factors potentially account for a large fraction of the observed socioeconomic inequality in health. However, the method of Wagstaff et al. (2003), like all decomposition methods, leaves the question of how education impacts the health income relationship unanswered.

The approach of Wagstaff et al. (2003) requires a number of assumptions to hold and these are particularly restrictive if we want to move away from descriptive analysis towards the more interesting question of cause and effect. The first assumption is a common and stringent assumption made by all regression based decomposition methods and it is the assumption of no general equilibrium effects. Let us consider the impact of education on health as an example of what this means in practice. Any estimates of the impact of education on health will be based on a partial equilibrium analysis. That is the estimates are valid for changes to individual characteristics as long as not too many individuals are affected so that wider changes in the economy start to ensue. As an example, let us raise the level of education so that everyone has a university degree. This will of course

impact the outcomes of those who would have had less than a university education otherwise. The partial equilibrium analysis may be valid if only a small number of people did not have a university education before the change. However, if there were a lot of individuals without a university education it is likely that the labour earnings returns will change due to this sudden large increase in supply of young adults with a degree. There could also be peer composition effects from more mixed classes at university which impact health related behaviours. Partial equilibrium based analysis assumes that these effects, even for large changes, are zero. The approach of Wagstaff et al. (2003) has the explicit aim of explaining inequality as a sum of its factor components. That is, how much inequality is due to each explanatory factor. Because it is based on partial equilibrium analysis, any causal interpretation has to be made on the assumption that there are no general equilibrium effects.

A second assumption made by the decomposition method of Wagstaff et al. (2003) is that health is a function linear in variables, not just parameters and we know that for many health variables this is a stringent assumption (Van Doorslaer et al., 2004a,b; Van Ourti et al., 2009; Van de Poel et al., 2009). This assumption is just as restrictive for descriptive decompositions as it is for decompositions aimed at answering questions of cause and effect, given the aim of the approach is to model the entire distribution of health, not just the mean. Third, the decomposition method of Wagstaff et al. (2003) holds the mean of health and the socioeconomic variable fixed. This means that we are only explaining the health part of the inequality index when most explanatory variables we can think of will also likely impact socioeconomic rank. This is less of an issue for descriptive decompositions but it means the approach is not ideally suited to the consideration of changes because for changes in inequality we are interested in the change in the mean of health and socioeconomic rank.

What should be clear from the above discussion is that the decomposition of socioeconomic related health inequality is not a solved problem. Indeed there is a need for a less parametric approach to the decomposition of socioeconomic related health inequality that allows us to quantitatively assess the importance of potentially important public policy levers, such as education, in determining the level of inequality. Indeed, the strict assumptions imposed by the decomposition method of Wagstaff et al. (2003) are potentially why very little research has looked at identifying the causal impact of important public health policy levers on measures of socioeconomic related inequality.

This thesis addresses this issue directly by developing a new decomposition method for socioeconomic related health inequality that makes much weaker parametric assumptions than those of existing methods.

Education as a public health policy lever

Study after study has documented the strong association between education and health in all its forms. Indeed some have gone as far as to suggest that in order to address the socioeconomic gradient in health we need to address the social determinants of health (Marmot et al., 2010). Education policy could potentially play an important part, but only if education *causes* differences in health. However, it is not clear that the well documented association between education and health is a causal relationship.

There are a number of theories that suggest that it could be a causal relationship. One of the most influential theories in health economics regarding the formation of health is the one by Grossman (1972) that states amongst other things that health is an increasing function of education. In this model individuals are assumed to produce their own health, using their own time and goods as inputs. Education is predicted to improve the efficiency of this production, reducing the time and resources needed to produce health and thereby raising the optimal level of health of the individual. In epidemiology, a number of theories suggest that the distribution of power, money and resources are driving inequalities in health (Marmot et al., 2010). These in turn could all be influenced by differences in education. Cutler and Lleras-Muney (2008) also review a number of additional theories that suggest a causal pathway between education and health.

The education gradient in health may, however, just reflect a missing third hard to observe variable that predicts both education and health. Both time preferences (and therefore willingness to invest in both education and health) (Fuchs, 1980) and innate ability (Bijwaard, 2015) have been suggested as potential candidates. These are hard to observe yet may explain why individuals who have higher levels of education also have better health. For instance those with high ability are more likely to find it easier to obtain higher levels of schooling *and* find it easier to maintain their health. Or, those who prefer now very much compared to the future (they have a high discount rate of the future) may also be less willing to spend time investing in their education *and* also spend time investing in their health if the pay-offs for these investments accrue a long time into the future. That education is associated with health may therefore reflect that we do not observe innate ability or time preferences. The association between education and health may also be due to reverse causality where current health is just a reflection of initial health and it is initial health endowments that determine educational achievement.

What the above discussion highlights is that whilst there is reason to believe education improves health, it is not clear that it in fact does. That study after study shows a strong association between education and health does not prove that differences in education *cause* differences in health. What is needed is a

convincing "instrument". This is a variable that induces variation in the explanatory variable (education) but has no direct effect on the outcome variable (health). A randomised experiment would do this, but at great cost. Instead quasi-experimental techniques are often used. A review by Grossman (2015) of the recent quasi-experimental research of the impact of education on health found that results pointed either to an increasing or a zero effect. It was hard to draw any conclusions as a result of this.

This thesis looks to help improve our understanding of the role of education in determining health and income related health inequality. A variety of quasi-experimental techniques are used to identify the impact of education on health. This analysis is then extended to assess the impact of education on income related health inequality using the new decomposition method developed as part of this thesis.

Minimum legal drinking ages as a public health policy lever

Education is not the only public policy tool available to policy makers that can potentially be used to improve health and reduce health inequalities. Rules, legislation and health information campaigns are all widely used in this regard, and one of the most researched pieces of legislation regarding alcohol consumption in the United States is the Minimum Legal Drinking Age (MLDA).

Episodic heavy drinking is very common amongst young adults aged between 16 and 30 in both the US (Carpenter & Dobkin, 2011) and in Sweden (Ramsted, M., et al. 2010). Over 40 per cent of young adults in Sweden reported drinking four or more cans of strong beer/bottle of wine or more or equivalent in one sitting in the previous month (Ramsted, M., et al. 2010). Heavy drinking can impair judgement, co-ordination, reaction time and vision. Unsurprisingly then, accident related deaths (motor vehicle related, homicides, suicides, alcohol related, narcotics related and other external causes) are the most common causes of death for this age group. The same causes also form a substantial proportion of hospital admissions. Can we design an MLDA in such a way as to minimise alcohol related health costs?

There is an active debate in the United States (Carpenter & Dobkin, 2011) and in Australia (Toumbourou et al., 2014; Lindo & Siminski, 2014) about what the optimal age the country's MLDA should be set at. This debate, however, ignores the possibility that an MLDA can be designed in more than one way. In fact, Sweden's MLDA is quite different to MLDAs imposed elsewhere in that it has two parts: one at 18 years of age for on-licence consumption and one at 20 years of age for off-licence purchasing. Perhaps Sweden's MLDA is better designed than MLDAs offered elsewhere?

This thesis assesses the impact of Sweden's MLDA on alcohol consumption and on health and also obtains an estimate of the causal impact of alcohol on health.

Aims

The overall aim of this thesis is to robustly assess the health and health inequality impacts of two important branches of public health policy; education and minimum legal drinking age laws. The thesis has the following specific aims:

First, provide a method of socioeconomic related health inequality decomposition that can be easily applied in combination with the tools from the treatment effects literature (Paper I).

Second, assess the impact of education on both the level of health and its socioeconomic related inequality (Papers 1, II and III).

Third, assess the impact of Sweden's MLDA on alcohol consumption and health and relate these findings to the wider literature on the impacts of MLDAs (Paper IV).

2. Methods

Measuring income related health inequality

The Concentration Index

As noted in the introduction, there is a concern that there are systematic differences in health that are related to socioeconomic status. In health economics, income is often used as a proxy for socioeconomic status because it allows a finer level of ranking compared to say education or social class. I follow this approach in this thesis using either income rank of the individual or the parents, depending on the age of the individual.

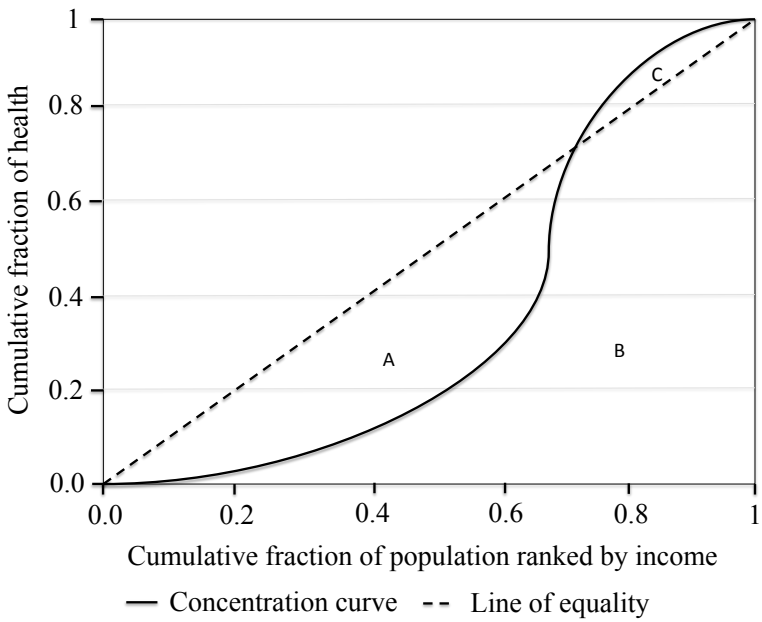

Figure 1. The concentration curve
Figure notes: The concentration curve plots the cumulative fraction of the population ranked by income against the cumulative fraction of health. The Concentration Index is calculated as *CI = 2(a-c)*.

In health economics socioeconomic related health inequality is commonly measured using the Concentration Index (CI). It is a summary index of the relationship between health and socioeconomic status (income) and is derived from the concentration curve (CC). The CC plots the cumulative fraction of the population ranked by socioeconomic status, proxied by income in our case, against the cumulative fraction of health (see Figure 1). The further the CC is away from the 45 degree line (line of equality, see Figure 1) the greater the level of inequality. The Concentration Index captures the degree of inequality by adding up the area between the CC and the line of equality. The area of the box is one. If the CC goes below the line of equality we take the area above the CC and below the line of equality and double it. If the CC goes above the line of equality we take the area below the CC and above the line of equality and double it. If the CC traces the line of equality then the CI is zero – there is no relationship between health and income in this case. The CI is therefore bounded between 1 (if area A in figure 1 was equal to all the area under the line of equality) and -1 (if area C in figure 1 was equal to all the area above the line of equality). A positive value means health is concentrated amongst the rich, and a negative value means health is concentrated amongst the poor.

Choosing an index involves a range of value judgements

The CI is a relative measure of income related health inequality, which means if everyone receives an equal proportional increase in health it does not change. However, there is no consensus as to whether a relative measure is of interest. An equal proportional increase in health would necessarily increase absolute inequality and this may be of concern. It is therefore prudent to consider both relative and absolute inequality (Kjellsson et al., 2015). The absolute version of the CI is given by multiplying the CI by the mean of health.

A further complication with measuring health inequality as an index is that when health is measured by a bounded variable, which many health measures are, such as obesity rates, cancer rates or death rates, the results can change depending on whether we measure health or ill-health. This was first illustrated by Clarke et al. (2003). This is an issue that only affects relative measures of inequality and a suggested solution is to consider both shortfalls and attainments as they represent the potential bounds of different value judgements (Kjellsson and Gerdtham, 2013a,b). In addition adaptations of the CI have been developed that are not affected by the choice of health or ill health of bounded variables and include the Erreygers Index (EI) (Erreygers, 2009) and the Wagstaff Index (WI) (Wagstaff, 2005). This thesis acknowledges these issues when measuring inequality.

A method for determining the causes of income related health inequality

The particular question we want to answer is: how is the health CI or its variants affected by public policy? Paper I of this thesis presents a new statistical method that allows one to answer the above question and requires very few stringent parametric assumptions. It is shown that any bivariate statistic, such as the CI, can be expressed in terms of individual influences on the statistic. The really useful part of this new approach is that it allows all versions of the CI to be calculated as the mean of all the individual influences. In statistics, the mean is well understood. Probability theory, the role of expectations and the Law of Iterated Expectations and, by extension, linear regression techniques are focussed on the mean. That we can express any bivariate statistic as a mean of all the individual influences then opens up all of the tools we have for investigating the mean, allowing us to apply them to the CI.

In standard analysis of the mean and under a linear setting we use Ordinary Least Squares (OLS) with our dependent variable (health) on the left hand side and our explanatory variables on the right hand side. Paper I derives the Recentered Influence Functions (RIF) for the common forms of the CI. Using the formulas presented in paper I yields each individual's (recentered) influence on the CI. In an OLS regression each individual's RIF value replaces health as the dependent variable and using this we can state to what extent education increases or decreases the health CI. This is RIF-I-OLS regression.

Identifying the causal impact of public policy on health and health inequality

This thesis considers the causal impact of two important public policy interventions on health and health inequality. The first is the impact of education; the second is minimum legal drinking age (MLDA) laws. The golden standard for any policy evaluation is a randomised trial. There are clear concerns that both the quantity of education and the MLDA are endogenous. That is, an important part of the simple association of our public health policies on health outcomes can plausibly be explained by hard to observe third factors (potential confounders) or that causality even runs the other way. Randomisation of education and drinking laws respectively would allow us to identify the impacts of these policies but is not feasible for a variety of reasons including and not limited to ethical concerns, costs and time. Instead this thesis relies on what are known as quasi-experimental

techniques; techniques that aim to replicate the conditions of an experiment using observational data.

Twins

In paper I monozygotic twins are used to get nearer to the causal effect of years of education on health and health inequality. Monozygotic twins (commonly known as identical twins) come from the same egg and are born with the same genetic make-up. The concern is that association between education and health is in part due to unobserved factors common within twins such as genetics, innate ability and early life factors. These unobserved factors are biasing the years of education estimates. To deal with this, differences within twin pairs are taken and this way unobserved factors that are common to both twins such as genetics or environmental exposure are differenced out of the equation yielding a less unbiased estimate. That is we used a within twin pair Fixed Effects (FE) strategy. The data used in paper I comes from the Swedish Twin Records and covers twins who took part in a telephone interview including a question on Self-Reported Health, conducted between years 1998 and 2002. Administrative records on education and income are then linked using each individual's unique personal identification number.

Difference-in-Differences

In paper II two compulsory school reforms are used to identify the causal impact of years of education on health. The reforms were rolled out progressively over time across municipalities. This resulted in individuals who were born in the same year but in different municipalities receiving a different amount of compulsory schooling. Similarly, individuals born in different years but in the same municipality could have gone to the same school but received a different number of years of compulsory schooling. This variation over birth cohorts and municipalities allows differences to be taken in years of schooling and health outcomes across municipalities and across birth cohorts. That is, we use the quasi-experimental technique of Difference-in-Differences (DiD). The assumption is that any remaining variation in years of education and health is then due to the reform. The data used in paper II comes from the Swedish Interdisciplinary Panel, a dataset combining various population based administrative records on income, education, mortality, hospital visits and more for all individuals in Sweden between 1930 and 1980 and their parents.

Regression Discontinuity Design

Papers II, III and IV utilise the quasi-experimental technique of Regression Discontinuity (RD) design. RD requires detailed data and a large sample combined with an explanatory variable of which the outcome variable is a smooth function except for a jump caused by some arbitrary rule. RD utilises this arbitrary rule. The assumption is that very close to rule cut-off, an individual below yet very near the cut-off will be very similar in observable and unobservable characteristics to someone just above the cut-off yet the person above the cut-off is subject to the rule change that the person below is not subject to. Assuming individuals either side of the cut-off are indeed similar we can identify the impact of the arbitrary rule on our outcomes of interest.

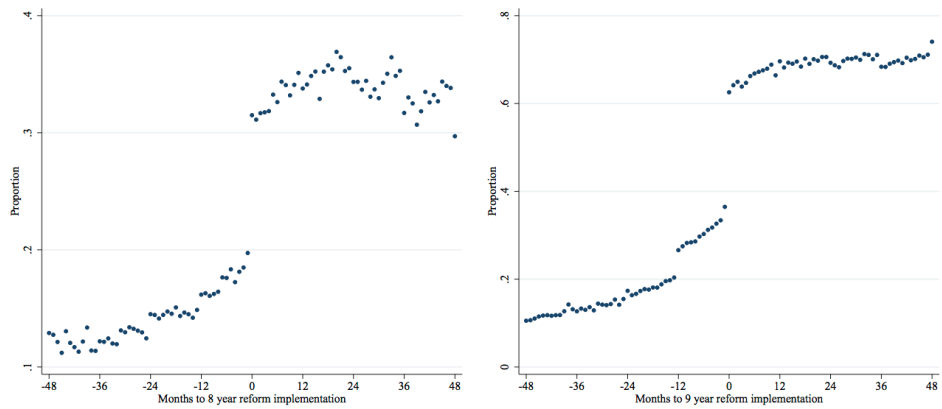

Figure 2. Paper II - Impact of two Swedish school reforms on minimum years of schooling
Figure notes: Scatter plots of the proportion with the new minimum years of schooling by age in months measured as months to reform implementation in their municipality. Left panel is for the 8 year reform, right panel the 9 year reform. Reform implementation is at time zero.

Paper II uses an individual's year and month of birth combined with the school year cut-off of the 1st of January and year of reform implementation to identify the impact of the reforms on years of schooling and later health outcomes. As shown in figure 2, the level of education increases smoothly with year of birth, reflecting the trend of increasing levels of education over time. At the reform year cut-offs however, there are clear jumps in schooling. It is this exogenous variation in schooling that allows identification of the impact of education on health.

In paper III, university attendance is a smooth function of how many credits a student achieved of a full program at upper secondary school (see Figure 3). There is a cut-off however for university eligibility at the 90 per cent of a full program. In figure 3 it can be seen that this eligibility rule leads to a clear jump in the probability of university attendance for females of about 10 percentage points. It is this exogenous jump in university attendance that we use to assess the impact of

university on medical care use. Paper III uses administrative data for education, income, hospital admissions and prescriptions for students graduating between years 2003 and 2005 and follows them up until 2013.

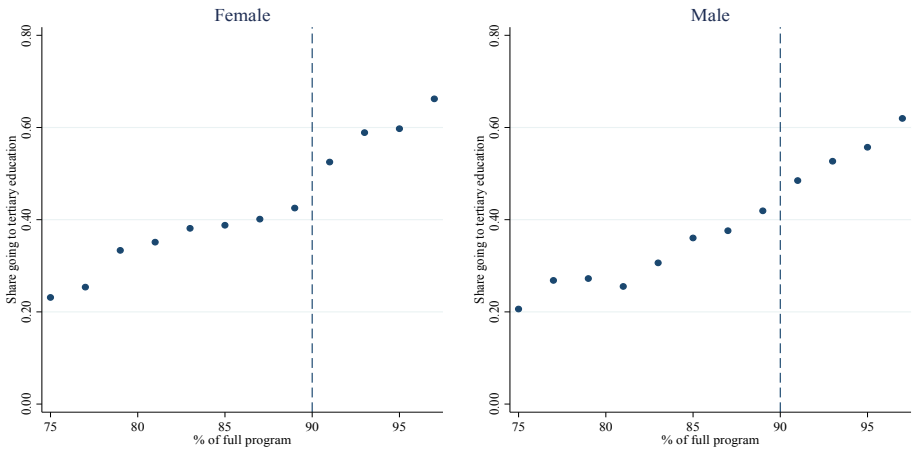

Figure 3. Paper III - Impact of university eligibility on university attendance
Figure notes: This figure plots a scatter of the share who attended a first term of university against percentage completed of a full program with a bin width of 2 percentage points (pp) of a full program (the size of the smallest course) in each bin for those graduating upper secondary school between the years 2003 and 2005. The cut-off for university is marked by the dashed vertical line at 90pp of a full program.

Paper IV uses an individual's exact age and Sweden's MLDA to identify the impact of the MLDA on both alcohol consumption and medical care use. In figure 4 it can be seen that alcohol consumption is a smooth function of age, increasing during the late teens and then flattening out in the mid twenties. There is also a clear jump in the quantity of pure alcohol consumed at 18 years of age, but not at age 20. Data on alcohol consumption patterns come from the Monitor Project survey and are for the years 2001 – 2012. Data on hospital admissions and deaths come from health administrative data for the whole population for the years 1969 - 2015.

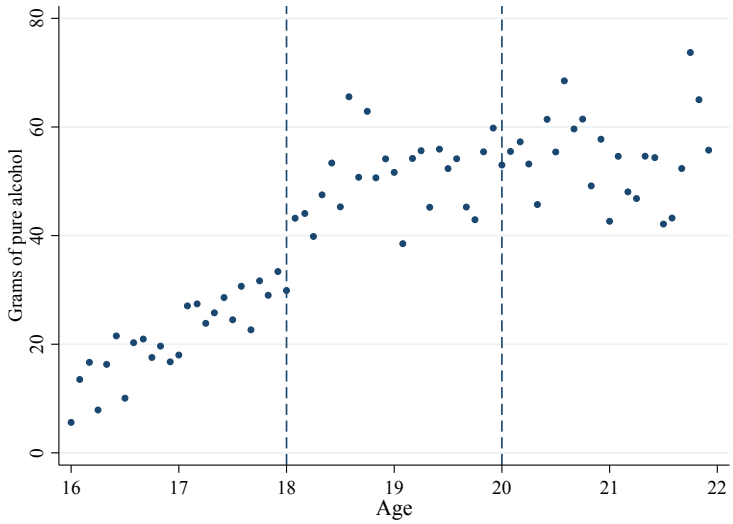

Figure 4. Paper IV - Impact of two Swedish MLDAs on quantity of pure alcohol consumed in last 30 days
Figure notes: This figure plots the scatter points of mean drinking behaviour by monthly age blocks. Data source is Monitor project survey 2001-2011.

Impacts on income related health inequality

The quasi-experimental techniques used in this thesis are all implemented in a linear setting. Papers I and III extend the analysis beyond the mean and consider the impact of years of schooling and university eligibility respectively on socioeconomic related health inequality using an extension of RIF-I-OLS. Paper I substitutes self-assessed health for the RIF of income related self-assessed health inequality and applies a within pair fixed effects regression to assess the importance of years of schooling on the level of inequality. Paper III substitutes medical care use with the RIF of parental income related medical use inequality and applies RD design to assess the importance of university eligibility on the level of inequality. In this way, more robust estimates of the impact of years of education and university eligibility on socioeconomic related health inequality are obtained. This is a key contribution of this thesis.

3. Results

The impact of education on health

Health

Figure 5 summarises the empirical evidence contained in this thesis on the impact of years of education on health, specifically: mortality, self-reported Fair or Bad Health (FBH) and a utility score between zero and one based on Self-Reported Health (SRH). Results from paper I using monozygotic twins and within pair fixed effects find no evidence of an impact of years of schooling SRH.

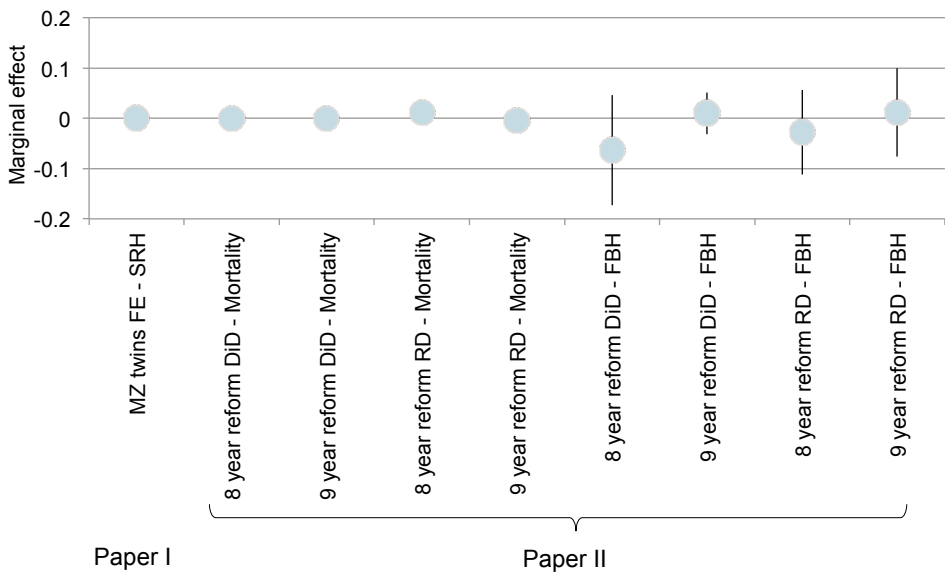

Figure 5. Education's impact on health
Figure notes: This figure presents coefficient estimates of years of education on health from paper I and paper II with corresponding 95% confidence intervals (Note that the confidence intervals are very small for the first 5 point estimates from the left-hand-side which is why they are hidden). *MZ twins FE* is monozygotic twins based within twin pair Fixed Effects. FBH is self-reported Fair or Bad Health. SRH is Self-Reported Health. See respective papers for details.

In paper II the impact of two different reforms on a variety of health outcomes including mortality and FBH are assessed. Two quasi-experimental strategies are used to identify the causal impact of education on health, DiD and RD. The two reforms were different but were rolled out across overlapping cohorts. Any observed differences in effects between the reforms are therefore due to the characteristics of the reforms. In figure 5 the Two Stage Least Squares (2SLS) estimates are presented from paper II and we find no impacts of education on mortality using DiD and RD and for either reform. The results for FBH, whilst less precisely estimated than those for mortality, find no convincing evidence of increased education leading to improved health. In paper II a number of sensitivity checks are performed where differences across genders and modelling strategies are considered and the results are robust to sub-group and modelling strategy.

Medical care use

Measures of health and medical care use do not necessarily measure the same thing. Health is often of larger interest, but medical care use can give us insights into the health production function of individuals. It also has the advantage of being objectively measured and available for the whole population of Sweden, both improving the precision of the estimates. Figure 6 summarises the findings in this thesis of the impact of education on medical care use. In paper II the causal impact of years of education on hospital days is considered and no evidence is found to support the hypothesis that years of education have a health improving impact (see first four estimates in Figure 6). More detailed analysis by cause of hospital visit is considered in paper II and the conclusions remain the same. In paper III no impact on frequency of all cause hospital admissions or prescriptions in general due to university eligibility is found. More detailed analysis by cause however finds a clear positive impact of university eligibility on the proportion of females who are prescribed contraceptives.

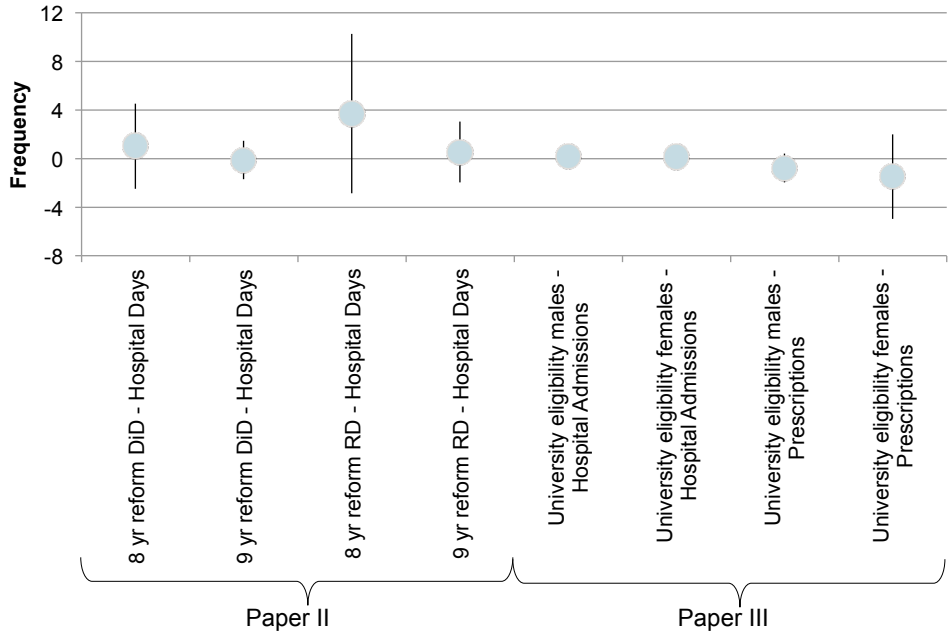

Figure 6. Education's impact on medical care use
Figure notes: This figure presents 2SLS based coefficient estimates of years of education on health from paper II (first 4 coefficients from the left hand side) and intention to treat coefficient estimates of university eligibility from paper III (last four coefficients on the right hand side) with corresponding 95% confidence intervals. See respective papers for details.

The impact of education on health inequality

Health

Paper I considers the impact of education on income related health inequality by decomposing the CI and its variants using the method developed in the same paper (RIF-I-OLS). Using within twin pair fixed effects together with RIF regression of income related SRH CI, no evidence is found of an impact of years of education on the CI. This conclusion also holds for the Erreygers Index and the Wagstaff Index.

Medical care use

Paper III considers the impact of university eligibility on the CI of parental income related medical care amongst young adults aged up to 30 years. Whilst a clear positive impact was found for the *level* of prescriptions of contraceptives for females no clear impacts were found for parental income related medical care use, either for all causes or specific causes including contraceptives for females.

The impact of minimum legal drinking age laws on health

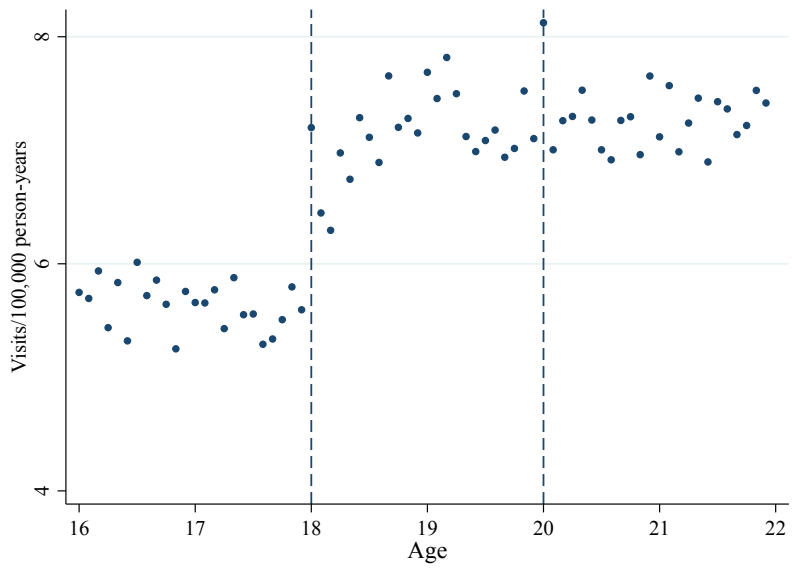

Figure 7. Swedish MLDA impact on alcohol related hospital admissions
Figure notes: This figure presents scatter points which are monthly age blocks of hospital visits/100,000 person-years for the years 1969-2015. See paper IV for details.

Paper IV considers the impact of Sweden's two-part MLDA on alcohol consumption and on health. The MLDA has a clear protective effect on alcohol consumption before 18 years of age. A clear jump in participation of alcohol consumption and quantity consumed is observed after turning 18. It appears that the quantity effect is a combination of increased participation (of about 6%) and also more frequent heavy drinking of about 16%. There is no clear evidence of a protective effect of the MLDA at 20 years of age in terms of alcohol consumption.

The jump in alcohol consumption at age 18 coincides with a clear jump in alcohol specific causes of hospital admission but not for mortality. Figure 7 shows the impact on alcohol specific causes of hospitalisation. There are clear birthday impacts at both 18 and 20. There is also a clear longer-term, non-birthday party impact of the MLDA at 18 where hospitalisations are estimated to increase by about 5%.

Whilst no clear impacts on alcohol consumption are observed at the MLDA of 20 years of age, clear protective impacts on hospitalisations are. Both hospitalisations due to homicides and self-harm jump after turning age 20. A drop in suicides is also found. The alcohol consumption data used, whilst very detailed, does not present any obvious explanations for these changes at age 20.

4. Discussion

Decomposing the Concentration Index

The first part of this thesis has presented a new method for the decomposition of socioeconomic related health inequality, RIF-I-OLS. This method can be used to answer questions of the form: could an increase in the level of education impact the CI? This type of decomposition based on RIF-I-OLS offers results derived under plausible assumptions. The value of RIF regression is that it can be used alongside the tools widely used in the treatment effects literature. Previous work that developed the idea of RIF regression has also laid out clearly what assumptions need to be made in order to approximate a random experiment (see e.g. Fortin et al. (2011), for a discussion of the issue of *identification* in decomposition). In addition, RIF-I-OLS can be used alongside Oaxaca-Blinder type techniques to decompose changes in the CI over time or differences between groups (e.g. gender) or across time periods following the line of argument of Fortin et al. (2011).

As outlined in the background a common and stringent assumption that is made by all regression based decomposition methods is the assumption of no general equilibrium effects. The assumption of no general equilibrium effects is a strong assumption, especially if large changes are considered. RIF-I-OLS makes the same assumption but because RIF-I-OLS is only valid for small changes this assumption is not a great threat to the validity of the results.

Another common feature of regression based decompositions is that they do not say anything about the channels leading to effects in our outcome measures. They are like black boxes where a change is observed in an explanatory variable and an effect is observed in our outcome measure but the economic mechanisms that produce this impact are hidden in the black box and cannot be seen. RIF-I-OLS can tell us how the CI may change with a change in education for example but it tells us nothing about the economic mechanisms that produce this change.

A potential misunderstanding of RIF-I-OLS is that it is only valid for individuals and not for sub-groups. This confusion comes about because the RIF value for each individual is the influence of the individual on the statistic. Using a RIF, one can remove an individual from the sample and very quickly calculate what the

statistic will be without that individual and therefore negating the need to recalculate the statistic for the new population. This is how a RIF works, but it is not a helpful way to understand how RIF regression works. RIF regression uses conditional distributions so thinking in terms of individuals can cause confusion. What RIF-I-OLS does is it calculates the conditional CI – an approximation of what the CI is for all subgroups. It therefore tells us how the CI would change if the population changed in the direction of a particular subgroup assuming that the CIs for all subgroups remain the same (no general equilibrium effects) and that the change is small.

RIF-I-OLS decomposition and existing methods for the decomposition of socioeconomic related health inequality (Wagstaff et al., 2003; Kessels & Erreygers, 2016) are all unable to speak to economic mechanisms that explain the inner workings of the decomposition results. The natural solution is not to extend these methods further but to take a step back and build a structural model of health. From there we can then create a measure of health inequality. This is the suggestion of Fleurbaey and Schokkaert (2009) and also Fortin et al. (2011). This is a good end goal but one that is ambitious and not often empirically feasible. Where this is not possible RIF-I-OLS can be used where exogenous variation in explanatory variables is identifiable to answer specific policy evaluation questions in conjunction with the tools from the treatment effects and policy evaluation literatures. This has been a key goal of this thesis, to show how the treatment effects literature and decomposition of the CI can be jointly applied to answer immediate policy relevant questions. In this way RIF-I-OLS can be used to identify the key forces underlying changes in the CI. This can then be complemented in the future with more structural type approaches to explain the economic mechanisms underpinning the decomposition results.

The role of education in determining health and health inequality

This thesis provides important evidence of the impact of education on health and medical care use.

The results from paper I indicate no clear impact of years of education on self-reported health or on various forms of CI when using within twins fixed effects and this is based on one of the largest twins datasets available worldwide. Twins based evidence of education's impact on health has the advantage that the differences in education between twins are across the spectrum of education from compulsory schooling all the way through to postgraduate studies. The estimated impacts therefore have a general external validity to the education question.

Various criticisms of results based on twin differences have been made however. A common critique is simply that twins are different and not representative of the general population. This may be true, but Gerdtham et al. (2016) who use the exact same data as that used in paper I find that the twins used are very similar to the general population across important measurable characteristics. Another critique is that research from epigenetics has shown that whilst twins are born with identical genetic make-ups, their genetics in fact evolve differently over time (see for e.g. Fraga et al., 2005). This is of concern if these differences also impact education and our health variable. There is also evidence that not even twins are born equal, where birth weight has shown to differ enough to explain substantial differences in education (Behrman et al. 1994). These are valid concerns but the potential biases that may occur from these differences all point to an overestimation of the impact of education on health yet no discernible differences are found in paper I.

A potentially more relevant concern to the twins based evidence of paper I is the issue of measurement error when taking differences within twin pairs. Griliches (1979) showed that any measurement error in the education variable will be exacerbated when a differencing method is applied to it and this will lead to downward biased results. The education variable used in paper I is derived from Swedish administrative records and research has shown this has a relatively low measurement error (see e.g. Holmlund et al., 2011). Even so, small measurement errors are magnified when differencing and could still cause a problem of downward bias in our estimates. Furthermore twins interact with each other so that there are likely to be strong peer effects where one twin's education will impact the other twin's health outcomes. To use the jargon, we cannot be sure the Stable Unit Treatment Value Assumption holds, and in this case the bias is again probably downwards. To be sure that we can rely on the conclusions from paper I it would be beneficial to confirm the findings using alternative data and or identification strategies.

Paper II assesses the causal impact of education on health using two compulsory school reforms to yield exogenous variation in years of schooling. The results show small and insignificant impacts of education on mortality and these are estimated with high precision. Results using survey data of self-reported health outcomes and behaviour also fail to find a positive relationship between education and health. The results of paper II therefore confirm the results of paper I.

The results of paper II provide an important contribution to the literature on the causal impact of education on health. Recent reviews of the literature on the causal impact of education on health have found it hard to draw conclusions from the evidence due to conflicting results (see Cutler and Lleras-Muney, 2012 and Grossman, 2015 for recent reviews). Results using compulsory school reforms have shown both improved health outcomes as well as very small or zero impacts

on health. Various suggestions have been given for this including analysis of different populations, in different time periods or using instruments that affect different sub-groups. Paper II is an important contribution to this wider literature because the nature of the Swedish school reforms allows many of these explanations for variation between studies to be pinned down and tested. Two school reforms that are different in character were rolled out on average just 7 years apart within municipalities in Sweden. Both reforms were rolled out progressively over time so that concerns about resource shocks due to teacher shortages for example or concerns about large general equilibrium effects of a whole cohort having an extra year of schooling do not apply. The reforms were overlapping in their roll out across Sweden and therefore students were entering similar labour markets and health systems. This allows for a clean comparison of the two reforms. In addition the paper uses a large dataset derived from population based administrative data, two identification strategies (DiD and RD), which assess the sensitivity of the results to the sub-groups analysed, and different modelling approaches. Analysis also considers various measures of health: mortality, self-reported health and health behaviours and medical care use (namely hospital admissions). The finding of no health improving effects of education is robust to school reform type, choice of DiD or RD, modelling approach and health outcome.

To date, Clark and Royer (2013) provide probably the most convincing evidence of the impact of education on health and they find zero or very small effects. The evidence provided in paper II confirms their findings of no or small effects of education on health but estimated for a different sample. Concerns that the results of Clark and Royer (2013) are specific to Britain, to the way the reforms were introduced or the cohorts they analysed appear to not be important. The results from paper II are similar to those of Clark and Royer (2013), but for Sweden based on two reforms rolled out progressively over time and for overlapping cohorts. Together, the results of paper II and of Clark and Royer (2013) provide important evidence that the role of education in determining health outcomes is small at the lower end of the education distribution.

The fact that there is convincing evidence that compulsory school reforms have a limited impact in determining health outcomes does not preclude education at any level having an impact on health. Indeed there is some evidence from the USA, using the Vietnam draft as an Instrumental Variable for college education, that shows college education leads to improved health behaviours and reduced mortality (De Walque, 2007; Buckles et al., 2016).

Paper III adds to the relatively limited literature on the impact of university/college education on health and considers the impact of university eligibility in Sweden on medical care use and medical care use inequality. The

identification strategy uses the fact that students need to achieve a pass rate of 90% of the upper secondary school program in order to go to university. Using RD the results show that females who achieve 90% of a complete program are much more likely to go to university. No clear overall impacts are found for hospital admissions or prescriptions but the proportion of females who received a prescription for contraceptives jumps at the 90% threshold. This result is a behaviour change relevant to the age group assessed (the observation window follows individuals up to age 30) and could be interpreted as a health investment, protecting against unwanted pregnancy. It could also be interpreted just as a fertility decision and a preference to delay childbirth amongst those who attend university. There are also indications that mental health of male students for this sub-population is negatively impacted by university attendance. No clear impacts are found for parental income related medical care use inequality using RD combined with RIF-I-OLS.

The overall conclusion that can be drawn from the evidence provided in this thesis is that the impact of education on health and on socioeconomic related health inequality is small. The impact is nearly always smaller than the OLS derived associations and possibly very small. Whilst there is a large body of evidence showing a clear association between education and health, it is far from clear that public policy should be orientated to increasing education levels in order to improve public health and reduce its socioeconomic related inequalities.

The effectiveness of Sweden's minimum legal drinking age policy

The evidence presented in this thesis casts doubt on the premise that education is an important public policy tool for improving public health. Paper IV considers an altogether different form of public policy, that of an MLDA.

Paper IV assesses the impact of Sweden's two part MLDA on alcohol consumption and health. It finds that while the MLDA at 18 years of age has similar protective effects in terms of alcohol consumption to that of the MLDA at 21 years of age in the United States, there are no negative impacts of the jump in alcohol consumption at age 18 on alcohol related causes of mortality. Jumps are, however, observed for alcohol related causes of hospital admissions. This suggests that young adults in Sweden make the transition to unrestricted alcohol without the large negative consequences observed in the United States. Potentially the two part MLDA is a reason for this alongside the other alcohol control policies in place in Sweden, notably the state controlled alcohol off-licence monopoly that restricts access to off-licence alcohol and that the blood alcohol content limit for driving a

motorised vehicle is set very low at 0.02 per cent (vs 0.08 in the United Kingdom and the United States).

5. Conclusion

In this introductory chapter I have presented the common theme for the thesis, *health, inequality and the impact of public policy*.

The decomposition of socioeconomic inequality in health is not a solved problem. This is not a controversial statement and has been made elsewhere (see e.g. Van Doorslaer et al., 2004a,b; Van Ourti et al., 2009; Van de Poel et al., 2009; Jones and Nicolás, 2006; Erreygers and Kessels, 2013; and Kessels and Erreygers 2016). This thesis has made explicit the restrictive parametric assumptions that need to be made for current decomposition methods to yield valid estimates and has proposed a less ambitious yet more believable alternative.

Using three different quasi-experiments to identify exogenous variation in education, no clear evidence is found for a health improving impact of education on health. No impact of education on health is found using variation in education identified by either a twins differencing strategy or variation in schooling induced by two major compulsory school reforms. Using eligibility rules for university education combined with RD, evidence is found that university education leads to greater contraceptive use amongst women, but this impact may just be a consequence of family planning rather than a health investment *per se*.

The same quasi-experimental techniques have been applied in combination with the new decomposition method developed as part of this thesis to assess whether differences in education can explain income related health inequality. Even though a clear association between education levels and income related inequality in health is observed, no clear impacts of education on income related inequality are observed once a more convincing control strategy is used.

Sweden's MLDA, however, does appear to have an impact on alcohol consumption and in particular episodic heavy drinking. The combination of the specific design of Sweden's two part MLDA and other alcohol control policies in place appear to both protect young underage adults and also mitigate the large negative health effects of increased alcohol consumption in the transition to full legal access to alcohol. In particular the large increases in mortality that accompany the large increases in drinking observed in the United States at age 21 when purchasing of alcohol is legalised are not observed in Sweden even though large increases in drinking in Sweden are observed.

POLICY RECOMMENDATIONS

No evidence has been found that increasing levels of education leads to improvements in health or changes in income related health inequality. Public health policies aimed specifically at health behaviours are potentially likely to be more effective. An example of which is the combination of Sweden's two-part MLDA policy, restrictive access to alcohol through the state run alcohol monopoly off-licence and a stringent blood alcohol limit for driving which has reduced the negative health consequences observed elsewhere of increased consumption of alcohol among young adults.

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Paper I

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A general method for decomposing the causes of socioeconomic inequality in health

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ABSTRACT

We introduce a general decomposition method applicable to all forms of bivariate rank dependent indices of socioeconomic inequality in health, including the concentration index. The technique is based on recentered influence function regression and requires only the application of OLS to a transformed variable with similar interpretation. Our method requires few identifying assumptions to yield valid estimates in most common empirical applications, unlike current methods favoured in the literature. Using the Swedish Twin Registry and a within twin pair fixed effects identification strategy, our new method finds no evidence of a causal effect of education on income-related health inequality.

1. Introduction

Socioeconomic differences in health are well documented across the western world (Deaton, 2003; Mackenbach et al., 2008, 2015). This awareness has led to a rapidly growing interest in the measurement and analysis of socioeconomic inequality in health. In terms of measurement, the dominant family of measures of socioeconomic inequalities in health are the various versions of the concentration index (CI) – a family of bivariate rank dependent indices. A bivariate rank dependent index summarises the relationship between cumulative health and socioeconomic rank, where a positive or negative socioeconomic gradient in health is represented by a positive or negative index value (Wagstaff et al., 1991; Fleurbaey and Schokkaert, 2009). These measures are bivariate because they relate an individual's level of health to her relative socioeconomic status. They are rank dependent because relative socioeconomic status is given by the socioeconomic rank of the individual.

Policymakers' and researchers' interest in socioeconomic inequality in health also extends beyond measurement through to explaining and understanding its underlying causes. One way to examine this issue is to decompose an inequality measure into a function of its (potential) causes. The dominant decomposition procedure to decompose a bivariate rank dependent index is the technique developed by Wagstaff et al. (2003) (WDW, onwards) which has been used extensively to explore the determinants of the well documented socioeconomic gradient (see, e.g., Leu and Schellhorn, 2004; Gomez and Lopez-Nicholas, 2005; Lauridsen et al., 2007; Hosseinpoor et al., 2006; McGrail et al., 2009; Morasae et al., 2012).[1] As well as being extensively applied, the WDW decomposition method has also been developed to expand its potential for application to a greater set of empirical situations such as health variables that are non-linear in nature (see, e.g.,

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[1] Gravelle (2003) is acknowledged for developing the same method although the explicit aim of his paper was not to decompose, but to standardise, the concentration index. The resulting methodology is nevertheless the same as that of WDW decomposition.

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Van Doorslaer et al., 2004a,b; Van Ourti et al., 2009; Van de Poel et al., 2009) and the inclusion of heterogeneous responses (Jones and Nicolás, 2006).

The health inequality toolbox is to a large extent adopted from the income inequality literature. The concentration index, for instance, is an adaptation of the Gini index, a popular index in the income inequality literature that measures the degree of income concentration. One important dimension in which measures of socioeconomic related health inequality differ from measures of income inequality, such as the variance or the Gini index, is that the latter consider a single distribution whereas the former consider the joint distribution of health and socioeconomic rank. Specifically to the issue at hand, bivariate rank dependent indices should be thought of as *two-dimensional* indices that consider the *covariance* between health and rank. Unfortunately, the leading decomposition method for bivariate rank dependent indices, the WDW decomposition method, is one-dimensional because it focuses on health but ignores rank (Erreygers and Kessels, 2013). That is, the WDW decomposition method explains the degree of variation in health rather than the covariance between health and rank. In response to this, Erreygers and Kessels (2013) and Kessels and Erreygers (2015) derive a set of two-dimensional decomposition methods, where rank and health are both estimated.[2] However, both the WDW decomposition and the alternatives proposed by Erreygers and Kessels (2013) and Kessels and Erreygers (2015) also suffer from another limitation: they can only *correctly* decompose one form of rank dependent index, yet there is an abundance of rank dependent measures used in the literature. So whilst one may wish to measure inequality in different ways, these approaches will yield the same results no matter which measure one chooses. We will illustrate later how the WDW method is only able to decompose absolute inequality measures (i.e., measures invariant to the addition or subtraction of an equal amount of health for all individuals in the population), which for example does not include the standard concentration index.

The various issues with the WDW methodology are closely linked to the general critique of decomposition methods raised by Fortin et al. (2011): many decomposition methods have focused on the derivation of procedures without first specifying the object of interest nor how to identify this object (i.e., stating what we want to estimate and the assumptions required to interpret the estimates). Indeed, both the derivation of the WDW method and the subsequent variations thereof have focussed on the procedure rather than identification of the object of interest. The discussion of identification has come second at best, potentially because these decompositions have been seen as an accounting exercise. As a consequence it is unclear how to actually interpret the parameters, and the so called contributions, within these decompositions. The relevance of this critique for the existing variations of the WDW decomposition is implicitly illustrated by Erreygers and Kessels (2013). Following a similar line of logic to that of the standard WDW decomposition, Erreygers and Kessels (2013) derived a set of two-dimensional decompositions by making small changes to the starting point of the procedure. These different methods yield a wide range of results, yet it is unclear which is preferred, and how to interpret the estimated coefficients. Erreygers and Kessels (2013) were unable to choose a preferred method because, as they noted themselves, they did not consider the identification issue.[3] In sum, the literature has shown that the WDW method is not only a one-dimensional decomposition of just one specific form of a bivariate rank dependent index but also just one possible method of many similar alternatives, the choice of which greatly affects the results.

We contribute to this literature by deriving and empirically illustrating an alternative regression based decomposition method for rank dependent indices that overcomes the criticisms of the decomposition methods currently available. This method aims to explain the causes of socioeconomic inequality, not by focussing on the variables that form the covariance, but by directly decomposing the weighted covariance of health and socioeconomic rank, i.e., the rank dependent index. This new approach builds on the concept of regression of a recentered influence function (RIF). A RIF is a concept that originates from the robustness literature of statistics that yields an approximation of the derivative (gradient) of a statistic. Intuitively, the RIF is a vector where each element corresponds to a particular individual's influence on the statistic. That is the RIF value for a specific individual tells us how the statistic would change if that individual were to be removed from the sample (weighted by the inverse of the sample size). The RIF is useful for decomposition because it allows any statistic to be expressed as a mean of the RIF vector and this allows all the regression tools for standard mean analysis to be used to link individual characteristics to a statistic. Importantly RIF regression already has a defined object of interest and it is clear how to identify it (Firpo et al., 2009): regressing the vector of RIF values on a set of covariates yields the unconditional partial effect of covariates on the statistic.

In this paper we apply the concept of RIF regression to a bivariate rank dependent index. Firpo et al. (2007, 2009) applied RIF regression to an income inequality question, estimating and decomposing RIFs for univariate measures such as the variance, the unconditional quantile, and the Gini index. The major contribution of this paper, and a key step forward for health inequality analysis, is that we derive the RIF for a general bivariate rank dependent index, and also specifically for familiar versions such as the concentration index and the adjustments suggested by Wagstaff (2005) and Erreygers (2009). Decomposition of the index is then performed by a two-step procedure of first computing the RIF of the rank dependent index, and then regressing the RIF on a set of covariates yielding the marginal effects of the covariates on the index.

The application of the RIF regression method to the decomposition of rank dependent indices has a few important benefits. First, the object of interest of the method is clear and therefore discussion of identification is much more straightforward. Second, the method directly decomposes the weighted covariance of health and socioeconomic rank. As a consequence it overcomes the critique of Erreygers and Kessels (2013) and it can be used to decompose all forms of bivariate rank dependent indices. Being able to decompose all forms of bivariate rank dependent indices is a key feature of this new method because each form of rank dependent index has a different set of underlying value judgements with respect to inequality (Allanson and Petrie, 2014; Kjellsson et al., 2015) and there remains no actual consensus as to which index is preferred. The ability to decompose several indices is therefore key for health inequality analysis.

A further benefit of RIF regression decomposition is that the results are familiar in their interpretation. Assuming a linear relationship means the RIF is the dependent variable in an OLS regression whose coefficients equal the marginal effect of covariates X on the rank dependent index. This interpretation is analogous to that of an OLS regression of a random variable. Indeed, a RIF decomposition of the

[2] In the more recent paper by Kessels and Erreygers (2015), they propose a structural equation modelling approach where rank and health are both estimated. This two-dimensional decomposition is one potential way to acknowledge the bivariate nature of these inequality indices, but the requirements of such a structural modelling approach is data demanding (requires two instrumental variables for health and for rank respectively), limiting the potential scope for such a solution. This solution also doesn't address the other issues raised in this paper.

[3] In the conclusion of their paper, Erreygers and Kessels (2013) call for an "axiomatic approach" to derive the most preferred method. We interpret this as a call for specifying the object of interest in beforehand, and then set up the assumptions needed to identify this object.

mean (assuming a linear function of the dependent variable) is simply OLS of a random variable (Firpo et al., 2009). As most researchers are familiar with OLS, assuming linearity makes the RIF regression straightforward to estimate and the conditions needed to obtain a causal parameter are well known. This familiarity makes RIF decomposition a useful tool, not only for descriptive analysis, but also in a policy evaluation framework.

In order to help the reader understand why RIF regression based decomposition is a useful addition to the analyst's toolkit for the analysis of bivariate rank dependent indices, a brief description of rank dependent indices and the standard WDW decomposition method is provided (in Section 2) before a discussion of the identifying assumptions of WDW decomposition (Section 2 again). Although the health inequality literature has previously highlighted that these identifying assumptions may be restrictive (Van Doorslaer et al., 2004a,b; Erreygers and Kessels, 2013; Gerdtham et al., 2016), they have never been summarised clearly in one place, and may therefore be unknown to practitioners. The literature suggests that the usefulness of the WDW decomposition should be questioned, as the violation of these conditions is potentially severe. The paper then presents a new method for decomposing a bivariate rank dependent index based upon RIF regression that requires fewer identifying assumptions. To help develop the intuition of this new method the concept of the RIF is briefly introduced before deriving the RIF for a general bivariate rank dependent index (Section 3). RIF decomposition is then discussed in detail (Section 4).

To illustrate the differences in interpretation between the RIF and the WDW decomposition we present an empirical example using the Swedish Twin Register (Section 5). The empirical example also highlights the importance of being able to decompose different forms of rank dependent index showing that the choice of index has bearing on the association between education and health inequality. We find no association of education with socioeconomic health inequality using RIF regression. To highlight how one can use the RIF decomposition for establishing causal relationships, we use a twin differencing strategy to attempt to isolate the effect of education on socioeconomic related heath inequality. The results suggest there is no causal effect of education on any common choice of bivariate rank based measure of health inequality.

Having illustrated RIF regression of a bivariate index we then discuss the relative merits of this new approach compared to WDW decomposition (Section 6) concluding that RIF-I-OLS will uncover the (causal) parameters of interest under common empirical conditions. Evidence from the literature and also presented in this paper suggests that when concern lies with covariates that are known to impact on the ranking variable and or the weighting variable, WDW decomposition is likely to yield biased results. Conversely, RIF regression does not require these identifying assumptions and this makes the RIF regression of a bivariate rank dependent index easier to interpret and a preferable descriptive decomposition tool. In addition, RIF regression is also well suited to policy evaluation. RIF regression allows the effect of a policy to be evaluated across a wide range of statistics, highlighting its potential in the field of program evaluation.

2. Preliminaries

2.1. A rank dependent index

The general term for a statistic, such as the mean, variance or the Gini for example, is a functional, $\nu(F)$, where F is a probability measure for which $\nu(F)$ is defined.[4] Let us define $H \in [0,+\infty)$[5] as a random variable of health with mean denoted as μ_H and with probability measure denoted as F_H. We rank each individual by a random variable for socioeconomic status, Y. The CDF of Y, F_Y, yields the fractional rank for each individual, which by definition has mean ½ (F_Y is uniformly distributed over the unit interval). The joint distribution of H and F_Y is given by F_{H,F_Y}. The functional for the general form of a rank dependent index (I) is then given by:

$$I = \nu^I(F_{H,F_Y}) = \nu^{\omega_I}(F_H)\nu^{AC}(F_{H,F_Y}), \tag{1}$$

where $\nu^{\omega_I}(F_H)$ is a weighting function specific to a particular form of rank dependent index, and the absolute concentration index (AC) is given by twice the covariance between H and F_Y:

$$AC = \nu^{AC}(F_{H,F_Y}) = 2cov(H, F_Y), \tag{2}$$

We refer to this as the absolute concentration index as it is invariant to the addition or subtraction of an equal amount of health for all individuals in the population.[6] The relative counterpart is the standard concentration index (CI), which is invariant to equi-proportional changes in health. The weighting functions for these common forms of rank dependent index are:

Absolute concentration index:

$$\nu^{\omega_{AC}}(F_H) = 1 \tag{3}$$

Concentration index:

$$\nu^{\omega_{CI}}(F_H) = \frac{1}{\mu_H} \tag{4}$$

Different choices of weighting function imply different value judgements, in this case a preference for absolute or relative inequality. The choice of index, and therefore the choice of weighting function, is more complex when the health variable of interest has both an upper and lower bound denoted as b_H and as a_H respectively, i.e., $H \in [a_H, b_H]$ (Wagstaff, 2005; Erreygers, 2009; Erreygers and Van Ourti, 2011; Kjellsson and Gerdtham, 2013a,b; Kjellsson et al., 2015). For such a variable, health can be represented as both attainments ($H - a_H$) and shortfalls ($b_H - H$), and the choice of which affects the value of the concentration index. One set of indices adjusted for bounded variables

[4] The symbol ν is used to signify a functional and comes from the class of statistics called ν-statistics.
[5] We define H in the general case as an unbounded measure without any loss of generalisability for bounded health variables.
[6] In the literature the absolute concentration index is sometimes called the generalised concentration index, although it is not a generalisation of the concentration index. We label it the absolute concentration index because it is an absolute measure of socioeconomic-related health inequality (it is not affected by the addition or subtraction of a certain amount of health).

assures that the level of inequality is the same irrespective of this representation. The weighting functions for two rank dependent indices that make this adjustment are:

Erreygers index:

$$\nu^{\omega_{EI}}(F_H) = \frac{4}{b_H - a_H} \tag{5}$$

Wagstaff index:

$$\nu^{\omega_{WI}}(F_H) = \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)} \tag{6}$$

The Erreygers Index (EI) is an absolute index adjusted for a bounded variable, whereas the underlying value judgement of the Wagstaff Index (WI) is more complex (Wagstaff, 2005; Kjellsson and Gerdtham, 2013a,b; Allanson and Petrie, 2014). It is also possible to define a concentration index that is invariant to either proportional changes in attainment or shortfalls of bounded health variables. Following Kjellsson et al. (2015), we denote these as:

Attainment-relative concentration index (ARCI)[7]

$$\nu^{\omega_{ARCI}}(F_H) = \frac{1}{(\mu_H - a_H)} \tag{7}$$

Shortfall-relative concentration index[8] (SRCI)

$$\nu^{\omega_{SRCI}}(F_H) = \frac{1}{(b_H - \mu_H)} \tag{8}$$

There exists no actual consensus as to which index is preferred, but the literature stresses that any choice of index represents a value judgement (Allanson and Petrie, 2014; Kjellsson et al., 2015). Given this lack of consensus it is arguably important that any decomposition analysis is able to encompass as broad a view as possible.

2.2. The standard decomposition

The leading decomposition method applied to I is the WDW decomposition method based on a linear regression of health. Assuming health, represented by h, an $n \times 1$ vector of drawings from H, is observed alongside covariates, X, and that health can be expressed as a linear in variables model in X, together yields the following regression equation:

$$h = \alpha + X'\beta + e, \tag{9}$$

where X is a $k \times n$ matrix, α is an intercept, β is a $k \times 1$ vector of regression coefficients, and e is a $n \times 1$ vector of error terms. Following Wagstaff et al. (2003), I can then be decomposed by substituting Eq. (9) into (1), yielding the following formula:

$$I = \nu^{I}(F_{H,F_Y}) = \nu^{\omega_I}(F_H)\sum_{k=1}^{K}\beta_k 2cov(X_k, F_Y) + \nu^{\omega_I}(F_H)2cov(e, F_Y), \tag{10}$$

where β_k is the regression coefficient corresponding to the kth regressor from the linear regression Eq. (9), $2cov(X_k, F_Y)$ is the absolute concentration index of the kth covariate X_k and $2cov(e, F_Y)$ is the absolute concentration index of e. The first part of the WDW decomposition formula, given by Eq. (10), expresses the change in $\nu^{I}(F_{H,F_Y})$ predicted by a change in either $cov(X_k, F_Y)$ or β_k, what we will call *marginal contributions*. The first part of equation (10) has also been used to express I as the proportion explained by X, "the explained part" (what we will refer to as *percentagewise contributions*), plus the second part of Eq. (10), as "the unexplained part". What can be immediately observed from Eq. (10) is that the WDW decomposition implicitly holds the weighting function, $\nu^{\omega_I}(F_H)$, constant and assumes that if one made any changes that impacted $\sum_{k=1}^{K}\beta_k 2cov(X_k, F_Y)$ this would have no impact on the weighting function. A consequence of this is that percentagewise contributions are the same no-matter which index one uses.

2.3. The identifying assumptions of WDW decomposition

To the best of our knowledge the identifying assumptions that underpin WDW decomposition, whilst not new to the literature, have never been stated explicitly in one place, neither in its application or otherwise. To be explicit we set out these assumptions below and then discuss each assumption in turn.

The identifying assumptions required by the WDW decomposition are:

I. The determinants of health do not determine rank (rank ignorability).
II. The determinants of health do not determine the weighting function (weighting function ignorability).
III. Health can be modelled as a function linear in variables X and an error term.
IV. Exogeneity: The errors from the health regression have zero conditional mean.

[7] Erreygers and Van Ourti (2011) first suggested this as the generalised version of the corrected concentration index.
[8] An index using this weighting function is equivalent to applying the (attainment-relative) concentration index representing the health variable in terms of shortfalls, or ill health.

If all the identifying assumptions above hold, WDW decomposition identifies both percentagewise and marginal contributions yielding results of potentially great empirical interest.[9] In most empirical applications identifying assumption IV – which OLS requires for causal interpretation – is not seen as a necessary condition and WDW decomposition is generally viewed as a "simple descriptive accounting exercise" based on some correlations from an OLS regression (Gerdtham et al., 2016). WDW decomposition is therefore generally thought of as yielding descriptive percentagewise contributions. However, even as a descriptive accounting exercise, this still requires the results to be interpreted in light of identifying assumptions I, II & III, which in empirical practice often are unreasonable to impose. This muddies the interpretation of the results.

The restrictiveness of *rank ignorability* (Identifying assumption I) has previously been pointed out by Erreygers and Kessels (2013) as well as in Kessels and Erreygers (2015). They criticise the WDW decomposition approach for being a *one-dimensional* decomposition (of a bivariate index) because it only decomposes one part of the covariance (health). Ignoring the association between the covariates and rank means that for any (causal) explanation of changes in covariates the income rank is assumed to remain the same even after the change. Indeed Erreygers and Kessels (2013) and Kessels and Erreygers (2015) both find important differences in the results when this assumption is relaxed. It is important to note that their results are based on an approach that still maintains the other identifying assumptions.

Assumption II, *weighting function ignorability*, is similarly restrictive because the weighting function, $\nu^{\rho_I}(F_H)$, is generally a function of health, and will by design be correlated with the covariates, as the covariates are predictors of health. As seen in Eqs. (3)–(8), the weighting functions of CI, WI, ARCI, and SRCI are all functions of mean health. Only absolute versions of the rank dependent index (such as AC and EI) have a constant weighting function. Because w*eighting function ignorability* requires the analyst to assume that the weighting function is unaffected by a change in these covariates, a WDW decomposition of any rank dependent index implicitly decomposes an absolute version of the index. In practice this restriction means that the WDW decomposition is only applicable to absolute inequality indices (even though it was developed for the relative concentration index).[10]

In regard to assumption III, *health is a function linear in variables*; there are few health outcomes that can truly be modelled in a linear way. It is common to find non-linear health functions: outcomes may be categorical (Underweight, normal, overweight, obese), censored at zero (doctor visits) or two-part decisions (quantity smoked) all of which are non-linear. The linearity assumption of WDW, however, requires more than the standard linearity assumption: To provide the popular interpretation of percentagewise contributions of each variable of interest, WDW requires the model to be not only linear in parameters, but linear in variables. Potential solutions have been proposed (see, e.g., Van Doorslaer et al., 2004a,b; Van Ourti, 2004; Van de Poel et al., 2009; Van Ourti et al., 2009), but they require the non-linear estimates to be translated back to the linear setting in order to yield percentagewise contributions or are applicable only to changes. With that said, the linearity assumption has not been found to be that restrictive in practice (Van Doorslaer et al., 2004b; Van de Poel et al., 2009). In addition, linearity is an assumption that empirical economists are often willing to make (especially in the policy evaluation literature). The flexibility and simplicity of methods such as OLS generally provide a powerful framework for empirical analysis. The available evidence does indicate that assumption III ranks as a less restrictive assumption compared to both I and II.

The underlying issue with the current available methods to decompose a bivariate rank dependent index is the critique raised by Fortin et al. (2011): many decomposition methods have not been explicit about what the parameter of interest is and the required identifying assumptions. The methods that are currently available have been developed with a focus on procedures with little thought given to identification. As set out in the introduction, Erreygers and Kessels (2013) implicitly illustrate the consequence of this ambiguity and derive quite a few alternative decomposition methods. A consequence of not defining the parameter of interest is that it is not immediately obvious how to interpret these various different methods of decomposition yet alone be able to choose a preferred method. However, the results obtained by Erreygers and Kessels (2013) vary quite dramatically depending on the method chosen and therefore the choice of method matters. Combined with the literature highlighting the implicit identifying assumptions of WDW, the findings of Erreygers and Kessels (2013) reveal that the results of the WDW decomposition are not as easily interpreted as once thought.

In the next section we derive a completely different approach to regression-based decomposition of a bivariate rank dependent index that allows two of the identifying assumptions of WDW decomposition to be relaxed simultaneously: rank and weighting function ignorability. Importantly, we explicitly state our parameter of interest and the assumptions required to identify this parameter. This method has the potential to identify the parameters of interest under much more common empirical conditions, yielding results that have a clear interpretation.

3. The RIF for a general bivariate rank dependent index

The RIF is derived from the influence function (IF), which originates from the robustness literature of statistics. Hampel (1974) introduced the concept of the IF with the original purpose to explore how various statistics are affected (or influenced) by particular observations, hence the name, influence function. The RIF has the same properties as the IF with the singular exception that the RIF has a different expected value to that of the IF. Firpo et al. (2009) developed the concept of the RIF, RIF regression and hence RIF decomposition. In this section we first introduce the concept of the IF and the RIF in a univariate setting, before deriving the RIF for a general *bivariate* rank dependent index.

3.1. The influence function and the recentered influence function

The influence function is a specific form of a directional derivative (or Gâteaux derivative). A directional derivative is used to find the influence of a perturbation or contamination in a distribution, for example from F_H towards a new distribution, on a statistic. The IF is

[9] It is worth noting that percentagewise contributions is a global parameter and requires the assumption that under large changes in the covariate there will be no (unaccounted for) general equilibrium effects.

[10] A Potential solution to weighting function ignorability has been developed by Van Ourti et al. (2009) but this still assumes rank ignorability and is applicable only for decomposition of changes.

the particular form of a directional derivative where the new distribution, denoted as δ_h, equals a cumulative distribution function for a probability measure that puts mass 1 at a particular value h:

$$\delta_h(l) = \begin{cases} 0 & \text{if } l < h \\ 1 & \text{if } l \geq h \end{cases}, \tag{11}$$

where l is a draw from H.[11] To define the IF of the functional $v(F_H)$ evaluated at point h, denoted as IF(h; v), we first define G_h as a mixing probability distribution of F_H and δ_h:

$$G_h = (1-\varepsilon)F_H + \varepsilon\delta_h, \tag{12}$$

where $\varepsilon \in (0, 1)$ is a probability, or a weight, representing the relative change in the population through the addition of δ_h. That is, G_h is a distribution that is ε away from F_H in the direction of δ_h. IF(h; v) is then defined as:

$$IF(h; v) = \left.\frac{\partial v(G_h)}{\partial \varepsilon}\right|_{\varepsilon=0} = lim_{\varepsilon\to 0}\frac{v(G_h) - v(F_H)}{\varepsilon}, \tag{13}$$

if the limit is defined for every point $h \in \mathbb{R}$, where $\mathbb{R}$ is the real line.[12] Intuitively speaking, the IF captures the (limiting) influence of an *individual* observation on the functional $v(F_H)$ (Wilcox, 2005) and this can be used to understand how the addition/subtraction of an observation would affect a statistic without having to re-calculate the statistic. In practice, calculating an IF yields an influence function value for each individual in the sample.

Having defined the IF it is now possible to define the RIF. One can think of the RIF in two ways. First, as a linear approximation of the functional, the RIF consists of the first two leading terms of a Von Mises linear approximation. The RIF is also a minor transformation of the IF, and is obtained from the IF by adding back the original functional, $v(F_H)$:

$$RIF(h; v) = v(F_H) + IF(h; v). \tag{14}$$

While the expectation of the IF is zero (Monti, 1991), the expectation of the RIF is equal to the original distributional statistic $v(F_H)$ (Firpo et al., 2009). This is a useful property because, as we discuss later, it allows standard regression tools for the mean to be applied to (and therefore decompose) any statistic.

To illustrate the two concepts, the IF and the RIF, assume the statistic of interest is the mean. The IF of μ_H equals $\text{IF}(h; \mu_H) = lim_{\varepsilon\to 0}((1-\varepsilon)\mu_H + \varepsilon h - \mu_H)/\varepsilon = h - \mu_H$. This states that adding or removing an observation will have an effect on μ_H equal to the distance between the observation, h, and the mean (standardised by the sample size). Adding the statistic, μ_H, to $IF(h; \mu_H)$ yields the RIF of the mean, $RIF(h; \mu_H) = \mu_H + (h - \mu_H) = h$.

3.2. The RIF for a general (bivariate) rank dependent index

As the rank dependent index, I, is a functional of the joint probability distribution F_{H,F_Y}, we need to extend the definitions in Eqs. (11)–(14) from a univariate to a bivariate setting. Let $G_{h,F_Y(y)}$ be a bivariate distribution function obtained by an infinitesimal contamination of F_{H,F_Y} in both h and $F_Y(y)$:

$$G_{h,F_Y(y)} = (1-\varepsilon)F_{H,F_Y} + \varepsilon\delta_{h,F_Y(y)}. \tag{15}$$

Here $\delta_{h,F_Y(y)}$ denotes a joint cumulative distribution function for a joint probability measure that gives mass 1 to $(h, F_Y(y))$ jointly:

$$\delta_{h,F_Y(y)}(l, r) = \begin{cases} 0 & \text{if } l < h \text{ or } r < F_Y(y) \\ 1 & \text{if } l \geq h \text{ and } r \geq F_Y(y) \end{cases}, \tag{16}$$

where l and r are draws from H and F_Y respectively. In analogy with Eq. (13), we then define the bivariate IF of $v^I(F_{H,F_Y})$ evaluated at point $(h, F_Y(y))$ as[13]:

$$IF(h, F_Y(y); v^I) = \left.\frac{\partial v^I(G_{h,F_Y(y)})}{\partial \varepsilon}\right|_{\varepsilon=0} = lim_{\varepsilon\to 0}\frac{v^I(G_{h,F_Y(y)}) - v^I(F_{H,F_Y})}{\varepsilon}, \tag{17}$$

given that this limit is defined for every point $(h, F_Y(y)) \in \mathbb{R}^2$, where $\mathbb{R}^2$ denotes the real plane. The RIF of I is then defined as:

$$RIF(h, F_Y(y); v^I) = v^I(F_{H,F_Y}) + IF(h, F_Y(y); v^I). \tag{18}$$

In Proposition 1 we state the expression of the RIF for a general bivariate rank dependent index for socioeconomic related health inequality, leaving the proof to Appendix A, before we present the RIF for the common forms of I that appear in the health inequality literature.

[11] Note that the definition of h is no longer an $n \times 1$ vector as defined previously for the WDW decomposition.

[12] Another way of checking whether the IF exists is to check if the functional is continuous (has no jumps or spikes) and the differential is bounded.

[13] Note that I is a covariance not of two random variables but a covariance of a random variable, H, and the ranking variable, F_Y, which is a function of a random variable. Deriving the IF is therefore more complicated than deriving the IF of a standard covariance because the ranking function is also affected by the infinitesimal contamination.

Proposition 1. *Let* $\nu^I(F_{H,F_Y}) = \nu^{\omega_I}(F_H)\nu^{AC}(F_{H,F_Y})$ *be a general rank dependent index, the AC be defined as* $\nu^{AC}(F_{H,F_Y}) = 2cov(H, F_Y)$ *and* F_{H,F_Y} *be the joint CDF of H and* F_Y *with corresponding pdf denoted as* f_{H,F_Y}. *Then the RIF for* $\nu^I(F_{H,F_Y})$ *is given by:*

$$RIF(h, F_Y(y); \nu^I) = \nu^I(F_{H,F_Y}) + IF(h; \nu^{\omega_I}) * \nu^{AC}(F_{H,F_Y}) + \nu^{\omega_I}(F_H) * IF(h, F_Y(y); \nu^{AC}),$$

where $IF(h; \nu^{\omega_I})$ *denotes the IF of the weighting function for I and* $IF(h, F_Y(y); \nu^{AC}) = -2\nu^{AC}(F_{H,F_Y}) + \mu_H - h + 2hF_Y(y) - 2\int^y \int^{+\infty} hf_{H,F_Y} dh dF_Y(z)$ *denotes the IF for AC.*

Proposition 1 shows that for any general rank dependent index, the RIF of *I* equals the sum of the original statistic, $\nu^I(F_{H,F_Y})$, and its IF, of which the IF is found by application of the product rule of $\nu^{\omega_I}(F_H)\nu^{AC}(F_{H,F_Y})$. The IF for the AC consists of terms familiar from standard inequality analysis; the AC, the mean of health, an individual's health, an individual's rank, and the absolute concentration curve co-ordinate of the individual, $\nu^{ACC}(F_{H,F_Y}(y)) = (2\int^y \int^{+\infty} hf_{H,F_Y} dh dF_Y(z))$. The RIF of any *I* follows from calculating the IF of the weighting function for the particular *I* in question and then slotting this into the formula for the RIF given in Proposition 1. Corollary 1 presents the formulas for the RIF of the specific versions of *I*, again leaving the proof to Appendix A.[14]

Corollary 1. *The RIFs for the AC, EI, CI, ARCI, SRCI and the WI are given by*:

$$RIF(h, F_Y(y); \nu^{AC}) = \nu^{AC}(F_{H,F_Y}) + IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{EI}) = \nu^{EI}(F_{H,F_Y}) + \frac{4}{b_H - a_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{CI}) = \nu^{CI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{\mu_H^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{ARCI}) = \nu^{ARCI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{(\mu_H - a_H)^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H - a_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{SRCI}) = \nu^{ARCI}(F_{H,F_Y}) + \frac{(-\mu_H + h)}{(b_H - \mu_H)^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{b_H - \mu_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{WI}) = \nu^{WI}(F_{H,F_Y}) + \frac{-(b_H - a_H)[(b_H + a_H - 2\mu_H)(h - \mu_H)]}{((b_H - \mu_H)(\mu_H - a_H))^2} * \nu^{AC}(F_{H,F_Y}) + \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)} IF(h, F_Y(y); \nu^{AC})$$

The RIF formulas may appear complex, however they are just a linearisation of the statistic. Practical implementation of RIF estimation is straight forward and to illustrate this we consider empirical estimation of the RIF where the empirical RIF for *I* is estimated using sample data as:

$$\widehat{RIF}(h, F_Y(y); \nu^I) = \widehat{\nu^I}(F_{H,F_Y}) + \widehat{IF}(h; \omega_I) * \widehat{\nu^{AC}}(F_{H,F_Y}) + \widehat{\nu^{\omega_I}}(F_H)\left[-\widehat{\nu^{AC}}(F_{H,F_Y}) + \widehat{\mu_H} - h_i + 2h_i\widehat{F_Y}(y_i) - \widehat{\nu^{ACC}}(F_{H,F_Y}(y_i))\right] \quad (19)$$

To empirically estimate the RIF, the data of *N* observations is first ordered using the ranking variable, *Y*, so that $y_1 \leq y_2 \leq \ldots \leq y_i \leq \ldots \leq y_N$. Then estimates of the *I*, $\widehat{\nu^I}(F_{H,F_Y})$, the AC, $\widehat{\nu^{AC}}(F_{H,F_Y})$, the weighting function, $\widehat{\nu^{\omega_I}}(F_H)$, and the mean $\widehat{\mu_H}$ are obtained using the formulas in Section 2. The estimate of the rank, $\widehat{F_Y}(y_i)$, and the absolute concentration curve coordinate, $\widehat{\nu^{ACC}}(F_{H,F_Y}(y_i))$, can be calculated as follows:

$$\widehat{F_Y}(y_i) = \frac{\sum_i^1 1}{N} \quad (20)$$

$$\widehat{\nu^{ACC}}(F_{H,F_Y}(y_i)) = \frac{\sum_i^1 h_i}{N}, \quad (21)$$

where the numerators are a sum that follow the orderings of the *i* values of *Y*.[15] Together these yield the empirical RIF. It is important to note that the formulas are the same for all empirical applications, no matter what form the health and socioeconomic ranking variable. Consequently, estimation of the RIF can be automated. To this end the Stata do file used in our empirical example of this paper is provided as an Appendix (see online supplementary material) that allows estimation of the RIFs derived in this paper and also provides a working example of how to decompose the RIF and yield bootstrapped standard errors.[16] For decomposition analysis (RIF regression) the empirical RIF is used as a dependent variable in a regression. We now turn to the concept of RIF regression.

[14] The formula for the RIF for the CI is very similar to the RIF for the univariate Gini. Indeed we show in Appendix B that if we derive the RIF for the univariate Gini from the covariance formula, as we have done in the proof of proposition 1, this is the same as presented in Firpo et al. (2007) where the RIF for the Gini has been derived from a formula for the Lorenz curve.

[15] The absolute concentration curve is a mapping of cumulative health and fractional rank (Wagstaff et al., 2003).

[16] A Stata ado file is also available that allows users to perform OLS based RIF regression of a number of forms of the concentration index and also save the RIF values to perform graphical analysis and is found at: https://sites.google.com/site/gawainheckley/home/stata-code?pli=1.

4. RIF regression decomposition

RIF regression is a method that allows us to decompose a RIF of any functional into a function of the sources of its variation, the covariates, X. Our focus is decomposition of I and hence RIF of I regression decomposition. Firpo et al. (2009) identify two parameters of interest that can be estimated using RIF regression: the marginal effect of covariates X on a functional, which is an individual effect, and the unconditional partial effect, which is a population effect measure. The latter captures the impact of a marginal location shift in a continuous covariate or the impact of marginal changes in the conditional distribution of a binary covariate holding everything else constant. Relating this to the topic of the paper, the unconditional partial effect measures how an equal marginal increase in education for everyone would impact on the bivariate rank dependent index (Note that RIF regression estimates *marginal contributions*, not percentagewise contributions as WDW decomposition results are often presented). In this section we show how RIF regression of I obtains these parameters and the assumptions required to identify them.

4.1. RIF regression

The recentering of the IF yielding the RIF implies that $v^I(F_{H,F_Y})$ can be expressed as an expected value of the RIF:

$$v^I(F_{H,F_Y}) = \int_{-\infty}^{\infty} RIF(h, F_Y(y); v^I) \cdot dF_{H,F_Y}(h, F_Y(y)) = E[RIF(H, F_Y; v^I)] \tag{22}$$

In order to link $v^I(F_{H,F_Y})$ to the covariates X, we follow Firpo et al. (2009) applying the law of iterated expectations to express $v^I(F_{H,F_Y})$ as a conditional expectation:

$$\begin{aligned} v^I(F_{H,F_Y}) &= \int_{-\infty}^{\infty} RIF(h, F_Y(y); v^I) \cdot dF_{H,F_Y}(h, F_Y(y)) = \int_{-\infty}^{\infty}\int_{-\infty}^{\infty} RIF(h, F_Y(y); v^I) \cdot dF_{(H,F_Y)|X}(h, F_Y(y)|X = x) \cdot dF_X(x) \\ &= \int_{-\infty}^{\infty} E[RIF(H, F_Y; v^I)|X = x] \cdot dF_X(x) \end{aligned} \tag{23}$$

where F_X is the CDF of X.[17] Thus, decomposing $v^I(F_{H,F_Y})$ boils down to a problem of estimating a conditional expectation, which can be solved by standard regression methods. For a general function of covariates X and an error term $\in$, denoted as $\lambda(X, \in)$, the conditional expectation of $RIF(h, F_Y(y); v^I)$ may then be modelled as:

$$E[RIF(H, F_Y; v^I)|X = x] = \lambda(X, \in) \tag{24}$$

The first parameter of interest, the marginal effect with respect to X, is given by the partial derivative of the regression estimates of (24):

$$\frac{dE[RIF(H, F_Y; v^I)|X = x]}{dx} = \frac{d\lambda(X, \in)}{dx} \tag{25}$$

The second parameter of interest is the unconditional I partial effect. For a continuous covariate, this captures the response of I to a small location shift in the covariate (unconditional on the other covariates). For a binary covariate, this captures the response of I to marginal changes in the conditional distribution of the binary covariate *given* the other covariates. The $k \times 1$ vector of unconditional I partial effects, denoted as $\gamma(v^I)$, is a vector of average partial derivatives expressed as:

$$\gamma(v^I) = \int_{-\infty}^{\infty} \frac{dE[RIF(H, F_Y; v^I)|X = x]}{dx} \cdot dF_X(x) = \int_{-\infty}^{\infty} \frac{d\lambda(X, \in)}{dx} \cdot dF_X(x) \tag{26}$$

The potential choice of regression methods one could use to model the conditional expectation of $RIF(h, F_Y(y); v^I)$ and recover these parameters is limitless, but the eventual choice will depend on the form one is willing to assume for the function $\lambda(.)$. Assuming $\lambda(.)$ to be linear and applying OLS to estimate the parameters, yields an estimator we refer to as RIF-I-OLS. We use RIF-I-OLS as our working example for illustration of the method, because it is both simple and attractive from an operational perspective. As is the case for standard OLS, the restriction to a linear in parameters functional form, still allows for a fairly flexible functional form by inclusion of non-linear or higher order transformations of the covariates.

[17] Noting that $F_{H,F_Y}(h, F_Y(y)) = \int F_{(H,F_Y)|X}(h, F_Y(y)|X = x) \cdot dF_X(x)$, which is substituted into the second equality.

4.2. RIF-I-OLS

RIF-I-OLS identifies our parameters of interest, the marginal effect and the unconditional *I* partial effect, under the following assumptions:

Additive linearity. Assuming a functional form linear in parameters with an additive error term for the regression model for the RIF of *I*, we may rewrite Eq. (24) as:

$$E[RIF(H, F_Y; \nu^I)|X = x] = X'\psi + \mu \tag{27}$$

Zero conditional mean. $E[\mu|X] = 0$. Assuming conditional mean independence of the error term means our coefficient estimates, ψ, have a meaningful interpretation.

Using ν^{AC} as an example, assuming a linear functional form implies the assumption that the sum of: health, the product of health and fractional rank, and the individual's position on the absolute concentration curve, can together be modelled as linear in parameters. As is the case for standard OLS, linearity implies that the marginal effects are constant along the distribution of *X* and the derivative of Eq. (27) with respect to the covariates *X* equals the coefficient ψ:

$$\frac{dE[RIF(H, F_Y; \nu^I)|X = x]}{dx} = \frac{d[X'\psi + \mu]}{dx} = \psi, \tag{28}$$

and the unconditional *I* partial effect equals ψ:

$$\gamma(\nu^I) = \int_{-\infty}^{\infty} \frac{d[X'\psi + \mu]}{dx} \cdot dF(x) = \psi \tag{29}$$

Thus, under the linearity and zero conditional mean assumptions, the marginal effect and the unconditional partial effect are the same and RIF regression is optimally estimated using OLS. The procedure of RIF-I-OLS first involves estimating the empirical RIF, as we outlined in the final part of Section 3. This yields empirical estimates of each individual's recentered influence on *I*. Then, using the empirical RIF as the dependent variable in an OLS regression we yield the unconditional *I* partial effects. In practical terms the distinction between the marginal effect and unconditional partial effect becomes important when one relaxes the linearity assumption. As RIF-I-OLS estimates are a first-order approximation of the effect of *X* on *I*, the unconditional *I* partial effect is a local effect estimate of a small change in *X*. That RIF-I-OLS is a local estimate implies that it should only be considered for relatively small changes. The definition of relatively small will depend on the empirical context, for example the degree to which the true functional form is non-linear and/or the importance of general equilibrium effects.

5. An empirical illustration of WDW decomposition and RIF-I-OLS

In this section we aim to empirically illustrate what the RIF function is, and how WDW decomposition and RIF-I-OLS compare in their interpretation. We also show how RIF-I-OLS is both a well-suited method for determining the causal effect of a covariate on *I* given a suitable identification strategy and a useful descriptive decomposition method when no causal inference can be made. The illustrative example presented here focuses on the effect of education on income-related health inequality controlling for age and gender and uses data on monozygotic ("identical") twins.

The data is a replica of the data used in Gerdtham et al. (2016). Performing a WDW decomposition, Gerdtham et al. (2016) find education to be significantly associated with a higher level of health and to significantly contribute to the level of inequality, however, this all but disappears when controlling for family and genetic fixed effects common to twin pairs using a twins differencing strategy. To see if these results hold subject to a theoretically less restrictive decomposition method, we extend the analysis by decomposing income-related health inequality using RIF-I-OLS. As in Gerdtham et al. (2016), we first apply a naïve selection on observables identification strategy using OLS and then a twin fixed effects identification strategy.[18] We use the former primarily to illustrate the difference in the interpretation of the results of the two methods but also because most decomposition studies tend to use OLS and even in this descriptive setting RIF-I-OLS has important advantages. We use the twin fixed effects identification strategy to highlight that RIF-I-OLS is well suited to reduced form causal impact analysis where RIF-I-OLS potentially has the most to offer. First, however, we introduce the data and illustrate the empirical RIF of *I* (focusing on EI in particular).

5.1. Data material

The data used in this empirical example is a subset from the Swedish Twin Registry consisting of respondents that took part in a telephone interview, including a question on self-assessed health, called Screening Across the Lifespan Twin study (SALT) conducted between the years 1998–2002. The final sample size includes 3328 twin pairs born between the years 1931–1958. The survey data is matched with registers from Statistics Sweden on annual taxable gross income (income from earnings, own business, parental leave benefits, unemployment insurance and sickness benefits) and education level. Register data should have relatively small measurement error, which is very important as measurement errors are magnified when differencing between twins, as we do here in the final part of this section. Income is measured as an average of gross income over ages 35–39 years.[19] The education variable is measured as years of schooling and ranges between 8 and 20 years of schooling.[20] To obtain a health measure appropriate for a rank dependent index, we

[18] We refer the reader to Gerdtham et al. (2016) for both an up-to-date discussion on the merits of twin design based studies in revealing the treatment effect of education and for more detailed discussion of the dataset and the twin based fixed effects methodology.

[19] This point is discussed further in Gerdtham et al. (2016).

[20] Years of schooling is imputed from register data using the highest educational degree obtained in the year 1990 as outlined in the appendix in Gerdtham et al. (2016).

Table 1
Variable descriptions, 1st moments and algorithm weights.

Variable	Description	Mean	Algorithm weight
Health	Health utility from TTO algorithm	0.916	
Health1	1 = Very Good Health (self assessed)	0.379	(Reference)
Health2	1 = Good Health (self assessed)	0.37	−0.0315
Health3	1 = Fair Health (self assessed)	0.169	−0.1414
Health4	1 = Poor Health (self assessed)	0.064	−0.3189
Health5	1 = Very Poor Health (self assessed)	0.018	−0.4817
Age4044	1= aged between 40 and 44 years	0.083	0.0109
Age4554	1= aged between 45 and 54 years	0.427	0.0179
Age5564	1= aged between 55 and 64 years	0.449	0.0235
Age6567	1= aged between 65 and 67 years	0.042	0.0193
Female	1 = female, 0 = male	0.551	0.0058
Schooling	Number of years in education	11.571	
Income	Gross income (35–39 years)[a]	199,145	
Constant		1	0.9589

Notes:
[a] Income is in 2010 prices, SEK.

cardinalise the categorical self-rated health measure using a linear algorithm from Burström et al. (2014) (see model 3, supplementary table 8 of their paper) that transforms self-rated health to a time trade-off (TTO) quality of life utility value. The algorithm values taken from Burström et al. (2014) are shown in column 4 of Table 1.[21] Summary statistics are also presented in Table 1.[22]

5.2. Empirical estimation of the RIF

The Erreygers index (EI) for estimated health utility scores is 0.03 (Table 2) indicating that higher health utility is more concentrated amongst the rich. The empirical RIF for EI of health utility score ranked by income is calculated as explained in Section 3 and the result is shown in a scatter plot in Fig. 1. Each scatter point in Fig. 1 is an individual's recentered influence value of EI plotted against their income rank. If an individual were to be removed from the sample, the influence on the statistic would be minus that individual's RIF value weighted by the inverse of the sample size. The figure shows that those at the extreme ends of the income distribution have greatest influence on the EI. This is similar to the findings in Monti (1991) for income concentration as measured by the Gini (a univariate rank dependent index): individuals whose income value is at the extremes of the income distribution have greatest influence on the Gini. As the EI is a bivariate index, health, in addition to the ranking variable, affects the degree of influence an individual has on EI. In this particular example those with very poor health (squares) *and* income levels at the extreme ends of the distribution are the ones with the greatest influence on EI.[23] This result is important to note for researchers and policy makers and whilst it may be known to some, the RIF allows it to be shown as a figure. Researchers estimating a rank dependent index as a measure of socioeconomic related health inequality need to be sure that the observations with the largest influence on the statistic are not miss-codings. Policy makers may want to focus attention towards those individuals they can help with most influence on inequality – the extreme poor with poor health in this instance.

5.3. Interpretation of RIF decomposition and comparison with WDW decomposition

To provide more information on the characteristics of the individuals that are influencing the statistic, either positively or negatively and to a greater or lesser extent, one may plot the RIF against another variable or turn to the RIF regression method.[24] Table 2 reports descriptive decomposition results of WDW decomposition of EI, and RIF-EI-OLS decomposition, in addition to results for RIF-I-OLS for AC, ARCI, SRCI, and WI alongside standard mean regression. In a descriptive RIF-I-OLS decomposition the estimated coefficients $\hat{\psi}$ may be interpreted as an association between the covariate and the influence on I, providing valuable information as to which groups of individuals influence the inequality index. If we (naively) assume the error term, ϵ, and covariates, X, are independent having controlled for selection on observables then the RIF-I-OLS parameter $\hat{\psi}$ identifies the (causal) unconditional I partial effects of a shift in the distribution of X on I. Thus, interpretation of $\hat{\psi}$ is similar to the interpretation of the coefficients in standard mean regression (the results of which are shown in column (1) of Table 2). Indeed, RIF decomposition of the mean of health, assuming a health function linear in parameters, is standard OLS (Firpo et al., 2009).

In the decomposition analysis, years of schooling enters the model as an explanatory variable alongside age, gender and interview year dummies (because each twin was not necessarily interviewed at the same time). We only control for age and gender because these variables are exogenous and predetermined before school was attended thereby avoiding the issue of "bad controls" (see 3.2.3 of Angrist and Pischke, 2008). Proceeding in this way allows us to interpret the education coefficient in a meaningful way. Even for descriptive analysis, care should be taken not to introduce mediators that may complicate interpretation. It is for this reason we do not include employment

[21] Health economists often value health states of people by the TTO method where respondents value quality of life in relation to length of life; respondents are asked to imagine living in a given state of health for (typically) ten years, and then to state the shorter amount of time in full health which makes them indifferent between the two options (Drummond et al., 2005). Reference categories are very good self rated health, age 18–24 years and female.

[22] Gerdtham et al. (2016) show that the Swedish Twin Registry data used here is fairly representative of Sweden's population more widely, which otherwise may be a concern for twin based datasets.

[23] The bivariate rank dependent index gives zero weight to those at the median rank (which is the mean ranking value) and increasing weight to those further away from the median. Health values at the mean (in this case those with good health or thereabouts) also have zero impact. This is because the covariance is driven by those furthest away from the mean of the two variables.

[24] One could for example plot a Lowess curve of the RIF and explanatory variable to visually assess a potential relationship and any functional form assumption. We did this for education but there was no real relationship by years of education and therefore do not report the results here.

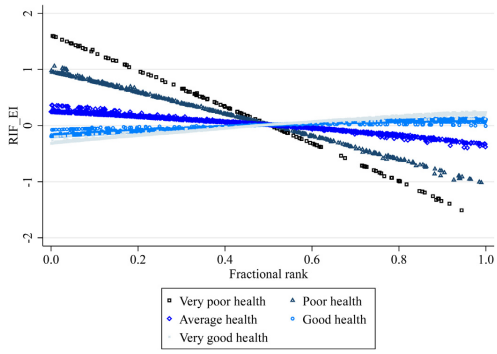

Fig. 1. Scatter plot of individual RIF of EI values plotted against individual's fractional income rank. Each scatter point represents an individual's recentered influence on EI plotted against their fractional income rank by health value.

Table 2
RIF-I-OLS and WDW decomposition estimates of years of schooling, age and gender on income related health inequality.

	OLS	RIF-I-OLS decomposition					WDW EI–OLS decomposition	
Statistic	Health	AC	EI	ARCI	SRCI	WI	Contribution	% contribution
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Years schooling	0.005***	0.000	0.001	0.000	0.009***	0.010***	0.008***	0.282***
	(0.000)	(0.000)	(0.001)	(0.000)	(0.003)	(0.003)	(0.001)	(0.035)
Age	−0.000*	0.000	0.001	0.000	0.002	0.003	0.001	0.022
	(0.000)	(0.000)	(0.001)	(0.000)	(0.002)	(0.002)	(0.000)	(0.014)
Male	0.009***	0.003***	0.014***	0.004**	0.051***	0.054***	0.006***	0.212***
	(0.002)	(0.001)	(0.005)	(0.001)	(0.016)	(0.017)	(0.002)	(0.050)
Constant	0.930***	−0.016	−0.066	−0.018	−0.181	−0.199		
	(0.018)	(0.015)	(0.058)	(0.016)	(0.167)	(0.183)		
Mean of RIF	0.916	0.007	0.030	0.008	0.089	0.098		
Observations	6656	6656	6656	6656	6656	6656	6656	6656
WTP FE	NO	NO	NO	NO	NO	NO	NO	NO

Notes: Each column represents a separate decomposition. Column 1 is OLS of the health variable, which is RIF decomposition of the mean assuming linearity in parameters and is optimally estimated using OLS. AC = absolute concentration index, EI = Erreygers Index, ARCI = Attainment relative concentration index, SRCI = Shortfall relative concentration index, WI = Wagstaff Index, WDW = Wagstaff, Van Doorslaer and Watanabe (2003) decomposition. The mean of RIF is the value of the statistic being decomposed. All decompositions control for year of interview fixed effects. Robust standard errors in parenthesis for RIF-mean-OLS and bootstrap standard errors in parenthesis for RIF-I-OLS, 999 repetitions with replacement. Bootstrap standard errors are calculated by bootstrapping the whole procedure (Both for RIF and WDW procedures). Testing null of the coefficient/contributions/% contribution: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

status, for example, as an explanatory variable. Employment status predicts health but it is also an outcome variable affected by education. Its inclusion complicates the interpretation of the education coefficient. The coefficient estimates from RIF-EI-OLS in column (3) of Table 2 suggest, if interpreted as the unconditional *I* partial effect, that if one made an equal marginal increase to the number of years of education for everyone in the population, this would have no discernible effect on EI. There also appears to be no age profile regarding EI.

Importantly and in contrast to the contribution estimates of WDW decomposition, RIF-I-OLS identifies the effect of the covariates *X* on the full statistic. That is, the parameter estimate $\hat{\psi}$ captures the effect of the covariates on the *product* of the AC (which is two times the covariance of the level of health and fractional rank) *and* the weighting function $\omega_I(h)$. The parameter estimates $\hat{\psi}$ presented in Columns 2–6 of Table 2 also vary between rank dependent indices depending on the weighting function. Education is found to be significantly associated with the RIF of WI and SRCI, but not with the RIF of AC, EI, or ARCI. That is, more educated individuals have larger influence on the inequality index when measured as WI and SRCI, but not when the AC, EI, or ARCI are considered. This highlights an important issue. The differences in weighting functions, and hence value judgements, among the inequality indices can also lead to important differences in the decomposition results. In this particular example the judgement of whether to consider attainment relative inequality or shortfall relative inequality has bearing on whether education has a potential impact.[25] It is worth noting that it is possible to identify the effect of a particular weighting function by comparing decomposition results for *I* with decomposition results for AC: AC has a constant weighting function, so any differences in the (standardised) decomposition results compared to those for the AC will be due to the weighting function.

[25] Note that the results divide the indices into two groups. On the one hand EI, AC, ARCI, and on the other hand WI and SRCI. This is a consequence of the high mean of the health utility index.

The last two columns in Table 2 report the results from WDW decomposition of EI. The interpretation is different from any standard form of mean decomposition and to RIF-I-OLS decomposition. The marginal interpretation of WDW ($\iota^{\omega_I}(F_H)\beta_k$) (as described in Eq. (10)) implies that a change in the covariance of the covariate and the socioeconomic rank (due to a change in the distribution of the covariate) affects EI by a factor of 4×0.005, with regard to education in this application. It also implies that a change in beta, the health return to the covariate of interest, affects EI by a factor of $4 * 2cov(X, F_Y)$. The procedure also summarises I as a summation of the contribution of each covariate, where these are the covariate-rank covariances weighted by a linear health-covariate correlation. Following the standard practice, we report the WDW decomposition results as contributions from the covariates in levels and percentagewise contributions of the total index. The results suggest that about 28% of the income-related inequalities in health is due to income-related inequalities in education. The contribution is statistically significant suggesting that eliminating income-related inequalities in education might reduce the EI of health, *assuming no change in the ranking variable and a linear health function*.[26] As the procedure ignores the potential impact of the covariates on the weighting function, $\iota^{\omega_I}(F_H)$, the percentagewise "contributions" are the same no matter the choice of I (only levels vary with the weighting function). That is, WDW decomposition of any inequality measure implicitly decomposes an absolute index such as EI or AC.

Whilst the results of the two decomposition methods are not directly comparable, as they have different units of measurement, they nevertheless draw contrasting conclusions. WDW decomposition finds a significant contribution due to education whereas RIF for the EI or AC – the most comparable basis, as WDW decomposition holds the weighting function constant and EI and AC have constant weighting functions – finds no significant effect of education. In this particular case we are focussing on a covariate that is well known to causally impact the level of income. Indeed using within twin pair fixed effects on slightly different sample of the same twins population Isacsson (1999) found a significant impact of years of schooling on income and this is a generally accepted finding (Card, 1999). It is hard to interpret the results of WDW decomposition when one knows that a key identifying assumption does not hold (rank ignorability). This is not an uncommon situation; most covariates that impact health also impact the ranking variable. It is our view that the results obtained from RIF regression in this kind of situation are much clearer in their interpretation. RIF-I-OLS results allow us to conclude that there is no *local* association of education with absolute income related health inequality, but there is a *local* association with relative short-falls income related health inequality.

5.4. The causal effect of education on income-related health inequality

In the previous section, our identification of the unconditional partial effects did not use twin fixed effects but instead (naïvely) relied on selection on observables to satisfy the assumption that the errors are independent of the covariates. To highlight the importance of causal inference in decomposition analysis we now apply a twins differencing strategy that allows unobserved heterogeneity common between twins to be differenced out. That is, we control for factors such as innate ability and early life factors common to both twins, which may invalidate the exogeneity assumption and yield biased parameter estimates. In the case of income-related health inequality the concern is specifically that this unobserved heterogeneity may be correlated with education and the weighted *covariance* of health and income rank.

To formally derive the within twin pair (WTP) fixed effect decomposition, we denote the RIF values of the jth twin pair, $RIF(h, F_Y(y); I)_{1j}$ and $RIF(h, F_Y(y); I)_{2j}$. Further, we let u_j denote unobserved factors that vary between twin pairs but not within pairs, such as genetic characteristics and certain early life environmental factors and e_{1j} and e_{2j} denote unobserved factors specific to each twin. Assuming a linear functional form for the RIF, we may write these as:

$$RIF(h, F_Y(y); I)_{1j} = X'_{1j}\psi + u_j + e_{1j} \tag{30}$$

$$RIF(h, F_Y(y); I)_{2j} = X'_{2j}\psi + u_j + e_{2j} \tag{31}$$

where X_{1j} is a $k \times n$ matrix of covariates for the first twin in the twin pair j, X_{2j} is for the second twin in the twin pair and ψ is a $k \times 1$ vector of unconditional I partial effects. Taking the difference yields the WTP estimator:

$$RIF(h, F_Y(y); I)_{1j} - RIF(h, F_Y(y); I)_{2j} = \left(X_{1j} - X_{2j}\right)' \psi_{WTP} + e_{1j} - e_{2j} \tag{32}$$

where ψ_{WTP} is the WTP estimator of the effect of education. The unobserved factors that are common to both twins such as genetics or environmental exposure captured by u_j will be differenced out of the equation yielding an unbiased OLS-estimator of $\hat{\psi}$ (given that these are the only sources of unobserved heterogeneity).[27] Applying the WTP approach to the RIF of EI using OLS yields the RIF-EI-WTPFE estimator.

Table 3 reports the monozygotic WTP fixed effects results for EI, AC, CI, and WI alongside standard mean fixed effects regression and WDW decomposition. The results for the RIF-I-WTPFE decomposition suggest that if one gave an equal marginal increase in the number of years of education to everyone in the population, this would have no discernible effect on any measure of I, nor the mean. It therefore appears that either education has no effect on income-related health inequality, or possibly better put: the variation in education that exists under an extensive egalitarian education system cannot explain the observed income-related health inequality.

[26] In the case of EI, the weighting function is a constant and therefore the condition that the weighting function is constant is not binding in this case. However WDW decomposition of CI and WI would also assume that the weighting function is a constant, which it is not.

[27] For a further discussion of potential sources of unobserved heterogeneity see Gerdtham et al. (2016).

Table 3
RIF-I-WTPFE and WDW-WTPFE decomposition estimates of years of schooling on income related health inequality.

	OLS-WTPFE	RIF-I-WTPFE decomposition					WDW -EI- WTPFE decomposition	
Statistic	Health (1)	AC (2)	EI (3)	ARCI (4)	SRCI (5)	WI (6)	Contribution (7)	% contribution (8)
Years schooling	0.001	−0.000	−0.002	−0.000	−0.004	−0.004	0.001	0.05
	(0.001)	(0.001)	(0.002)	(0.001)	(0.009)	(0.007)	(0.002)	(0.058)
Constant	0.930***	0.020	0.081	0.022	0.256	0.277		
	(0.034)	(0.029)	(0.117)	(0.032)	(0.452)	(0.401)		
Mean of RIF	0.916	0.007	0.030	0.008	0.089	0.098		
Observations	6656	6656	6656	6656	6656	6656	6656	6656
WTP FE	YES	YES	YES	YES	YES	YES	YES	YES

Notes: Each column represents a separate decomposition. Column 1 is simply OLS with FE of the health variable. AC = absolute concentration index, EI = Erreygers Index, ARCI = Attainment relative concentration index, SRCI = Shortfall relative concentration index, WI = Wagstaff Index, WDW= Wagstaff, Van Doorslaer and Watanabe (2003) decomposition. The mean of RIF is the value of the statistic being decomposed. All decompositions control for year of interview fixed effects. Robust standard errors in parenthesis for OLS-WTPFE and bootstrap standard errors in parenthesis for RIF-I-WTPFE and WDW, 999 repetitions with replacement. Bootstrap standard errors are calculated by bootstrapping the whole procedure (Both for RIF and WDW procedures). Testing the null of the coefficient/contributions/% contribution: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

6. Discussion

Having introduced and illustrated both the WDW decomposition and RIF-I-OLS decomposition, we now compare the two approaches by summarising the underlying identifying assumptions and differences in interpretation. For clarity, we start by giving a side-by-side comparison of the identifying assumptions of the two approaches.

WDW identifying assumptions:
I. The determinants of health do not determine rank.
II. The determinants of health do not determine the weighting function.
III. Health can be modelled as a function linear in variables X and an error term.
IV. Exogeneity: The errors from the health regression have zero conditional mean.

RIF-I-OLS identifying assumptions:
I. I is differentiable and the differential is bounded.
II. $RIF(h, F_Y(y); I)$ can be modelled as a linear in parameters function of X and an additive error term
III. Exogeneity: The errors from the RIF OLS regression have zero conditional mean.

It is clear from the comparison that RIF-I-OLS requires fewer, and less restrictive, identifying assumptions than WDW decomposition. The first condition for RIF-I-OLS holds as shown in the proof. Exogeneity is of huge importance for causal inference and is common to both methods–but both methods may be used as descriptive exercises without this assumption. Linearity is also common to both methods, but this is an assumption often applied in wider empirical practice and the immediate available evidence suggests this is not a particularly limiting assumption to impose (Van de Poel et al., 2009, Van Doorslaer et al., 2004b, Firpo et al., 2009). This fits with the perceived wisdom that OLS generally provides a good approximation. However, the remaining identifying assumptions of WDW decomposition (rank and weighting function ignorability) are often restrictive as illustrated in this paper and in Erreygers and Kessels (2013). When concern lies with covariates that are known to impact on the ranking variable and the weighting function WDW is likely to yield biased results, which is not true for RIF-I-OLS.

An additional benefit of RIF regression is that it is familiar in its interpretation. The results in Tables 2 and 3 highlight how education's effect on income-related health inequality as estimated by RIF-I-OLS can be shown alongside its effect on mean health in a consistent manner. Similar to mean OLS regression coefficients, the RIF-I-OLS coefficients should be interpreted as how a marginal shift in the distribution of a covariate, e.g., education, influences the inequality index. The interpretation of RIF-OLS estimates and mean OLS regression estimates are similar because RIF-OLS estimates of the mean are in fact exactly the same as mean OLS estimates. We can use this fact to illustrate the difference between WDW decomposition and RIF-I-OLS. WDW is based on a mean OLS regression of health. The contribution of covariate k in WDW decomposition corresponds to its coefficient in an OLS regression on the mean of health - weighted by the weighting function and twice the covariance between covariate k and rank, i.e., $\beta_k \nu^{\omega_I}(F_H) 2cov(X_k, F_Y)$. WDW is therefore equivalent to a RIF-OLS decomposition of the mean of health weighted by two functions that are themselves not decomposed. In comparison, RIF-I-OLS estimates the impact of covariates on the index itself, the weighted covariance between health and rank, and therefore decomposes all parts of the index.

As a result of not imposing weighting function ignorability, RIF-I-OLS has the benefit that it allows the analyst to assess the impact of covariates on different forms of I. RIF-I-OLS includes the impact of the covariates on the weighting function and therefore the importance of the covariates may differ between particular indices. Indeed, we illustrate this in our empirical application based on the simple correlations (not WTP fixed effects), where we find that education had no association with the AC, EI and ARCI, but had a significant association with WI and SRCI. Whereas WDW decomposition only allows for decomposing an absolute index, RIF-I-OLS allows researchers to explore how the policy impacts on the level of inequality and how this differs depending on the particular value judgement and hence the particular inequality index policy makers sympathise with. We view this as a necessary part of any inequality analysis because there is no consensus as to which inequality measure is preferred.

In this paper we have highlighted the identifying assumptions of the WDW decomposition method and shown they rarely hold in practice and that this makes interpretation difficult. It appears that the central issue with WDW decomposition is that the parameter of interest is not clear and consequently neither are the conditions under which it will be identified. As Erreygers and Kessels (2013) implicitly show, this results in many potential decomposition methods that can yield very different results. Erreygers and Kessels (2013) conclude with a warning that until it is understood which form of WDW type of decomposition is preferred, all decomposition methods

should be used with caution. It is our view that this conclusion should be made a little more explicit and that decomposition methods that *are unclear as to what they estimate and what the necessary identifying assumptions are* should be used with caution. Our approach differs from currently available decomposition methods for bivariate rank dependent indices: first, we are clear as to what our parameter of interest is (the unconditional *I* partial effect); second, we derive a decomposition method that yields this parameter, based on RIF regression.

On a much simpler level RIF also allows useful graphical presentation of the data. In the empirical example we showed who influence the statistic most – those with very poor health and very low income. Those with very good health have very little impact on the statistic no matter their income rank. This may not be immediately obvious to practitioners and policy makers and is a great way to illustrate who would have greatest impact if targeted. RIF also allows the statistic to be plotted against another covariate and simple bivariate plots can show any potential relation that may be of interest.

The RIF approach does have its limitations; the leading one is that it is a local approximation. It is therefore not reasonable to calculate percentagewise contributions using RIF regression. The usefulness of a local estimate should, however, be placed into the larger context of the overall aims of decomposition analysis and the available alternative approaches. The approach suggested by Kessels and Erreygers (2015) potentially solves the rank ignorability issue but ignores the weighting function assumption. It also requires a structural model, but if one were to have a structural model to hand it may not be preferable to decompose a bivariate index of health inequality. Instead, Fleurbaey and Schokkaert (2009) convincingly make the case for a structural model approach to be used for analysing fair and unfair inequalities in health. As a road map for the health inequality literature this may very well be the goal or ideal that we all should be aiming for. However, if no structural model is available or feasible it may still be of huge interest how a policy change (which is most often a marginal one) impacts both average health and health inequality. RIF of *I* regression allows this reduced form type of analysis to be made without the need for restrictive assumptions making it a useful addition to a health economist's toolkit.

7. Conclusion

In this paper we have summarised the literature that has identified the identifying assumptions required by WDW decomposition and presented evidence that these assumptions can be important for the decomposition results. Causal analysis using WDW decomposition is therefore troublesome. Even when WDW decomposition is interpreted purely as a descriptive accounting exercise the evidence suggests that results from the WDW decomposition will be difficult to interpret if one is concerned about the rank ignorability and weighting function assumptions. We have introduced an alternative rank dependent index decomposition method that simultaneously relaxes the rank and weighting function ignorability assumptions. This alternative is based on a RIF regression. We have extended the RIF concept from a univariate setting to a general bivariate rank dependent index, providing a method that yields the unconditional *I* partial effect of a shift in the distribution of X on the inequality index and has strong links to the program evaluation literature. This new decomposition approach is simple to estimate and the interpretation resembles that of standard conditional mean analysis. Our empirical application using the Swedish Twin Registry found a discrepancy between the results of the two methods: WDW decomposition finds a significant association of education and income related health inequality, RIF regression finds no such association. In this example we know that education impacts the ranking variable (income) and therefore interpretation is muddied by the assumptions imposed by WDW decomposition. In comparison, interpreting the results from RIF regression is much clearer and the results suggest there is no *local* impact of education on income related health inequality. In an attempt to illustrate RIF-I-OLS's close link to the treatment effects literature, we used linear WTP fixed effects and found little evidence that (twin differences in) education causally impact income-related health inequality in Sweden.

Finally, it is worth noting that the usefulness of the RIF regression goes beyond the estimation of unconditional *I* partial effects using OLS. One can for example use instrumental variables for endogenous variables by adding control functions as per Rothe (2010) to obtain consistent estimates of the marginal effects. RIF regression also allows Oaxaca-blinder type decompositions of between group/time differences to be decomposed for statistics other than the mean under some further identifying assumptions (Fortin et al., 2011). We have not discussed these in any great detail but they highlight the potential of our suggested decomposition method and its applicability to a wide range of empirical questions.

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Appendix A. Derivation of the *RIF* for a general rank dependent index (*I*), the IF for the AC and the RIFs for AC, EI, CI, ARCI, SRCI and WI.

Proposition 1. *Let* $\nu^{I}(F_{H,F_Y}) = \nu^{\omega_I}(F_H)\nu^{AC}(F_{H,F_Y})$ *be a general rank dependent index, the AC be defined as* $\nu^{AC}(F_{H,F_Y}) = 2cov(H, F_Y)$ *and* F_{H,F_Y} *be the joint CDF of H and* F_Y *with corresponding pdf denoted as* f_{H,F_Y}*. Then the RIF for* $\nu^{I}(F_{H,F_Y})$ *is given by:*

$$RIF(h, F_Y(y); \nu^I) = \nu^I(F_{H,F_Y}) + IF(h; \nu^{\omega_I}) * \nu^{AC}(F_{H,F_Y}) + \nu^{\omega_I}(F_H) * IF(h, F_Y(y); \nu^{AC}),$$

where $IF(h; \nu^{\omega_I})$ *denotes the IF of the weighting function for I and* $IF(h, F_Y(y); \nu^{AC}) = -2\nu^{AC}(F_{H,F_Y}) + \mu_H - h + 2hF_Y(y) - 2\int^y \int^{+\infty} hf_{H,F_Y} dhdF_Y(z)$ *denotes the IF for AC.*

Proof. To show $RIF(h, F_Y(y); \nu^I) = \nu^I(F_{H,F_Y}) + IF(h; \nu^{\omega_I}) * \nu^{AC}(F_{H,F_Y}) + \nu^{\omega_I}(F_H) * IF(h, F_Y(y); \nu^{AC})$, we first apply the definition of the IF given by Eq. (17) to *I* yielding:

$$IF(h, F_Y(y); \nu^I) = \frac{d}{d\varepsilon}\left[\nu^{\omega_I}(F_H)\nu^{AC}(F_{H,F_Y})\right]\Big|_{\varepsilon=0} \tag{A1}$$

Applying the product rule to Eq. (A1) yields:

$$IF(h, F_Y(y); \nu^I) = IF(h; \nu^{\omega_I}) * \nu^{AC}(F_{H,F_Y}) + \nu^{\omega_I}(F_H) * IF(h, F_Y(y); \nu^{AC}) \tag{A2}$$

As per Eq. (18), adding the functional $\nu^I(F_{H,F_Y})$ to Eq. (A2) yields the RIF for $\nu^I(F_{H,F_Y})$.

To show that $IF(h, F_Y(y); \nu^{AC}) = -2\nu^{AC}(F_{H,F_Y}) + \mu_H - h + 2hF_Y(y) - 2\int^y \int^{+\infty} hf_{H,F_Y} dhdF_Y(z)$, we first note that the absolute concentration index can be written as:

$$\nu^{AC}(F_{H,F_Y}) = 2cov(H, F_Y) = 2\int hF_Y dF_{H,F_Y} - 2\int hdF_{H,\infty}\int F_Y dF_{\infty,F_Y} \tag{A3}$$

Eq. (A3) states that AC is a functional of the joint probability distribution F_{H,F_Y} and the probability distribution F_Y. Substituting $\nu^{AC}(G_{h,F_Y})$ and $\nu^{AC}(F_{H,F_Y})$ for $\nu^I(G_{h,F_Y})$ and $\nu^I(F_{H,F_Y})$ in the formula for the bivariate IF given by Eq. (17) yields:

$$IF(h, F_Y(y); \nu^{AC}) = lim_{\varepsilon\to 0}\frac{2\int hG_y dG_{h,F_Y(y)} - \int hdG_{h,\infty}\int G_y dG_{\infty,F_Y(y)} - cov(H, F_Y)}{\varepsilon} \tag{A4}$$

Substituting G_y as defined in Eq. (12) and $G_{h,F_Y(y)}$ as defined in Eq. (15) into Eq. (A4) yields:

$$IF(h, F_Y(y); \nu^{AC}) = lim_{\varepsilon\to 0} 2\frac{\left[\int h((1-\varepsilon)F_Y + \varepsilon\delta_y)d((1-\varepsilon)F_{H,F_Y} + \varepsilon\delta_{h,F_Y(y)}) - \int hd((1-\varepsilon)F_{H,\infty} + \varepsilon\delta_{h,\infty})\int((1-\varepsilon)F_Y + \varepsilon\delta_y)d((1-\varepsilon)F_{\infty,F_Y} + \varepsilon\delta_{\infty,F_Y(y)}) - cov(H, F_Y)\right]}{\varepsilon} \tag{A5}$$

Which after taking the limit and re-arranging yields:

$$\begin{aligned} IF(h, F_Y(y); \nu^{AC}) = 2\Bigg[&-2\left(\int hF_Y dF_{H,F_Y} - \int hdF_{H,\infty}\int F_Y dF_{\infty,F_Y}\right) + \int hdF_{H,\infty}\int F_Y dF_{\infty,F_Y} - \int hd\delta_{h,\infty}\int F_Y dF_{\infty,FY} \\ &+ \int hF_Y d\delta_{h,F_Y(y)} - \int hdF_{H,\infty}\int F_Y d\delta_{\infty,F_Y(y)} + \int h\delta_y dF_{H,F_Y} - \int hdF_{H,\infty}\int \delta_y dF_{\infty,F_y}\Bigg] \end{aligned} \tag{A6}$$

Term by term Eq. (A6) is equal to:

$$-2\int hF_Y dF_{H,F_Y} + 2\int hdF_{H,\infty}\int F_Y dF_{\infty,F_Y} = -\nu^{AC}(F_{H,F_Y}), \tag{A7}$$

$$\int hdF_{H,\infty}\int F_Y dF_{\infty,F_Y} = \frac{\mu_H}{2}, \tag{A8}$$

$$-\int hd\delta_{h,\infty}\int F_Y dF_{\infty,F_Y} = -\frac{h}{2}, \tag{A9}$$

$$\int hF_Y d\delta_{h,F_Y(y)} = hF_Y(y), \tag{A10}$$

$$-\int hdF_{H,\infty}\int F_Y d\delta_{\infty,F_Y(y)} = -\mu_H F_Y(y), \tag{A11}$$

$$\begin{aligned} -\int hdF_{H,\infty}\int \delta_y dF_{\infty,F_Y} &= -\mu_H\int^{+\infty}\int^{+\infty}\delta_y f_{\infty,F_Y} dhdF_Y(y) = -\mu_H\int_y^{+\infty}\int^{+\infty} 1f_{\infty,F_Y} dhdF_Y(z) = -\mu_H\int^{+\infty}\int^{+\infty} 1f_{\infty,F_Y} dhdF_Y(y) \\ &+ \mu_H\int^y\int^{+\infty} 1f_{\infty,F_Y} dhdF_Y(z) = -\mu_H + \mu_H F_Y(y), \end{aligned} \tag{A12}$$

$$\begin{aligned} \int h\delta_y dF_{H,F_Y} &= \int^{+\infty}\int^{+\infty} h\delta_y f_{H,F_Y} dhdF_Y(y) = \int_y^{+\infty}\int^{+\infty} h\delta_y f_{H,F_Y} dhdF_Y(z) = \int^{+\infty}\int^{+\infty} hf_{H,F_y} dhdF_Y(y) - \int^y\int^{+\infty} hf_{H,F_Y} dhdF_Y(z) \\ &= -\mu_H - \int^y\int^{+\infty} hf_{H,F_Y} dhdF_Y(z). \end{aligned} \tag{A13}$$

Together these yield:

$$IF(h, F_Y(y); \nu^{AC}) = -2\nu^{AC}(F_{H,F_Y}) + \mu_H - h + 2hF_Y(y) - 2\int^{y}\int^{+\infty} hf_{H,F_Y}\,dh dF_Y(z), \tag{A14}$$

This completes the proof. □

Corollary 1. *The RIFs for the AC, EI, CI, ARCI, SRCI and the WI are given by:*

$$RIF(h, F_Y(y); \nu^{AC}) = \nu^{AC}(F_{H,F_Y}) + IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{EI}) = \nu^{EI}(F_{H,F_Y}) + \frac{4}{b_H - a_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{CI}) = \nu^{CI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{\mu_H^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{ARCI}) = \nu^{ARCI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{(\mu_H - a_H)^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H - a_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{SRCI}) = \nu^{ARCI}(F_{H,F_Y}) + \frac{(-\mu_H + h)}{(b_H - \mu_H)^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{b_H - \mu_H} IF(h, F_Y(y); \nu^{AC})$$

$$RIF(h, F_Y(y); \nu^{WI}) = \nu^{WI}(F_{H,F_Y}) + \frac{-(b_H - a_H)[(b_H + a_H - 2\mu_H)(h - \mu_H)]}{((b_H - \mu_H)(\mu_H - a_H))^2} * \nu^{AC}(F_{H,F_Y}) + \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)} IF(h, F_Y(y); \nu^{AC})$$

Proof. To show the result of Corollary 1, Proposition 1 states the IFs for the weighting functions for the AC, EI, CI, ARCI, SRCI and WI need to be calculated. The weighting functions for both the AC and EI are constants, therefore the IFs for their weighting functions will be zero and we can plug in straight away the functions we need into the formula for the RIF of I. The IF for the CI weighting function is:

$$IF(h; \omega^{CI}) = \frac{d}{d\varepsilon} \frac{1}{\left[(1-\varepsilon)\int h dF_H + \varepsilon h\right]} - \frac{1}{-\int h dF_H}|_{\varepsilon=0}. \tag{A15}$$

Differentiating Eq. (A15) and taking the limit with respect to ε and noting that $\int h dF_H = \mu_H$ gives us:

$$IF(h; \omega^{CI}) = \frac{\int h dF_H - h}{\int h dF_H \int h dF_H} = \frac{(\mu_H - h)}{\mu_H^2} \tag{A16}$$

Substituting Eq. (A16) into the formula for the RIF for I yields the RIF for CI:

$$RIF(h, F_Y(y); \nu^{CI}) = \nu^{CI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{\mu_H^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H} IF(h, F_Y(y); \nu^{AC}) \tag{A17}$$

The IF for the ARCI weighting function is:

$$IF(h; \omega^{ARCI}) = \frac{d}{d\varepsilon} \frac{1}{\left[(1-\varepsilon)\int (h - a_H) dF_H + \varepsilon (h - a_H)\right]} - \frac{1}{-\int (h - a_H) dF_H}|_{\varepsilon=0} \tag{A18}$$

Differentiating Eq. (A18) and taking the limit with respect to ε gives us:

$$IF(h; \omega^{ARCI}) = \frac{\int (h - a_H) dF_H - (h - a_H)}{\int (h - a_H) dF_H \int (h - a_H) dF_H} = \frac{(\mu_H - h)}{(\mu_H - a_H)^2} \tag{A19}$$

Substituting Eq. (A19) into the formula for the RIF for I yields the RIF for ARCI:

$$RIF(h, F_Y(y); \nu^{ARCI}) = \nu^{ARCI}(F_{H,F_Y}) + \frac{(\mu_H - h)}{(\mu_H - a_H)^2} \nu^{AC}(F_{H,F_Y}) + \frac{1}{\mu_H - a_H} IF(h, F_Y(y); \nu^{AC}) \tag{A20}$$

Following a similar argument as for ARCI, the IF for the SRCI is given by:

$$IF(h; \omega^{SRCI}) = \frac{\int (b_H - h) dF_H - (b_H - h)}{\int (b_H - h) dF_H \int (b_H - h) dF_H} = \frac{(-\mu_H + h)}{(b_H - \mu_H)^2} \tag{A21}$$

Substituting Eq. (A21) into the formula for the RIF for I yields the RIF for SRCI:

$$RIF(h, F_Y(y); \nu^{SRCI}) = \nu^{SRCI}(F_{H,F_Y}) + \frac{(-\mu_H + h)}{(b_H - \mu_H)^2} * \nu^{AC}(F_{H,F_Y}) + \frac{1}{b_H - \mu_H} IF(h, F_Y(y); \nu^{AC}) \tag{A22}$$

The IF for the WI weighting function is given by:

$$IF(h;\omega^{WI}) = \frac{d}{d\varepsilon}\left[\frac{b_H - a_H}{\left(b_H - \int hd\left((1-\varepsilon)F_H + \varepsilon\delta\right)\right)\left(\int hd\left((1-\varepsilon)F_H + \varepsilon\delta\right) - a_H\right)} - \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)}\right]|_{\varepsilon=0} \tag{A23}$$

Expanding gives us:

$$IF(h;\omega^{WI}) = \frac{d}{d\varepsilon}\left[\frac{b_H - a_H}{(b_H(1-\varepsilon)\mu_H + b_H\varepsilon h - b_H a_H - (1-\varepsilon)^2\mu_H^2 - (1-\varepsilon)\varepsilon h\mu_H + (1-\varepsilon)a_H\mu_H - (1-\varepsilon)\varepsilon h\mu_H - \varepsilon^2 h^2 + \varepsilon a_H h)} - \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)}\right]|_{\varepsilon=0} \tag{A24}$$

Differentiating with respect to ε and taking the limit w.r.t. ε yields:

$$IF(h;\omega^{WI}) = \frac{-(b_H - a_H)[(b_H + a_H - 2\mu_H)(h - \mu_H)]}{((b_H - \mu_H)(\mu_H - a_H))^2} \tag{A25}$$

Substituting Eq. (A25) into the formula for the RIF for I yields the RIF for WI:

$$RIF(h, F_Y(y); \nu^{WI}) = \nu^{WI}(F_{H,F_Y}) + \frac{-(b_H - a_H)[(b_H + a_H - 2\mu_H)(h - \mu_H)]}{((b_H - \mu_H)(\mu_H - a_H))^2}\nu^{AC}(F_{H,F_Y}) + \frac{b_H - a_H}{(b_H - \mu_H)(\mu_H - a_H)}IF(h, F_Y(y); \nu^{AC}), \tag{A26}$$

This completes the proof. □

Appendix B. Linking proposition 1 and corollary 1 to Essama-Nssah and Lambert (2012) and Firpo et al. (2007)

The (R)IF for a univariate rank dependent index, the Gini index (a measure of the concentration of one variable), has been derived in Essama-Nssah and Lambert (2012) and Monti (1991) (only for the IF) and reported in Firpo et al. (2007). If a univariate setting is assumed, where individuals are ranked by health instead of income (i.e., F_H is substituted for F_Y), our derivation of the RIF of the concentration index coincides with previous derivations of the (R)IF of the Gini. As Essama-Nssah and Lambert (2012) show that their result is the same as shown in Firpo et al. (2007), we only need to link our results to the latter.

The IF for the AC is given by proposition 1:

$$IF(h, F_Y(y); \nu^{AC}) = -2\nu^{AC}(F_{H,F_Y}) + \mu_H - h + 2hF_Y(y) - 2\int^{y}\int^{+\infty} hf_{H,F_Y}dhdF_Y(z) \tag{B1}$$

If in deriving Eq. (B1) we had used F_H as the ranking variable instead of F_Y we would have got the IF for the absolute Gini index (AG):

$$IF(h, F_H(h); \nu^{AG}) = -2\nu^{AG}(F_{H,F_H}) + \mu_H - h + 2hF_H(h) - 2\int^{h}\int^{+\infty} hf_{H,F_H}dhdF_H(z) \tag{B2}$$

Similarly to how the RIF of CI was derived in Appendix A, we find the RIF of the Gini index (GI) equals:

$$RIF(h;\nu^{GI}) = -\frac{h - 2\mu_H}{\mu_H}\nu^{GI}(F_{H,F_H}) + \frac{1}{\mu_H}IF(h;\nu^{GI}) \tag{B3}$$

Rearranging yields

$$\begin{aligned} RIF(h;\nu^{GI}) &= -\frac{h - 2\mu_H}{\mu_H}\nu^{GI}(F_{H,F_H}) + \frac{1}{\mu_H}\left[-2\nu^{AG}(F_{H,F_H}) + \mu_H - h + 2hF_H - 2\int^{h}\int^{+\infty} hf_{H,FH}dhdF_H(z)\right] \\ &= -\frac{h - 2\mu_H}{\mu_H}\nu^{GI}(F_{H,F_H}) + -2\nu^{GI}(F_{H,F_H}) + 1 - \frac{h}{\mu_H} + \frac{2}{\mu_H}hF_H - \frac{2}{\mu_H}\int^{h}\int^{+\infty} hf_{H,F_H}dhdF_H(z) \end{aligned} \tag{B4}$$

Note: Firpo et al. (2007) denote the Lorenz ordinate as:

$$\frac{1}{\mu_H}\int^{h}\int^{+\infty} hf_{H,F_H}dhdF_H(z) = \frac{1}{\mu_H}q(\alpha, F_H) \tag{B5}$$

where α is the fractional rank. Firpo et al. (2007) also denote the area under the Lorenz curve as:

$$R(F_H) = \int_0^1 q(\alpha, F_H)d\alpha \tag{B6}$$

The Gini index equals the area between the line of equality and the Lorenz curve:

$$\nu^{GI}(F_{H,F_H}) = 1 - 2R(F_H) \tag{B7}$$

Substituting Eqs. (B5)–(B7) into Eq. (B5) yields:

$$RIF(h;\nu^{GI}) = -\frac{(h-2\mu_H)(1-2R(F_H))}{\mu_H} - 2 + 4R(F_H) + 1 - \frac{h}{\mu_H} + \frac{2}{\mu_H}(hF_H - q(\alpha, F_H))] \tag{B8}$$

which after re-arranging yields the expression presented in Firpo et al. (2007):

$$RIF(h;\nu^{GI}) = 1 + \frac{2hR(F_H)}{\mu_H} - \frac{2}{\mu_H}(h(1-F_H) + q(\alpha, F_H))] \tag{B9}$$

This completes the proof. □

Appendix C. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jhealeco.2016.03.006.

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Erratum to "A general method for decomposing the causes of socioeconomic inequality in health" [Journal of health economics 48 (2016) (89-106)]

February 15, 2018

I would like to thank Toshiaki Aizawa for pointing out the following typos in the paper. Fortunately they have no impact on the final derivations, the conclusions or the Stata code but they may lead to confusion. The typos are as follows:

1. On page 95,

   Equation (19), a 2 is needed in front of $\hat{v}^{AC}(F_H, F_Y)$. That is, it should read as follows:

   $\hat{RIF}(h, F_Y(y); v^I) = \hat{v}^I(F_H, F_Y) + \hat{IF}(h; ^I)\hat{v}^{AC}(F_H, F_Y) + \hat{v}^{wI}(F_H)[-2\hat{v}^{AC}(F_H, F_Y) + \hat{\mu}_H h_i + 2h_i\hat{F}_Y(y_i) - \hat{v}^{ACC}(F_H, F_Y(y_i))]$

2. Page 95 again,

   Equations (20) and (21), the lower and upper limits should be the other way around and should be as follows:

   $\hat{F_Y(y_i)} = \frac{\sum_i^1 1}{N}$
   $\hat{v}^{ACC}(F_H, F_Y(y_i)) = \frac{\sum_i^1 h_i}{N}$

3. Page 103,

   Equation (A13), there should not be a negative sign in front of μ_H.

4. Page 104,

   Equation (A15), there should not be a negative sign in front of $\int h dF_H$.

Paper II

The long-term impact of education on mortality and health: Evidence from Sweden

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Abstract

There is a well-documented large positive correlation between education and health and yet it remains unclear as to whether this is a causal relationship. Potential reasons for this lack of clarity include estimation using different methods, analysis of different populations and school reforms that are different in design. In this paper we assess whether the type of school reform, the instrument and therefore subgroup identified and the modelling strategy impact the estimated health returns to education. To this end we use both Regression Discontinuity and Difference in Differences applied to two Swedish school reforms that are different in design but were implemented across overlapping cohorts born between 1938 and 1954 and follow them up until 2013. We find small and insignificant impacts on overall mortality and its common causes and the results are robust to regression method, identification strategy and type of school reform. Extending the analysis to hospitalisations or self-reported health and health behaviours, we find no clear evidence of health improvements due to increased education. Based on the results we find no support for a positive causal effect of education on health.

Keywords: Health returns to education, demand for medical care

JEL Classification: *I12, I18, I26*

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1 Introduction

The existence of an education gradient in health has been documented across various countries and for a variety of health measures including mortality, disability and various measures of morbidity (see for example, Mackenbach et al. 2003, 2008; Marmot et al. 2012; O'Donnell et al. 2013). A range of theories has been posited to explain the existence of this gradient including the suggestion that education has a causal effect through its impact on health production, or through impacting differences in financial resources, preferences or self-empowerment, understanding of information or better access to information (see for e.g. Cutler and Lleras-Muney 2006; Grossman 2006; Mackenbach 2012 for overviews). To test these theories we need credible instruments. A substantive part of the more recent empirical literature has used differences in compulsory schooling as a source of exogenous variation for years of education. Two recent reviews of the literature on the impact of education on health (Cutler and Lleras-Muney, 2012; Grossman, 2015) have found the literature hard to summarise.

In this paper we address this lack of clarity in the literature looking at the causal effect of education on health. We do this by pinning down many of the potential reasons that have been given to explain the differences in the findings in the literature. Suggestions for differences in results across studies include estimation on different populations and for different periods (Cutler and Lleras-Muney, 2012) or that the instruments used affect different groups (Grossman, 2015; Clark and Royer, 2013). Clark and Royer (2013) arguably provide the most convincing evidence so far from Britain's compulsory schooling law changes using month of birth and Regression Discontinuity (RD) design and find small or zero impacts of education on health. However, the reforms were implemented overnight, nationwide. The reforms were also enacted during very different periods (1947 and 1972). It may be that overnight implementation of the reforms reduced the wage returns to schooling because of the sudden change in supply of more educated workers, and this could impact a key potential channel for education to influence health. There is also cause for concern that the cohorts of 1947 and 1972 are not comparable. For example, Gathmann et al. (2015) provide indicative evidence that results based on pre WWII cohorts show larger impacts on mortality than results based on post WWII cohorts and this may be due to different base level mortality rates.

In this paper we consider the long-term impact of education on mortality and wider measures of health. We want to know whether the type of school reform has an impact on the

results, holding other variables constant. To this end we use two school reforms in Sweden that increased years of schooling for cohorts born very close together in time but different in nature. The first reform increased minimum years of schooling from 7 years to 8 years but only for those who were not eligible or unwilling to take the academic track. This was rolled out to about half of Sweden's municipalities. The second reform increased minimum years of schooling from 7 or 8 years (depending on municipality) to 9 years and introduced a new national comprehensive school system involving a change in peer groups and the introduction of a new national curriculum. The 9 year reform was rolled out nationally but phased in across municipalities and has been found to have had a sizeable impact on years of schooling (Meghir and Palme, 2005; Holmlund, 2007; Lundborg et al., 2014; Hjalmarsson et al., 2015). In fact by utilising a methodological improvement in the measurement of years of schooling we show that the 9 year reform had impacts on years of schooling twice that of previous estimates. This paper is the first paper to use the 8 year reform. It was rolled out extensively across Sweden and we show that it had a sizeable impact on years of education.

The particular set-up assessed in this paper is rather unique because the reforms are overlapping in the sense that they occur on average 7 years apart within municipalities. This means the reforms impacted individuals from similar backgrounds who entered similar labour market structures under a similar health system. That is, under similar labour market structures any returns to education should be similar for similarly aged cohorts. For each reform, the nature of their phased roll out means we can compare two groups born in the same year but who received different amounts of compulsory schooling based on where they were born. An advantage of this paper is therefore that we are able to pin down the impacts of the reforms separate of cohort effects. Also, because the reforms were rolled out over time, the impacts of the reforms are less likely to suffer the same level of general equilibrium effects that may be a concern if the reforms were rolled out nationwide overnight.

To help further isolate potential variables that explain differences across studies we employ both difference-in-differences (DiD) and RD design to identify the causal impact of the reforms on health. RD uses the cut-off for the school year, the 1st of January, combined with reform year for each municipality. Using birth date in years and months, those too old and therefore born before the reform year cut-off are not assigned reform status and those born on or after are. There are as many cut-offs as there are years of reform implementation and we average

over these to estimate the overall impact of the reform. Our DiD strategy compares across cohorts in municipalities that didn't implement the reform to those that did. To assess if modelling approach matters, we perform analysis of mortality using both linear regression and Cox proportional hazard regression for lifetime duration analysis.

Our data is based on the universe of Swedes born between 1932 and 1959. We link our various administrative records together using each individual's unique personal identification number enabling us to assess the mortality and health outcomes of about 1.2 million individuals. We consider the reform status of individuals born between 1938 and 1954. Mortality data is provided by the Swedish Cause of Death Database and our observation period follows our individuals up to the end of 2013 which means our oldest cohort born in 1938 and subject to reform is 75 years old when our period of observation ends. We also consider leading causes of death. We complement this analysis using Swedish hospital administrative data and a large survey (The Swedish Health and Living Standards Survey) considering self-reported health and health related behaviours. The survey data allows us to consider both contemporaneous health and health related behaviours and therefore gives us the potential to pick up effects using a more sensitive measure of health and also explain the pathways of any effects we find. Compared to previous Swedish studies which only consider the 9 year reform using DiD (Spasojevic, 2010; Lager and Torssander, 2012), the data we use has a much longer follow-up with more up to date data, more outcome variables and a larger sample size and we analyse this data using RD in addition to DiD. Meghir et al. (2017) also make the same contributions as we do over and above those of prior Swedish studies. In comparison to Meghir et al. (2017), our major contribution is that we introduce a new reform which has the novelty of allowing us to perform instrumental variable analysis of the causal effect of education on health, compare two different types of school reform across over-lapping cohorts. Further, we utilise a better measure of schooling that captures the full effect of the reform which dramatically changes the interpretation of the 9 year reform and we introduce new survey data measuring self-reported health and health behaviours.

In the following section we describe the two Swedish compulsory schooling reforms. In section 3 we introduce the data and in section 4 we outline our empirical strategy. In section 5 we present the results. Our findings show that there was a sizeable increase in years of education due to both reforms but these increases did not lead to an improvement in life

expectancy or health. The results are robust to reform, modelling choice, identification strategy and health outcome. We then discuss the results in section 6 and argue that the results are not only robust internally, they also have validity beyond the Swedish context. Finally, we conclude in section 7.

2 The Swedish compulsory schooling reforms

During the 1950s and 1960s in Sweden, a large number of municipalities raised the minimum years of compulsory schooling from 7 to 8 years gradually over a period between 1941 to 1962 (affecting birth cohorts born between 1927 to 1948). This is illustrated in figure 1 (See "Old primary school 8-Year"). We call this the 8 year reform. From 1948 to 1969 municipalities then gradually replaced the old system with a new comprehensive school system that also raised the minimum years of schooling to 9 years (affecting birth cohorts born between 1938 and 1958). Again, this is illustrated in figure 1 (See "New Compulsory school"). We call this the 9 year reform. This section provides some background information on these reforms, leaving a more detailed description to Appendix A.

Prior to both reforms, students attended a common compulsory primary school (*Folkskola*) up to and including the 6^{th} grade. After the sixth grade, good performing students (defined by an assessment) had the option to switch to an academic educational track and study at a three year junior secondary school (*Realskola*).[1] Those who continued on at primary school studied up to seventh grade. Attending junior secondary school allowed students to continue to higher secondary school (*Gymnasium*) and later university, an option denied to those who stayed on at primary school. Only a small minority went on to junior secondary school, the vast majority of students stayed on and completed compulsory education at primary school.[2]

The 8 year reform was a simple extension of the minimum years of schooling within the municipality for those students who did not choose to go to junior secondary school. The reform was seen as an opportunity to give more time for the students to learn without any specific changes to the curriculum. It is therefore a credible instrument for years of education. The 9 year reform introduced the Swedish comprehensive school system and was very different in character to the 8 year reform, as not only did it increase the minimum years of schooling

[1] Students could also start junior school after the 4^{th} grade and study a four year track.
[2] For the cohorts we consider the share was 16% (cohort 1938) and 30% (cohort 1951).

within the municipality to 9 years (from 7 or 8 years) it also postponed tracking of students with the aim of fostering greater equality of opportunity (Holmlund, 2007). The reform also introduced a new national curriculum. The removal of early tracking is likely to have broadened the peer group mix in the new comprehensive school system as higher ability students who would have gone to junior secondary school now shared the same class as their lower ability peers for longer compared to students under the old system. The curriculum changes may well have led to quality changes in schooling.

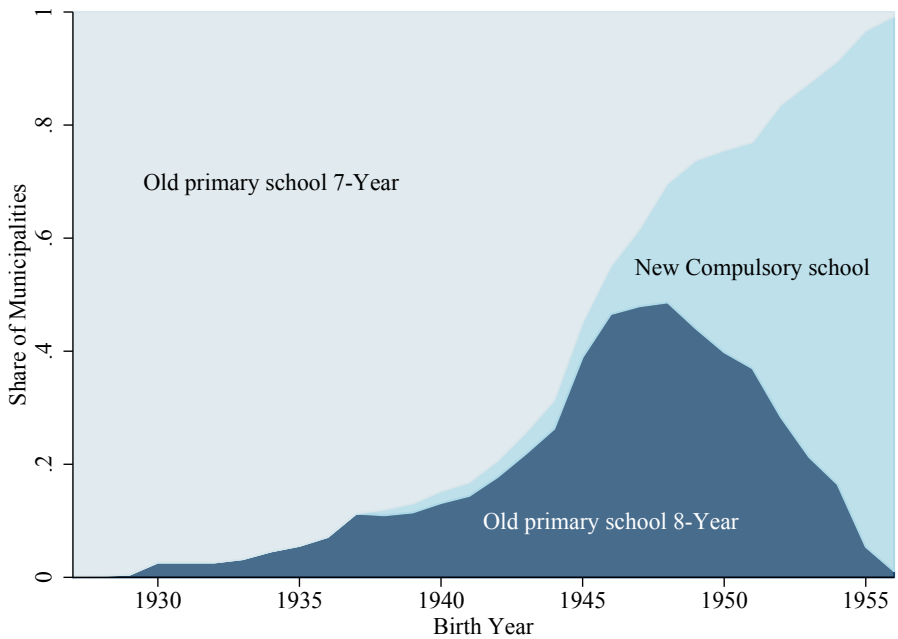

Fig. 1: Share of municipalities by length of compulsory education

Notes: This figure shows the proportion of municipalities in Sweden who have the 7 year old primary school system, the 8 year old primary school system and the new 9 year compulsory school system by birth cohort.

Both reforms were rolled out at municipality level over time and this phased roll out was at the discretion of the municipalities. The number of municipalities who had implemented an 8^{th} compulsory year gradually increased from 33 in 1946/47 to 207 in 1958/59. The early birds in this development tended to be more urban and included most of the larger cities. Smaller municipalities followed and in the end more than half of Sweden's more than 1,000 municipalities had introduced a mandatory 8^{th} grade before implementing the comprehensive

9 year school reform. The previous literature referred to the 8 year extension as a rare phenomenon mainly occurring in the largest cities (Holmlund, 2007). In fact the majority of municipalities had rolled out a compulsory schooling length of eight years before the 9 year comprehensive school was introduced. Figure 1 illustrates this development.[3] Both reforms were rolled out in such a way that within the same school older students were under the old regime and younger students were under the reformed compulsory schooling regime. For the 8 year reform this simply meant that younger cohorts studied a year longer than older cohorts. For the 9 year reform this meant two curricula were being taught within the same school.

The roll out of the reforms was not random. The 8 year reform roll out was chosen by the municipalities themselves, both whether or not to implement at all and the timing. The 9 year reform in its early phase was introduced with explicit intention to evaluate the policy reform. The process became less strict later on. In section 4 we set out how we control for this to identify the exogenous variation in schooling we are after.

3 Data

To quantify the impact of the two education reforms on health outcomes we employ both population based administrative data and survey data. The full population administrative data is drawn from the Swedish Interdisciplinary Panel (SIP)[4]. We consider the universe of those born in Sweden between 1932 and 1959, who survived to 16 years of age, and had not emigrated from Sweden by 2012.[5] These cohorts consist of 2,789,494 individuals.

To identify individuals as exposed or unexposed to the reforms we assign treatment status based on year of birth and place of residence. Information on the timing of the 8 year reform in each municipality was gathered from the Swedish National Archives. For the introduction of the 9 year reform we rely on a dataset as used in Hjalmarsson et al. (2015), of which an earlier version is described in detail in Holmlund (2007). Information on municipality

[3] In the early years of the introduction of the 9 year comprehensive school reform (between 1949 and 1962), the reform was introduced as a *social* experiment in certain areas (Marklund, 1982). The National School Board chose the areas from a group of applicants to form a representative set based on observable municipality characteristics (Holmlund, 2007). In 1962 the Swedish parliament finally decided that all municipalities should be obliged to offer the new comprehensive school system and by 1969 all municipalities were to have the new system in place.

[4] This is based upon Statistics Sweden's Multiple Generation dataset to which all other datasets are then linked using personal identifiers. It is administered at the Centre for Economic Demography, Lund University, Sweden. The present study was approved by the *Lund University Regional Ethics Committee, DNR 2013/288.*

[5] Very limited intergenerational information is available before 1932 explaining our chosen start point

of residence which we use to infer reform assignment is obtained from the 1960 and 1965 censuses.[6] In the empirical analysis we consider the reform status of cohorts born between 1938 and 1954 and use cohorts born before and after only as control groups. Our endpoint for the analysis, the cohort born in 1954, is chosen because this is the last cohort questioned in the 1970 census for which we have enough years to measure their years of schooling. Both reforms were rolled out in different parts at different times within the cities of Stockholm, Gothenburg and Malmo and therefore we exclude those resident in these cities. From the original sample we have reform assignment for both reforms for 2,108,696 individuals.

Data on schooling is obtained from the 1970 census and this is combined with post schooling attainments from the Education administrative database. We derive a measure of years of education by assigning the years typically associated with different types of schooling (from the census in 1970) and post-schooling qualifications from Education administrative database and take the sum as an approximation for the total years of education. This approach is an important innovation in how to measure years of education in Sweden as it allows us to capture the effect of the 8 year reform on years of education and also better captures the effect of the 9 year reform. Previous Swedish population administration data based studies have approximated years of education by the average length associated with the highest educational qualification (Hjalmarsson et al., 2015; Lager and Torssander, 2012; Lundborg et al., 2014). We cannot use this approach for the 8 year reform because there is no information on whether an individual went to 7 year or 8 year primary school. These are clumped together in the same category in the variable capturing highest educational qualification. Using just administrative based information on highest qualification also means the impact of the 9 year reform is under-reported as this approach cannot distinguish between someone who attained more than

[6]For cohorts born between 1943 and 1948, we assume that place of residence in 1960 is also the municipality where they went to school. For cohorts born on or after 1949 we follow the suggestion of Holmlund (2007) and use the place of residence as recorded in the 1965 census. For those born before 1943 we use place of residence of the mother (father if information is missing for the mother and if both parents are missing we use own place of residence in 1960) as recorded in the 1960 census. An alternative approach would be to use the *place of birth* as an approximation of place of residence during childhood. In theory this approach has a nice intention to treat interpretation and avoids being susceptible to potential parental choice of reform assignment, itself linked to the child's ability. However, for the cohorts we consider births were increasingly occurring at a hospital and before 1947 the hospital was recorded as the place of birth which often did not coincide with the place of residence of the parents. Up to 1947 therefore, the place of birth becomes increasingly uninformative as a measure of place of residence for the child at birth. From 1947 this practice changed and the place of birth of the child was recorded as the place of residence of the mother (Holmlund, 2007). In figure B.1 in the appendix we show that the misclassification from hospital recording is sizeable. As near half of our cohorts are born before 1947 and Meghir and Palme (2005) provide evidence that inter-municipality migration is very small (and therefore parental response to the reforms is small if there is one at all), we do not follow this approach.

compulsory schooling but had different amounts of compulsory schooling e.g. two people who received vocational training but received different amounts of compulsory schooling would be given the same number of years of education using just the information from the administrative data on highest educational qualification. Our new method captures the impact of the 9 year reform more accurately as it distinguishes schooling and post schooling achievements when calculating years of schooling.[7] How we construct our years of education variable is a key contribution of the paper, as it not only allows us to capture the impact of the 8 year reform, it also improves the measurement of the impact of the 8 year reform as we shall show later.[8] Our final sample size for those we also have information on years of education is 2,022,174.

Data on cause of death and cause of inpatient care at hospital is obtained from the Swedish Cause of Death database and the Hospital Patient database (stays at a hospital of more than 24 hours) and merged using a personal identifier to our main dataset. The mortality data covers the years from 1964 and cause specific information is from 1969 and runs up to 2013. We consider the whole observation period and therefore measure the impact on death up to age 75 for the oldest cohort.[9] The inpatient data covers the years 1987 up to 2012. We therefore consider the impact on hospitalisations up to age 74. Underlying cause in both datasets is recorded according to the 7^{th}, 8^{th}, 9^{th} and 10^{th} versions of the International Classification of Diseases (ICD) depending on year of death/hospital admission. The data also includes date of death, date of hospital visit (admission and discharge) and length of hospital visit. We consider the most common causes of death and hospital visits and also the number of hospital days from inpatient records (see appendix B table B.1 for variable ICD codings). In table 1 we present the means of our administrative data based outcomes variables by reform and whether treated or untreated. We include only those born within 10 years of

[7] We still have downward measurement error for cohorts born after 1951 because the years of schooling measure in the census measures schooling up to age 19. For cohorts born 1952-1954 we impute years of schooling using the modal years of schooling measured in the census corresponding to each level of further education achieved as measured in the Education administrative data from earlier cohorts (1948-1950). For cohorts after 1954 we assign 7 years of schooling for those with post schooling Swedish Education Nomenclature 2000 (SUN2000) classification of less than 200 and 9 years with a SUN2000 classification of 200-300. This adjusts for the downward bias of measuring individuals' schooling before they have reached 19 but will still underestimate the reform effect.

[8] This is particularly important for interpretation of prior studies that used the 9 year reform as an Instrumental Variable for years of schooling as they will have biased their causal estimates upwards using years of education based on highest qualification from the Education administrative database.

[9] We consider death by 2013 so that we capture as much data as possible. We could consider death by a certain age, but then we would lose a lot of information given most deaths are for older ages.

reform implementation as this is the population we use in our main analysis. We observe that on average for our 8 year reform sample the untreated have 9.4 years of schooling, 19% have died by 2013 and predominantly of cancer and have spent 30 days in hospital whereas the treated population have 10.9 years of schooling, only 9% have died by 2013 and have spent 21 days in hospital. For the 9 year reform sample on average the untreated are again less educated, more have died by 2013 and are more likely to have had a hospital visit and for longer compared to those treated. For both reforms, the treated are younger and this explains a large part of the differences in health outcomes between our treated and untreated groups. We control for this in our analysis.

Table 1: Descriptive statistics - administrative data

Variable:	8 Year Reform		9 Year Reform	
	Untreated	*Treated*	*Untreated*	*Treated*
Years of education	9.4	10.9	10.1	11.5
Dead	0.199	0.090	0.128	0.063
Proportion dead due to:				
Cancer	0.080	0.035	0.050	0.021
Circulatory Disease	0.050	0.019	0.029	0.012
External Causes	0.010	0.011	0.011	0.012
Other	0.059	0.026	0.038	0.019
Days at hospital	29.7	21.3	24.0	20.3
Proportion who had a hospital visit due to:				
Cancer	0.111	0.066	0.084	0.051
Circulatory Disease	0.156	0.083	0.113	0.062
External Causes	0.121	0.092	0.106	0.092
Other	0.313	0.269	0.284	0.293
Observations	215,846	318,557	640,093	607,715

Notes: This table shows the means for education and health outcomes for those treated and not treated by each reform and born within 10 years of the first cohort impacted by the reform.
Source: SIP. Own calculations.

The survey data stems from the Swedish survey on living standards (ULF) (Statistics Sweden, 2008).[10] The survey includes self-reported health and health behaviour variables which we consider as valuable complements to population based administrative data on cause of death. We consider binary indicators for smoking behaviour, obesity, self-reported anxiety or worry and self-reported fair or bad health (in contrast to good).[11] In table 2 we present the means of our education and health outcomes. The years of education means correspond

[10] The ULF survey is a well respected survey used for a wide range of research topics and in recent years has formed the Swedish part of the European Union Statistics on Income and Living Conditions (EU-SILC).

[11] We define a binary variable *bad or fair health* equal to one if self-reported health is reported as fair or poor. Smoke Daily is a binary indicator, indicating one if smoked daily in the past 30 days prior to interview, zero otherwise. Anxiety is a binary variable, one indicating whether the individual self-reported having heightened anxiety, concern or worry, zero otherwise. Obese is a binary indicator derived from information on height and weight creating a Body Mass Index (BMI), one indicating a BMI of 30 or more, zero otherwise.

to those for the administrative data in table 1 which suggests the sampling frame of the survey data does well to represent the population estimate. Whilst self-reported *fair or bad health* and obesity are more common amongst the untreated samples, *smoke daily* and *anxiety, concern* are actually more common amongst the treated population. The survey itself is carried out by face-to-face interviews of a randomly selected sample of the population. The sample size is about 7,500 individuals per year and data is reported for years 1980 through to 2012. We therefore have 32 years of data. Information from different Swedish censuses and on education attainment from the Education administrative data is linked to individuals in the survey.

Table 2: Descriptive statistics - survey data

Variable:	8 Year Reform		9 Year Reform	
	Untreated	*Treated*	*Untreated*	*Treated*
Years of education	9.5	10.9	10.2	11.5
N	3,360	4,840	9,847	9,306
Fair or bad health	0.261	0.183	0.213	0.166
N	3,349	4,832	9,830	9,294
Smoke daily	0.226	0.276	0.255	0.268
N	3,325	4,813	9,776	9,257
Obese	0.129	0.090	0.105	0.083
N	2,139	2,857	6,004	5,510
Anxiety, concern etc	0.149	0.150	0.146	0.149
N	2,256	3,132	6,437	6,050

Notes: This table shows the means for education and health outcomes for those treated and not treated by each reform and born within 10 years of the first cohort impacted by the reform.
Source: ULF-Survey. Own calculations.

4 Empirical Strategy

4.1 Identifying the impact of the reforms

In this section we outline the two empirical identification strategies we use to identify the impact of the reforms on health outcomes: DiD and RD design. The purpose of using two identification strategies is that it provides a sense of how robust the findings are. The two methods rely on different identifying assumptions to identify the causal impact of the reforms and potentially estimate the impact for different populations of compliers. We are therefore able to assess if this is important to the conclusions we draw.

The impacts of the reforms on education, mortality, hospitalisations and self-reported health outcomes are modelled in a linear setting, using either OLS or a Linear Probability

Model (LPM) depending on the outcome variable, as is standard for both DiD and RD. Our DiD empirical strategy utilises the fact that the education reforms were introduced slowly over time across municipalities in Sweden. Two individuals born in the same year but one resident in a reform municipality and the other not have different exposures to compulsory schooling. This provides us with variation in reform exposure both over time and across municipalities. However, the implementation was not random as discussed in Holmlund (2007). To control for this we difference across municipalities and across birth cohorts using dummy variables for both. Our linear DiD model is:

$$H_{i,c,m} = \beta_0^{DiD} + \beta_1^{DiD} Z_{c,m} + \beta_2^{DiD} C + \beta_3^{DiD} M + \beta_4^{DiD} trend_m + \epsilon_{i,c,m}; \tag{1}$$

where i indicates an individual, c the birth cohort, m the municipality, and $Z_{c,m}$ is a variable equal to one for individuals assigned as exposed to the reform using their date of birth and place of residence, zero otherwise. $H_{i,c,m}$ is our outcome of interest, C is a vector of birth year cohort dummies, M is a vector of municipality dummies, $trend$ is a vector of municipality specific trends and β_0^{DiD} is a constant term. The coefficient β_1^{DiD} measures the impact of the reforms on our outcome measures. We estimate equation (1) separately for each reform.

The empirical strategy utilising RD design involves identifying the reform effect within municipalities based on the year the school reform was introduced and the cut-off date for the school year, which is the 1st of January. Individuals born before the reform year cut-off are not assigned as exposed to the reform and those born on or after the cut-off are assigned as exposed. The forcing variable, $T_{i,m}$, is birth date measured in years and months from the reform cut-off date in their municipality. Our linear RD model takes the form:

$$H_{i,c,m} = \beta_0^{RD} + \beta_1^{RD} Z_{c,m} + f(T_{i,m}) + \mu_{i,c,m}; \tag{2}$$

where i, c, m, $Z_{c,m}$ are as for equation (1). The coefficient β_1^{RD} captures the average impact of the reforms across all of the municipality level cut-offs. The identifying assumption is that the outcome variable is a smooth function of our forcing variable and that after adequately modelling this function, $f(T_i)$, any jump found at the cut-off is due to the education reform and not some other unobserved variable. To capture the birth cohort relationship with the outcome variable, we model $f(T_i)$ using a polynomial in years-months from the cut-off, T_i,

estimated separately either side of the cut-off combined with dummies for gender and month of birth, to control for seasonality effects.[12] To choose our preferred function for T_i we followed the approach of Imbens and Lemieux (2008) and included progressively higher order polynomials in T until the additional polynomials were insignificant. At the same time care was taken to not over-fit the model, a concern raised by Gelman and Imbens (2017). We found that a second order polynomial was sufficient for all outcomes.

It is potentially of concern that we include individuals much older or younger than the first cohorts impacted by the reform within a municipality. To deal with this we use a bandwidth of up to 10 years for both our DiD and RD regressions so that only those born up to 10 years before or after the first cohort impacted by the reform are included in the analysis.[13] We also test the sensitivity of bandwidth choice and whether municipality level trends are important.

We consider mortality as our main health outcome. In addition to modelling mortality using an LPM we also consider time until death during the observation period using a Cox proportional hazard model (Cox and Oakes, 1984) together with our DiD and RD identification strategies. In this way, we model the conditional probability of dying in the next period given survival to the current period. By considering the survival nature of our data we use more information, potentially increasing efficiency. It also allows us to deal with censoring because of survival beyond the sample period of 2013 and is also a natural choice when considering causes of death. If a particular cause of death is reduced by the reforms, by construction this means other causes of death will be increased for a given level of mortality. Cox models, under the independent competing risks assumption, deal with this. In our application of the Cox model we estimate duration until death, d, and using DiD we stratify on municipality of residence and include dummies for birth cohort which gives us:

$$I_{1,i,c,m}(d|X) = I_{0,m}(d)exp[\delta_0^{DiD} + \delta_1^{DiD}Z_{c,m} + \delta_2^{DiD}\gamma_c + \delta_3^{DiD}trend_z]; \tag{3}$$

where $I_{0,m}$ is the baseline hazard stratified by municipality, cohort specific fixed effects are given by γ_c, time trends for municipalities that roll out the reform in the same year are given by $trend_z$, and subscripts i, c and m, and $Z_{c,m}$ are as for equation (1).[14] δ_1^{DiD} is the reform

[12] For analysis of the survey data we also include survey year dummies to control for age and year effects.

[13] We say up to ten years because for the earliest cohort we only have cohorts up to 6 years older to compare to and for later cohorts we only have cohorts up to 5 years younger to compare to.

[14] For estimation to be feasible we have to limit the municipality trends estimation to municipalities that

impact on mortality. For our RD design, the Cox model takes the form:

$$I_{1,i,c,m}(d|X) = I_0(d)exp[\delta_0^{RD} + \delta_1^{RD} Z_{c,m} + f(T_{i,m})]; \tag{4}$$

where I_0 is the baseline hazard and subscripts i, c and m, and variables $Z_{c,m}$ and $f(T_{i,m})$ are as for equation (2). The coefficient δ_1^{RD} is the impact of the reform on mortality within each municipality averaged over all municipalities.

All of the models we have outlined above are reduced form models. We also apply Two Stage Least Squares (2SLS) using our linear equations (1) and (2) as the first stages with years of education YE in place of H as the dependent variable and instrumented with reform status. Our second stages are:

$$H_{i,c,m} = \alpha_0^{DiD} + \alpha_1^{DiD}\widehat{YE}_{i,c,m} + \alpha_2^{DiD} C + \alpha_3^{DiD} M + \alpha_4^{DiD} trend_m + v_{i,c,m}; \tag{5}$$

$$H_{i,c,m} = \alpha_0^{RD} + \alpha_1^{RD}\widehat{YE}_{i,c,m} + f(T_{i,m}) + u_{i,c,m}; \tag{6}$$

where α_1^{DiD} and α_1^{RD} are the coefficients on years of education and are our coefficients of interest. Both α_1^{DiD} and α_1^{RD} are identified by the variation in years of education that comes from the variation generated by the school reforms.

To identify our 2SLS coefficients we need to assume that the reforms affected our health outcomes only via their effects on years of education (the exclusion restriction) and that reform exposure is as good as random given our control strategies. The exclusion restriction would be violated if the reforms had other impacts on students over and above their impact on years of education that then impacted on health. For the 8 year reform the education system remained the same and so did the curriculum. However, for the 9 year reform, in addition to the increase in years of compulsory schooling, both the curriculum and the school system were changed. Prior to the 9 year reform, students were selected into different schools based on their academic ability. The 9 year reform abolished this and instead, students were kept in the same school and classes until the ninth grade.[15] Tracking has been found to impact educational achievement and later life outcomes (Betts et al., 2011) suggesting that its removal

roll out the reform in the same year rather than model trends for each individual municipality.

[15]There were some exceptions, where tracking was used for some subjects but overall students were much more mixed.

may have had an impact on educational quality. The removal of tracking will also have changed the peer group mix that students were exposed to, potentially impacting learning, health related behaviours and even assortive mating. Peer effects have been found for health outcomes and health related behaviours such as drinking, smoking and drug use (see for e.g. Sacerdote et al. 2011 for an overview on peer effects). In addition to the change in tracking, the 9 year reform coincided with the introduction of a new national curriculum. Although we have no evidence of the impact on quality this change made, it seems reasonable to assume that it had some impact on the variation in quality of schooling across the municipalities.

In order to use the 9 year reform as an IV we have to assume that both the changes to the tracking system and the introduction of a national curriculum had no impact on schooling quality or peer effects that could in turn impact our health outcomes. A number of articles have made this claim (Spasojevic, 2010; Lundborg et al., 2014) whilst others view this as controversial and focus on estimating the reduced form effect of the reforms (Meghir and Palme, 2005; Meghir et al., 2017). In this paper we take the latter view but present IV estimates based on the 9 year reform as a way of quantitative comparison to the 8 year reform, a reform which we argue more convincingly meets the exclusion restriction requirements.

4.2 First stage results and diagnostic tests

Both of our identification strategies build upon our method of treatment status assignment performing well. In addition to this and the exclusion restriction, our 2SLS estimates require our education reform impacts on schooling to be as good as random given our control strategies. For our DiD estimates our control strategy hinges on the assumption that conditional on birth cohort and municipality fixed effects, exposure to treatment is as good as random. For our RD estimates our control strategy hinges on the assumption that conditional on our modelling of age relative to the first cohort within the municipality impacted by the reform, there are no jumps in the error term at the cut-off. In this case, any jumps we do find in years of schooling at the cut-off can then be assumed to be as good as random. In this section we establish the existence of a first stage and provide some diagnostic tests to assess the plausibility of our identification assumptions.

In figure 2 we present the raw data of the probability of having achieved 8 years of old primary schooling (left hand side panel) and 9 years of schooling (right hand side panel)

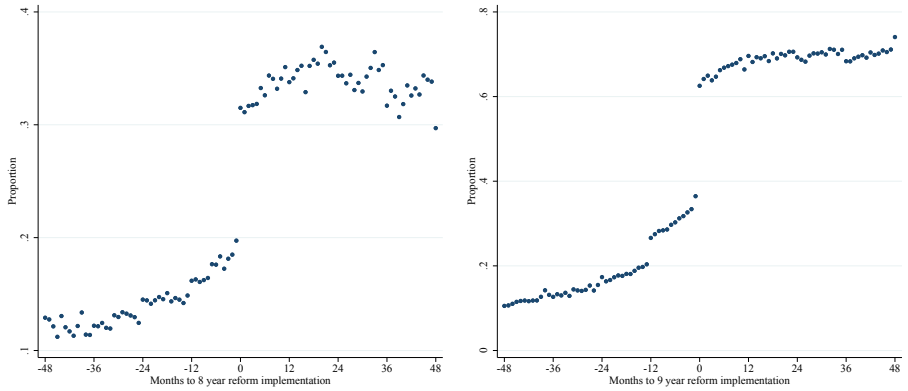

Fig. 2: Effect of the reforms on the proportion with the new minimum years of schooling

Notes: Scatter plots of the proportion with the new minimum years of schooling by age in months measured as months to reform implementation in their municipality. Left panel is for the 8 year reform, right panel the 9 year reform. Reform implementation is at time zero. *Source:* SIP. Own calculations.

against birth cohort measured in months relative to the first cohort impacted by each reform respectively. For each monthly bin the proportion with 8 years/9 years of schooling is plotted. We see that there is an increasing trend with time until exposure and that at the cut-off there are clear jumps in the proportion with the new minimum years of schooling. Note that it is entirely expected that a proportion of students have the new compulsory schooling before reform implementation. Students who repeat a grade would naturally receive an extra year of schooling. For both reforms it is also documented that there was partial roll out that was non-mandatory prior to the reform becoming mandatory. We also see a jump in the proportion with the new compulsory years of schooling in period t-1 and this is much clearer for the 9 year reform. Hjalmarsson et al. (2015) suggest that the pre-reform increase in schooling is due to either measurement error in the exposure variable or due to pupils being in the wrong grade based on their age due to choosing to repeat a grade. Hjalmarsson et al. (2015) cite evidence that grade repetition was not a common occurrence for those in the old 7 year primary school system but grade repetition and dropping out was for those who were tracked into the junior secondary school. Those at junior secondary school who were born a year too early but had dropped out would have normally gone back to old primary school, but because of the reform they would have instead been caught by the 9 year school reform and would as a consequence be a year older than their peers in the same class. This last

explanation fits with what we see in the data. There is possibly a small jump in t-1 for the 8 year reform and this fits with the reported observation that grade repetition was not very common in the old primary school system. There is however a clear jump in t-1 for the 9 year reform, and this is quite likely in part driven by dropouts from junior secondary school.[16]

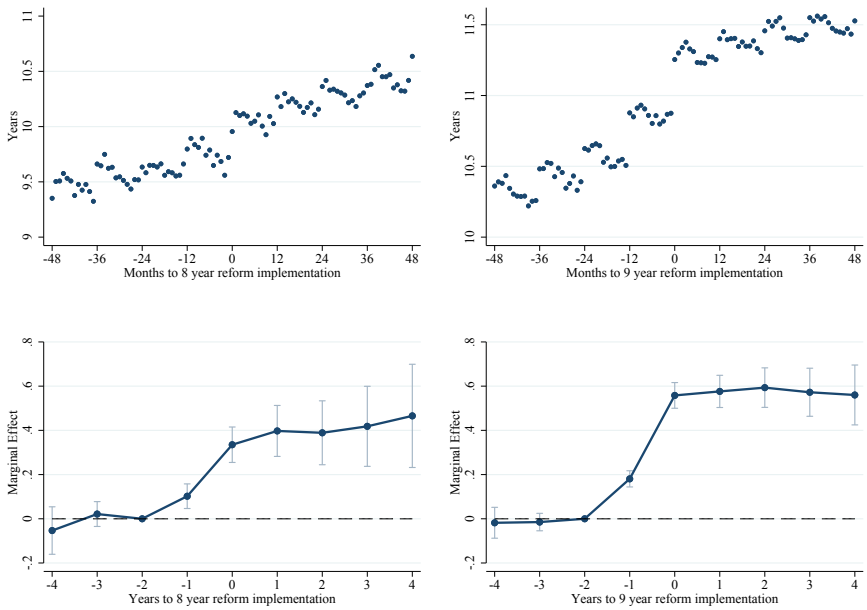

Fig. 3: Effect of the reforms on years of education

Notes: Top two panels: Scatter plots of mean years of schooling by age in months measured as months to reform implementation in their municipality where the first cohort impacted is at zero. The bottom two panels: plot regression coefficients of an individual's birth year relative to the first reform cohort in their municipality on years of education (spikes represent the 95% confidence interval for each coefficient estimate). Municipality and birth year fixed effects and municipality level time trends are controlled for, a bandwidth of 10 years is used and clustered standard errors are estimated at the municipality level. Category 4 is four or more years after the first reform cohort. The reference category is *two years before the first reform cohort* (t-2).
Source: SIP. Own calculations.

In figure 3 the top two panels present the raw data in a similar fashion to that of figure

[16]In the appendix we also show figures equivalent to figure 3 but for the proportion with just 7 years of schooling, just 8 years of schooling and just 9 years of schooling. These all confirm the jump at the cut-off. They also confirm the pre-reform jump in t-1. The jump in t-1 for the 9 year reform coincides with a clear drop in 8 years of old primary school in t-1 suggesting it is driven by individuals dropping out of junior secondary school after one year in municipalities who had introduced the 8 year reform. We can also see that there is measurement error in the exposure variable as after the 8 year reform there are still some with 7 years schooling. Similarly for the 9 year reform there is still a proportion with old primary school after reform exposure. This is partly explained by partial implementation in the municipality where exposure is given as 1 if just part of the municipality enacted the reform.

2, this time with years of education on the y-axis. The bottom two panels of figure 3 are event study graphs from our DiD regressions and show the conditional marginal effect and the corresponding 95% confidence interval for each year cohort relative to the first cohort impacted by the reform (t=0) on years of education. The reference cohort is the cohort born two years before the first treated birth cohort. The estimates are from a regression controlling for municipality and birth cohort fixed effects and standard errors are clustered at the municipality level. From figure 3 we can see a jump in the average years of schooling due to both reforms and that this jump is larger for the 9 year reform.

Table 3 presents the regression results of the impact of the reforms on years of education for all individuals, and also split by gender. Column (1) in table 3 presents the results for the 8 year reform on years of education for all individuals and we find an increase of 0.23 years of schooling using RD and 0.27 years of schooling using DiD. Column (4) presents the results for the 8 year reform for all individuals and we find an impact of 0.39 years using RD and 0.53 years using DiD. Note that the 9 year reform estimates presented here are much larger than previously documented (see e.g. Holmlund 2007; Lundborg et al. 2014; Meghir et al. 2017) and the reason is because we use a different measure of years of education which better captures the impact of increased compulsory schooling on years of education. Indeed, using a schooling measure calculated in the same way as Holmlund (2007) we find that the impact of the 9 year reform on years of schooling is 0.3 years using DiD and 0.25 years using RD, both much smaller than our preferred estimates presented in table 3 (see model (7) of table B.3 in the appendix for the administrative data based education variable results).

Table 3: Compulsory schooling reforms' impact on education

	(1)	(2)	(3)	(4)	(5)	(6)
	8 year reform			9 year reform		
	All	Females	Males	All	Females	Males
Difference in Difference	0.272***	0.227***	0.316***	0.523***	0.493***	0.551***
	(0.023)	(0.029)	(0.026)	(0.019)	(0.019)	(0.023)
F-stat	140.64	59.44	147.39	779.52	688.97	576.09
N	534,403	264,237	270,166	1,247,808	613,317	634,491
Regression Discontinuity	0.230***	0.208***	0.251***	0.392***	0.349***	0.433***
	(0.023)	(0.031)	(0.030)	(0.023)	(0.027)	(0.024)
F-stat	101.29	44.25	72.36	299.31	172.71	317.89
N	534,403	264,237	270,166	1,247,808	613,317	634,491

Notes: This table shows the impact of the 8 year and 9 year school reforms on years of education. Each coefficient is from a separate regression by reform, method and group. DiD specification includes birth cohort and municipality fixed effects and municipality level time trends. Regression discontinuity estimates have separate second polynomials in the running variable either side of the cut-off and a full set of dummy variables for month of birth and gender. Bandwidth of up to 10 years is used for both DiD and RD. Robust standard errors clustered by municipality level (for DiD) and by the running variable (for RD) are in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

The 9 year reform had a larger impact on years of education compared to the 8 year reform. This makes sense because the 9 year reform increased years of schooling by two years for students who were in municipalities only offering 7 years of primary school and one year for those offering 8 years, whereas the 8 year reform was just a single year increase for all municipalities affected. The 8 year reform was also predominantly rolled out in urban areas that had less potential compliers as more students in urban areas went to junior secondary school. The results split by gender show that the impact for males is slightly larger across modelling strategies and reforms. The F-statistic results suggest across the board that we have a strong first stage, based on the the rule of thumb for weak instruments of an F-statistic above 10 (Stock et al., 2002). In table B.3 in the appendix we investigate whether the results in table 3 are sensitive to our choice of bandwidth and inclusion of municipality specific linear trends (DiD only). Using bandwidths with a range of 2 years to up to 12 years we find that the point estimates change only slightly (see columns (1-6) in table B.3). Inclusion of trends in our DiD estimates makes little difference to the point estimates.

The event study graphs in figure 3 show increases in years of education for the birth cohort born a year before the first treated birth cohort (t-1) although this is much smaller for the 8 year reform. Hjalmarsson et al. (2015) find the same pattern when including lags of the treatment in their assessment of the 9 year reform. We also find that our RD estimates are consistently smaller than our DiD estimates. The increase we observe for the t-1 cohort in the

figure explains most of the difference between our RD and DiD estimates. The RD estimates only capture the impact of the reforms in the reform period whereas the DiD estimates capture some of the pre-reform treatment differences. We illustrate this by dropping the t-1 cohort as a sensitivity test (see model (8) table B.3 in the appendix) and find that the gap between the RD and DiD estimates largely disappears.

We conclude that both the 8 year and 9 year reforms lead to increased years of education, that the increases were slightly larger for males and that the 9 year reform actually had much more bite than previous research has suggested. Our RD estimates are smaller than our DiD estimates and this is likely due to DiD capturing more compliers to the reform. This is a consequence of them capturing different sub-populations.[17]

We have shown that the reforms coincide with substantial increases in years of education using both DiD and RD and therefore that our method of reform assignment is working well. In addition to a strong first stage regression, our 2SLS estimates require exposure to reform to be as good as random, conditional on our control strategies. This may be violated if selective migration to and from reform municipalities occurred, either to escape or gain access to the reform. In previous work assessing the 9 year reform, both Meghir and Palme (2005) and Holmlund (2007) have tested for selective migration and have found that it was not a problem. We are not able to test it for the 8 year reform but make the assumption that the results of both Meghir and Palme (2005) and Holmlund (2007) apply to the earlier reform as well. We view this as a plausible assumption given that the 8 year reform was just a pure years of schooling change and would have provided much less of a reason to move compared to the comprehensive 9 year school reform - a reform that itself led to very limited selective migration.

Our estimates are robust to the inclusion of lags and leads and various forms of model specification but there may still be concern that our error term remains correlated with our explanatory variables, in particular reform assignment. In figure 4 we perform an RD diagnostic test of manipulation of the forcing variable in the spirit of McCrary (2008) by

[17] We have also tested whether seasonal variation in years of schooling changes after the reforms. We find no impact for the 8 year reform and a small negative impact for the 9 year reform on years of schooling but not on later health outcomes. This is consistent with work looking at the impact of school starting age on longer term labour market outcomes, that also finds both an impact on years of schooling but also no impact on later life outcomes (Fredriksson and Öckert, 2014). Including separate monthly dummies each side of the threshold however comes at a severe loss of efficiency, we therefore choose to model month effects without a reform interaction.

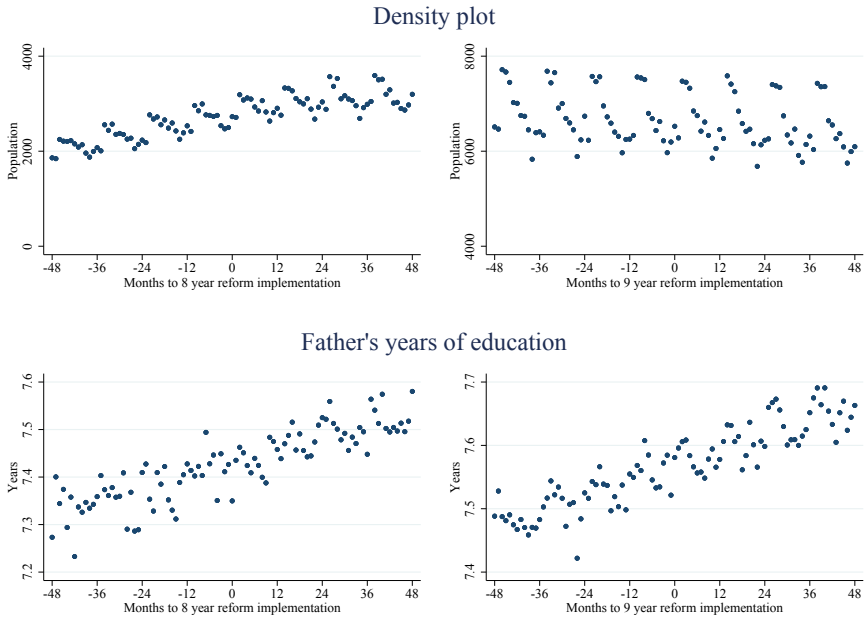

Fig. 4: Diagnostic tests

Notes: Top two panels: Density plots of age measured as the distance in months to reform in their municipality (the first cohorts to be impacted are at zero). The bottom two panels: Placebo tests of reform status on father's years of education - plotted as mean father's years of schooling in monthly bins of months to reform.
Source: SIP. Own calculations.

plotting the population density by age relative to the first cohort in the municipality impacted by the reform (top two panels). We observe no obvious jump at the cut-off point and therefore no clear changes in the fertility timing decisions around the reform. We also present scatter plots of father's education by age relative to the first reform birth cohort and again see no clear jump in father's years of schooling at the cut-off. In addition we perform a batch of balancing tests of predetermined characteristics and reform assignment in order to assess our exclusion restriction in table 4. The results show that when we control only for birth cohort fixed effects (columns 1 and 4) our predetermined characteristics are predicted by reform status. The correlations also go the way we might expect: the reforms were introduced earlier in areas where parents were more educated and had better jobs. The inclusion of municipality fixed effects and municipality specific time trends and hence our DiD strategy (see columns 2 and 5) reduces the size of the coefficients bringing them down to zero. Similarly in columns

(3) and (6) using RD to identify the impact of the reforms we find the size of the coefficients tends towards zero and they are insignificant. Whilst this evidence is just indicative that our reforms are not correlated with our error terms, they provide certain credibility to our strategies. In sum, we have shown that our assignment method works well and that with the application of our DiD and RD strategies we have provided support to our claim that reform exposure is as good as random.

Table 4: Diagnostics: Balancing test for differences in predetermined characteristics by reform status

	(1)	(2)	(3)	(4)	(5)	(6)
	8 year reform			9 year reform		
	OLS	DiD	RDD	OLS	DiD	RDD
Panel A: Mother						
Years of Education	0.095*	-0.009	-0.009	0.074**	-0.002	0.005
	(0.039)	(0.007)	(0.009)	(0.026)	(0.004)	(0.006)
Blue collar worker	0.033***	0.002	0.001	0.002	-0.001	-0.000
	(0.007)	(0.002)	(0.003)	(0.005)	(0.001)	(0.002)
White collar worker	0.032***	0.004	-0.002	0.019**	0.000	-0.000
	(0.008)	(0.002)	(0.002)	(0.006)	(0.001)	(0.001)
No occupation	-0.068***	-0.006*	0.002	-0.021*	0.000	-0.001
	(0.012)	(0.002)	(0.003)	(0.009)	(0.002)	(0.002)
Panel B: Father						
Years of Education	0.118	-0.005	-0.011	0.128***	0.005	0.005
	(0.065)	(0.011)	(0.014)	(0.038)	(0.007)	(0.009)
Blue collar worker	0.043***	-0.001	0.004	0.010	-0.000	-0.004
	(0.012)	(0.003)	(0.004)	(0.008)	(0.002)	(0.002)
White collar worker	0.060***	-0.001	0.000	0.032**	0.002	0.005*
	(0.017)	(0.003)	(0.003)	(0.012)	(0.002)	(0.002)
No occupation	-0.004*	0.001	0.002	0.001	0.000	-0.000
	(0.002)	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)

Notes: This table shows impact of reform status on various predetermined characteristics. Columns (1) and (4) are simple associations controlling for year of birth. Columns (2) and (5) are estimates from a DiD regression. Columns (3) and (6) are estimates from a RD design regression using a 2nd polynomial in age from reform estimated either side of the cut-off, and dummies for month of birth and gender. All estimates use a bandwidth of up to 10 years and robust standard errors clustered at the municipality level (age on months level for RD) and these are shown in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

5 Results

5.1 Mortality

5.1.1 All cause mortality by 2013

In this section we analyse the impact of the school reforms on mortality risk by 2013 (dying by age 75). In figure 5 we show the risk of dying by 2013 and we observe no impact of the

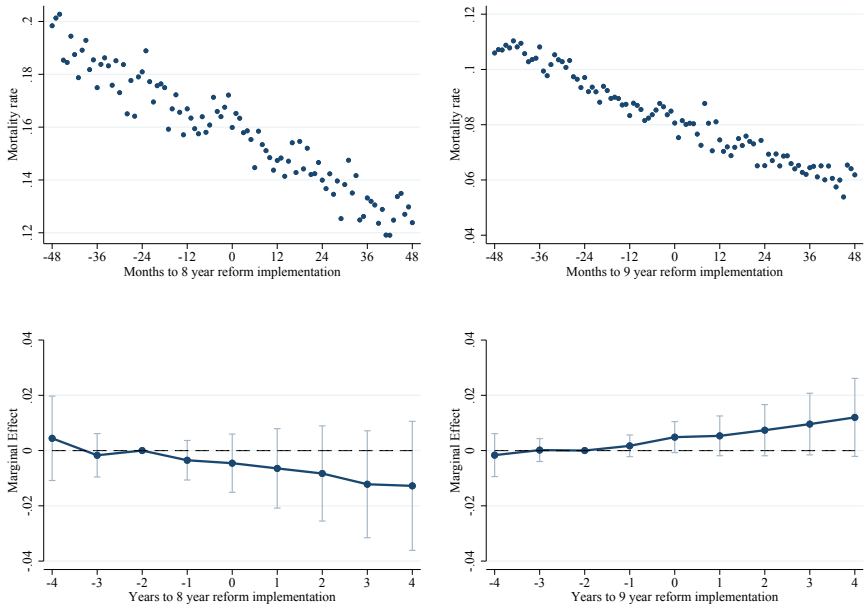

Fig. 5: Impact of the reforms on mortality by 2013

Notes: These figures plot the relationship between time to reform and mortality. The top two figures are raw data scatter plots. The bottom two figures are event study figures of coefficients from DiD regression. See notes for figure 3.
Source: SIP. Own calculations.

reforms on overall mortality in the raw data or in the event study figures.

In table 5 we present the LPM regression results for mortality. In column (1) we present the linear association of years of education with mortality probability controlling for year of birth only. This is estimated only for those not treated and with pre or post reform minimum years of schooling. This is to give an idea as to the strength of the education gradient in health for the sub-populations impacted by the reforms. The estimates confirm the finding in the wider literature that education is positively (negatively) associated with health (mortality). Columns (2) and (3) present our Reduced Form (RF) and 2SLS (IV) results respectively using our DiD identification strategy. Columns (4) and (5) present our RF and IV RD based results respectively.

Table 5: Regression results: OLS estimates and reform effects on overall mortality.

	OLS (1)	RF-DiD (2)	IV-DiD (3)	RF-RD (4)	IV-RD (5)
PANEL A: Females and Males					
8 Year Reform Impact	-0.0167***	-0.0003	-0.0011	0.0027	0.0119
	(0.0013)	(0.0022)	(0.0082)	(0.0025)	(0.0112)
9 Year Reform Impact	-0.0100***	-0.0003	-0.0005	-0.0016	-0.0041
	(0.0006)	(0.0011)	(0.0021)	(0.0014)	(0.0034)
PANEL B: Females					
8 Year Reform Impact	-0.0163***	0.0024	0.0106	0.0029	0.0139
	(0.0017)	(0.0029)	(0.0131)	(0.0033)	(0.0161)
9 Year Reform Impact	-0.0099***	0.0001	0.0002	-0.0022	-0.0064
	(0.0007)	(0.0014)	(0.0028)	(0.0017)	(0.0048)
PANEL C: Males					
8 Year Reform Impact	-0.0096***	-0.0028	-0.0088	0.0026	0.0104
	(0.0019)	(0.0031)	(0.0097)	(0.0039)	(0.0159)
9 Year Reform Impact	-0.0063***	-0.0009	-0.0016	-0.0010	-0.0022
	(0.0008)	(0.0017)	(0.0031)	(0.0018)	(0.0042)

Notes: This table presents the OLS, reduced form and 2SLS regression estimates on mortality. Mortality is modelled using an LPM of death by 2013. Sample sizes/No. of deaths: Panel A, 8 year reform (534,403/71,640); Panel A, 9 year reform (1,247,808/120,382); Panel B 8 year (264,237/28,613); Panel B 9 year (613,317/47,531); Panel C 8 year (270,166/43,723); Panel C 9 year (634,491/72,851). Each coefficient estimate is from a separate regression. Column (1) is the association of years of education with mortality controlling for year of birth and the sample is limited to those not treated and with pre or post reform minimum years of schooling. Columns (2) and (3) are reduced form and 2SLS regression estimates using our DiD specification which includes birth cohort and municipality fixed effects, municipality level time trends and a bandwidth of up to 10 years. Columns (4) and (5) are reduced form and 2SLS regression estimates using regression discontinuity and have separate second polynomials in the running variable either side of the cut-off and a full set of dummy variables for month of birth, control for gender and bandwidth of up to 10 years. Robust standard errors clustered by municipality level (for DiD) and by the running variable (for RD) in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

In Panel A of table 5 the results are for all individuals. The reduced form estimates for the 8 year reform are -0.03 percentage points using DiD and 0.27 percentage points using RD and are much smaller than the OLS correlations. The standard errors rule out even moderately large sized effects. The IV estimates for the 8 year reform are -0.11 percentage points using DiD and 1.2 percentage points using RD (relative impacts of -0.8% and 8.9% respectively) with corresponding 95 percent confidence interval for DiD of -1.5 to 1.4 percentage points and using RD of -1 to 3.3 percentage points. The OLS estimate of column (1) is both larger and more negative than both IV point estimates and lies outside both the DiD and RD 95% confidence intervals. Testing for endogeneity of years of schooling in the OLS estimates, we reject the OLS estimates based on the RD IV results but not using the DiD results.[18] The 9 year reform reduced form impacts are similar in magnitude to those found for the 8 year

[18]The test used is a test based on difference-in-Sargan statistics (C-statistic).

reform but the standard errors are half the size compared to those from the 8 year reform regressions. As noted in section 4.1 there are strong arguments for not using the 9 year reform as an IV, but if we do we reject the OLS estimates using DiD but not RD.

In panels B and C of table 5 we split the results by gender as there are both biological and social differences between the genders that could potentially lead to different health responses to the reforms. The OLS correlations are stronger for females than for males but in general, we find no clear gender specific differences in our reduced form estimates or our causal IV estimates.

5.1.2 All cause mortality sensitivity analysis

In table 6 we present alternative estimates to the LPM results of table 5, this time based on Cox regression, modelling the proportional hazard function of the probability of dying in the next period. In column (1) of table 7 we confirm the LPM findings of table 5, that there is a significant positive association between education and health and that this is stronger for females. The reduced form estimates using Cox proportional hazard regression for all cause mortality (columns 2 to 5) mirror the LPM findings; we find no significant impact of either reform on mortality, that the impacts are very close to zero and there are no discernible differences in response to the reforms between the genders.

In the appendix, tables B.4 and B.5, we present sensitivity analysis that assesses the robustness of the results to choice of bandwidth and removal of trends in the LPM and the Cox DiD analysis and find the conclusions are unaffected by these modelling choices. In our analysis of the impact of the reforms on education in section 4.2 we found there to be measurement error in our reform assignment and a positive jump in years of schooling for individuals one year too old and that this was much larger for the 9 year reform. This lead to a large discrepancy between the RD estimates and the DiD estimates, but including a doughnut in our regressions (removing the t-1 cohort) removed the difference between the RD and DiD estimates. Our LPM and Cox DiD and RD estimates are in absolute terms very close to one another. However we test if controlling for the t-1 cohort affects our conclusions in column (8) of tables B.4 and B.5 in the appendix and the conclusions remain the same for the 8 year reform and for the 9 year reform using DiD. Using RD we find that our LPM results show a mortality reducing effect, however not for the Cox regression results suggesting

this is not robust to modelling strategy. We conclude that we find no evidence of a causal effect of years of schooling on mortality and that this general result is robust to modelling choices, type of school reform and identification strategy.

Table 6: Cox proportional hazard estimates of survival till 2013

		8 year reform		9 year reform	
	COX-PH	COX-DiD	COX-RD	COX-DiD	COX-RD
	(1)	(2)	(3)	(4)	(5)
Females and Males					
Reform Impact	0.9150***	0.9905	1.0136	0.9888	0.9925
	(0.0059)	(0.0169)	(0.0190)	(0.0130)	(0.0160)
N	138,460	534,403	534,403	1,247,808	1,247,808
No. Deaths	30,633	71,640	71,640	120,382	120,382
Females					
Reform Impact	0.9013***	1.0112	1.0285	0.9970	0.9828
	(0.0094)	(0.0296)	(0.0319)	(0.0206)	(0.0254)
N	67,737	264,237	264,237	613,317	613,317
No. Deaths	12,105	28,613	28,613	47,531	47,531
Males					
Reform Impact	0.9561***	0.9790	1.0039	0.9821	0.9989
	(0.0080)	(0.0196)	(0.0264)	(0.0167)	(0.0176)
N	70,723	270,166	270,166	634,491	634,491
No. Deaths	18,528	43,027	43,027	72,851	72,851

Notes: This table presents the impact of the compulsory school reforms on cause specific mortality. Each coefficient estimate is from a separate regression. Column (1) is from a Cox proportional hazard regression of the impact of years of schooling for those not treated and with pre or post reform minimum years of schooling. Columns (2) and (4) are regression results from out DiD specification which includes birth cohort fixed effects, municipalities that reformed in the same year level time trends stratified by municipality and a bandwidth of up to 10 years. Columns (3) and (5) are RD estimates and have separate second polynomials in the running variable either side of the cut-off and a full set of dummy variables for month of birth and gender and a bandwidth of up to 10 years. Robust standard errors clustered by municipality level (for DiD) and by the running variable (for RD) in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

5.1.3 Cause specific mortality by 2013

To test whether the reforms had competing impacts by cause of death that potentially offset each other or whether diseases more amenable to health related behaviours, specifically cancer, circulatory diseases and external causes show a response to the reforms we consider some leading causes of mortality by 2013. Figure 6 presents the raw data for the average within municipality relationship between birth cohort and cause specific mortality and there are no discernible jumps at the reform cut-offs.

In table 7 we present the Cox independent competing risks regression estimates for leading causes of mortality. The RF Cox estimates presented in columns (2-5) are all smaller than those found in column (1). For both the 8 year and 9 year reforms we find no cause specific

impacts. The potential exception is the impact of the 9 year reform on deaths due to other causes. However, we only find a significant positive impact using DiD and a negative but insignificant impact using RD. It is therefore not robust to identification strategy.

Table 7: Cox proportional hazard independent competing risk results: Impact of the reforms on causes of mortality

		8 year reform		9 year reform	
	COX-PH	COX-DiD	COX-RD	COX-DiD	COX-RD
	(1)	(2)	(3)	(4)	(5)
Cancer					
Reform Impact	0.9526***	1.0221	1.0167	1.0232	0.9851
	(0.0103)	(0.0291)	(0.0280)	(0.0215)	(0.0289)
No. Deaths	11,831	28,255	28,255	45,079	45,079
Circulatory disease					
Reform Impact	0.8659***	0.9603	0.9776	1.0077	0.9747
	(0.0109)	(0.0334)	(0.0395)	(0.0298)	(0.0403)
No. Deaths	8,091	16,792	16,792	26,086	26,086
External causes					
Reform Impact	0.9199***	0.9578	0.9403	0.9483	0.9917
	(0.0258)	(0.0535)	(0.0690)	(0.0336)	(0.0464)
No. Deaths	1,527	5,650	5,650	13,834	13,834
All other causes					
Reform Impact	0.9101***	0.9772	1.0449	0.9399***	1.0270
	(0.0108)	(0.0291)	(0.0354)	(0.0218)	(0.0307)
No. Deaths	9,184	20,943	20,943	35,383	35,383
N	138,460	534,403	534,403	1,247,808	1,247,808

Notes: See notes for table 6
Source: SIP. Own calculations.

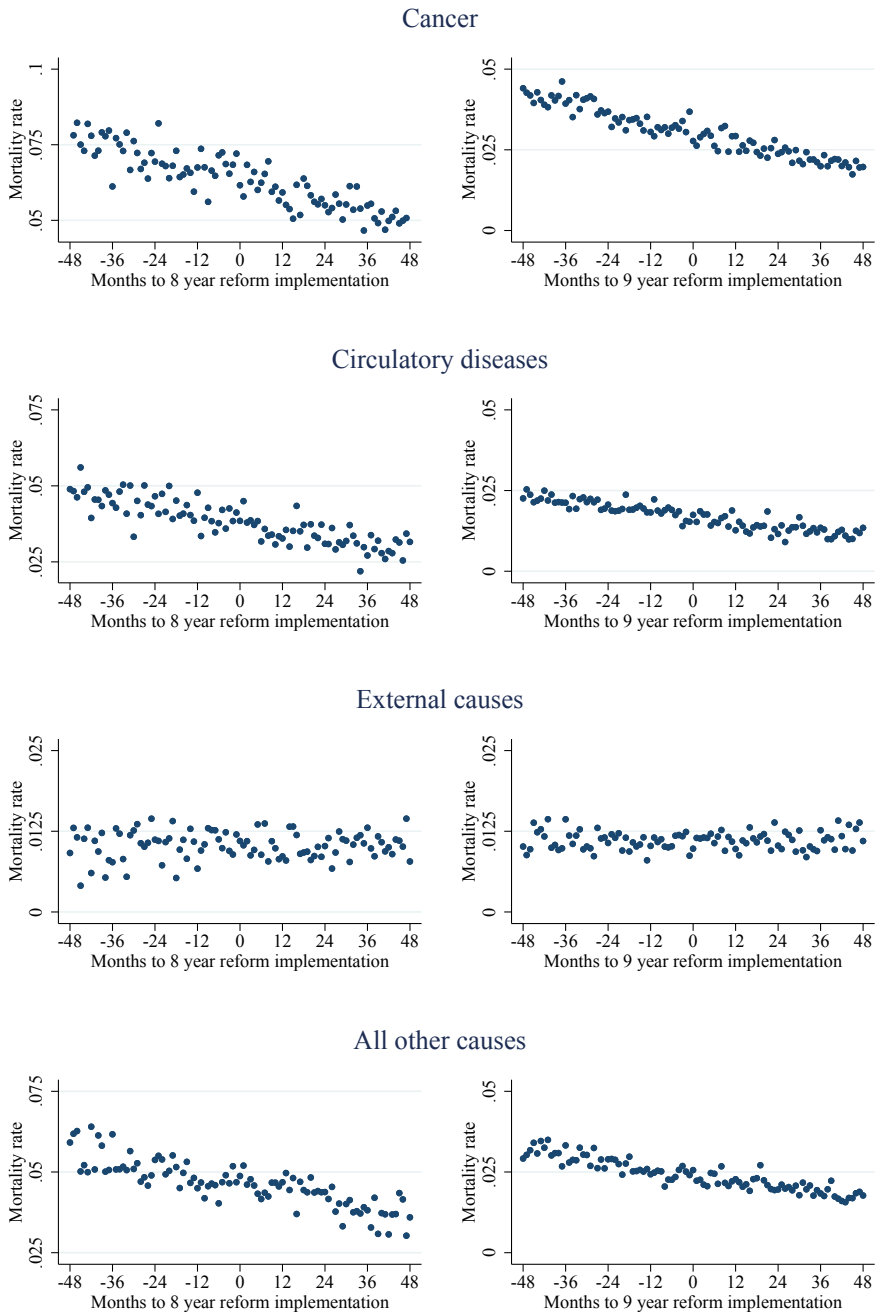

Fig. 6: Impact of the reforms on cause specific mortality by 2013

Notes: Scatter plots of cause specific mortality rate by age in months measured as the age difference of each individual from the first birth cohort in their municipality to be impacted by the reform (the first cohorts to be impacted are at zero).
Source: SIP. Own calculations.

5.2 Hospital admissions

5.2.1 All cause days admitted to hospital

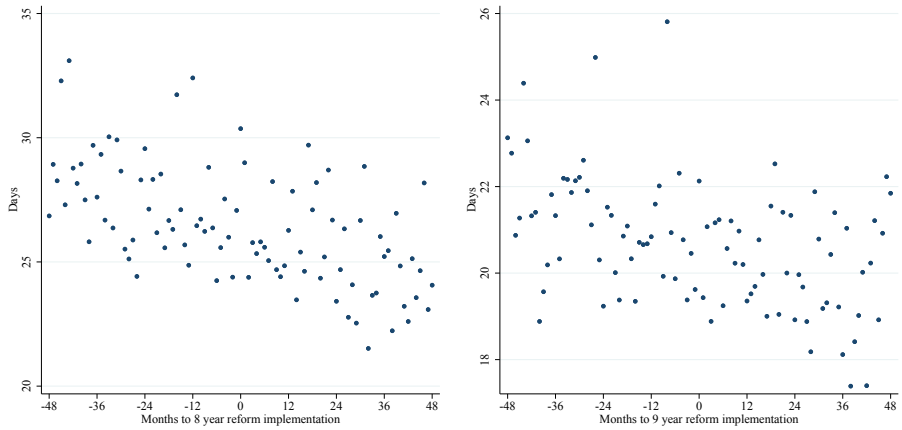

Fig. 7: Impact of the reforms on days admitted to hospital by 2012

Notes: This figure plots the relationship between birth cohort and first cohort impacted by the reform and days admitted to inpatient care (a stay over-night). See notes for figure 5.
Source: SIP. Own calculations.

In this section we assess the impact of the school reforms on a potentially more sensitive measure: inpatient hospital admissions. Specifically, days admitted to inpatient hospital care and cause specific inpatient hospital admissions are considered. In the previous section we found no impact on overall mortality over the observation period. This means we can assess other health measures without concern for mortality impacts affecting our results. Figure 7 presents the relationship between age relative to the first reform cohort and days admitted to hospital. We find no visually discernible impact of the reforms on days admitted to hospital at the reform cut-offs (0 represents the first cohorts in the municipality impacted by the reforms).

In table 8 we present the regression results for days in hospital for both reforms. Column (1) of table 8 shows the simple association of years of education for those not treated and with pre or post reform minimum years of schooling. There is a substantial and highly significant negative relationship between years of schooling and days admitted to hospital. The OLS results suggest that for an additional year of schooling, individuals will have about 1.5 fewer days at hospital, or a 6 to 7% reduction. The 8 year reform reduced form point estimates

using both DiD and RD are found in columns(2) and (4) and are equal to 0.27 and 0.85 days for DiD and RD respectively (relative impacts of 1.1% and 3.4% respectively) and are insignificant, small and positive. The 8 year reform IV point estimates are 1 and 3.7 days for DiD and RD respectively and also insignificant (increases of 4.1% and 14.9% respectively). The reduced form point estimates for the 9 year reform are -0.07 and 0.2 days for DiD and RD respectively and insignificant. The corresponding IV estimates report increases of -0.6% and 2.4% and are substantially smaller than the OLS correlations. Although both our 8 year and 9 year reform based IV estimates are quite different to the OLS estimates, we are unable to reject the OLS estimates.

5.2.2 Cause specific hospital admissions

Hospital admissions can occur for a variety of reasons and differences in education levels may push the quantity of medical care use in different directions. For example particular admissions due to health shocks are potentially more likely amongst those who have invested less in their health which may be a function of having received less education. On the other hand admissions that are in themselves health investments such as screening and examinations or preventative care or early detection may be a function of having received more education. To assess if there are counteracting effects of education we consider three leading reasons for hospital admission: cancer, circulatory diseases and external causes. In figure 8 we present the raw data as scatter plots of the probability of admission due to specific causes by age relative to the first cohort impacted by the reform in each municipality. Eyeballing the data, there are no obvious jumps at the reform implementation cut-offs in the hospitalisation rates by cause.

In table 8 column (1) we see that the association of years of education is negative and significant for circulatory diseases, external causes and all other causes but not for cancer. We test for jumps using RD in table 8 alongside DiD regression and our reduced form estimates (columns (2) and (4)). The results using the 8 year reform find no significant impact of the reform on the probability of inpatient care due to the specific causes we consider except for external causes, where using RD our IV estimate of -1.6 percentage points is nearly four times as large as our OLS estimate and twice as large as our DiD IV estimate. The results using the 9 year reform find evidence of an impact of the reform on the probability of inpatient

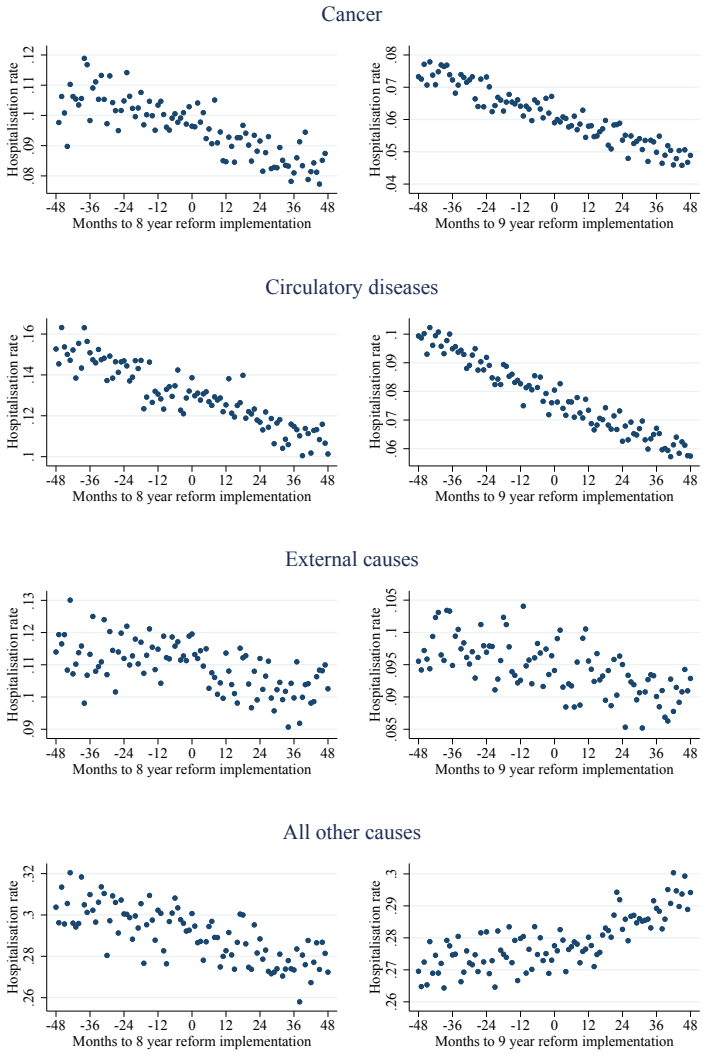

Fig. 8: Impact of the reforms on probability of hospital admission by cause by 2012

Notes: Scatter plots of the probability of hospital admission by age in months measured as the age difference of each individual from the first birth cohort in their municipality to be impacted by the reform (the first cohorts to be impacted are at zero).
Source: SIP. Own calculations.

care, through a reduction in other causes related admissions of -1.5 percentage points using our IV estimate. We should note that we make no adjustment for multiple hypothesis testing here and any attempt to do so would remove any significance we have found here. In general we do not find convincing evidence of an impact of education on cause specific inpatient care

that is robust to identification strategy.

5.2.3 Sensitivity analysis of impact on hospital admissions

In the appendix table B.6, we test the sensitivity of these results to bandwidth choice and inclusion and removal of linear trends in the DiD regression specification. In table B.6 we estimate the reduced form results for days of inpatient care of table 8 varying the bandwidth between 2 to up to 12 years (see columns (1) - (7) in table B.6). Compared to the results for mortality and years of education, the inpatient days results are more sensitive to bandwidth choice but the choice of a 10 year bandwidth does not impact the conclusions. The exclusion of municipality specific linear trends in our DiD analysis makes little difference to the estimates. Inclusion of a dummy for the t-1 cohort increases the coefficient size across all model types and both reforms, but also the standard errors. In summary we conclude that there is no evidence of an impact of either reform on hospital admissions.

Table 8: Linear regression results: Impact of the reforms on inpatient hospital admissions

	Mean (1)	OLS (2)	RF-DiD (3)	IV-DiD (4)	RF-RD (5)	IV-RD (6)
Panel A: 8 Year Reform						
Days at hospital	24.71	-1.4768***	0.2745	1.0083	0.8469	3.6832
		(0.3283)	(0.4855)	(1.7969)	(0.7659)	(3.3420)
Probability of hospital admission due to:						
Cancer	0.08	-0.0008	0.0004	0.0014	0.0003	0.0014
		(0.0010)	(0.0017)	(0.0064)	(0.0017)	(0.0074)
Circulatory diseases	0.11	-0.0101***	0.0022	0.0082	0.0024	0.0106
		(0.0012)	(0.0017)	(0.0064)	(0.0024)	(0.0107)
External causes	0.10	-0.0045***	-0.0023	-0.0085	-0.0036**	-0.0158**
		(0.0010)	(0.0019)	(0.0071)	(0.0018)	(0.0078)
Any other cause	0.29	-0.0128***	-0.0034	-0.0125	-0.0028	-0.0121
		(0.0015)	(0.0026)	(0.0095)	(0.0035)	(0.0151)
N		138,460	534,403	534,403	534,403	534,403
Panel B: 9 Year Reform						
Days at hospital	22.17	-1.6405***	-0.0707	-0.1354	0.2070	0.5287
		(0.1684)	(0.4180)	(0.7986)	(0.4980)	(1.2650)
Probability of hospital admission due to:						
Cancer	0.07	0.0005	-0.0004	-0.0007	-0.0009	-0.0024
		(0.0004)	(0.0009)	(0.0018)	(0.0011)	(0.0029)
Circulatory diseases	0.09	-0.0089***	0.0017*	0.0033*	0.0006	0.0015
		(0.0005)	(0.0010)	(0.0020)	(0.0014)	(0.0035)
External causes	0.10	-0.0051***	0.0005	0.0009	-0.0001	-0.0002
		(0.0005)	(0.0012)	(0.0023)	(0.0016)	(0.0040)
Any other cause	0.29	-0.0110***	-0.0010	-0.0019	-0.0061**	-0.0156***
		(0.0007)	(0.0017)	(0.0033)	(0.0024)	(0.0060)
N		397,961	1,247,808	1,247,808	1,247,808	1,247,808

Notes: This table presents the impact of the compulsory school reforms on inpatient hospital admissions. All coefficients are from separate regressions. See notes for table 5.

5.3 Health and health related behaviours

In this section we consider the impact of schooling on health outcomes and health related behaviours. These health measures are arguably more sensitive to the potential mechanisms in which education may influence health compared to mortality and hospital visits. So whilst mortality and hospital visits are outcomes that are objectively measured and available for the whole population, they require major health events to occur making them relatively insensitive measures of the impact of education on health. Even though we have found no impact on mortality or hospitalisations up to the age of 75, it is still possible that we may see an impact in more sensitive measures such as health behaviours or in self-reported measures of current health.

In table 9 we present the regression estimates of the school reforms on various self-reported

health outcomes and health related behaviours using our survey data. Column (1) shows the simple OLS correlation estimates of years of education on health for those untreated and with years of education equal to or one more year than the legal minimum. Education is found to be associated with lower probability of having fair or bad health and a lower probability of being obese for our population. The cohorts we use in this analysis therefore exhibit the same positive education gradient in self-reported health and health related behaviours that is observed for mortality and hospital admissions in this paper and that has also been observed more widely in the literature.

The reduced form estimates are found in columns (2) and (4) of table 9. These are modelled in the same way as for mortality and hospital admissions with the addition of dummies for survey year but the removal of municipality specific trends. Even though we observe a strong and significant effect of both reforms on years of education we find significant impacts of neither the 8 year reform nor the 9 year reform on health or health behaviours.

Focussing on the health variables where there is a significant correlation observed in column (1) between education and health (fair or bad health and obesity), we find that the 8 year reform based IV results show quite large relative drops and in the same direction as the OLS estimates. Our DiD and RD based IV results for fair and bad health report relative drops of -30% and -13% respectively and are large in comparison to the OLS results of a relative effect of -6%. For obesity our DiD and RD based IV results find relative impacts of -47% and -6% respectively. However, our estimates are not precisely estimated and this is with over 30 years of survey data.[19]

The reduced form estimates for the 9 year reform using both DiD and RD on *fair or bad health* imply small positive effects of the reform which is in contrast to the OLS regression estimates. Incidentally, our results indicate that the findings of Spasojevic (2010) who finds a causal impact of the 9 year reform on self-reported health are not robust to model specification. Whilst we are using a different dataset, we mirror her analysis on our dataset. In her paper she only controls for cohort fixed effects (our column 1 results) and the results in column (2) show that controlling for differences across municipalities which are also correlated to background characteristics (see table 4) explain away her findings.

We test the sensitivity of these results to using a smaller bandwidth, found in the appendix,

[19]The 8 year reform is a strong instrument using RD but not quite so strong using DiD. See table B.2 in the appendix.

table B.7, and the results show our conclusions remain the same. We find no clear impact of the reforms on measures of self-reported health or health behaviours.

Table 9: Education effects on self-reported health and health behaviours

	OLS (1)	RF-DiD (2)	IV-DiD (3)	RF-RD (4)	IV-RD (5)
Panel A: 8 Year Reform					
Fair or bad health	-0.019*	-0.021	-0.063	-0.017	-0.028
	(0.010)	(0.018)	(0.056)	(0.027)	(0.043)
N	2,691	8,181	8,181	8,181	8,181
Mean	0.31	0.21	0.21	0.21	0.21
Smoke daily	-0.008	0.001	0.004	-0.004	-0.006
	(0.009)	(0.021)	(0.061)	(0.026)	(0.041)
N	2,661	8,138	8,138	8,138	8,138
Mean	0.24	0.26	0.26	0.26	0.26
Obese	-0.029***	-0.022	-0.052	-0.005	-0.007
	(0.009)	(0.019)	(0.048)	(0.030)	(0.035)
N	1,683	4,996	4,993	4,996	4,996
Mean	0.15	0.11	0.11	0.11	0.11
Anxiety, concern etc.	-0.004	-0.007	-0.018	-0.029	-0.033
	(0.011)	(0.022)	(0.055)	(0.032)	(0.037)
N	1,776	5,388	5,385	5,388	5,388
Mean	0.17	0.15	0.15	0.15	0.15
Panel B: 9 Year Reform					
Fair or bad health	-0.018***	0.005	0.010	0.004	0.012
	(0.004)	(0.011)	(0.021)	(0.016)	(0.045)
N	12,866	19,124	19,122	19,124	19,124
Mean	0.28	0.19	0.19	0.19	0.19
Smoke daily	-0.007*	-0.011	-0.021	-0.012	-0.032
	(0.004)	(0.014)	(0.025)	(0.016)	(0.045)
N	12,741	19,033	19,030	19,033	19,033
Mean	0.27	0.26	0.26	0.26	0.26
Obese	-0.010***	0.001	0.002	0.013	0.036
	(0.003)	(0.011)	(0.020)	(0.012)	(0.033)
N	7,935	11,514	11,496	11,514	11,514
Mean	0.13	0.09	0.09	0.09	0.09
Anxiety, concern etc	-0.001	-0.015	-0.026	0.009	0.026
	(0.004)	(0.014)	(0.023)	(0.018)	(0.054)
N	8,468	12,487	12,472	12,487	12,487
Mean	0.16	0.15	0.15	0.15	0.15

Notes: This table presents the impact of compulsory school reforms on self-reported health and health behaviours. See notes for table 5. Note that a full set of dummy variables for survey year are included in all regressions and DiD regressions are modelled without municipality trends.
Source: ULF-Survey. Own calculations.

6 Discussion

Our findings show that across two major school reforms that led to clear and substantial increases in years of education we observe only small and generally insignificant changes in mortality and other measures of health. Our IV point estimates for the 8 year reform find that an additional year of education yields a -0.01 and 1.2 percentage point change in mortality using DiD and RD respectively and the lower bound of our confidence intervals allows for a 1.7 percentage point reduction in mortality.

The research that most closely aligns to that of ours is that of Lleras-Muney (2005), Mazumder (2008) and Clark and Royer (2013). Our IV estimates are much smaller than those of Lleras-Muney (2005) and are more in line with the findings of Clark and Royer (2013) for Britain, and Mazumder (2008) for the USA. The findings of Clark and Royer (2013) are potentially the most convincing evidence gathered so far. However, there are concerns that the results of Clark and Royer (2013) are based upon two reforms in Britain that impacted very different cohorts (1947 and 1972) and both reforms were implemented overnight nationwide. The cohorts are likely to have been very different, both in terms of their own characteristics but also in terms of the health and labour market structures they were exposed to and there may have been large general equilibrium effects of a nationwide roll out of increased compulsory schooling that potentially reduce the earnings effect of these reforms. Our results from Sweden are based on two reforms, different in design, that were rolled out over time and with overlapping cohorts and evaluated using two different identification strategies and we still find only small or zero impacts on health. Our findings together with those of Clark and Royer (2013) suggest that the timing of the reforms and the nature of their roll out has little bearing on the results. Our own findings are also consistent across the two reforms and suggest it is not specific features of the Swedish compulsory school setting that explain the Swedish results because the two reforms were in fact quite different.

Whilst our findings for the 9 year reform are similar to those of Meghir et al. (2017), who also study the impact of the 9 year reform on mortality in Sweden, our results add a large sense of robustness to their findings. We have shown that previous estimates of the impact of schooling have been downward biased because of the measure of years of education used and in fact the 9 year comprehensive school reform had a much greater effect on years of education than previous estimates suggest. We have also extended the analysis of the 9 year

reform to include self-reported measures of health and health related behaviours. The final and leading contribution of our paper is that we introduce another school reform that allows us to instrument the effect of education on health. The 9 year reform has been argued to not be a pure years of schooling reform (Meghir and Palme, 2005) and therefore analysis using the 9 year reform has to be kept to considering the reduced form impacts. This is not to say that analysis of the 9 year reform is not of interest, it is just that the theories that we are testing often relate to years of education (Grossman, 2015).

The results of this paper and of Meghir et al. (2017) stand in contrast with previous Swedish research of Lager and Torssander (2012) and Spasojevic (2010) who find small health improving impacts of the 9 year reform. In the appendix C we have attempted to replicate the results from Lager and Torssander (2012) who found a small but significant reduction in mortality due to the 9 year reform. Unfortunately we are unable to replicate their results exactly, but the analysis highlights how our conclusion is robust to their reform assignment and a different observation sample. We have also shown that we can repeat the significant finding of a causal impact of the 9 year reform on self-reported health outcomes of Spasojevic (2010) using a different and larger dataset. However, this result is not robust to a DiD or RD identification strategy.

The nature of our study is that we have been able to pin down quite a few variables that potentially explain the differences in impacts of compulsory school reforms found in the literature. We start with two reforms that both have a substantial impact on years of schooling. The reforms themselves were also different to each other and were rolled out in a way that concerns about resource shocks such as lack of teachers and schools and even general equilibrium effects that may be the case with the reforms in Britain, for example, do not apply in the same way to the Swedish reforms. That both reforms were rolled out during similar time periods further implies that the cohorts exposed to the two reforms later acted under the same welfare and labour market institutions. The clean comparisons of the two reforms further strengthen the internal validity of our findings. We have used a large dataset that includes a whole range of health outcomes. Different econometric methods have been used to estimate the impact on mortality. Two identification strategies have been used to assess the sensitivity of estimating across different sub-groups and under different identifying assumptions. Our finding of no or small mortality impacts of education are robust to modelling

strategy, both LPM and Cox proportional hazard regression, identification strategy, either DiD or RD design, and school reform type.

The results presented here have strong internal validity. But how relevant are the findings outside of Sweden? Like the National Health Service (NHS) in Britain, the Swedish health care system has universal coverage and is publicly provided with access at the point of need paid for through taxation. Whilst universal provision removes the direct role of financial resources in determining health care quality, a channel which improved education could influence, there is still plenty of scope for the more educated to achieve better health. Any publicly provided health care system has to prioritise resources and more educated individuals are potentially more likely to be able to manipulate the system to their advantage, either through knowing how the system works, being more aware of the services that are available or through the ability to convince doctors of the need for treatment. Medical services also only in part determine health outcomes. Health behaviours and investments are also very important in determining health outcomes and these are also potentially impacted by education through better understanding and knowledge. Financial resources may also play a role in determining our health behaviours and health investments. To understand how education may impact health we need to understand which of these potential channels are channels that matter and this means the results of this paper, due to their high internal validity, are of importance.

The reform cohorts we have considered in this paper were born across the span 1938-1954. Like Clark and Royer (2013) who considered the school reform of 1947, some of these cohorts were impacted by the Second World War (WWII). Children in Britain during the war were moved out of the big cities and lived with members of the extended family or even volunteers in the countryside. Sweden was neutral during WWII and was much less affected in general and there were no specific policies to move children out of the large cities.[20] So whilst WWII was an unusual time, it is unlikely to have impacted the external validity of the results drawn here because life for children in Sweden during the war was largely unaffected.

We argue therefore that our results are not specific to the cohorts we consider. The results also help us understand which economic channels, if any, education has an impact on health. Our result, that we are not able to identify an effect of education on mortality,

[20]During WWII it was possible for schools to cancel classes in case of a threat. However, any time lost had to be caught up later on. Also if a teacher was called for military service a substitute teacher had to be called in. Historical sources suggest no educational disruptions in Sweden during the period of WWII (see Bhalotra et al. (2016)).

hospital admissions or self-reported health, results that are internally very robust, is therefore an important finding and of relevance beyond Sweden.

7 Conclusion

The literature documenting the education gradient in health is vast yet the causal effect literature using compulsory school reforms as instruments for education has produced results that have been difficult to summarise. In this paper we have been able to pin down many of the potential explanations for differences in results across studies and have shown using a large dataset with a long follow-up period that the causal impact of education on mortality is small. This is also true for hospital admissions and other health measures. For mortality we can rule out impacts of years of education larger than -1.7 percentage points. These results hold across econometric technique and identification strategy. We argue that the conclusions we draw from the results of this paper are not specific to the Swedish context, rather they have a more general relevance.

Compulsory school reforms have provided a powerful way to assess the causal impact of education on health. They often impact a large population and can provide exogenous variation in years of schooling under certain parametric assumptions. They also impact a sub-population that is often a public health policy focus: the lowly educated who live short lives and have poorer health outcomes generally. However, the mounting evidence suggests compulsory schooling laws as policy levers for public health improvements may not be very effective. It is important to note that we do not conclude that education has no impact on health outcomes. Evidence has been found that compulsory schooling reforms can have an impact on health across generations (Lundborg et al., 2014). There is also hope found further up the education ladder, where evidence using the Vietnam draft as an instrument to incentivise college attendance in the USA (Buckles et al., 2016; Grimard and Parent, 2007; De Walque, 2007) has found a mortality reducing impact and an improvement in health related behaviours. Experience from the compulsory schooling literature suggests the results from the Vietnam draft induced college attendance need to be replicated for other populations, health outcomes and identification strategies before any firm conclusions can be drawn however.

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Appendix A - Background to the Swedish school reforms

In this appendix we provide some background information on the Swedish school system, the two school reforms and their interrelation used in this paper.

A.1 The Swedish School System

Long before compulsory schooling was introduced by law on a nationwide level in Sweden, a large fraction of the population had basic reading and writing capabilities, and a notable share of all parishes had introduced some kind of primary school on a voluntary basis (Lindmark, 2015). Regulations announced in the mid to late 19th century came to imply both the right to cost-free primary schooling and an obligation to take part in the schooling offered (Fredriksson, 1971). Specifically the 1882 legal statue of *Folkskolan* stated that every parish had to offer primary schooling by an approved teacher, that school attendance was compulsory for all children, and that children should start primary school the year they turn seven years old (Edgren, 2011).

The country was divided into school districts (generally corresponding to a parish, and later a municipality) and the local school board was responsible for the organization of elementary education. To overcome differences in content and format across school districts, a national central education plan was introduced in 1919 (Paulsson, 1946). These guidelines were published by the Ministry of Ecclesiastical Affairs and included time tables and syllabuses for compulsory schooling. The ministry also appointed school inspectors responsible for yearly evaluations of a number of school districts (Fredriksson, 1971). Completion rates were high and more than 90 per cent of all pupils finished compulsory schooling with full curriculum (Fredriksson, 1950).

In the 1920s elementary schooling in Sweden was compulsory for six years, but the central education plan provided curricula also for seven years of schooling and in 1920 a clause was introduced in the primary school code (paragraph 47 mom. 4) that stated a seventh school year could be made compulsory in a school district (Fredriksson, 1950). At the time Sweden applied a tracking system, where good performing students (defined by an assessment) could select to switch to an academic educational track and study at a four or three year long junior secondary school (*Realskola*) after the fourth or the sixth year of elementary education, respectively. The alternative was to continue and finish basic compulsory schooling. Attending junior secondary school allowed students to continue to higher secondary school (*Gymnasium*) which was a prerequisite for University. During the first half of the twentieth century, the Swedish school system was highly selective and the vast majority of people only completed compulsory education Björklund et al. (2004).[21]

In 1936 the national Government decided that a seventh year of schooling should be compulsory. The law came into force on July 1, and the decision to extend compulsory schooling by an extra year was taken by the school board of the school district. It was stipulated that seven years of schooling had to be implemented across the whole country over a twelve-year period, before 1949 (Fischer et al., 2013). The reform was seen as a new epoch, especially among teachers, because previously Folkskola had remained the same since 1882,

[21] See e.g.Centralbyrån (1977) for yearly numbers of students matriculating to lower and higher secondary schooling. For the cohorts of interest in this study matriculation to lower secondary schooling increased over time (16 percent of cohort 1938 and 30 percent of cohort 1951).

offering six years of compulsory education (Folkskollärarförbund, 1949).[22] With the bill of 1936, school districts were also allowed to introduce an eight year of compulsory schooling, but for this they needed to send in a formal application and to be given the king's consent.

A.2 The 8 Year Reform

With the start of the World War II, the Swedish political debate came to place a large focus on how to best foster democratic members of society. More education was seen as one of the main components for fulfilling this goal (Edgren, 2011). Thus, despite the on-going national implementation of the seventh year of compulsory schooling, a reform work was initiated and assigned to a new expert commission (*Skolutredningen* later replaced by *Skolkommissionen*) in 1940. This was the first governmental commission with a real mission to investigate primary and secondary education together, and with an aim to replace the tracking system with a unified comprehensive school system tying compulsory schooling closer to secondary education.[23]

Between 1940-1948 the commission continuously released reports evaluating the current school system and developing proposals and guiding principles for the future compulsory school (Marklund, 1982). Although the main focus of the commission's work was to postpone tracking decisions to higher grades, and by that improve equality of opportunity, and despite the on-going implementation of the seven-year compulsory schooling, there were also continued efforts to further extend compulsory schooling within the old system. The commission's work discussed an eight year extension of *Folkskola*, and in 1945 the Minister of Ecclesiastical Affairs proposed a bill introducing compulsory eight year schooling (without changing tracking options), but no action was taken by the Government (Fredriksson, 1971).

As stated above one of the main arguments for extending compulsory education was *democratic fostering*. This motive was not new. The 7 year reform was also motivated by fostering democracy including universal suffrage which was argued to place great demands on members of society, wherefore a solid education is necessary (Ecklesiastikdepartementet, 1935). The on-going war made this argument even more important in the debate. Specifically an eight-year extension was believed to improve student performance with respect to elementary skills in reading, writing and math, but also other subjects. An extension would also allow for the introduction of foreign languages (English) as a subject. In addition to theoretical arguments an extension was further justified by social and ethical arguments and that an eight-year could fill a supportive and nurturing role for young people that have not established on the labour market (SOU, 1945).

A second argument for extending compulsory education was induced by international *benchmarking* -- that Sweden was lagging behind (Waldow, 2013). Compared to other countries few students matriculated to higher levels of education, and the time spent in compulsory education was still quite modest. For example, compulsory education in the US endured at least until age 16, in Germany there were Volkschule or Hapuptschule until the age of 15 and in the UK students generally had nine years of compulsory schooling in the late 1930s.

[22] For a detailed review of the background and the implementation of the seventh compulsory year, see Fischer et al. (2013).

[23] Since the 1890's there had been a quite heated debate about the rational of the the so-called parallel system where student took different tracks. The main argument in the debate for that all students should have to complete the very same basic education before continuing to secondary schooling, was that it created inequalities (Morawski, 2010).

A third argument for extending compulsory schooling was the *increasing specialization of the labour market* and the *increased complexity* of society and societal life, implying a need to significantly increase educational goals of Folkskolan. Finally, the economic and societal *duality* that existed between urban and rural areas was brought forward to motivate a general 8 year reform or a compulsory school reform. Specifically with respect to education, the rural areas of the country was falling behind, e.g. smaller shares of students matriculating to junior secondary education in rural compared to urban areas (Centralbyrån, 1977). With a general implementation of the 8 year reform such differences could decrease.

The main arguments of the proponents of the 8 year reform to why the realization of an eight-year extension was seen as preferred compared to a comprehensive school reform was that there was (i) no large demand from students nor from the parties of the labour market for a 9 year comprehensive reform, and that (ii) the supply of teachers was too limited for a comprehensive reform, but also that the teachers generally had too limited education (SOU, 1945).

Likely spurred by the political debate some municipalities applied and got consent from the king and took the opportunity to implement a mandatory eight-year of Folkskola (Folkskollärarförbund, 1943).[24] The first two municipalities to implement an eight mandatory year were Kävlinge and Mariestad in the school year of 1941/42. The number of municipalities offering an eighth year gradually increases in the next-coming decade: In 1946/47 there were 33 and in 1958/59 207 municipalities, respectively. A characteristic of the municipalities introducing a mandatory eight-year in this time period is that they were urban and most of the larger cities of Sweden were early birds in this development. Consequently a quite large share of all students in the country had eight years of compulsory schooling: in the school year 1948/49 this was 16 per cent and in the school year 1951/52 this was 25 per cent (Folkskollärarförbund, 1952)

All municipalities introducing the eighth year followed the *main form* curriculum requiring full time reading and a teacher with an appropriate teacher degree.[25] Normative and binding curricula regarding the eight-year were missing in the early period, but the curriculum and hourly plan presented in the proposal of Skolutredningen in 1946 generally became the norm for the school districts that implemented an eight-year of Folkskola. The mandatory subjects in the eight grade were the same as in seventh grade, but local preferences could to some extent be met (Fredriksson, 1971).

A.3 The 9 Year Reform

In 1948, the expert commission proposed to replace the compulsory primary and the junior secondary school with a nine-year compulsory comprehensive school. The expert commission however wanted to evaluate the new school form before introducing it to all schools across the country. The reform was therefore introduced during an assessment period where the 9 year comprehensive school was introduced in different locations at different points in time.

[24] Only in a few cases a municipality did not get the permission to implement the extension. The reason was that the district asked to do an isolated change and only introduce the change in a separate school in a municipality (Fredriksson, 1971).

[25] The alternative to the main form were *exception forms*, characterised by half time reading or that the teacher did not have an appropriate teachers degree. In the early 1940's more than 90 percent of all pupils in Sweden went to a school that were assigned to the main forms (Fredriksson, 1950).

Starting from 1949/1950 the 9 year reform was rolled out at the municipality level.[26] For the first year of the roll-out of the reform 14 municipalities are selected to participate in the assessment. [27] The evaluation period was not run as a random experiment, but the National School Board chose the areas from a group of applicants to form a representative set based on observable municipality characteristics. Municipalities participating in the early assessment period were compensated with earmarked money from the central government for the increased costs following the expansion of mandatory education (Holmlund, 2008). After the assessment period, the national parliament decides to permanently introduce the 9 year reform to all schools the country in 1962. Seven years later, by 1969, all municipalities were obliged to have the new comprehensive school running (Marklund, 1982) and *Folkskolan* was fully disconuated.

The reform reshaped the entire school system and compared to the old tracking system students were kept in the same school type for nine years. Besides extending compulsory education from seven or eight to nine years and postponing tracking, the educational reform also came with a change in the national curriculum implying English and civics became a compulsory subject, but there were no major changes to the total number of hours or the distribution of hours taught in different subjects (Richardson, 1992).

The educational reform was also pedagogical. The commission proposal of of 1948 was very clear on that the traditional school and its working methods were obsolete. Specifically whole-group teaching and questions-response methods should be replaced by more individualized and activating elements, pandering students drive and independence (Marklund, 1982).[28]

Based on the principles of the final report of *Skolkommissionen* a new educational plan for schools to follow is released 1962 (Lgr 62). The pedagogical key concepts of the plan are individualization and activity learning (Larsson, 2011). The pedagogical fundament on the special position of the individual and that the school should foster independent individuals did not meet any major objections (Marklund, 1989). However the first reform municipalities experienced difficulties in getting accurate work material and text books (Marklund, 1982).

A.4 Comparing the Two reforms

Based on the above it is evident that Sweden experienced a continuous roll-out of extending the compulsory amount of schooling from 6 to 9 years over a period of 40 years, and that the 8 year reform and the 9 year reform were implemented across overlapping cohorts. On average the 8 year and the 9 year reform was 7 years apart in a municipality.

Both reforms introduced change in the extent of compulsory schooling. As regards the exact definition of treatment it however seems that the two reforms differ somewhat. Treated

[26]The comprehensive school system is introduced throughout the whole municipality, or in certain schools within a municipality. At the time there were 1037 municipalities in Sweden.

[27]Municipalities had to show interest in the reform and also report on various issues, such as e.g. population growth, local demand for education, tax revenues and school situation, and all municipalities that took part in the first year of assessment were required to have eight year comprehensive schooling. The 14 first-movers were selected out of 144 municipalities.

[28]The emphasis on the importance of the need of new working methods can also be assigned to the aim that education should foster democratic societal members. As discussed by Richardson (1978) there is also a change regarding the view of the individual in the late 1940's. The development of the individual now matters more than the societal development. An essential feature of the report by the commission is that that the school should be more pupil centred and less subject-matter oriented. Another novel perspective is the view that parents, not the school, are responsible for the pupil.

students of the 8 year reform faced no significant school system changes, nor any changes in working methods in class. Thus, any effects from the 8 year reform should mainly be driven by changes in the amount of time spent in education.[29] With the abolishment of the tracking system the 9 year reform implied a fundamental change of the complete school system and the reform also came with a new curriculum program and methods. Any effects from the 9 year reform can thus be driven by changes in the amount of time spent in education, by that the new system kept students together in the same school until the ninth grade, and/or changes in curricula, working methods and pedagogics.

As discussed above schools and teachers initially faced some problems in that they lacked appropriate teaching materials corresponding to the new curricula and teaching methods of the comprehensive school. According to Marklund (1982) teachers degrees of freedom with respect to novel and open-ended activities were also limited by that students and parents that wanted *Realskola* but instead had to undergo compulsory schooling in the comprehensive system, translated their ambitions and goals for the former school type to the latter. Together this suggest that the first part of the 9 year reform likely was more similar to the 8 year reform. Also the first period of the 9 year reform was more similar to the previous school system in the sense that most schools still streamed students into different classes according to their choices regarding languages or vocational training and harder and easier courses in some subjects (Marklund, 1982).

The two reforms were gradually implemented across municipalities. The timing of implementation in individual municipality was based on a mixture of local and national decisions. As regards the wider institutional context, we are unaware of any reforms that might have coincided with the 8 year or the 9 year school reform at the local level. During the assessment period of the 9 year reform it was only municipalities that showed interest in the reform that could be selected implying reform implementation was not random. Previous studies suggest that 9 year reform was implemented earlier in municipalities with higher incomes and with higher average education, see e.g. Lundborg et al. (2014). Regarding the 8 year reform the early-birds tended to be more urban and most of the larger cities implemented a mandatory eight year. Smaller municipalities followed and in the end more than half of all municipalities had introduced a mandatory 8th grade before implementing the comprehensive 9 year school reform.

A.5 Reform data and Validation

The reform data for the 9 year reform was generously shared by Helena Holmlund and we rely on a dataset as used in Hjalmarsson et al. (2015), of which an earlier version is described in detail in Holmlund (2008). The dataset encompasses information on the year a specific school district introduced the new comprehensive school.

While the 9 year reform has previously been used in several economic applications, this paper is the first to use the 8 year reform and the reform data on the timing of the year of introduction of the eight year in each municipality was purposively collected from archives and digitized by the authors. Various official sources provide aggregate information on the development of the implementation on the 8 year reform. To check the accuracy of the gathered reform data we perform checks to confirm that the collected information conform with aggregate official statistics. For example, information on the share of school districts in the

[29] See e.g. discussion by Orring et al. (1962) on that all earlier reforms than the 9 year reform more or less left the fundamental work of schools unaffected.

country that had eight years of compulsory schooling in certain years from Skolöverstyrelsen (1955) and from Centralbyrån (1977), respectively, suggest our data conform with aggregate statistics.

The decision to introduce eight years of compulsory schooling was made on the municipal level, and the assumption is that schools within the same district implemented the reform in the same year. Theoretically there could however be discrepancies between schools within municipalities. We believe the assumption is valid since official sources state that the change generally applied to a whole district (see e.g. Fredriksson (1971) and Skolöverstyrelsen (1955)). Moreover, aggregate figures on the share of all students taking on the extra year of compulsory education in certain years (Centralbyrån, 1977) suggest that there should be no major deviations from this rule.

Appendix B Online tables and figures

Table B.1: ICD codes used to define causes of death and hospitalisation

Diagnosis	ICD 10 code	ICD 9 code	ICD 8 code	ICD 7 code
Cancer	C00-D48	140-239	140-239	140-239
Circulatory disease	I	390-459	390-458	400-468
External causes	S,T,V,W,X,Y	800-999,E800-999	800-999,E800-999	800-999,E800-999

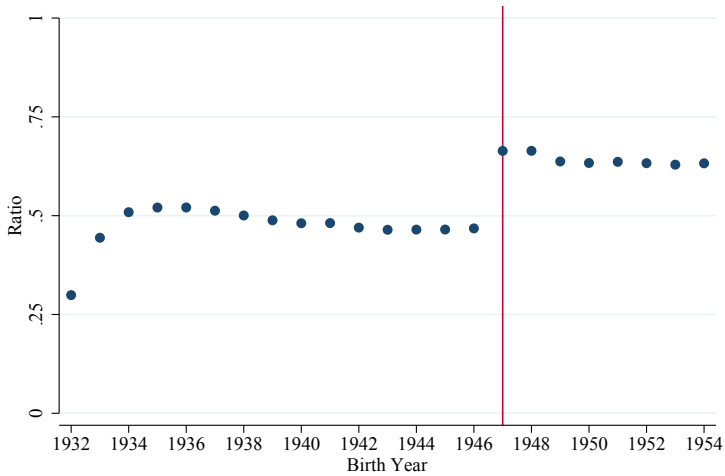

Fig. B.1: Correlation between place of birth and place of residence over time

Notes: Scatter plots of correlation between place of birth and place of residence as recorded in the 1960 census over time. The vertical line at 1947 indicates when place of birth was changed from being recorded as municipality of the hospital to being recorded as place of residence of the mother.
Source: SIP. Own calculations.

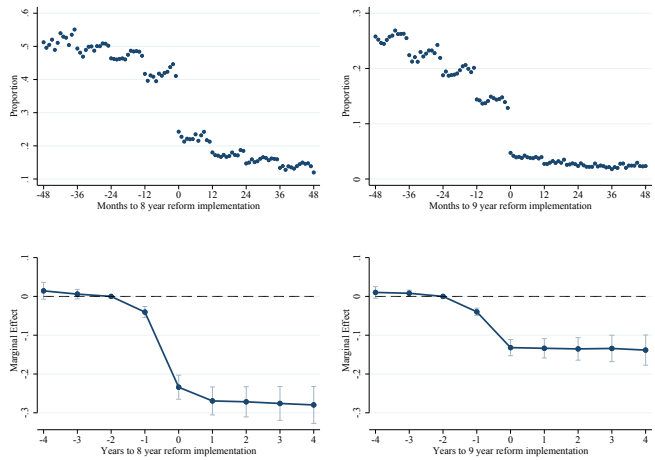

Fig. B.2: Impact of the reforms on leaving school with 7 years of old primary school

Notes: Top two panels: Scatter plots of proportion with 7 years of old primary schooling by age in months measured as months to reform implementation in their municipality where the first cohort impacted is at zero. The bottom two panels: plot regression coefficients of an individual's birth year relative to the first reform cohort in their municipality on proportion with 7 years of old primary schooling (spikes represent the 95% confidence interval for each coefficient estimate). Municipality and birth year fixed effects and municipality level time trends are controlled for, a bandwidth of 10 years is used and clustered standard errors are estimated at the municipality level. Category 4 is four or more years after the first reform cohort. The reference category is "two years before the first reform cohort" (t-2).
Source: SIP. Own calculations.

Table B.2: Compulsory schooling reforms' impact on education (ULF survey)

	(1)	(2)
	8 YEAR REFORM	9 YEAR REFORM
Difference in Difference	0.333***	0.530***
	(0.124)	(0.074)
F-stat	7.20	50.90
N	8,200	19,153
Regression Discontinuity	0.627***	0.355***
	(0.160)	(0.105)
F-stat	15.45	11.56
N	8,200	19,153

Notes: This table shows the impact of the 8 year and 9 year school reforms on years of education. Each coefficient is from a separate regression by reform, method and group. The DiD specification includes birth cohort, survey year and municipality fixed effects and an observation window of up to 10 years before and after the first cohort impacted by the reform. Regression discontinuity estimates have separate second polynomials in the running variable either side of the cut-off and a full set of dummy variables for month of birth, survey year and gender. Robust standard errors clustered by municipality level (for DiD) and by the running variable (for RD) are in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: ULF-Survey. Own calculations.

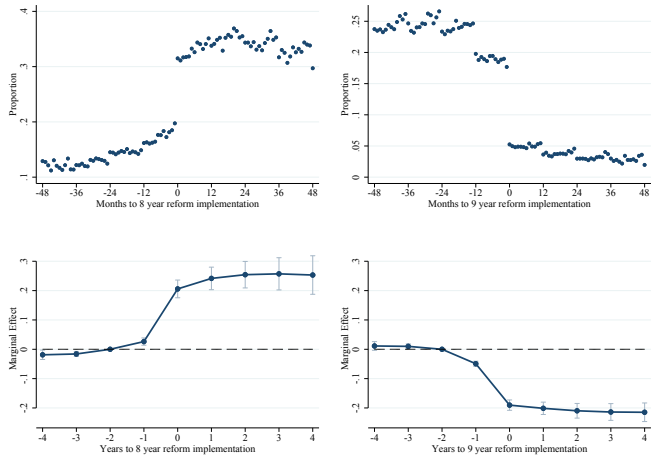

Fig. B.3: Impact of the reforms on leaving school with 8 years of old primary school

Notes: Top two panels: Scatter plots of proportion with 8 years of old primary schooling by age in months measured as months to reform implementation in their municipality where the first cohort impacted is at zero. The bottom two panels: plot regression coefficients of an individual's birth year relative to the first reform cohort in their municipality proportion with 8 years of old primary schooling. See notes for table B.2
Source: SIP. Own calculations.

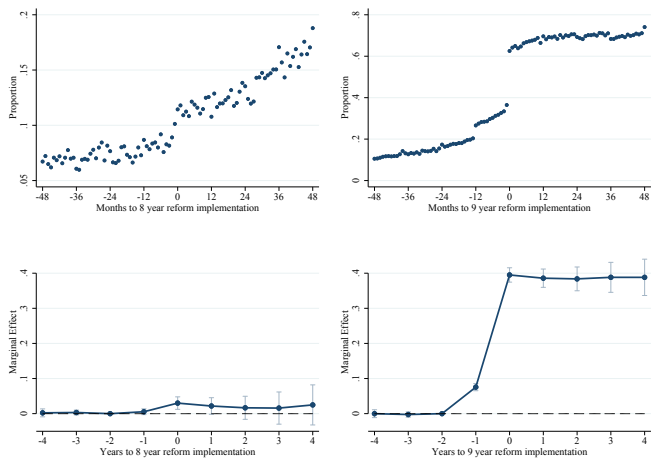

Fig. B.4: Impact of the reforms on leaving school with 9 years of old primary school or 9 years of comprehensive school

Notes: Top two panels: Scatter plots of proportion with 9 years of old primary schooling or new comprehensive schooling by age in months measured as months to reform implementation in their municipality where the first cohort impacted is at zero. The bottom two panels: plot regression coefficients of an individual's birth year relative to the first reform cohort in their municipality on 9 years of old primary schooling or new comprehensive schooling. See notes for table B.2
Source: SIP. Own calculations.

Table B.3: Sensitivity analysis: Reforms' impact on education

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: 8 Year Reform								
DiD	0.223***	0.221***	0.245***	0.271***	0.281***	0.292***	0.033	0.310***
	(0.031)	(0.027)	(0.023)	(0.024)	(0.024)	(0.025)	(0.021)	(0.027)
DiD with municipality trends	0.159***	0.218***	0.242***	0.264***	0.272***	0.281***	0.031	0.294***
	(0.033)	(0.025)	(0.022)	(0.023)	(0.023)	(0.024)	(0.020)	(0.025)
RD	0.264***	0.241***	0.240***	0.225***	0.230***	0.204***	0.017	0.285***
	(0.047)	(0.036)	(0.028)	(0.025)	(0.023)	(0.022)	(0.025)	(0.030)
N	143553	251958	352728	447649	534403	608642	534403	506676
Panel B: 9 Year Reform								
DiD	0.353***	0.438***	0.485***	0.511***	0.533***	0.546***	0.306***	0.630***
	(0.022)	(0.018)	(0.018)	(0.017)	(0.018)	(0.019)	(0.014)	(0.020)
DiD with municipality trends	0.259***	0.402***	0.460***	0.500***	0.523***	0.538***	0.299***	0.615***
	(0.024)	(0.018)	(0.019)	(0.018)	(0.019)	(0.019)	(0.014)	(0.022)
RD	0.194***	0.231***	0.298***	0.357***	0.392***	0.435***	0.249***	0.529***
	(0.031)	(0.020)	(0.021)	(0.022)	(0.023)	(0.024)	(0.015)	(0.021)
N	332517	599339	856337	1070486	1247808	1384702	1247808	1180536
Bandwidth (years)	2	4	6	8	10	12	10	10
Admin. data based educ. measure							✓	
Drop $t-1$ cohorts								✓

Notes: This table presents sensitivity analysis of the impact of the reforms on years of education. Model (5) is as in table 3 and Models 1 to 6 are as model (5) but vary the bandwidth. Model (7) is as (5) but uses the administrative data based measure of years of schooling as used in prior research. Model (8) is as model (5) but is a sandwich/donut estimator, removing individuals up to one year too old to be eligible for the reform. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

Table B.4: Sensitivity analysis: LPM estimates of impact of the reforms on mortality by 2013

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: 8 Year Reform								
DiD	0.0016	0.0017	0.0018	0.0012	0.0005	-0.0001	-0.0010	0.0001
	(0.0035)	(0.0031)	(0.0027)	(0.0024)	(0.0021)	(0.0021)	(0.0020)	(0.0024)
DiD with municipality trends	0.0024	0.0004	0.0012	0.0002	-0.0000	-0.0003	-0.0015	-0.0001
	(0.0043)	(0.0034)	(0.0028)	(0.0025)	(0.0022)	(0.0022)	(0.0022)	(0.0027)
RD	-0.0078*	-0.0005	0.0000	0.0018	0.0012	0.0027	0.0067***	0.0042
	(0.0040)	(0.0040)	(0.0034)	(0.0032)	(0.0028)	(0.0025)	(0.0026)	(0.0037)
N	143553	198598	251958	352728	447649	534403	608642	534403
Panel B: 9 Year Reform								
DiD	0.0020	0.0022	0.0013	0.0001	-0.0006	-0.0002	0.0001	-0.0002
	(0.0019)	(0.0016)	(0.0015)	(0.0013)	(0.0012)	(0.0011)	(0.0010)	(0.0012)
DiD with municipality trends	0.0021	0.0024	0.0013	-0.0004	-0.0010	-0.0003	0.0001	-0.0005
	(0.0023)	(0.0018)	(0.0016)	(0.0014)	(0.0012)	(0.0011)	(0.0011)	(0.0012)
RD	-0.0014	-0.0007	0.0016	0.0031*	0.0018	-0.0016	-0.0041***	-0.0057***
	(0.0020)	(0.0019)	(0.0019)	(0.0016)	(0.0015)	(0.0014)	(0.0014)	(0.0020)
N	332517	466287	599339	856337	1070486	1247808	1384702	1247808
Bandwidth (years)	2	3	4	6	8	10	12	10
Dummy for t-1 cohorts								✓

Notes: This table presents sensitivity analysis of the LPM estimates of the reforms on death by 2013. Model (5) is as in table 5. See notes for table B.3. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Source: SIP. Own calculations.

Table B.5: Sensitivity analysis: Cox proportional hazard regression results of the reforms' impact on mortality

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: 8 Year Reform								
DiD	1.0155	1.0104	1.0114	1.0020	0.9942	0.9918	0.9856	0.9882
	(0.0296)	(0.0256)	(0.0225)	(0.0196)	(0.0172)	(0.0169)	(0.0163)	(0.0194)
DiD with municipality trends	1.0077	0.9995	1.0064	1.0003	0.9937	0.9905	0.9851	0.9894
	(0.0338)	(0.0264)	(0.0222)	(0.0197)	(0.0171)	(0.0169)	(0.0163)	(0.0194)
RD	0.9497*	1.0012	1.0074	1.0159	1.0119	1.0136	1.0210	1.0093
	(0.0297)	(0.0305)	(0.0261)	(0.0245)	(0.0210)	(0.0190)	(0.0185)	(0.0253)
N	143553	198598	251958	352728	447649	534403	608642	534403
Panel B: 9 Year Reform								
DiD	1.0266	1.0195	1.0061	0.9890	0.9839	0.9922	0.9982	0.9864
	(0.0245)	(0.0207)	(0.0182)	(0.0160)	(0.0143)	(0.0130)	(0.0126)	(0.0139)
DiD with municipality trends	1.0130	1.0148	1.0030	0.9872	0.9827	0.9888	0.9887	0.9844
	(0.0279)	(0.0216)	(0.0187)	(0.0160)	(0.0142)	(0.0130)	(0.0127)	(0.0138)
RD	0.9769	0.9906	1.0138	1.0269	1.0043	0.9925	0.9905	0.9754
	(0.0244)	(0.0234)	(0.0229)	(0.0199)	(0.0182)	(0.0160)	(0.0154)	(0.0192)
N	332517	466287	599339	856337	1070486	1247808	1384702	1247808
Bandwidth (years)	2	3	4	6	8	10	12	10
Dummy for t-1 cohorts								✓

Notes: This table presents sensitivity analysis of the Cox PH estimates of the reforms on survival till 2013. Model (5) is as in table 7. See notes for table B.3. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$
Source: SIP. Own calculations.

Table B.6: Sensitivity analysis: Reforms' impact on inpatient days admitted to hospital

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: 8 Year Reform								
DiD	-1.0262	-0.5604	0.4691	0.7164	0.3372	0.2160	0.5121	0.4649
	(1.2257)	(0.9418)	(0.6379)	(0.5575)	(0.5086)	(0.4776)	(0.4835)	(0.6359)
DiD with municipality trends	-1.0365	-0.9562	-0.0454	0.4792	0.3770	0.2745	0.5651	0.6246
	(1.5446)	(0.9809)	(0.6723)	(0.5860)	(0.5329)	(0.4855)	(0.4839)	(0.6596)
RD	1.0865	0.2052	-0.4837	-0.2628	0.4911	0.8469	0.2609	1.9432*
	(1.1899)	(0.9675)	(0.9021)	(0.8472)	(0.8229)	(0.7659)	(0.7066)	(0.9906)
N	143553	198598	251958	352728	447649	534403	608642	534403
Panel B: 9 Year Reform								
DiD	0.4357	0.6149	0.0387	-0.0503	0.0622	-0.0561	-0.0273	0.0784
	(0.7895)	(0.7215)	(0.6081)	(0.5374)	(0.4671)	(0.4192)	(0.3890)	(0.4658)
DiD with municipality trends	-0.3990	0.6067	0.1252	0.0280	0.0887	-0.0707	-0.0244	0.0958
	(0.8890)	(0.7354)	(0.6252)	(0.5397)	(0.4661)	(0.4180)	(0.3877)	(0.4594)
RD	1.4492*	0.4467	0.1299	0.1559	0.1706	0.2070	-0.0244	1.1018*
	(0.7926)	(0.9088)	(0.7857)	(0.6385)	(0.5507)	(0.4980)	(0.4647)	(0.6199)
N	332517	466287	599339	856337	1070486	1247808	1384702	1247808
Bandwidth (years)	2	3	4	6	8	10	12	10
Dummy for t-1 cohorts								x

Notes: This table presents sensitivity analysis of the regressions estimates of the reforms on hospital admissions by 2013. Model (5) is as in table 8. See notes for table B.3. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Source: SIP. Own calculations.

Table B.7: Sensitivity analysis: Education effects on self-reported health and health behaviours for different bandwidth choice (5 year bandwidth)

	OLS (1)	RF-DiD (2)	IV-DiD (3)	RF-RD (4)	IV-RD (5)
Panel A: 8 Year Reform					
Fair or bad health	-0.028**	-0.017	-0.049	0.002	0.003
	(0.013)	(0.022)	(0.063)	(0.039)	(0.047)
N	2,857	5,047	5,045	5,047	5,047
Mean	0.31	0.22	0.22	0.22	0.22
Smoke daily	-0.012	0.006	0.017	-0.051	-0.060
	(0.010)	(0.023)	(0.061)	(0.031)	(0.037)
N	2,827	5,024	5,022	5,024	5,024
Mean	0.25	0.26	0.26	0.26	0.26
Obese	-0.025***	-0.011	-0.027	0.013	0.012
	(0.009)	(0.023)	(0.058)	(0.038)	(0.034)
N	1,799	3,092	3,070	3,092	3,092
Mean	0.14	0.11	0.11	0.11	0.11
Anxiety, concern etc	-0.004	-0.008	-0.021	-0.029	-0.025
	(0.010)	(0.028)	(0.067)	(0.036)	(0.033)
N	1,894	3,322	3,307	3,322	3,322
Mean	0.17	0.15	0.15	0.15	0.15
Panel B: 9 Year Reform					
Fair or bad health	-0.020***	0.006	0.011	-0.021	-0.079
	(0.004)	(0.014)	(0.027)	(0.016)	(0.074)
N	13,868	13,049	13,036	13,049	13,049
Mean	0.27	0.18	0.18	0.18	0.18
Smoke daily	-0.005	-0.025	-0.047	0.003	0.012
	(0.004)	(0.018)	(0.034)	(0.022)	(0.079)
N	13,731	12,988	12,975	12,988	12,988
Mean	0.27	0.28	0.28	0.28	0.28
Obese	-0.010***	0.006	0.014	0.014	0.071
	(0.003)	(0.015)	(0.033)	(0.017)	(0.090)
N	8,553	7,697	7,639	7,697	7,697
Mean	0.13	0.09	0.09	0.09	0.09
Anxiety, concern etc	-0.001	-0.006	-0.013	0.010	0.044
	(0.003)	(0.017)	(0.036)	(0.021)	(0.095)
N	9,126	8,417	8,363	8,417	8,417
Mean	0.16	0.15	0.15	0.15	0.15

Notes: This table presents the impact of compulsory school reforms on self-reported health and health behaviours. A 5 year bandwidth is used in DiD and RD regressions instead of 10 years as used in 9 to assess sensitivity to bandwidth choice. See notes for table 5.
Source: ULF-Survey. Own calculations.

Appendix C Reconciliation with Lager and Torssander (2012)

Our results differ to those of Lager and Torssander (2012) who also look at the impact of the 9 year reform on mortality in Sweden. They found a small and statistically significant mortality reducing effect of the 9 year comprehensive school reform for the population aged 40 plus. There are two obvious differences between our paper and theirs. In their paper they make some adjustments to reform assignment based on the observation that a large proportion of individuals in certain municipalities had the minimum years of schooling one or two years before the reform was officially implemented and adjust the reform assignment accordingly. The other major difference is that they have data only up to 2007 and make some slightly different sample restrictions. In table C.1 rows 1 to 4 we move slowly between our main results towards their specification, reform assignment and sample selection. Column (1) is for the sample aged 40 plus and column (2) is for all ages. In row 2 we, like them, model the hazard without trends. This has next to no impact on the coefficients or the standard errors. In row 3 we use their reform assignment code that they kindly shared with us. This increases the impact slightly. In row 4 we attempt to replicate the sample restrictions of Lager and Torssander (2012) (cohorts born between 1943-1955, observed in 1960 and 1965 censuses, followed up to 2007, not emigrated). The results are essentially the same as for row 3 but the standard errors have increased. Column 5 are the actual results from Lager and Torssander (2012) where they find a small but significant reduction in mortality due to the school reform. Their result is not substantially different from our row 4 replication, but we are unable to repeat their significant finding exactly or their sample size. What we can conclude is that the results of this current paper are robust to reform assignment methodology and sample selection and suggest that the impact of education on health is really quite small.

Table C.1: Replication of Lager and Torssander (2012): Cox proportional hazard estimates

	9 Year Reform	
	From aged 40	All ages
	(1)	(2)
Row 1*: own sample, own reform assignment, trends*		
9 year reform	0.9826	0.9888
	(0.0132)	(0.0130)
Row 2*: As per Row 1, no trends*		
9 year reform	0.9862	0.9922
	(0.0134)	(0.0130)
Row 3*: As per Row 2, LT (2012) reform assignment*		
9 year reform	0.9794	0.9859
	(0.0136)	(0.0133)
N	1,242,843	1,247,808
No. Deaths	115,417	120,382
Row 4*: As per Row 3, LT (2012) sample restrictions*		
9 year reform	0.9769	0.9896
	(0.0194)	(0.0160)
N	1,280,550	1,304,807
No. Deaths	73,909	98,166
Row 5*: Results from LT (2012)*		
9 year reform	0.96	0.98
	[0.93-0.99]	[0.95-1.01]
N	1,200,519	1,247,867
No. Deaths	65,329	92,351

Notes: This table presents the impact of the 9 year compulsory school reform on mortality. See text for details. Robust standard errors clustered by municipality level in parentheses. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. 95% confidence intervals in brackets.

Paper III

Could easier access to university improve health and reduce health inequalities?

Gawain Heckley, Martin Nordin and Ulf-G Gerdtham*†

Abstract

This paper estimates the impact of university education on medical care use and its income related inequality. We do this by exploiting an arbitrary university eligibility rule in Sweden combined with regression discontinuity design for the years 2003-2013 for students who graduated 2003-2005. We find a clear jump in university attendance due to university eligibility. This jump coincides with a positive jump in prescriptions for contraceptives for females but also a positive jump in mental health related hospital admissions for males. Analysis of the inequality impact of tertiary eligibility finds no clear impact on medical care use by socioeconomic status of the parents. The results imply that easing access to university for the lower ability student will lead to an increase in contraceptive use without increasing its socioeconomic related inequality. At the same time, the results highlight that universities may need to do more to take care of the mental health of their least able students.

Keywords: Health returns to education, demand for medical care, causes of health inequality, Regression Discontinuity Design, Concentration Index

JEL Classification: *I14, I23, I24, I26*

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1 Introduction

The relationship between education and health is of fundamental interest and has consequently received a great deal of empirical attention. This literature finds its theoretical origins in the demand for health model of Grossman (1972) and more recently Grossman (2000). These models include health as part of an individual's human capital and they emphasise that health capital is not only determined by medical care but also potentially other factors such as knowledge capital, commonly proxied by years of education. The education gradient in health is observed in nearly every country (Mackenbach et al., 2003; Van Doorslaer and Koolman, 2004) and has prompted some to focus on education as a means of raising health levels and reducing socioeconomic related disparities in health (Marmot, 2005; Marmot et al., 2010, 2012).

In this paper we investigate whether access to university education for the student at the margin of university eligibility (and therefore of relatively low ability) shows improved health. For this group we observe a significant association between university attendance and frequency of hospital admissions and the number of prescriptions prescribed. The concern with any association of education and health is that the relationship may be due to reverse causality. In human capital models, our initial endowment of human capital affects our ability to invest further in our human capital, which means those with poor health and therefore low levels of health capital are less able to invest in their knowledge capital. As a consequence, the associations noted widely in the literature may just be due to health's impact on education. There may also be a third hard to observe variable that explains both our knowledge capital and our health capital. This could be some form of innate ability as suggested by **?** or time preferences as suggested by Fuchs (1982) where those who prefer today much more than tomorrow are more likely to consume their human capital early.

A review of the recent empirical research investigating the causal link between education and health by Grossman (2015) finds that there is either a positive impact or a zero or very small impact. This is illustrated if we consider the recent quasi-experimental evidence that uses changes to the compulsory education system as instruments. Research in the US (Lleras-Muney, 2005), in Germany (Kemptner et al., 2011; Jürges et al., 2011), in Italy (Atella and Kopinska, 2014), in the Netherlands (Van Kippersluis et al., 2011) and in France (Etilé and Jones, 2011) has found a positive impact on health. Other studies of education system changes in Britain (Clark and Royer, 2013; Braakmann, 2011), in France (Albouy and Lequien, 2009), in Germany (Pischke and Von Wachter, 2008), in Sweden (Lager and Torssander, 2012; Meghir et al., 2012) and in Denmark (Arendt, 2008) have found a small or no effect on health. Both Cutler and Lleras-Muney (2012) and **?** have suggested that the margin being estimated is very important for the interpretation of the results and is possibly the leading explanation for the large variation in results.

The impact of university education on health is one margin that has received relatively little empirical attention yet is of great potential interest. Cunha et al. (2010) have shown that there are potential complementarities between early and late life interventions. It is therefore useful to consider whether university education for low ability students can be effective in improving health outcomes. The evidence that does exist uses the Vietnam draft as a quasi-experiment and finds that university education reduces smoking initiation and increases cessation (De Walque, 2007; Grimard and Parent, 2007). It has also been found to lead to a reduction in mortality (Buckles et al., 2016).

In this paper we present new findings of the impact of university education by exploiting quasi-experimental variation caused by an arbitrary rule in Sweden that states that students must have a pass mark for at least 90% of their courses that make up a program in order to go on to university. This rule leads to a large jump in the proportion of students who go on to study at university of 8 to 9 percentage points (pp) for females and 2pp to 4pp for males. It is this arbitrary rule that allows us to identify the impact of university eligibility on various medical care use outcomes using Regression Discontinuity (RD) design. The marginal group affected by the eligibility rule are individuals who are towards the lower end of the education distribution (46th percentile and 42nd percentile for males and females respectively, who were enrolled on the academic stream at upper secondary school). The margin we estimate is therefore of particular policy interest because it captures the potential egalitarian impact of increasing access to higher education for lower ability individuals and or individuals from lower ranking socioeconomic groups.

Our results consider individuals who graduated from upper secondary school between 2003 and 2005. The data we use is based on population based administrative records of inpatient and outpatient hospital admissions (2003-2013) and prescriptions (2005-2013) linked using a personal identifier to education records. The results show that university eligibility leads to a significant and substantial jump in university attendance. Previous research using the same eligibility rule (Nordin et al., 2017) has found that the impact of university eligibility on years of education is in the region of 0.2 to 0.3 years which is similar in scale to those found for a Swedish compulsory school reform (Hjalmarsson et al., 2015). We find that this jump in university eligibility leads to no clear overall impact on hospital admissions or prescriptions. However, when we consider specific cause of hospital admission and prescription receipt we find a positive jump in the probability of prescription receipt for contraceptives for females. For males we find an increase in hospital admissions for mental disorders and a reduction in prescriptions for pain relief related medicines.

We also consider the impact of university eligibility on socioeconomic related health inequality. This analysis is complementary to our analysis of the mean using OLS. There could quite plausibly be mean preserving effects on health that are correlated with socioeconomic status or even impacts just on socioeconomic status that change the covariance of health and socioeconomic status. This

analysis is a key contribution of the paper. Socioeconomic inequality in health has received a great deal of public health and political interest as witnessed by the large amount of work done by various health inequality commissions (Marmot, 2005; Marmot et al., 2010, 2012). The work of these commissions and by others (e.g., Kunst et al. (2005); Shkolnikov et al. (2011); Mackenbach et al. (2015); Hu et al. (2016)) has shown that socioeconomic related health inequalities are observed in almost every country and that not only have they persisted over time but they have even increased across most western countries.

The question we specifically address is: can we use access to university education as a policy lever to reduce the observed concentration of hospital care use or prescriptions amongst young adults from poorer or richer families? To this end we employ the novel decomposition technique of Heckley et al. (2016) and we find that university eligibility overall has no clear impact on the concentration of hospital admissions and prescriptions in young adults from either poorer or richer family backgrounds. Inequality increasing impacts are found for medical examinations at hospital amongst females but the effects are offset by males. Additionally, even though a clear impact was found for mean contraceptive prescription receipt, this jump did not coincide with a change in parental income related inequality of prescriptions for contraceptives.

Overall our findings suggest that increasing access to university should increase female contraceptive use and not have a detrimental impact on socioeconomic related health inequality. However, the increase observed in mental health hospital admissions for males just crossing the eligibility threshold suggests universities should do more to help their least able students with the pressures of university life.

The rest of the paper is structured as follows. In section 2 we introduce the Swedish education system and the eligibility rule we consider. In section 3 we introduce our measures of socioeconomic inequality. In section 4 we introduce the data material we use for the analysis and in section 5 we explain our empirical approach and test the identifying assumptions we make. Section 6 presents the results for medical care use and section 7 concludes.

2 The Swedish education system

In this section we briefly outline the Swedish educational system and the eligibility rule for university that we use to identify the impact of eligibility on medical care use.[1] In Sweden in order to be able to attend university a student needs to achieve eligibility through passing at least 90% of a full program at upper secondary school. This can also be achieved by completing complementary adult

[1]The system we describe here was in place between the years 1997 to 2010. During this period the system was slightly tweaked in 2003

studies after upper secondary school but the cut-off we consider is university eligibility as achieved at graduation from upper secondary school. We choose to use university eligibility defined at end of upper secondary school because it is a well-defined and hard to manipulate rule that leads to a jump in university attendance, as we shall show later. In general, upper secondary school is for three years and students start upper secondary school aged 16 and graduate at age 19.[2] There are two streams at upper secondary school: the academic stream with the explicit aim of going to university after graduating from upper secondary school and the vocational stream with an explicit focus of getting a job once graduated. In this paper we focus on students graduating from the academic stream because this is where the university eligibility threshold has largest bite (Nordin et al., 2017). Students can choose their preferred stream. A full program consists of 2500 course credits for both types of tracks.[3] To receive a diploma of eligibility for university a student needs to pass at least 90 percent of full program i.e. receive 2250 credits. A program is a sum of courses and courses can give either 50, 100, 150, 200 or 250 course credits (with some exceptions for even larger courses). The courses that make up a program are graded on four levels: fail, pass, pass with distinction and pass with special distinction. To receive the course credits, the student has to at least pass the course but the credits received are not impacted by how well one passes. We choose to investigate the period starting in 2003 because the diploma of eligibility for university is much more clearly defined compared to previous years.

In figure 1 we show the impact of barely passing the cut-off point on the probability of enrolling in university for cohorts graduating between 2003 to 2005. The raw data is graphed as scatter plots of the proportion who attended a first term course of university by the number of achieved credits at upper secondary school in bins of 2pp of a full program wide. The vertical dashed line represents the cut-off of university eligibility (2250=2500*0.9). Figure 1 is for men and women studying the academic track. In both figures, the probability of enrolling in university increases with the percentage completed of a full program and follows a smooth function. At the university eligibility cut-off, however, there is a positive jump in the probability of enrolling in university. From just eyeballing the data it can be seen that the probability of enrolling in university is around 10pp higher for females and there is potentially a small jump for males passing the marginal course on the academic track. Nordin et al. (2017) show that the jump for those on the vocational track is much smaller and is why we choose to focus on the academic track students. Note that the

[2]The large majority of students who complete their compulsory schooling choose to continue their studies at upper secondary school with only 1.7% of students choosing not to continue with their studies. Whilst all students are able to continue their studies at upper secondary school, there is an eligibility requirement. Those students who do not pass this eligibility requirement enter what is called an individual program with the aim to transfer to the standard upper secondary school program at some point.

[3]Whilst a large proportion of students went on to study at upper secondary school a large proportion end up dropping out: for the period under consideration in this paper the drop out rate is about 25%.

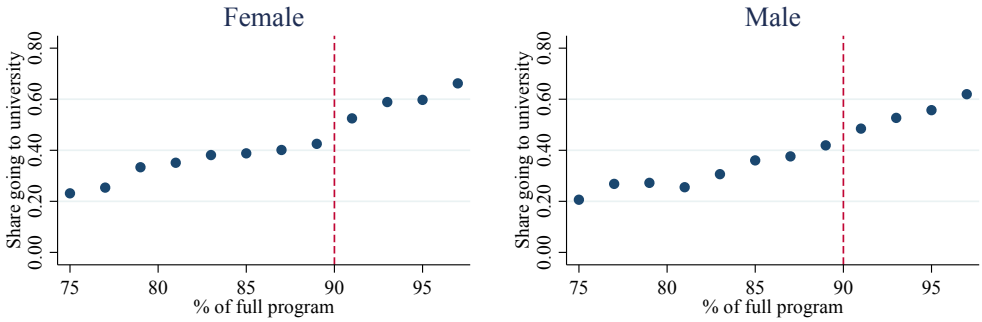

Fig. 1: Impact of university eligibility on university attendance by gender

Notes: This figure plots a scatter of the share who attended a first term of university against percentage completed of a full program with a bin width of 2pp of a full program (the size of the smallest course) in each bin for those graduating upper secondary school between the years 2003 and 2005. The cut-off for university is marked by the dashed vertical line at 90pp credits.

cut-off and forcing variable are defined at a point in time, graduation from upper secondary school. Even though students can achieve university eligibility after completing upper secondary school by complementing their studies to achieve university eligibility we still observe a jump using our definition of the cut-off. That is, students who fail to achieve university eligibility at completion of upper secondary school are less likely to go to university even though they could later on achieve eligibility by complementing their grades.

Students coming in to the final term of their upper secondary school program often have seven to eight courses of varying credit size to complete, the smallest worth just 2pp of a full program. A key identifying assumption for regression discontinuity of the eligibility threshold is that those at the margin of university eligibility will not have precise control over whether they cross the threshold. Given the typical course size is 4pp of a full program and that students often require about 32% of course credits in their final term in order to finish, a bandwidth of 4pp represents the impact on eligibility of just one course out of eight in the final term. Precise manipulation of the threshold would require the more motivated students to understand in advance how many courses they need to pass, and which particular courses they need to focus on in order to just cross the eligibility threshold, which appears quite a high stakes gamble. It would seem unlikely that students are willing to stake eligibility for university by focussing on just one or even two courses. The teachers grade the courses themselves and may also be aware that a particular student is near the eligibility threshold and mark up the grades for the marginal student so they achieve eligibility. This may happen, but for teachers to be able to manipulate the threshold *exactly* they need to know what the student is likely to achieve in the other seven or so courses they are enrolled in *and*

collude with the other teachers so that the marginal student crosses the threshold exactly but no more. This form of manipulation by the teachers then has to have a link between the students and the outcomes we consider, perhaps a preference towards the students with higher ability. The information requirements seem very onerous for this degree of collusion to happen so precisely. It is this lack of precise control that allows us to identify the impact of university eligibility on health and education outcomes.

3 Measuring health inequality

An explicit aim of this paper is to study the impact of university eligibility on medical care use inequality, specifically socioeconomic related medical care use inequality. It is our view that it is important that our inequality analysis yields results based on the full distribution of socioeconomic status and that they are comparable with future work.[4] Specifically, we want to know how university eligibility increases or decreases the concentration of health amongst the richest/poorest individuals. To this end, we use the health Concentration Index (CI) as our measure of socioeconomic health inequality, a measure popular in health economics. The health CI captures the degree to which health is concentrated in higher or lower ranking socioeconomic groups (Fleurbaey et al., 2011). The health CI considers two variables: a health variable and a socioeconomic ranking variable and yields an index that can vary continuously between minus 1 and plus 1. A CI value of minus 1 would relate to a situation where all hospital admissions are concentrated in the lowest income individual, 0 would be where hospital admissions are equally distributed across the income distribution, and 1 would be where all hospital admissions are concentrated in the highest income individual. That is negative values infer a pro-poor concentration of health, positive a pro-rich concentration.

More formally, health is represented by the random variable H with corresponding mean, μ_H, and socioeconomic status is represented by the random variable Y. Socioeconomic fractional rank is given by the Cumulative Distribution Function of Y, F_Y. There are many ways to formulate the health CI: one of them is as a weighted covariance between H and F_Y yielding:

$$v_{CI} = \frac{2}{\mu_H} COV(H, F_Y); \tag{1}$$

Erreygers and Van Ourti (2011) argue that use of the health CI is appropriate if the health variable is of ratio scale, which means it does not have a finite upper bound. For health variables not of ratio scale, such as binary variables, a modified version is preferred. We are interested in relative

[4]This is in principle the same argument that has been made for using the CI to compare across countries and over time (see for e.g. Wagstaff et al. (1991)) - it produces a standardised measure. In this sense, our estimated impacts will also be comparable across future studies who look at education's impact on CI.

inequality (a proportional change in everyone's health does not impact the index) and therefore follow Kjellsson et al. (2015) and consider two variants of the health CI, the attainment relative concentration index (ARCI) and the short-fall relative concentration index (SRCI):

$$v_{ARCI} = \frac{2}{\mu_h - a_H} COV(H, F_Y); \tag{2}$$

$$v_{SRCI} = \frac{2}{b_h - \mu_H} COV(H, F_Y); \tag{3}$$

where a_H is the lower bound of H and b_H is the upper bound of H.[5] The ARCI and SRCI are relative measures of socioeconomic related health inequality that yield different measures of inequality depending on whether we measure health in terms of attainments (from the lower bound) or in terms of short-falls (from the upper bound). Which one is preferred is up to the individual reader and therefore we present both.

We can capture the impact of university eligibility on the CI using the approach of Heckley et al. (2016). The results will tell us to what extent university eligibility increases or decreases the concentration of health amongst the richest/poorest individuals. This approach means we capture the inequality aspects of university eligibility on a measure that is comparable with future studies and considers the whole socioeconomic distribution (rather than say just comparing the lowest socioeconomic status group vs the highest).

[5] As Kjellsson and Gerdtham (2013) note, the choice of socioeconomic health inequality index involves an array of value judgements. We have chosen to consider relative concentration of health inequality. We could also have considered absolute health inequality, but choose to limit our interest to relative changes.

4 Data

Table 1: Descriptive statistics

	Female	*Male*
Outcomes		
University attendance	0.580	0.521
	(0.004)	(0.004)
Frequency of hospital admissions	10.438	5.407
	(0.118)	(0.072)
Probability of being admitted to hospital due to:		
External causes	0.280	0.409
	(0.004)	(0.004)
Mental disorder	0.327	0.111
	(0.004)	(0.003)
Examinations	0.515	0.274
	(0.004)	(0.004)
All other causes	0.915	0.754
	(0.002)	(0.003)
Frequency of prescriptions	35.232	13.473
	(0.451)	(0.255)
Probability of receiving a prescription for:		
Contraceptives	0.848	0.000
	(0.003)	(0.000)
Psycholeptics	0.298	0.159
	(0.004)	(0.003)
Painkillers	0.349	0.234
	(0.004)	(0.003)
All other causes	0.966	0.873
	(0.002)	(0.003)
Years of education	13.46	13.22
	(0.01)	(0.01)
Compulsory school grades	216.18	207.40
	(0.35)	(0.28)
Father's education	10.28	10.91
	(0.04)	(0.03)
Mother's education	10.51	11.07
	(0.04)	(0.03)
Father's income	1333	1439
	(6.97)	(9.14)
Mother's income	781	827
	(3.84)	(3.70)
Observations	12652	15686

Notes: This table shows descriptive statistics for those graduating from upper secondary school between the years 2003 and 2005 and who have completed between 82% and 98% of a full program (a bandwidth of 8pp either side of the university eligibility threshold of 90%). Standard errors are shown in parenthesis

We use administrative register data on all students who graduated from upper secondary school between the years 2003 and 2005 and had previously graduated from Swedish compulsory school.[6] We combine education register data on final grades from compulsory school, grades from upper

[6]We need information on prior grades as a check and these are only available for those who attended the Swedish school system prior to starting upper secondary school. We also do not want to include individuals who have immigrated to Sweden during secondary school age. We consider the years 2003 onwards because in the years prior to 2003 it was much easier to re-take courses over the summer after graduating (from 2003 onwards, this is much less common) and as a consequence it is much harder to define whether a student achieved university education eligibility at graduation - our cut-off. We view measurement error and the potential for manipulation of the cut-off to be a significant threat to our identification strategy before the 2003 graduation year.

secondary school and data on higher education attendance and outcomes. This is then matched with administrative register data on labour market outcomes from the Longitudinal Integration Database for Health Insurance and Labour Market Studies (LISA) from Statistics Sweden (SCB) and administrative register data on hospital admissions and prescriptions is from the patient register and prescriptions register, respectively, both provided by the Swedish Board of Health (Socialstyrelsen). We also use the Multi-generational Register from Statistics Sweden that links the individuals to their parents who themselves are linked to their labour market and health outcomes. The population and housing censuses from years 1985 and 1990 provide us with parental education and income during the early childhood of the students we are following.

Our sample starts off with 128,751 students who graduated from upper secondary school between the years 2003 and 2005 and had previously graduated from Swedish compulsory school. We remove pupils who finish more than one year later (1.3 percent) or more than one year in advance (only 12 observations).[7] Keeping students who finish at age 18 or 20 has no impact on the results in this study.

Table 1 reports descriptive statistics for the sample analysed in this paper. Here we report the statistics for those with percentage of a completed program that lies within 8pp above or below the university eligibility cut-off. We split the sample by gender because there are important differences in education patterns and labour market and health decisions between genders. This leads to sample sizes of roughly 12,000 to 15,000 by gender very near to the cut-off.

Our medical care use variables are hospital admissions and prescriptions. Both the total number of hospital admissions and the total number of prescriptions since graduating and up to 2013 (our last period of observation) are considered. We also consider the probability of admission and the probability of prescription receipt by 2013 by the most common causes amongst young adults (aged 20 - 30). We consider causes of hospital admissions and prescriptions because they can be both as a consequence of a change in health status and due to investment decisions to raise current or future health levels and these two behaviours are potentially counterbalancing. We therefore consider hospital admissions and prescriptions by diagnosis (International Classification of Diseases (ICD10 codes) and drug type (Anatomical Therapeutic Chemical (ATC) Classification System codes).

Under preventative health actions we consider hospital admissions due to examinations (ICD10 code Z0-Z39) and prescriptions for contraceptives (ATC code female only). Under health consequences we have hospital admissions due to external causes (ICD10 codes S,T or if coded as external

[7] We exclude those on the individual program as they cannot gain university eligibility. Most students start upper secondary school aged 16 and graduate at age 19. It is not uncommon for students to finish upper secondary school at an older age (12.0 percent) than the typical graduation age of 19. A small share finish at a younger age (2.8 percent). There are many common and valid reasons for graduating older than 19 years of age: retaking courses, study breaks, changing programs or studying abroad. Students who graduate before the age of 19 have typically also started compulsory schooling before the mandatory starting age.

and M or main diagnosis missing), mental disorders (ICD10 code F, Z55, Z56, Z59, Z60, Z64, Z65, Z70-Z73) and for prescriptions we have psycholeptics (ATC codes N5, N6) that treat depression, anxiety and sleep disorders amongst others, and painkillers (ATC code N2).[8] Finally we consider university attendance in the first term, defined as a binary variable where unitary corresponds to attendance, zero otherwise.

The inequality outcomes we consider are the CI of frequency of hospital admissions (sum of admissions from graduation up to 2013) and the CI of frequency of prescriptions (again, the sum of prescriptions from graduation up to 2013). To measure the CI we need to rank individuals by their socioeconomic status and we choose a measure of family income as the measure of socioeconomic status for these young adults. In this way we capture a degree of intergenerational persistence. We calculate family income as the average of the income of the mother and father as reported in the 1985 and 1990 censuses. We take an average over years to remove temporal changes in income and get nearer to a measure of lifetime income of the parents. We use years 1985 and 1990 as these were defined during the childhood of the individuals we consider and therefore predetermined.

Background characteristics highly correlated with our health outcomes are used and include parental education in 1990 defined as years of education, age at migration and year of graduation from upper secondary school.[9] Dummies are defined for first-generation immigrant and second-generation immigrant and are region of origin specific.[10] We also define a dummy for whether the parents are of mixed origin or not and whether only one parent is an immigrant.

5 Method

5.1 Identifying the impact of university education eligibility

To estimate the effects of university eligibility on our health outcomes we use an RD design as our identification strategy. As shown in figure 1, the proportion going on to university is a smooth and increasing function of the percentage completed of a full program at upper secondary school. However, there is also a discontinuity caused by an arbitrarily chosen rule, the university eligibility threshold at 90% of a full program. We use individuals very close and either side of this cut-off that are just 1 or 2 completed courses apart on the assumption that they are likely to be very similar in all observable and unobservable ways except that those who are above the threshold have access to university education, and those below do not. This allows us to then assess the impact of university

[8] There are strong overlaps between the causes of hospital admissions and the causes for prescriptions. Painkillers are potentially linked to external causes related hospital admissions through the treatment of injuries requiring ongoing pain relief. Mental disorders related hospital admissions are likely to be linked in some way to psycholeptics.

[9] Where education information is not available, dummy variables are included indicating missing education information.

[10] Nordic countries, EU28, Non-EU28 countries and Russia, North America and Oceania, Africa, Asia and South America.

eligibility on educational and health outcomes. The general formulation for the regression equations we estimate is the following:

$$y_i = \alpha + \beta Eligible_i + f(\%fullprogram_i) + \varepsilon_i; \tag{4}$$

In this model y_i represents the various health outcomes we consider for individual i, *Eligible* is a binary treatment indicator variable equal to unity for those who have passed 90% or more of a full program and therefore eligible for university, zero otherwise and $\%fullprogram$ is measured in terms of distance from the eligibility threshold in percentage points of a full program. The functional form for the forcing variable, $f(\%fullprogram)$, is a local low ordered polynomial of $\%fullprogram_i$ and an interaction of $Eligibility_i * f(\%fullprogram_i)$ so that we have different trends either side of the cut-off. We follow the standard practice and add increasingly higher order polynomials until they become insignificant but also taking special care not to have too high a polynomial as argued by Gelman and Imbens (2017) and find a single polynomial is sufficient. The coefficient β is the discontinuous effect of university eligibility on the outcome variable assuming that our functional form absorbs any potential relationship between $\%fullprogram_i$ and ε_i.

The estimated impact of university eligibility on university attendance will be an Intention To Treat (ITT) parameter. Not all students who gain university eligibility having just graduated from upper secondary school go on to higher education. Some who do not gain eligibility go on to study at adult college and gain eligibility later. Eligibility at the end of upper secondary school therefore only impacts the probability of university attendance, it does not determine it. There is also potentially a pay-off to university eligibility without even going on to higher education. It may raise the esteem of the individual and it may be seen as a valid cut-off for employers to consider given its importance to universities. Our analysis therefore focusses on the reduced form impact of university eligibility on health outcomes.

We vary the bandwidth size between 4pp, 8pp and 16/8pp of a full program. This allows us to assess the sensitivity of the results to bandwidth choice.[11] Due to the fact that we have a large sample size so close to the cut-off, we are able to have small bandwidths. The inclusion of linear trends either side of the cut-off means we are in effect modelling a Local Linear Regression (LLR) with a rectangular kernel, the recommended approach of Imbens and Lemieux (2008).

When estimated equation 4, in some specifications we will add pre-determined characteristics. There are two reasons for this. First, as we expand the bandwidth we are including more observations

[11] We model bandwidth by running our linear regressions on the sample within the bandwidth. The discrete nature of the credit score means we are unable to non-parametrically choose the optimal bandwidth as recommended in general by Imbens and Lemieux (2008). 4pp is the smallest course size so makes a natural minimum bandwidth. 8pp is the largest bandwidth on the left hand side because any larger and we would have to model 100% of a completed program which is a very large jump.

that are not close to the cut-off and the inclusion of covariates may eliminate some bias that results from the inclusion of these observations (Imbens and Lemieux, 2008). Second, it provides an additional test of our identifying assumption that the error term is a smooth function crossing over the eligibility threshold.

5.2 The impact of university eligibility on university attendance

In this section we present the estimates of the effect of university eligibility on university attendance and the results of various diagnostic tests. In figure 1 we saw that there is a jump in the proportion who attend university at the university eligibility cut-off for females. The RD results are shown in table 2. Model (1) is a simple OLS of credit score on university attendance using only those within 8pp of the university eligibility threshold. It shows a strong positive correlation between university eligibility and university attendance. Model (2) shows our RD results using a bandwidth of 4pp and confirms there is a positive jump in the proportion attending university, 8pp for females and 2pp for males. Model (3) is as per (2) but with double the bandwidth of 8pp. Model (4) is as per (3) but with double the left-hand side bandwidth of 16pp. Models (5 & 6) are as per models (3 & 4) but with the addition of predetermined covariates.[12] The results for females across models (2-4) are stable to the choice of bandwidth and suggest university eligibility leads to a jump in university attendance in the range of 8pp to 10pp. The impact is much smaller for males in the range 2pp to 4pp.

Table 2: Impact of university eligibility on university 1st term attendance

	(1)	(2)	(3)	(4)	(5)	(6)
Bandwidth	8pp	4pp	8pp	16pp/8pp	8pp	16pp/8pp
Female						
Tertiary Eligibility	0.218***	0.0830***	0.0953***	0.0772***	0.0917***	0.0668***
	(0.0289)	(0.00494)	(0.0119)	(0.0130)	(0.0131)	(0.0127)
N	12652	4730	12652	13523	12652	13523
Male						
Tertiary Eligibility	0.188***	0.0175***	0.0285***	0.0415***	0.0293***	0.0429***
	(0.0318)	(0.00361)	(0.00562)	(0.00849)	(0.00646)	(0.00816)
N	15686	6644	15686	17148	15686	17148
Polynomial	0	1	1	1	1	1
Covariates	N	N	N	N	Y	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on first term university attendance for those graduating between years 2003 and 2005. Each estimate is from a separate regression. See text for details for each model (1-6). Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

The final analysis of this section considers the credit score distribution of the covariates as a

[12] Upper secondary graduation year, compulsory school grades, mother's and father's education and income plus dummies for missing education and income, dummies for world region of origin for first generation migrants and dummies for origin of parents for second generation migrants, age of migration and a dummy for whether one parent is a migrant.

test of our identifying assumption. The key identifying assumption is that the students and or their teachers are not able to manipulate the final credit scores in a systematic way that is linked to other important characteristics that determine health and medical care use. Our first diagnostic test of manipulation is that we include covariates in the regression estimates in models (5 & 6) in Table 2 and the impact of the inclusion of these covariates is very small. The inclusion of the covariates (models 5 & 6) leads to a small reduction in the estimated impacts for females and a small increase for males compared to estimates from models (3 & 4). The fact that we find a small impact of these covariates suggests that unobserved characteristics are in fact a smooth function over the cut-off.

Figure 2 presents four visual tests of cut-off manipulation. The top panel of figure 2 is a histogram of the population density by credit score plotted with bins of 4pp as suggested by Lee and Lemieux (2010) as a test of manipulation in the spirit of McCrary (2008). If there is a jump in the population just above the cut-off this is a sign of individuals manipulating their position around the threshold violating our identification assumption. The discrete nature of our data means this test is not ideally suited to our data but we observe no obvious jump in the density at the university eligibility cut-off. The second panel of figure 2 shows the final grade plotted against credit score. The third panel shows compulsory grades plotted against credit score. The final (fourth) panel shows the number of failed courses by final achieved credit score. These are all visual tests of whether individuals are trying to manipulate whether they cross the university eligibility threshold. For upper secondary grades we would expect if manipulation were occurring to see a jump in overall grade just above the threshold because of students trying harder in a number of courses to ensure they do not fall the wrongside of the threshold. For compulsory school grades we would expect the more able students to find it easier to manipulate the threshold and therefore observe a jump in compulsory school grades at the threshold. Finally, we consider the number of failed courses. Students can take more courses than needed for a full program and we therefore could expect to see a jump in the number of failed courses at the threshold as a consequence of students trying to maximise their chances of crossing the threshold. We observe no clear jumps in any of our visual diagnostic tests for females or males.

In table 3 we present results from a batch of balancing tests using RD that assess whether the covariates and our diagnostic test variables are equally distributed either side of the cut-off. Models (1) and (4) are OLS of the simple association of university eligibility and the covariate and show that university eligibility is highly correlated with all our diagnostic test variables and covariates. However, using our RD specification to isolate the impact of university eligibility in models (2-3 & 5-6) the coefficients all substantially reduce towards zero and nearly always lose statistical significance. We find evidence of a small jump in compulsory school grades at the cut-off using our largest bandwidth but not the smaller bandwidth. Whilst the jump is statistically significant it is

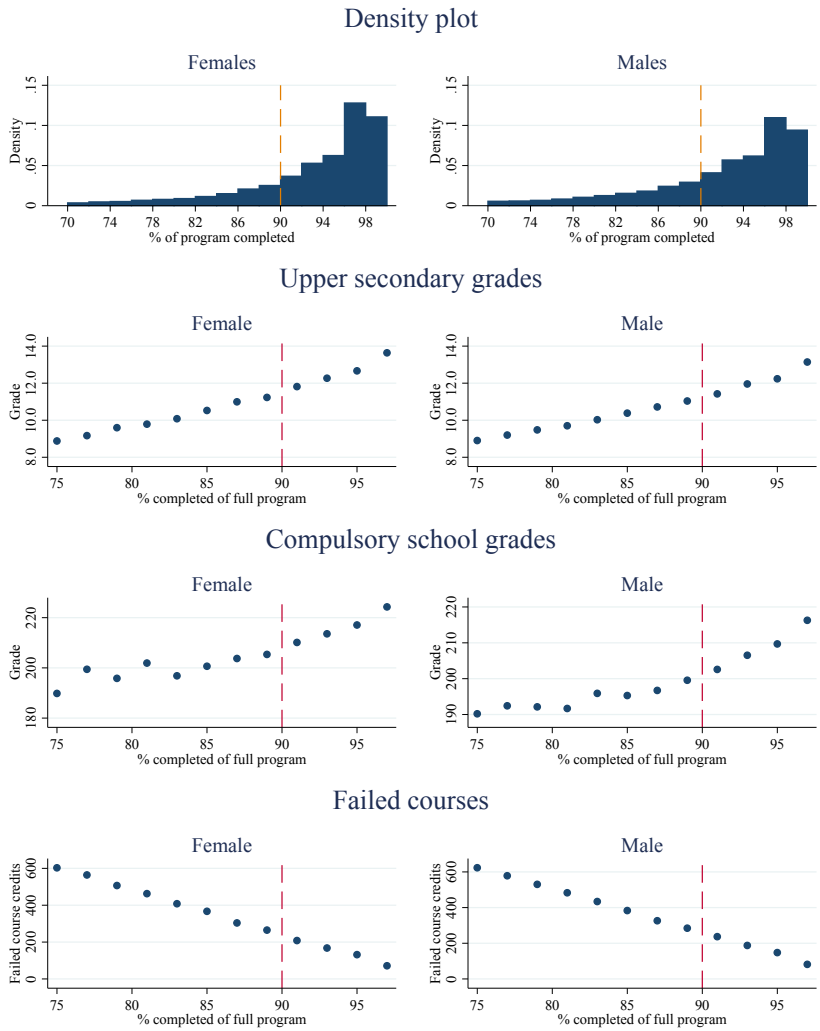

Fig. 2: Diagnostic tests

Notes: These figures plot various diagnostic tests using percentage of a completed program as the running variable shown in bins of 2pp of a program. For panels 2-4 we present the mean for each bin. The dashed vertical line is the 90% cut-off for university eligibility. See text for further details.

rather small in relative terms and represents a jump of less than 1pp (320 credits is the maximum). Our RD results also show mother's and father's education to jump significantly for males but this time for the smaller bandwidth but not the larger bandwidth. The sign has reversed compared to the naive OLS estimates of model (4). This suggests the data is very sensitive to how it is modelled

for this particular variable making conclusions difficult beyond that overall the potential differences appear small and possibly insignificant. Note also we have not made any adjustment for multiple hypothesis testing here which would pull down the significance levels reported here.

In sum, the fact that our estimates of the impact of university eligibility on university attendance are stable across different model specifications and also with and without the inclusion of covariates suggests that both our observed covariates and the covariates we do not observe are a smooth function across the cut-off and that the jumps we observe are due to the policy effect. Our diagnostic tests add further evidence that we find no compelling evidence of manipulation. Altogether, this suggests that the jumps we observe in university attendance are primarily driven by the arbitrary rule and not by unobserved factors resident in the error term.

Table 3: RDD based diagnostic tests

	(1)	(2)	(3)	(4)	(5)	(6)
Bandwidth	8pp	8pp	16pp/8pp	8pp	8pp	16pp/8pp
	Female			Male		
Upper secondary school grades						
Tertiary Eligibility	2.038***	0.0723	0.158*	1.722***	0.0141	0.0677
	(0.397)	(0.0991)	(0.0820)	(0.357)	(0.0503)	(0.0532)
Failed Upper secondary school course credits						
Tertiary Eligibility	-191.5***	-5.541	-6.094	-192.2***	3.793	1.980
	(34.34)	(3.935)	(3.847)	(37.32)	(3.978)	(3.014)
Compulsory school grades						
Tertiary Eligibility	16.81***	0.503	2.589**	12.98***	1.680	1.805**
	(3.498)	(1.089)	(1.176)	(2.744)	(1.013)	(0.803)
Mother's education						
Tertiary Eligibility	0.625***	-0.116*	0.0321	0.582***	-0.277**	-0.0651
	(0.154)	(0.0647)	(0.0948)	(0.181)	(0.124)	(0.132)
Father's education						
Tertiary Eligibility	0.564***	-0.166	0.0254	0.495***	-0.275**	-0.108
	(0.174)	(0.203)	(0.144)	(0.170)	(0.136)	(0.157)
Father's income						
Tertiary Eligibility	111.5***	-8.198	19.57	88.89***	-1.721	-15.84
	(25.87)	(14.72)	(22.23)	(25.77)	(20.35)	(16.26)
Mother's income						
Tertiary Eligibility	50.77***	-20.61*	4.395	46.26***	-9.585	-0.492
	(13.81)	(11.37)	(15.93)	(12.69)	(9.977)	(7.148)
N	12652	12652	13523	15686	15686	17148
Polynomial	0	1	1	0	1	1

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on a batch of diagnostic variables and pre-determined characteristics for those graduating between years 2003 and 2005 and who were enrolled on the academic stream. Each estimate is from a separate regression. Models (1) and (4) are simple OLS associations of university eligibility and the variable being tested using a bandwidth of 8pp. Models (2) and (5) use a linear trend in course credits either side of the cut-off and bandwidth of 8pp of a full program either side of the cut-off. Models (3) and (6) are as models (2) and (5) but with a bandwidth of 16pp before the cut-off and 8pp after. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

5.3 Estimating the distributional impact of university education

To determine whether university eligibility increases the concentration of medical care use amongst the rich or poor we combine the concept of Recentered Influence Function (RIF) regression with RD. RIF regression allows any statistic to be linked to individual characteristics. We use the results of Firpo et al. (2009) and Heckley et al. (2016) in order to estimate the marginal effect of university eligibility on the CI, ARCI and SRCI.

We shall use linear RIF regression of the CI, which is very similar in approach to standard OLS regression. In an OLS regression we have a vector of health outcomes on the left hand side as the dependent variable and explanatory variables on the right hand side. RIF regression swaps out the vector of health outcomes and replaces these with a vector of influences on a statistic, in our case the CI. The mean of the vector of RIFs of the CI is the CI, which means under a linear setting and by the Law of Iterated Expectations (LIE) we can link each individuals characteristics to the CI using regression e.g. using OLS. The coefficients from our regression are the marginal effects. The difference between OLS of the mean and OLS of a RIF is that RIF-OLS only has a marginal interpretation - that is, we cannot calculate contributions and they are local estimates. The complication with CI marginal effects interpretation is that inequality can be concentrated amongst the rich (positive CI) or the poor (negative CI) and therefore the interpretation of the signs of the coefficients and whether the covariate is inequality increasing or decreasing depends on the value of the CI.

More precisely, we write the RIF of a statistic as RIF(v), where v represents any summary statistic of a distribution (e.g. in our case, the mean, CI, ARCI or SRCI). Firpo et al. (2009) show that the LIE can be applied to a RIF and therefore individual characteristics can be linked to the statistic of interest. This is RIF regression and it requires estimating the following:

$$E[RIF(v)|X = x] = E[\lambda(X, \epsilon)|X = x] \tag{5}$$

The choice of regression method depends on the form we want to assume for $\lambda(X, \epsilon)$ and in principle this choice is limitless. RD design lends itself very well to RIF regression because RD can essentially be thought of as a non-parametric method under certain conditions and therefore the parametrisation of the function $\lambda(X, \epsilon)$ is uncontroversial. In our analysis we are using a small bandwidth with linear regression either side of the cut-off, which is the equivalent to running the non-parametric regression technique of local linear regression with a rectangular kernel. To be precise, the RIF RD

regression we estimate is the following linear regression:

$$RIF(v)_i = \alpha + \beta Eligible_i + f(\%fullprogram_i) + \epsilon_i \tag{6}$$

The parameter β from equation 6 will be the marginal effect of university eligibility on the CI, ARCI and SRCI. The functional form for the forcing variable, $f(\%fullprogram_i)$ will be as for equation 4.

6 Results

6.1 The impact of university eligibility on hospital admissions and prescriptions

In this section we present the estimates of the effect of university eligibility on hospital admissions and prescriptions during early adulthood (aged between 20 and 30). Figure 3 depicts completed credit profile of mean frequency of hospital admissions and prescriptions for the years since graduation up to 2013, split by gender. The data indicate no clear jumps in either hospital admissions or prescriptions at the 90% threshold.

This is confirmed in tables 4 and 5. All regression results from here on in will use regression models (1), (3), (4) and (6) from table 2. Model (1) in tables 4 and 5 is the simple association of university attendance without modelling the credit score and a bandwidth of 8pp. Model (2) is as per model (1) but now includes a linear trend estimated either side of the cut-off. Models (3) and (4) are as per model (2) but add a larger bandwidth to the left hand side.[13] Model (4) also includes covariates strongly associated with the outcome variable.

In model (1) of table 4 we can see that there is a significant negative association between university attendance and frequency of hospital admissions for females but not for males. The RD results for hospital admissions in table 4, however, show a positive jump in the frequency of hospital admissions for females according to model (2) of about 0.7 but this becomes insignificant and much smaller when increasing the bandwidth as modelled in models (3) and (4), although it remains positive. For males however the results are very sensitive to modelling choice and insignificant. Overall this suggests that university eligibility does not lead to a decrease in hospital admissions which is implied by the naive associations of model (1).

Turning to prescription receipt, we see in table 5 that the naive OLS regressions of university attendance and frequency of prescription receipt show no significant association for males or females.

[13] We cannot have a larger bandwidth on the right hand side because we would then have to model the huge jump at 100% of a completed course.

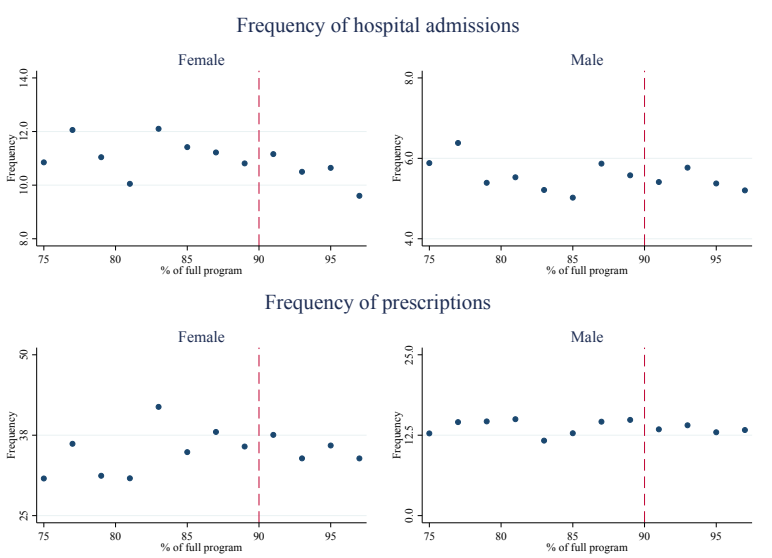

Fig. 3: Impact of university eligibility on the frequency of hospital admissions and prescriptions

Notes: These figures plot a scatter of the mean of hospital admission frequency and prescription frequency since graduation up to 2013 against percentage completed of a full program with a bin width of 2pp of a full course in each bin for those graduating upper secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

The RD results in table 5 are substantial in size relative to the OLS estimates of model (1) but are not at all stable to model specification and in the main not significant. As can be seen from the raw data in figure 3 there is no clear trend between frequency of prescriptions and credit score and therefore the results are sensitive to the noise in the data.

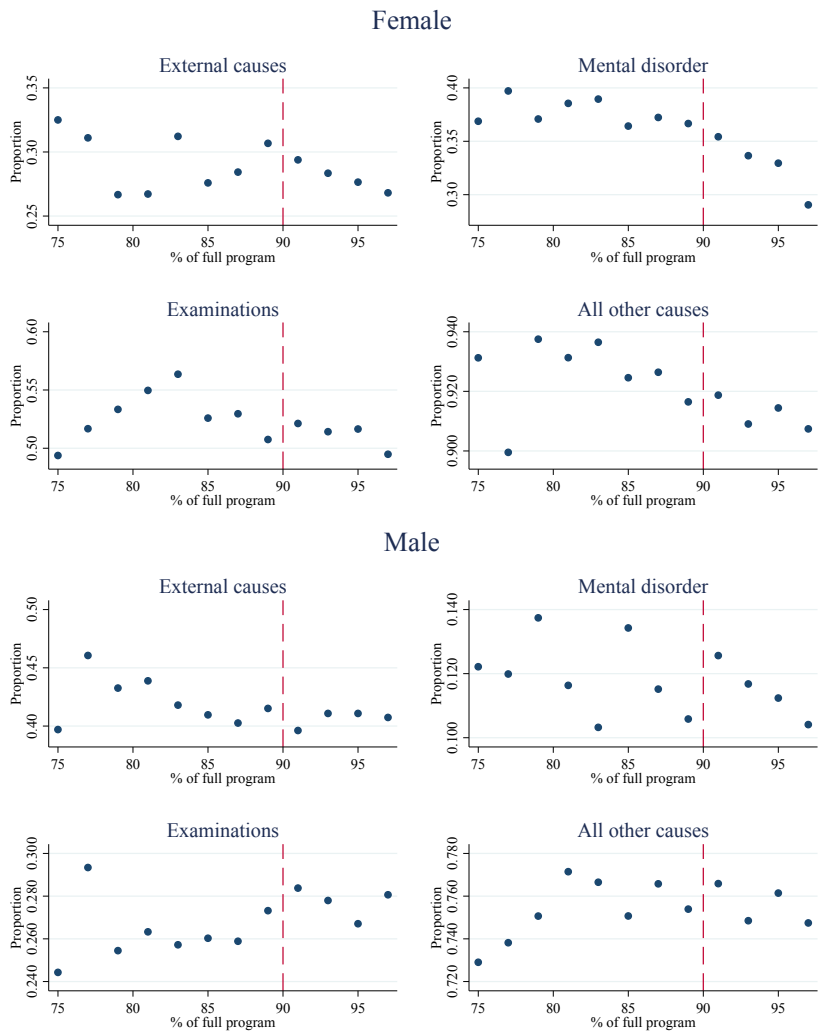

Fig. 4: Impact of university eligibility on the probability of hospital admission by diagnosis

Notes: This figure plots a scatter of average frequency of hospital admissions since graduation and up to 2013 by diagnosis against percentage completed of a full program with a bin width of 2pp of a full course in each bin for those graduating upper secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

Table 4: Impact of university eligibility on hospital admissions, by diagnosis

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
			Females	
Number of admissions (mean: 10.44)	-0.831***	0.738***	0.120	0.209
	(0.163)	(0.159)	(0.267)	(0.281)
Probability of hospital admission due to:				
External causes (mean: 0.28)	-0.021***	-0.008	-0.001	0.001
	(0.008)	(0.013)	(0.012)	(0.011)
Mental disorder (mean: 0.33)	-0.010	-0.001	-0.007	-0.006
	(0.009)	(0.005)	(0.005)	(0.005)
Examinations (mean: 0.51)	-0.028***	0.030***	0.001	0.006
	(0.007)	(0.004)	(0.012)	(0.012)
All other causes (mean: 0.92)	-0.016***	0.003	-0.006	-0.004
	(0.003)	(0.004)	(0.006)	(0.007)
N	12652.000	12652.000	13523.000	13523.000
			Males	
Number of admissions (mean: 5.41)	-0.119	-0.239	0.188	0.185
	(0.168)	(0.276)	(0.286)	(0.294)
Probability of hospital admission due to:				
External causes (mean: 0.41)	-0.092***	-0.008	-0.004	-0.004
	(0.006)	(0.009)	(0.010)	(0.010)
Mental disorder (mean: 0.11)	0.012**	0.018*	0.017***	0.018***
	(0.006)	(0.010)	(0.005)	(0.005)
Examinations (mean: 0.27)	0.004	0.002	0.010	0.011
	(0.007)	(0.007)	(0.008)	(0.008)
All other causes (mean: 0.75)	-0.017***	0.005	-0.004	-0.004
	(0.006)	(0.008)	(0.009)	(0.008)
N	15686.000	15686.000	17148.000	17148.000
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on hospital admissions by diagnosis since graduation and up to 2013 for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression. Model (1) is a simple correlation of university attendance and health. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

In figure 4 we present the credit score distribution of the *probability* of hospital admission by leading cause. In general there appears to be a downward trend in our causes of hospital admission with credit score and no clear jumps are observed for the causes we consider. The potential exception is mental disorders for males that appears to show a positive jump in cases for those reaching university eligibility, but the data appears quite noisy. In table 4 column (1) we present simple correlations of university attendance and hospital admissions by cause and in general the coefficients are negative and significant, confirming the widely documented education gradient in health and health care. The RD estimates for the probability of hospital admission by cause are found in table 4 models (2) to (4). We find that the jump in mental disorders for males is robust to model specification and lies in the range of 1.7pp to 1.8pp. These suggest a relatively large impact of university eligibility on mental disorder related admissions of about 10% (proportion who have a

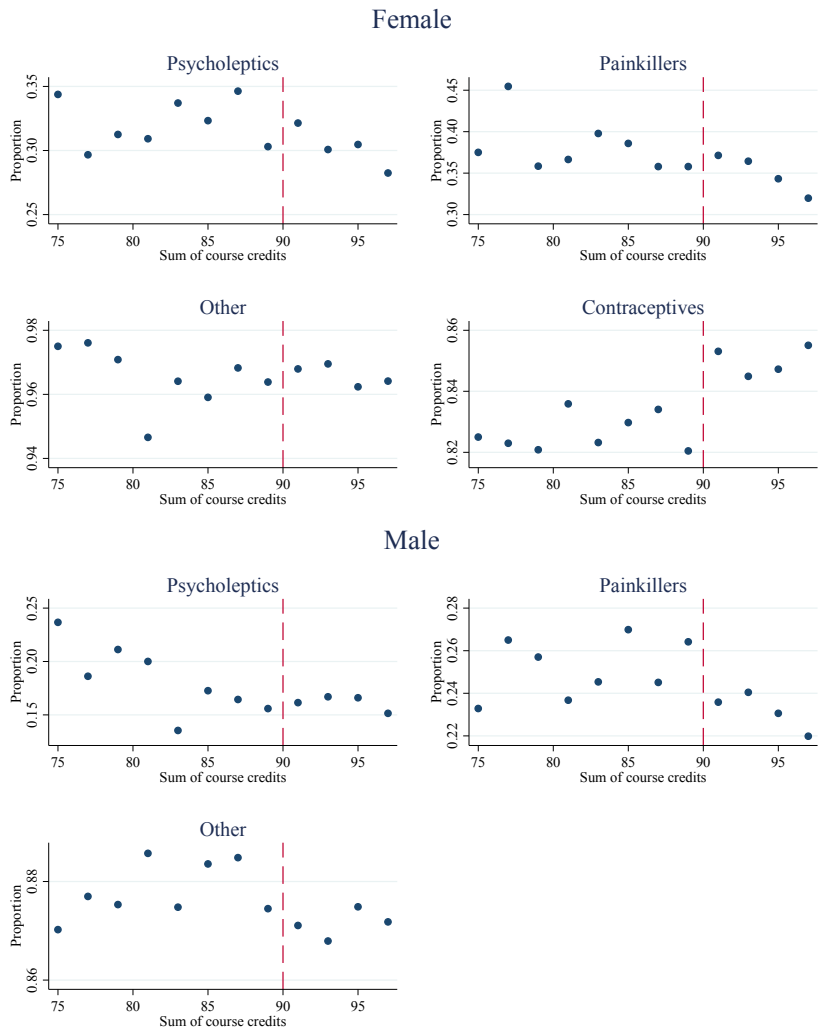

Fig. 5: Impact of university eligibility on the probability of prescription by cause

Notes: These figures plot a scatter of percentage completed of a full program with a bin width of 2pp against the probability of receiving a prescription since graduation and up to 2013 by main cause 2010-2013 in each bin for those graduating upper secondary school between the years 2003 and 2005. See notes for figure 1

mental disorder related admission is 11% for this group). No other results are robust to model choice.

Table 5: Impact of university eligibility on prescription receipt, by diagnosis

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
		Females		
Frequency of prescriptions (mean: 35.23)	0.200	2.000	-1.508	-1.299
	(0.901)	(1.579)	(1.771)	(1.913)
Probability of prescription due to:				
Psycholeptics (mean: 0.3)	0.012*	0.018	0.001	0.001
	(0.006)	(0.017)	(0.018)	(0.020)
Painkillers (mean: 0.35)	-0.047***	0.032***	0.019*	0.020**
	(0.002)	(0.007)	(0.010)	(0.009)
Other (mean: 0.97)	-0.001	0.001	0.005	0.006*
	(0.004)	(0.004)	(0.003)	(0.003)
Contraceptives (mean: 0.85)	-0.023***	0.019**	0.018**	0.021**
	(0.005)	(0.008)	(0.007)	(0.009)
N	12652	12652	13523	13523
		Males		
Frequency of prescriptions (mean: 13.47)	0.763	-2.518***	-0.791	-0.814
	(0.485)	(0.495)	(0.614)	(0.601)
Probability of prescription due to:				
Psycholeptics (mean: 0.16)	0.029***	0.003	0.026***	0.027***
	(0.005)	(0.012)	(0.008)	(0.008)
Painkillers (mean: 0.23)	-0.044***	-0.021**	-0.019**	-0.019*
	(0.006)	(0.009)	(0.009)	(0.010)
Other (mean: 0.87)	-0.021***	-0.008	-0.011**	-0.011**
	(0.005)	(0.006)	(0.005)	(0.005)
N	15686	15686	17148	17148
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on frequency of prescriptions and probability of prescriptions since graduation and up to 2013 by category for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression. Model (1) is a simple correlation of university attendance and health. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

We turn now to the specific causes for prescription receipt. We now consider prescriptions that are both preventative related (contraceptives (women only)) but also health outcome related in nature (psycholeptics, painkillers). We depict the credit score profile of prescriptions by cause and split by gender in figure 5. In the figure we see that females observe a clear positive jump in contraceptives and that males observe a drop in painkillers. In table 5, model (1) shows the association of university attendance with the probability of cause specific prescription receipt and we find there is in general a significant negative association between university attendance and prescription receipt. The RD results in table 5 models (2) to (4) for cause specific prescription receipt confirm that women who pass the eligibility threshold see an increase in contraceptive related prescriptions in the range 1.8pp to 2.1pp and that males see a drop in probability of receiving

painkiller related prescriptions in the range of -1.9pp to -2.1pp. The results are stable across modelling strategies (2 to 4) and statistically significant suggesting that these results are robust to specification. Less stable results are also found for female painkiller prescription receipt where university eligibility is estimated to increase the probability of prescription receipt rather than reduce it as found for the OLS results.

In summary, we find that a large jump in university attendance due to university eligibility amongst females also coincides with no clear impact on hospital admissions but a clear positive impact on the proportion receiving a prescription for contraceptives. For males we find a jump in university attendance due to university eligibility but this is smaller than found for females and may also coincide with both an increase in hospital admissions due to mental disorders and a reduction in prescriptions for pain related medication.

6.2 The impact of university eligibility on health inequality

The analysis of the previous section focussed on the mean of our medical care use outcome variables. In this section we present the impact of university eligibility on the CI of parental income related medical care use inequality. We calculate the level of inequality for the whole population of students graduating upper secondary school between 2003 and 2005. The CI for parental income related frequency of hospital admission inequality is -0.012 and the CI for frequency of prescriptions is 0.021 (results shown in tables 6 and 7). That is, hospital admissions are concentrated more amongst young adults from poorer backgrounds. The opposite is the case for frequency of prescriptions. We link course credits and university eligibility to the CI using RIF regression and present the marginal effects in figure 6. There are no obvious trends in percentage completed of a full program and income related concentration of medical care use. There are also no clear jumps in income related hospital admission inequality or income related prescription inequality at the university eligibility threshold.

Model (1) of tables 6 and 7 presents the association of university attendance and parental income related medical care use inequality. These associations give us an idea as to how university attendance is linked to an increased or decreased concentration of medical care use amongst young adults with poor or rich backgrounds. The slight complication with interpretation of the coefficients in tables 6 and 7 is that a negative coefficient is only inequality reducing if the CI is positive. If the CI is negative then a negative coefficient implies a worsening of inequality, and vice versa. We see from model (1) in table 6 that university attendance is associated with a reduction in the CI for females and an increase for males but these associations are not particularly significant. For the CI of prescription frequency we find university attendance to increase inequality for females but

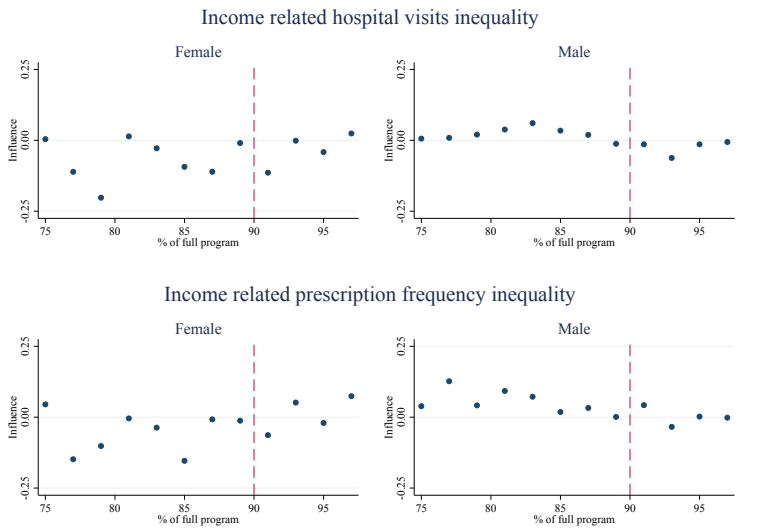

Fig. 6: Impact of university eligibility on income inequality of the probability of a hospital admission and a prescription

Notes: These figures plot a scatter of the mean frequency of hospital admissions and prescriptions (years 2010-2013) and mean (recentered) influence on the CI of hospital admissions and prescriptions against percentage completed of a full program using a bin width of 2pp of a full course for those graduating upper secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

reduce it for males, but again these associations are not significant. The RD estimates found in tables 6 and 7 of the impact of university eligibility on the CI confirm our observations from 6 that university eligibility leads to no clear impact on parental income related medical care use inequality.

In tables 6 and 7 we also present RD results by cause of hospital admission and cause of prescription (supporting figures are found in the appendix, A.1 and A.3). For the probability of a hospital admission we use the ARCI as our measure of relative inequality because we are interested in relative inequality but now need to account for the bounded nature of our binary health variable. We assess the sensitivity of our results to this choice of measure by also looking at the SRCI in the appendix. Just as frequency of hospital admissions inequality was found to be concentrated amongst the poor, so are the probabilities of admission due to various causes also concentrated amongst the poor. Model (1) of table 6 presents the naive associations between university attendance and ARCI and suggests the university attendance is inequality reducing for females and increasing for males, with the exception of the ARCI of external cause related and examination related hospital admissions for males. However, these associations are only statistically significant for the ARCI of examination related and mental disorder related hospital admissions for females. Examining the

raw data in A.1 there appear to be small jumps for hospital admissions due to examinations. The RD results are shown in columns (2-4) in table 6 and confirm that there are jumps in the ARCI of hospital admissions for females due to examinations in the range of -0.023 to -0.033. Because the ARCI is negative (-0.013), university eligibility therefore increases the concentration of examinations amongst poor young adult females. There are also jumps in the ARCI of hospital admissions for males due to examinations in the range of 0.033 to 0.046. This suggests that university eligibility reduces the concentration of examinations amongst poor young adult males. No other stable and significant results are found for ARCI of hospital admissions.

We now turn to the particular causes of the ARCI of parental income related prescription receipt inequality. The CI of frequency of prescriptions finds a pro-rich concentration of prescriptions for young adults in Sweden. The pro-rich concentration is driven by contraceptives and *all other causes* of prescriptions whereas prescriptions for psycholeptics and painkillers are found to be more concentrated amongst the poor. In table 7 model (1) shows the association of university attendance with ARCI of cause specific prescriptions and only the ARCI for contraceptives finds a significant association (an inequality increasing association). In the appendix, figure A.3 depicts the relationship between percentage of a completed course and average effect on ARCI of prescriptions by cause. No clear trends between percentage of a completed course and the marginal impact on ARCI are observed for any cause specific prescription probability with the potential exception of contraceptives that show an increasing trend. At the 90% threshold no clear jumps are observed for females but potentially a negative jump for males for ARCI of prescriptions for other causes. This is largely confirmed in 7 models (2) to (4). Females observe no jumps at the 90% threshold that are stable to modelling specification or significant. In general this is also true for men with the exception for any other cause that sees an inequality reducing jump at the threshold that is relatively large compared to the level of ARCI and is stable to modelling specification.

Sensitivity analysis of our choice of ARCI over SRCI is found in the appendix (A.1 and A.2 and figures A.2 and A.4). The conclusions are not affected by our choice of ARCI over SRCI. To sum up, we find that there is a parental income concentration of medical care use. Hospital admissions are concentrated amongst the poor and prescriptions are more concentrated amongst the rich with the exception of psycholeptics and painkiller related prescriptions. University eligibility is found to increase hospital admission inequality through females yet reduce it through males and also reduce prescriptions for other causes inequality through males.

Table 6: Impact of university eligibility on parental income related hospital admissions, by diagnosis

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
			Females	
Frequency of hospital admissions (CI=-0.012)	0.037*	-0.046	-0.033	-0.042
	(0.022)	(0.057)	(0.049)	(0.043)
Probability of admission due to:				
Mental disorders (ARCI=-0.033)	0.065***	-0.088	-0.024	-0.037
	(0.021)	(0.058)	(0.060)	(0.046)
External causes (ARCI=-0.005)	0.002	-0.021	-0.017	-0.018
	(0.019)	(0.013)	(0.017)	(0.015)
Examinations (ARCI=-0.013)	0.040**	-0.023***	-0.027***	-0.033***
	(0.020)	(0.006)	(0.010)	(0.009)
All other causes (ARCI=-0.001)	0.007*	-0.012**	0.004	0.002
	(0.004)	(0.004)	(0.007)	(0.006)
N	12652.000	12652.000	13523.000	13523.000
			Males	
Frequency of hospital admissions (CI=-0.012)	-0.005	-0.009	-0.049	-0.050*
	(0.014)	(0.025)	(0.030)	(0.028)
Probability of admission due to:				
Mental disorders (ARCI=-0.033)	-0.017	0.046*	0.007	0.002
	(0.017)	(0.025)	(0.036)	(0.033)
External causes (ARCI=-0.005)	0.012	-0.013	-0.021	-0.019
	(0.020)	(0.011)	(0.013)	(0.016)
Examinations (ARCI=-0.013)	0.001	0.046***	0.034**	0.033**
	(0.010)	(0.014)	(0.017)	(0.015)
All other causes (ARCI=-0.001)	-0.003	0.003	-0.006	-0.006
	(0.008)	(0.009)	(0.009)	(0.008)
N	15686.000	15686.000	17148.000	17148.000
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on concentration index of hospital admission frequency and the attainment relative concentration index of hospital admission probability by diagnosis since graduation and up to 2013 for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression and captures the marginal effect on the inequality index. Model (1) is a simple correlation of university attendance and health inequality. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. For simplicity of application we use empirical standard errors that do not account for the fact that the RIF is an estimated function. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Table 7: Impact of university eligibility on parental income related prescription receipt admissions

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
			Females	
Frequency of prescriptions (CI=0.021)	0.028	-0.047	-0.009	-0.024
	(0.023)	(0.044)	(0.047)	(0.039)
Probability of prescription due to:				
Psycholeptics (ARCI=-0.001)	0.026	-0.064***	0.008	-0.008
	(0.016)	(0.017)	(0.038)	(0.035)
Painkillers (ARCI=-0.017)	0.025	-0.034**	0.003	-0.001
	(0.019)	(0.016)	(0.022)	(0.018)
Other (ARCI=0.003)	-0.002	-0.000	-0.001	-0.001
	(0.002)	(0.005)	(0.004)	(0.004)
Contraceptives (ARCI=0.013)	0.055***	0.011	0.030	0.007
	(0.008)	(0.026)	(0.025)	(0.013)
N	12652.000	12652.000	13523.000	13523.000
			Males	
Frequency of prescriptions (CI=0.021)	-0.018*	0.025	0.009	0.006
	(0.010)	(0.030)	(0.031)	(0.027)
Probability of prescription due to:				
Psycholeptics (ARCI=-0.001)	0.007	0.074***	0.012	0.011
	(0.012)	(0.021)	(0.033)	(0.033)
Painkillers (ARCI=-0.017)	-0.020	-0.025	-0.065**	-0.065**
	(0.015)	(0.029)	(0.032)	(0.032)
Other (ARCI=0.003)	-0.002	-0.008***	-0.013***	-0.013***
	(0.003)	(0.003)	(0.003)	(0.004)
N	15686.000	15686.000	17148.000	17148.000
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on concentration index of prescription frequency and the attainment relative concentration index of prescription probability by diagnosis since graduation and up to 2013 for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression and captures the marginal effect on the inequality index. Model (1) is a simple correlation of university attendance and health inequality. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. For simplicity of application we use empirical standard errors that do not account for the fact that the RIF is an estimated function. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

7 Discussion

In this paper we have shown that university eligibility leads to a sharp positive discontinuity in the proportion attending university. Previous analysis using the same eligibility rule (Nordin et al., 2017) has shown that this jump in university eligibility leads to coinciding jumps in years of schooling of about 0.3 years for female and 0.2 for males and an increase in the probability of achieving 15 years of schooling (equivalent to a bachelor's degree) of about 10pp for females and 3pp for males. We find that this discontinuous jump in university level educational attainment for females coincides with no clear impact on hospital admissions but a clear positive impact on the proportion receiving a prescription for contraceptives of about 1.8pp. For males the jump in university attendance due to university eligibility is smaller than found for females and we find a

possible increase in hospital admissions due to mental disorders of about 1.7pp and a reduction in prescriptions for pain related medication of about 1.9pp.

We have also considered the impact of university eligibility on socioeconomic inequality in health, specifically how university eligibility has impacted the CI of family income related health inequality. No overall impact is found on the concentration of hospital admissions or prescriptions with family income, but specific impacts were found for hospital admissions for examinations (inequality increasing for females, decreasing for males) and for prescriptions for any other reason (inequality decreasing for males only).

Our results assessing the level of health appear to fit alongside those of De Walque (2007) and Grimard and Parent (2007) who find a protective impact of education on health (reduces smoking initiation and increases likelihood of cessation), and Buckles et al. (2016) who find a negative impact on mortality, all using the Vietnam draft as an Instrumental Variable for university attendance. The jump we find in contraceptive prescriptions for females can be interpreted as a jump in health investments. This fits with the evidence showing college graduates choosing to smoke less. The jump also could be linked to a preference to delay child birth. The impacts found for males are harder to interpret because they could be either due to impacts of health on medical care use e.g. university has increased stress and anxiety so they are more likely to visit hospital, or that they are now more aware of their condition and get themselves seen to. A similar argument can be made for the results for pain-killers.

Our results looking at the socioeconomic inequality aspects of university eligibility have not yielded any clear impacts. There appear to be competing effects of university eligibility on examination related hospital admission inequality where females see an increase in inequality but males a decrease. We do not find a strong impact on contraceptive prescription inequality which suggests that increasing access to university education is unlikely to worsen contraceptive use related inequality.

The results presented in this paper are based on RD design that has a very high level of internal validity. But are the results specific to Sweden? The Swedish welfare state and health care system is very comprehensive and is similar in its coverage and provision to that of the National Health Service (NHS) in Britain. Both systems offer universal coverage and use doctors as gatekeepers to the medical system that should in theory minimise shopping for best treatments. A small difference between the NHS in Britain and Sweden's health care system is that in Sweden patients are required to pay a small out of pocket payment to visit a doctor or use any hospital service. There is therefore a financial element to the participation decision. But this is small, about 150 SEK (roughly $18 in 2018 prices) depending on where one lives in Sweden. This means that one potential channel for education to impact health, via financial resources, is more limited in Sweden. However, we would

expect changes in health related behaviours to be related to education and these will independently impact the demand for health care. Financial resources can also impact health via other channels than medical care, through improved access to better diet, resources for increased activity and so on. It is important to understand all the channels by which education may affect health.

The period in which we consider the impacts on health are fairly short after the intervention, up to 8 - 10 years after graduating from upper secondary school. Early adulthood health outcomes are of interest as they allow us to understand the mechanisms by which potential changes to education could impact health immediately and later on in life. The objective health outcomes we consider, hospital admissions and prescriptions, represent health processes, behaviours and investments. The human capital models that predict the importance of education in determining our health capital do not state the timescale over which these investments might take place. It is therefore of interest to know if and how and when we see a difference in an individual's health capital investments.

A particular limitation of the data we have used is that we do not consider the impact of university eligibility on primary care use. This is because there is no national dataset that captures primary care use. Primary care use is likely to be relevant for young adults and their health investments. The impacts found for prescriptions are quite likely to be reflected in the primary care use data as the majority of prescriptions are made by the local GP and not doctors at hospitals. However, this type of analysis will remain difficult until someone manages to corral all 21 health regions in Sweden to join up and create a unified administrative system.

We conclude that university education for low ability students leads to an increase in contraceptive use amongst females. We also find that the changes in the levels of medical care use do not impact family income related medical care use and that overall any potential increases in ease of access to university are unlikely to impact overall income related medical care use amongst young adults. The results suggest caution, however, as we also find indications that male mental health issues jump for those achieving university eligibility and this suggests that universities need to take particular care of the mental health of their least able students.

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Appendix

A The impact of university eligibility on hospital admissions and prescription rates, detailed sub-group analysis

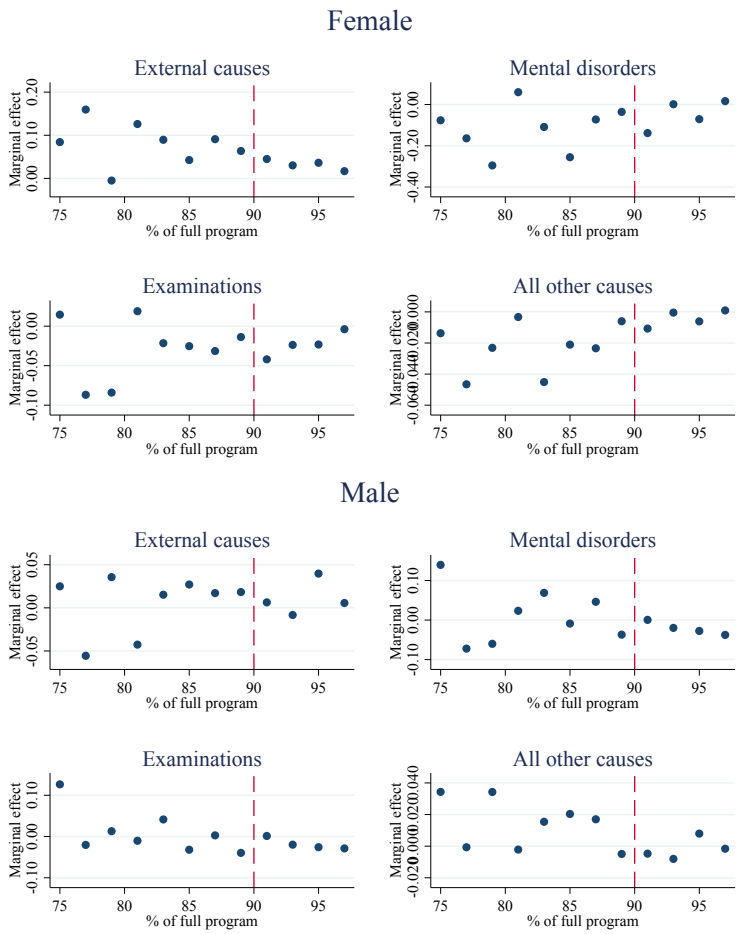

Fig. A.1: Impact of university eligibility on the attainment relative concentration index of *income related* inequality of hospital admission by diagnosis

Notes: These figures plot a scatter of the recentered influence function of attainment relative concentration index income related inequality of frequency of hospital admissions against the final achieved course credits with a bin width of 2pp of a full course in each bin for those graduating from upper secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

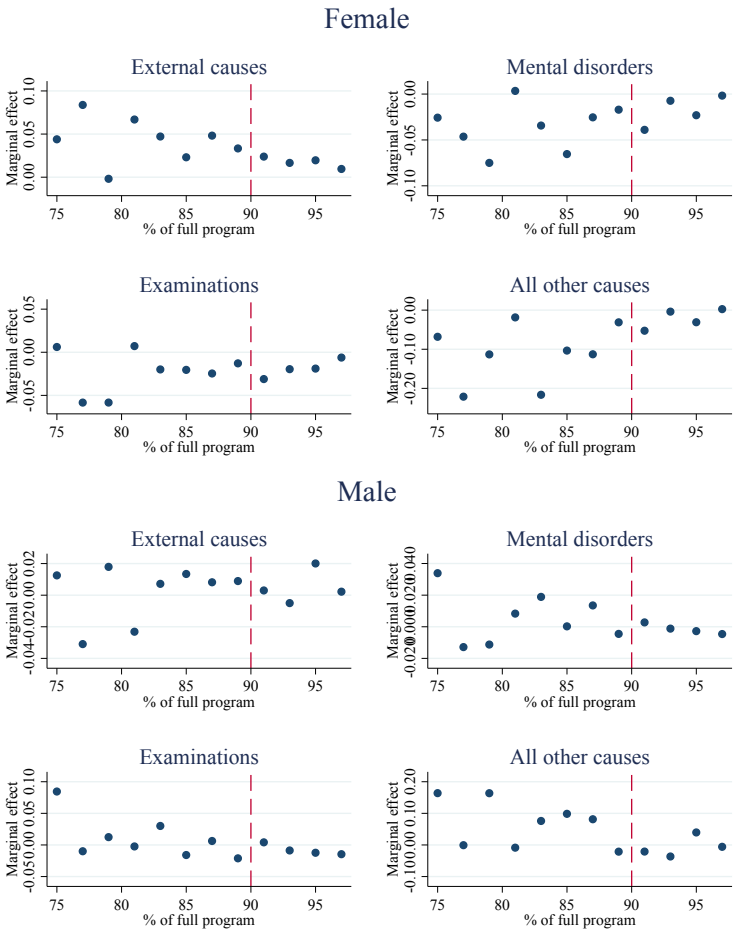

Fig. A.2: Impact of university eligibility on the short-fall relative concentration index of *income related* inequality of hospital admission by diagnosis

Notes: These figures plot a scatter of the recentered influence function of short-fall relative concentration index income related inequality of frequency of hospital admissions against the final achieved course credits with a bin width of 2pp of a full course in each bin for those graduating upper from secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

Table A.1: Impact of university eligibility on short-fall relative concentration index of parental income related hospital admission probability by cause

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
			Females	
Examinations (SRCI=-0.008)	0.026**	-0.016***	-0.017***	-0.021***
	(0.012)	(0.004)	(0.006)	(0.006)
Mental disorders (SRCI=-0.007)	0.015***	-0.019	-0.005	-0.008
	(0.005)	(0.013)	(0.013)	(0.010)
External causes (SRCI=-0.002)	0.001	-0.011	-0.008	-0.009
	(0.010)	(0.007)	(0.009)	(0.008)
All other causes (SRCI=-0.003)	0.036*	-0.054**	0.019	0.010
	(0.018)	(0.020)	(0.035)	(0.028)
N	12652.000	12652.000	13523.000	13523.000
			Males	
Examinations (SRCI=-0.008)	0.000	0.029***	0.021*	0.020**
	(0.007)	(0.009)	(0.011)	(0.010)
Mental disorders (SRCI=-0.007)	-0.004	0.009*	0.001	-0.000
	(0.004)	(0.005)	(0.008)	(0.007)
External causes (SRCI=-0.002)	0.007	-0.006	-0.011	-0.010
	(0.010)	(0.006)	(0.007)	(0.008)
All other causes (SRCI=-0.003)	-0.013	0.013	-0.027	-0.029
	(0.036)	(0.042)	(0.043)	(0.039)
N	15686.000	15686.000	17148.000	17148.000
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on concentration index of prescription frequency and the attainment relative concentration index of prescription probability by diagnosis since graduation and up to 2013 for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression and captures the marginal effect on the inequality index. Model (1) is a simple correlation of university attendance and health inequality. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

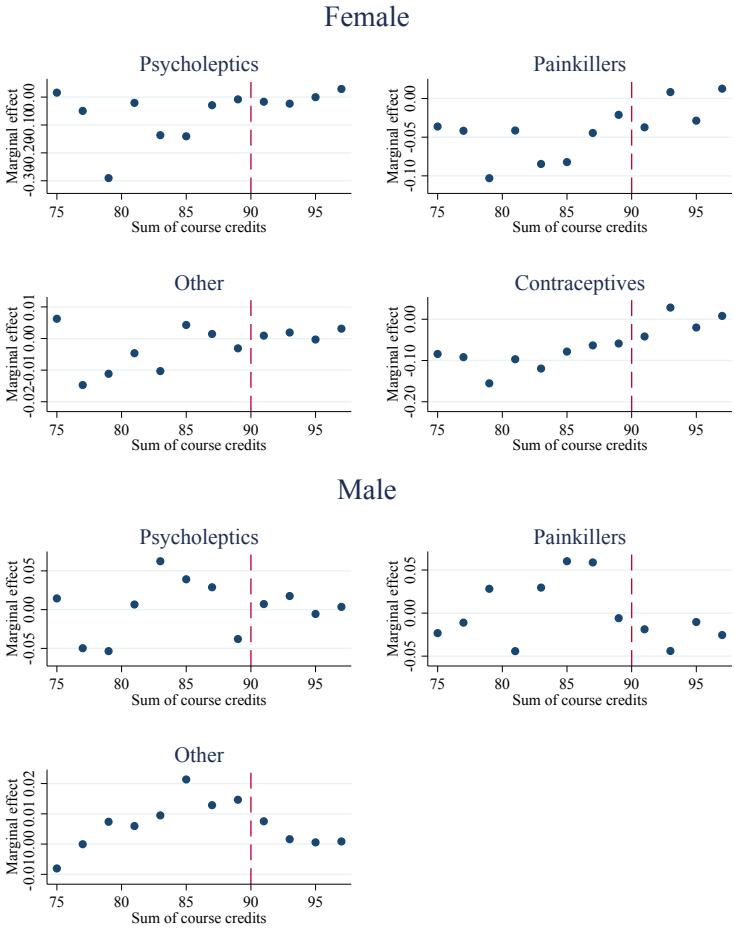

Fig. A.3: Impact of university eligibility on the attainment relative concentration index of *income inequality* of prescription receipt by cause

Notes: These figures plot a scatter of the recentered influence function of attainment relative concentration index income related inequality of frequency of prescriptions against the final achieved course credits with a bin width of 2pp of a full course in each bin for those graduating upper from secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

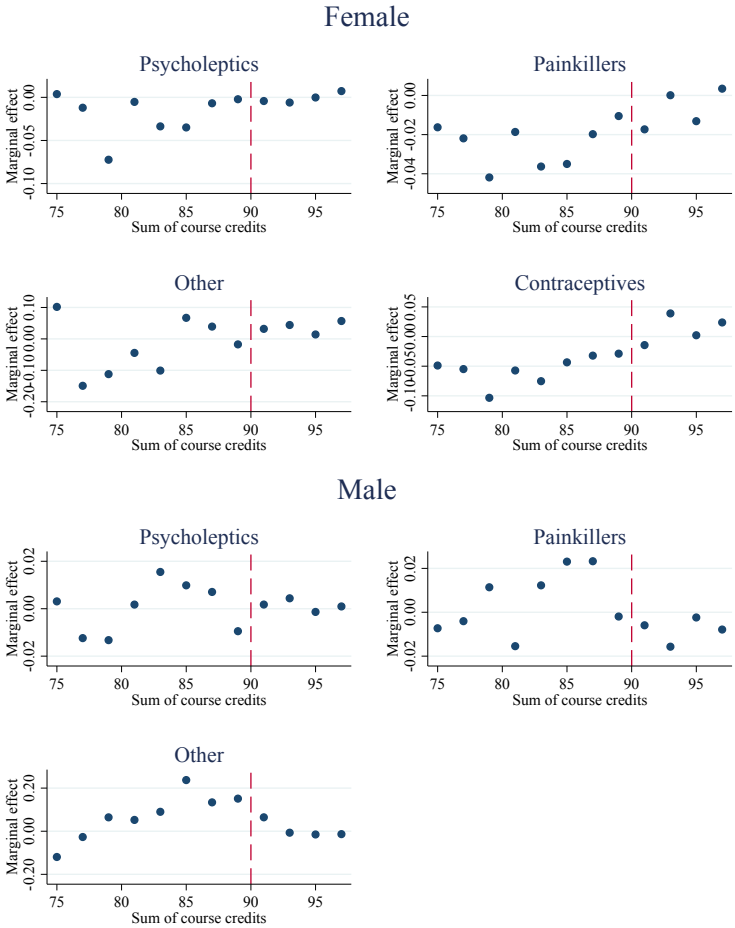

Fig. A.4: Impact of university eligibility on the short-fall relative concentration index of *income inequality* of prescription receipt by cause

Notes: These figures plot a scatter of the recentered influence function of short-fall relative concentration index income related inequality of frequency of prescriptions against the final achieved course credits with a bin width of 2pp of a full course in each bin for those graduating from upper secondary school between the years 2003 and 2005 (academic stream). See notes for figure 1.

Table A.2: Impact of university eligibility on short-fall relative concentration index if parental income related prescription probability by cause

	(1)	(2)	(3)	(4)
Bandwidth	8pp	8pp	16/8pp	16/8pp
			Females	
Contraceptives (SRCI=0.01)	0.041***	0.010	0.024	0.006
	(0.006)	(0.020)	(0.019)	(0.010)
Psycholeptics (SRCI=-0.000)	0.006	-0.016***	0.002	-0.002
	(0.004)	(0.004)	(0.009)	(0.009)
Painkillers (SRCI=-0.007)	0.011	-0.014**	0.001	-0.001
	(0.007)	(0.006)	(0.008)	(0.007)
Other (SRCI=0.034)	-0.022	-0.006	-0.007	-0.016
	(0.019)	(0.062)	(0.046)	(0.047)
N	12652.000	12652.000	13523.000	13523.000
			Males	
Psycholeptics (SRCI=-0.000)	0.002	0.018***	0.003	0.003
	(0.003)	(0.005)	(0.008)	(0.008)
Painkillers (SRCI=-0.007)	-0.006	-0.009	-0.024*	-0.024*
	(0.006)	(0.011)	(0.012)	(0.012)
Other (SRCI=0.034)	-0.029	-0.103***	-0.161***	-0.162***
	(0.031)	(0.035)	(0.042)	(0.044)
N	15686.000	15686.000	17148.000	17148.000
Polynomial	0	1	1	1
Covariates	N	N	N	Y

Notes: This table shows the regression discontinuity estimates of the impact of university eligibility on concentration index of prescription frequency and the attainment relative concentration index of prescription probability by diagnosis since graduation and up to 2013 for those graduating between years 2003 and 2005, academic stream only. Each coefficient is from a separate regression and captures the marginal effect on the inequality index. Model (1) is a simple correlation of university attendance and health inequality. Models (2) and (3) use a linear trend in credits either side of the cut-off but different bandwidths. Model (4) is as model (3) and also includes covariates as outlined in table 2. Robust standard errors clustered at number of credits achieved are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$

Paper IV

Too young to die: Regression discontinuity of a two-part minimum legal drinking age policy and the causal effect of alcohol on health*

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Abstract

This study examines the impact of Sweden's unique two-part Minimum Legal Drinking Age (MLDA) policy on alcohol consumption and health using regression discontinuity design. In Sweden on-licence purchasing of alcohol is legalised at 18 and off-licence purchasing is legalised later at 20 years of age. We find an immediate and significant 6% jump in participation and a larger increase in number of days drinking at age 18 of about 16% but no large jumps at age 20. No discernible increases in mortality at age 18 or 20 are found but hospital visits due to external causes do see an increase at both 18 and 20 years. Compared to previous findings for single MLDAs the alcohol impacts we find are smaller and the health impacts less severe. The findings suggest that a two-part MLDA can help young adults in their transition to unrestricted alcohol and help contain the negative health impacts that have been observed elsewhere.

Keywords: Alcohol consumption, regression discontinuity, minimum legal drinking age

JEL Classification: *I12, I18*

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1 Introduction

Worldwide, a common alcohol consumption control policy aimed at young adults is a Minimum Legal Drinking Age (MLDA). Even though MLDAs are widespread and long established policies, the impact of MLDAs on alcohol consumption, health and crime remains a much discussed issue. For example, in the United States there has recently been a high profile debate regarding lowering the MLDA from 21 to 18 years because some believe the current MLDA of 21 years is actually more harmful to health than an MLDA of 18 years (Carpenter and Dobkin, 2011). In Australia there has recently been a debate calling for a rise in the MLDA of 18 to 21 (Toumbourou et al., 2014; Lindo and Siminski, 2014). In India it is currently up to each province to decide the MLDA but there is discussion as to whether the federal government should set an India wide MLDA (ADD, 2015) and more widely there are still 18 countries (mainly low income countries) that have no MLDA at all (WHO, 2014).

Many countries have an MLDA in order to protect young adults in their transition to adulthood because alcohol is a poison with a range of negative biological and behavioural consequences, where heavy drinking is noted to immediately and negatively impair vision, balance and judgement. Heavy drinking is highly prevalent among young adults where, according to the data in this current study, over half of all Swedes aged 18-21 report heavy drinking at least once a week and this is similar in other countries, notably the United States (Carpenter and Dobkin, 2009). Among young adults, alcohol related causes of death that are often associated with heavy drinking are homicides, motor vehicle related accidents, falls and alcohol and narcotics specific related deaths (Miller et al., 2007). It has therefore been deemed important that we provide young adults with the best conditions to allow them to make the transition to unrestricted alcohol access in a safe manner. MLDAs have been part of a policy mix to help reduce the harmful consequences of alcohol by delaying legal access to alcohol until young adults are mature enough to understand the negative consequences of heavy consumption.

The hope is that the way an MLDA has been designed minimises the widely documented costs that can come with increased access to alcohol. Yet, as Carpenter and Dobkin (2011) note, we do not have the evidence required to define the optimal policy design for an MLDA. Whilst a later MLDA restricts access to young adults there is potentially a downside in that under-age drinking in uncontrolled environments is in effect encouraged and this in turn leads to reckless drinking (this is the concern in the United States). However, if an MLDA is introduced too early then young adults may not be mature enough to make more informed decisions in light of the negative consequences of alcohol consumption, which in turn may lead to excessive drinking. Instead of the evidence we need to balance this trade-off, we have to rely on evidence of the local effects of the existing MLDAs

on alcohol consumption and health. Even within this limited sphere the existing evidence is fairly limited and focuses on one form of MLDA - a complete ban on the purchasing of alcohol on or off premises. Primarily this evidence comes from the United States and concerns a single MLDA at age 21 years.

In this paper we add to the literature on the impacts of an MLDA on alcohol and health by examining the impact of Sweden's two-part MLDA of 18 years for on-licence purchasing of alcohol (consumption of alcohol at restaurants, bars and clubs) and 20 years for off-licence purchasing of alcohol using regression discontinuity design (RDD). This policy is fairly unique internationally and has not been assessed previously. Its assessment allows us to examine the potential differing effects of a two-part MLDA on alcohol consumption patterns and health and compare these effects to those documented for single MLDAs in the literature. The evidence we present here will therefore contribute to the evidence base as to what form an optimal MLDA policy should look like. An additional contribution of this paper is that we establish for young adults the causal effect of alcohol on mortality and hospital admissions. Alcohol consumption is determined by so many hard to observe factors that also potentially determine health outcomes and this means that any strategy relying on controlling for observables will almost certainly be biased. Variation in alcohol consumption that is exogenous is also hard to find because it is unethical to run randomised experiments that involve determining an individual's alcohol consumption. Using our identification strategy of RDD however, we are able to identify the causal effect of alcohol on health under plausible and testable assumptions. Evidence of the causal relationship between alcohol consumption and health outcomes is relevant for the development of public health policy.

Sweden's two-part MLDA was introduced in 1917 when it became illegal to sell alcohol on-premise to under-18s and to sell off-premise to under-21s, which was reduced to under-20s in 1969 and so it has remained up until today (Johansson, 2008). The design of the Swedish MLDA allows legal access to alcohol for 18-19 year olds but under the supervision of the licencee. This should minimise the health risks compared to what may have occurred if 18-19 year olds were not in a controlled environment and able to drink as much as they pleased. Indeed, it is the duty of the licencee to not sell alcohol to an individual who is deemed to be "noticeably under the influence of alcohol" (SFS, 2010). At age 18, in addition to legal availability of on-licence drinking it is also legal to purchase low alcohol content beer (max 3.5% volume) from general stores. Sweden has a state-run off-licence (Systembolaget) and this is the only outlet where one can buy alcohol over 3.5% in volume.[1] The state-owned monopoly off-licence has restricted hours and has a specific duty to encourage a controlled and healthy attitude to alcohol. The two year age gap between the two MLDAs was chosen in order to reduce the potential peer effect of elders purchasing alcohol for

[1] This is not to say that home brew and illegal purchasing of alcohol is not prevalant

minors (Johansson, 2008). In sum, at age 18 there are three restrictions in play that are removed at age 20: alcohol is not as readily available, is only legally available under supervision of a licencee and at a substantially higher price than at the off-licence.

We assess the impact of Sweden's MLDA on alcohol consumption using the Monitor Project survey of alcohol consumption patterns covering the years 2001-2011. Impacts on health are assessed using individual administrative data from the Ministry for Health that records the causes of death and hospital admission and exact age at death/admission for the years 1969-2015 (2001-2015 for outpatient data). The results show very different impacts at age 18 and at age 20. We find that on-licence availability at age 18 leads to a 6% increase in participation and the proportion heavy drinking and an 8% increase in those drinking more than the recommended weekly amount (risky drinking) compared to those who haven't quite turned 18 years of age. The largest impacts were found for the number of days drinking, lying in the range of between 16% - 22% depending on alcohol type. For those who had just turned 20 years of age we find a substitution away from home brew and illegally purchased alcohol but no overall impact on quantity, frequency or intensity. The jump at 18 in alcohol consumption coincides with a jump in hospital admissions due to external causes, largely driven by alcohol and homicide causes. At age 20, even though we find no clear jump in alcohol consumption we find a small negative fall in mortality, driven by a 14% fall in suicides and an increase in hospital admissions due to external causes driven by increases in self-harm and homicide related hospital admissions. The changes in alcohol consumption at 18 are less than observed for the United States at age 21 and for Australia at age 18 suggesting that the two-part MLDA does help control the behaviour of young adults when making the transition to an unrestricted alcohol regime. The impact of the two-part MLDA on health outcomes suggests that this more controlled transition to an unrestricted alcohol regime has helped avoid the increases in mortality observed in other countries although costs remain in terms of impact on medical care services.

Beyond considering a novel alcohol control policy, this paper contributes to the literature interested in the impacts of MLDA on alcohol consumption and alcohol's impact on health in several ways. First, our results present the impact of an MLDA in a new environment and therefore, in part, add to the robustness of previous findings. Second, compared to previous studies we make use of unusually detailed data on alcohol consumption patterns that allows analysis split by alcohol type. We are therefore able to give a more detailed picture as to the impact of the policy on alcohol consumption patterns. Third, we are able to split the results for alcohol consumption and health outcomes by gender, which has not been done previously regarding the effects of the MLDA. There are important consumption and biological differences between the genders and therefore any response to the MLDA in alcohol consumption and its impact on health may differ by gender. Fourth, our health outcome data is based on individual level registry data and therefore has correspondingly

low measurement error. We have combined this data with information on exact date of birth to be able to identify a discontinuity with high potential precision.

The rest of the paper is organised as follows: Section 2 reviews the relevant literature. Section 3 describes the data in detail before Section 4 sets out the empirical strategy. Section 5 presents the results on alcohol consumption patterns and health outcomes including various sensitivity checks. Section 6 summarises and concludes the paper.

2 Literature review

As Wagenaar and Toomey (2002) note in their review of the impact of MLDAs on alcohol consumption and traffic accidents, the MLDA is the most well-studied alcohol policy in the United States of America. The authors review the literature from 1970 - 2000 and find the evidence points to a reduction in alcohol consumption and traffic related accidents. Of the more robust statistical approaches reviewed, these largely relied on variation in the MLDA between states. In the 1970s and 1980s various states experimented with lowering the MLDA from 21 to anything as low as 18. Various studies used this natural experiment to assess the impact of MLDA on alcohol consumption and its impact on mortality. However, as Carpenter and Dobkin (2009) note, there remains a concern that unobserved heterogeneity remains and therefore the estimates are biased. It is possible that state level preferences influenced the decision to lower the MLDA and that these preferences are also linked to attitudes towards low age alcohol consumption and the risks of drink driving.

The more recent literature on the impact of MLDAs on alcohol consumption and the causal effect on health has largely relied on the quasi-experimental statistical technique of RDD. This non-parametric technique provides a local treatment effect and is deemed to have very high internal validity and as a consequence has received a great deal of attention in the economics literature in terms of refinement in its application (Hahn et al., 2001; Imbens and Lemieux, 2008; Lee and Lemieux, 2010; Imbens and Kalyanaraman, 2011). In a reduced form set-up sharp RDD yields the local impact of the rule change on the outcome of interest. If one is willing to stipulate a structural relationship between two variables then RDD can also be used as an instrument in a Wald type estimator set-up as used by Carpenter and Dobkin (2009). In fact, as utilized by Carpenter and Dobkin (2009), the reduced form estimates and the first stage estimates can come from two entirely different datasets making causal inference a more tractable proposition from an empirical perspective. The empirical conditions of the MLDA lend themselves to RDD and recent research into the impact of MLDAs on various health outcomes has yielded some convincing results.

In the United States the MLDA of 21 years of age has been found to lead to an increase in the number of drinking days by 21% for those turning 21 and that this increased alcohol consumption

causes an increase in the mortality rate of 9% (Carpenter and Dobkin, 2009). The authors have detailed information on the causes of death and find that road accident related deaths increase at the cut-off as well as external causes due to alcohol and suicide. Unfortunately the data on alcohol consumption patterns was not ideally suited to the RDD set-up as the questions related to how individuals had drunk in the last 12 months. The fact that the authors find a positive effect implies that the effect would have been larger if data with a greater level of precision had been available. Indeed Yörük and Yörük (2011) use better suited alcohol data combined with RDD, that asks about alcohol consumed in the last month since interview, and find a much larger impact on the number of days drinking alcohol of about 27% for those turning 21. This suggests that the causal estimates of Carpenter and Dobkin (2009) are overstated because a larger first stage estimate will reduce the final Wald type causal estimate. The impact of the United States MLDA at age 21 on additional health related outcomes other than mortality is considered by Yörük and Yörük (2011, 2013, 2012, 2015) who consider smoking and marijuana use, psychological wellbeing and risky sexual behaviour respectively. Beyond the significant discrete increase in alcohol consumption associated with the MLDA of 21, they find no discernible spill over effects of the discrete increase in alcohol consumption on smoking, marijuana use, psychological well being or risky sexual behaviour. Other evidence from the United States finds that the MLDA at age 21 reduces hard drug use (Deza, 2015).

Whilst the majority of evidence is from the United States there is also evidence from other countries. This is important as it helps us understand to what extent institutional and cultural differences impact on the effect of an MLDA and also how different ages at which an MLDA is imposed matters. Evidence from Canada using RDD has found the MLDA of 19 (except in Alberta, Manitoba and Quebec where it is 18) results in a discrete jump in mortality just after the MLDA age of about 14% which is in line with the evidence from the United States (Callaghan et al., 2014). In another paper Callaghan et al. (2013) also look at inpatient hospitalisations in Canada and find that hospital admissions jump at the MLDA. Evidence from Australia (Lindo et al., 2016) has found that the MLDA at 18 does not lead to a large increase in motor vehicle related accidents unlike in the United States at age 21 despite observing a near doubling in reported days drinking for those just turning 18 and increased hospitalisations due to alcohol and homicides. The authors claim this is due to a large and focussed campaign aimed at reducing drink driving. The evidence suggests that the existence of an MLDA does delay the negative health impacts of alcohol consumption by delaying the alcohol consumption levels that are associated with unrestricted alcohol access and that this impact is observed for different ages of MLDA implementation. The evidence also suggests that the institutional setting is important in determining how the MLDA impacts health outcomes.

3 Data

3.1 Alcohol data

To quantify the effect of the MLDA on alcohol consumption patterns we use survey data collected as part of the Monitor project on drugs and alcohol (SoRAD, 2015). This is a repeated cross-sectional survey performed by monthly telephone interviews of roughly 1,500 individuals per month.[2] The data covers the period 2001-2011, individuals are aged between 16 and 82 years of age and the data includes detailed questions regarding an individual's drinking patterns. The forcing variable in our analysis is age and for the Monitor project data we have year and month of birth and year and month of interview through which we create age to the nearest month at the time of interview.[3] The outcomes we consider are whether an individual drank or not and various patterns of consumption: heavy drinker; risky drinker; quantity of pure alcohol; drank unregulated alcohol; and frequency (days drinking) and intensity (number of drinks) of drinking occasions, all of which cover the last 30 days since interview.[4] The definition of heavy drinker is the same for men and women, which is a weakness of the variable as it is well known that women have a lower tolerance threshold which is why we also consider another definition - risky drinker which accounts for this alcohol tolerance difference. Whilst the risky drinker variable can be considered better than the heavy drinker variable in that it accounts for gender differences, they are still quite distinct in what they capture. Simply, a risky drinker may not be a heavy drinker and vice versa, which is why we consider both variables. Frequency is defined as the number of drinking days in the past 30 days and intensity is defined as the average amount consumed per drinking occasion in the past 30 days.[5] The alcohol outcome variables are described in table 1. Background variables strongly

[2]Interviewees are chosen at random by their telephone number and then the individual who most recently had their birthday is asked to respond to the questionnaire.

[3]Whilst it would be desirable to have exact age at interview it is not clear in our case if this would reduce measurement error due to the retrospective nature of the alcohol questions. The nature of questions regarding alcohol consumption is that they have a recall period. The relevant recall period for the analysis of the MLDA should arguably tie in with the level of detail thought to be required for the forcing variable in the RDD analysis. In the case of the Monitor project data the alcohol questions cover the period of the last 30 days before interview. 30 days was chosen by the project as it gives a good picture of each individual's drinking patterns that are heavily influenced by the day of the week and time from pay day which would be lost if questions related to yesterday or the previous seven days (Ramstedt et al., 2009). As a consequence of the recall period, exact date of birth would not lead to a particular improvement in the accuracy of our estimates of the MLDA on alcohol consumption.

[4]A drinker is defined as someone who had an alcoholic drink in the last 30 days before interview. A heavy drinker, as defined by the Monitor project study, is someone who in the last 30 days has had one or more episodes where the quantity of alcohol drunk was at least: 1 bottle of wine (75cl), 5 shots of spirit (25cl), 4 cans of strong beer/cider (>3.5%) or 6 cans of low alcohol content beer (3.5%). A risky drinker is defined as someone who on average in the past 30 days drank more than the weekly-recommended limit of 21 cl (14 cl) of pure alcohol if male (female). Quantities of alcohol have been converted into centilitres of pure alcohol to allow easier comparability across alcohol types by multiplying in litre terms: low alcohol content beer by 3.5%, beer and cider by 4.62%, wine by 12.8% and spirits by 38% (Standard measures are provided by CAN (2015) and converted to % volume measures (1 cl pure alcohol is 7.8 grams of alcohol). Unregulated alcohol is defined as illegally smuggled alcohol or homebrew.

[5]There are more observations for the heavy and risky drinker variables than for the frequency and intensity variables because heavy and risky are combined over the alcohol types, whereas frequency and intensity are alcohol type specific.

associated with alcohol consumption patterns are included in some of the regressions and include gender, employment status (employed, unemployed or inactive) and county of residence.

Table 1: Alcohol consumption sample sizes near the cut-offs

VARIABLE	DEFINITION (IN LAST 30 DAYS)	MEAN (16 - 19)	N	MEAN (18 - 21)	N
Drinker	1 = Drank alcohol	0.68	9160	0.83	8970
Heavy Drinker	1 = Drank heavily	0.36	7602	0.51	7033
Risky Drinker	1 = Drank more than recommended level	0.64	9160	0.80	8970
Quantity	Quantity of pure alcohol consumed	34.32	9160	50.86	8970
Unregulated	1 = Drank smuggled alcohol or home-brew	0.17	9160	0.16	8970
Frequency	No. of days drinking	9.50	6086	10.49	7292
Freq Beer	No. of days drinking beer	3.63	3514	4.41	4582
Freq Low Alc. Beer	No. of days drinking low alc. beer	3.35	2136	3.84	2244
Freq Wine	No. of days drinking wine	2.66	2358	3.12	3556
Freq Cider	No. of days drinking beer	2.58	2134	2.77	2575
Freq Spirit	No. of days drinking spirits	2.76	3236	2.97	4331
Freq Heavy drinking	No. of days heavy drinking	3.64	2703	4.01	3562
Intensity Beer	Average cl pure alcohol/occasion	8.69	3514	8.94	4582
Intensity Low Alc. Beer	Average cl pure alcohol/occasion	4.89	2136	4.35	2244
Intensity Wine	Average cl pure alcohol/occasion	4.61	2358	5.00	3556
Intensity Cider	Average cl pure alcohol/occasion	5.29	2134	5.33	2575
Intensity Spirits	Average cl pure alcohol/occasion	7.14	3236	6.57	4331

Notes: All alcohol variables correspond to the previous 30 days before date of interview. Sample sizes are calculated for two years either side of the cut-offs and give a representation of the sample size used in the analysis.
Source: Monitor Project Survey Data. Own calculations.

A concern for the question at hand is the potential for discontinuities in unobservables at the cut-off, and in particular under-reporting of alcohol participation/consumption due to social desirability bias. The concern is that those who are under 18 may report lower levels of consumption or non-participation than actually occurred due to its illegality and this behaviour may abruptly change after turning 18 (this should not really be an issue for the 20 years of age cut-off given it is acceptable to drink in pubs and bars already just not purchase from the off-licence). If this does occur the results would be biased. There are a few reasons as to why we think this is not the case in this instance. First, previous research considering the impact of MLDA in the United States provides convincing evidence that this is not a problem (Carpenter and Dobkin, 2009). Second, we observe a jump in health outcomes suggesting that the discrete jump in alcohol consumption we observe is real and not due to desirability bias.

3.2 Health data

To quantify the impact of the MLDA on various health measures we use two population based administrative register data sources from Sweden. Information on mortality is provided by the Swedish cause of death administration dataset. This dataset captures the universe of deaths for the years from 1969-2015 and includes information on cause of death and exact age at death

(Socialstyrelsen, 2015a). We use an extract that is for all individuals aged 12-26 years of age. There may also be an impact on hospital use due to an increase in alcohol consumption possibly directly due to poisoning or indirectly due to violence and accidents. We therefore use population based inpatient and hospital based outpatient administrative data that includes information on cause of visit and exact age at hospital visit (Socialstyrelsen, 2015b). This dataset also captures the universe of hospital admissions for individuals admitted between 12 and 26 years of age for the years from 1969-2015 for inpatient care and for the years 2001-2015 for outpatient care.[6] The patient data includes information on cause of hospitalisation and exact age at registration at the hospital and also exact age at discharge. Causes of death and hospitalisation are outlined in table A.1 in appendix A and are causes that are commonly linked to alcohol use for young adults - that is external causes and specific sub-set of external causes (motor vehicle related accidents, homicides, suicides, alcohol related causes and narcotics related causes). Internal causes are defined as all other causes not defined as due to external causes. We view internal causes as causes not immediately impacted by alcohol consumption. We define someone as having a particular cause if any of the recorded diagnoses mention the particular cause of interest, that is we do not just use main diagnosis. Background variables strongly associated with alcohol consumption, gender, whether foreign born and county of residence, are included in some regressions. Controls are also included for birthday and day after birthday and for counties that simultaneously introduce out of pocket (OOP) medical care costs alongside changes in MLDA.[7] Indicative sample sizes are shown in table 2.

[6]Inpatient care is for visits of 24 hours or more and outpatient care includes emergency room visits and visits to a specialist at a hospital

[7]At age 18 the counties of Stockholm and Gotland introduce small OOP costs and Skåne does so from 2012. All other counties introduce a small OOP cost at age 20 except those that did so at 18 and Sörmland, Jönköping, Kronoberg and Västmanland who start OOP the calendar year a person turns 20. In 2017 these OOP costs were about 150sek, which equated to about 18 dollars

Table 2: Health outcomes descriptive statistics

VARIABLE	N (16 - 19)	N (18 - 21)
Deaths:		
Any cause	9,407	11,765
Internal causes	2,727	2,972
External causes:	6,680	8,793
Alcohol related	181	308
Motor vehicle related accident	3,194	3,712
Suicide	1,578	2,465
Homicide	200	281
Narcotics related	139	352
Other external cause	1,388	1,675
Hospital visits:		
Any hospital visit	6,950,229	7,157,609
Internal causes	4,946,119	5,114,487
External causes:	1,198,843	1,229,338
Alcohol related	62,109	71,319
Motor vehicle related accident	112,455	111,074
Self-harm	43,084	47,732
Homicide	51,393	61,705
Narcotics related	47,838	79,366
Other external cause	881,964	858,142

Notes: Sample sizes are calculated for 2 years either side of the cut-offs and give a representation of the sample size used in the analysis.
Source: Death and Patient administrative data. Own calculations.

4 Empirical strategy

In order to estimate the impact of Sweden's two-part MLDA on alcohol consumption and on health we use RDD as our identification strategy. Figure 1 depicts the age profile of four different binary measures of participation: drank in the last 30 days, drank heavily in the last 30 days, drank a risky amount in the last 30 days and drank unregulated alcohol in the last 30 days. In addition figure 1 also depicts the age profile of total alcohol consumed in the last 30 days and the frequency of drinking occasions (given participation). The raw data is graphed as scatter plots of the mean by age measured in months. The MLDA cut-offs at 18 years and 20 years are indicated by the dashed vertical lines. As can be seen in the figure, alcohol consumption has a strong age profile that is a smooth profile increasing during the late teen years and then tailing off somewhat during the early 20s. However, there is an arbitrary chosen rule, the Swedish MLDA that leads to potential jumps in behaviour because of legalised access and a jump in ease of access to alcohol consumption at ages 18 and 20 and as a result there may also be a jump in alcohol related health outcomes at these cut-offs. In figure 1 we observe a clear jump in participation, risky drinking and in total quantity consumed at age 18 years but no positive jumps at age 20.

RDD allows us to test whether these jumps are statistically significant or just part of the age profile of alcohol consumption. The general formulation for the regression equation we estimate is the following:

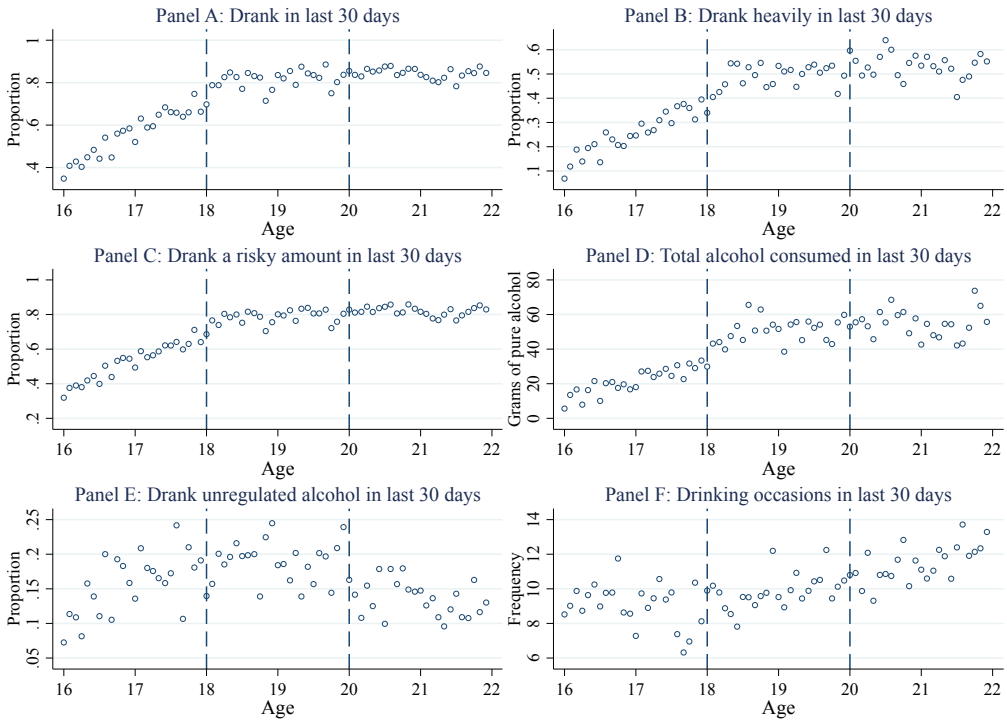

Fig. 1: Drinking behaviours by age

Notes: This figure plots the scatter points of mean drinking behaviour by monthly age blocks.

Source: Monitor Project Survey Data. Own calculations.

$$y_i = \alpha + \beta MLDA18_i + f(age_i) + \varepsilon_i; \tag{1}$$

$$y_i = \alpha + \beta MLDA20_i + f(age_i) + \varepsilon_i; \tag{2}$$

In this model y_i represents the various alcohol consumption patterns or health outcomes we consider for individual i, MLDA is a binary variable equal to unity for ages above the MLDA threshold (which is either 18 or 20 depending on which threshold we are investigating) and age is measured in months from the MLDA threshold using the alcohol data and days from the MLDA threshold using the data on health outcomes. The identification strategy we use here is a functional form for age, $f(.)$ that eliminates the potential endogenous relationship between age and the error term, ε.

The key identifying assumption is that near the MLDA threshold the relationship between age and the error term is a smooth function with no jumps or spikes as we cross the threshold but there is a discontinuity for the outcome variable. We follow Imbens and Lemieux (2008) and use a Local Linear Regression (LLR) in place of $f(.)$. The functional form for the forcing variable (age_i) then becomes a smoothed linear function and we choose to estimate this separately either side of the cut-off. The coefficient β is the discontinuous effect of the MLDA on the outcome variable assuming that our functional form absorbs any potential relationship between age_i and ε_i. LLR is preferred to parametric polynomial regression because it puts greater effort into estimating the curve near the cut-off whereas polynomial regressions focus on the sample population, which is not the focus of our analysis. Following Imbens and Lemieux (2008) we use a rectangular kernel and estimate on the entire sample, but use a bandwidth as determined by the optimal bandwidth algorithm of Imbens and Kalyanaraman (2011) (IK from here on in) which calculates the optimal bandwidth for each LLR equation.[8] It is preferred because it optimises the trade-off between accuracy and efficiency. In addition to the IK bandwidth estimates we present the estimates from a LLR using a bandwidth of half and double the IK optimal bandwidth size. Varying the bandwidth allows us to assess how sensitive the results are to bandwidth choice and therefore give a sense of the robustness of the results to model specification. Presenting alternative bandwidth choices also highlights immediately the accuracy efficiency trade-off of the bandwidth selection. We choose to use bandwidths that are the same size either side of the cut-off.

Equations (1) and (2) applied to health outcomes are reduced form regression equations and applied to alcohol consumption are first stage regression equations that capture the intention to treat (ITT) impact of the MLDA. It is also possible to use these estimates as part of an instrumental variables strategy assessing the causal effect of alcohol consumption on health. This is performed by dividing the estimate of discontinuity in health by the estimate of the discontinuity in alcohol yielding a Wald type IV estimator.

As mentioned above, our identification strategy builds upon the assumption that any jumps observed in our outcome variables at the cut-offs are purely due to the rule changes, once we have fully controlled for the smooth relationship between age and the outcome variable. There are, however, potential confounders with our MLDA cut-offs. For alcohol consumption and the MLDA at 18, a potential confounder is the minimum legal age for purchasing of tobacco which is also legalised at 18.[9] We can expect a jump in tobacco use due to this policy and this may independently impact on an individual's drinking behaviour through peer effects or potential complementary effects between alcohol and tobacco consumption. However, we find no jump in smoking as shown in figure

[8] The LLR are estimated over the full sample, the use of LLR ensures the estimates are local to the discontinuity.

[9] Individuals also have the right to vote and generally are deemed to have become an adult at age 18 but it is hard to imagine that these factors impact alcohol consumption nor the health outcomes under consideration.

B.1 or in table B.1 found in appendix B.

Another potential confounder with the MLDA at 18, specifically related to accidents, is that it is possible to gain a driving licence once turned 18 years of age (it is legal to drive under supervision from the age of 16). Alcohol consumption is known to increase motor vehicle related accidents in the United States which suggests this is an important confounder. We deal with this by looking at accidents by cause separating out motor vehicle related accidents. A potential confounder with hospital visits is a small (roughly 18 U.S. Dollars in 2017 prices) OOP payment required by the local health service. In our analysis we test if confounding is an issue by considering the regions who do not introduce an OOP cost on the birthday coinciding with MLDA as a sensitivity.

Beyond the potential problem of confounding it is of concern in RDD that individuals may be able to manipulate which side of the cut-off they are on. In the Monitor data this would require individuals to misreport their age discontinuously either side of the MLDA cut-offs. The incentives to manipulate the cut-off are not clear in this instance. We view it as highly unlikely that manipulation occurs as the age of the person is asked before any other question on the questionnaire. Due to the sampling methodology we are unable to test for manipulation of the forcing variable as suggested by McCrary (2008). However we illustrate the consequence of the sampling methodology in appendix C figure C.1 by way of a histogram of age just to show that this type of manipulation test isn't possible due to the sampling frame of the survey (there is a greater representation of those who have recently had a birthday reflecting the greater probability of being chosen due to the sampling frame). For the registry data or survey data on health outcomes that we discuss below, manipulating the cut-off is not an issue as these record the birth date from the individual's Swedish personal identification number, which is an official record, of which the first eight digits are the individual's birth date.

Lastly, we consider the age distribution of the covariates in the Monitor data as a diagnostic test of confoundedness. If there are jumps in the covariates at the 18 years of age and 20 years of age cut-offs this would indicate that there are other factors occurring at the same time confounding the results. In figure 2 we present the raw data for gender, unemployed and employed. LLR estimates are found in appendix C table C.1. It appears concerning that a significant at the 5% level negative jump in the proportion employed is observed at the 18 years of age cut-off, but when eyeballing the data it is there is no negative jump at the cut-off. What we do observe in the raw data in figure 2 is a large change in the slope at both cut-offs. A large bandwidth will more likely under these conditions mistake a large change in slope for a discontinuity at the cut-off and this appears to be what is happening here. Taken together the evidence suggest that there is a discontinuity in the trend of our background covariates but not a jump and that the covariates are smooth functions across both cut-offs.

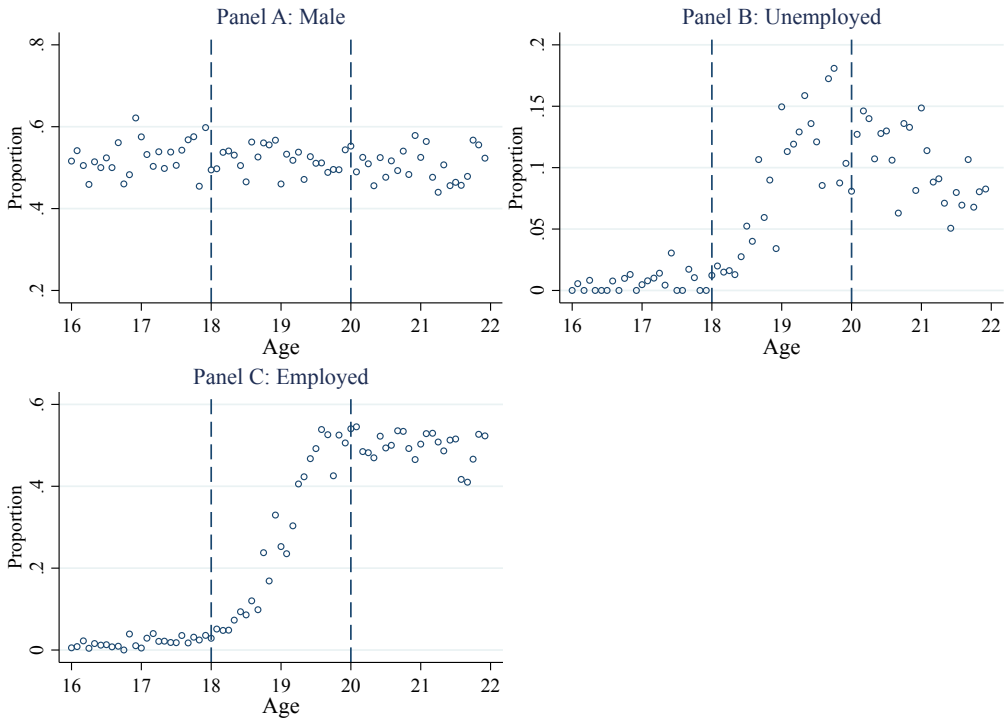

Fig. 2: Diagnostic tests: Covariates by age

Notes: This figure plots the scatter points of the mean of the covariate by monthly age blocks. The MLDA cut-offs at 18 years and 20 years are indicated by the dashed vertical lines. Male is 1 if male, 0 otherwise. Employed is 1 if employed, 0 otherwise. Unemployed is 1 if unemployed, 0 otherwise.

Source: Monitor Project Survey Data. Own calculations.

5 Results

5.1 Alcohol consumption

This section presents the estimates of the effect of Sweden's MLDA at both 18 years of age and 20 years of age on alcohol participation and patterns of consumption. As we have already seen in figure 1, there is a jump in participation, risky drinking and quantity consumed at 18 years of age and a drop in the proportion drinking unregulated alcohol at age 20. The effects of the MLDA on alcohol consumption estimated by LLR are shown in table 3. Model (1) uses the optimal IK bandwidth with a rectangular kernel and confirms there is a positive jump in participation (6 percentage points), risky drinking (8 percentage points) and quantity consumed (11.6 grams of pure alcohol) at age 18, all significant at the 1 percent level. The LLR results for model (1) also find a significant jump in the proportion who had a heavy drinking episode at 18 (6 percentage points) and a reduction in the

proportion who drank unregulated alcohol at age 20 (5 percentage points). Models (2) and (3) halve and double the size of the bandwidth used in model (1) respectively in order to test the sensitivity of the estimates to bandwidth choice. The impacts observed for participation, risky drinking, quantity and unregulated alcohol appear robust to bandwidth choice. Having had a heavy drinking episode appears much more sensitive to choice of bandwidth and this fits with what we observe in the raw data in figure 1 where it is not clear there is a jump in the proportion who participated in heavy drinking.

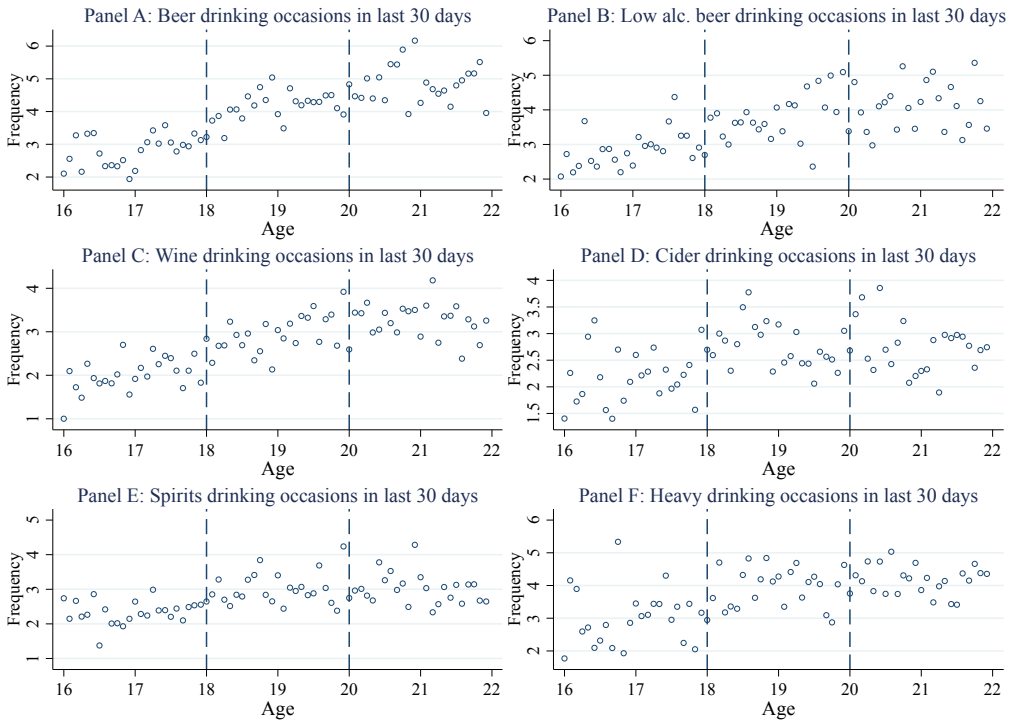

Fig. 3: Drinking frequency by age

Notes:. The scatter points are monthly age blocks representing the proportion reporting the drinking behaviour. *Source:* Monitor Project Survey Data 2001-2011 (heavy drinking frequency is asked from 2003 onwards). Own calculations.

In figure 3 we show the relationship between frequency of alcohol consumption and age for those who participate in drinking (we ignore those whose frequency is zero and therefore remove the participation effect). Figure 3 shows potential jumps in frequency of wine, cider and spirits and frequency of heavy drinking at the 18 years of age cut-off and a negative jump in frequency of low alcohol beer drinking occasions at the 20 years of age cut-off. In table 4 the LLR results

Table 3: LLR regression estimates of MLDA impact on alcohol participation

	(1)	(2)	(3)	(4)
Drank alcohol in last 30 days				
MLDA 18	0.0645***	0.0854**	0.0599***	0.0569**
	(0.0234)	(0.0370)	(0.0184)	(0.0236)
Bandwidth	1.596	0.798	3.192	1.596
MLDA 20	0.0179	0.0219	0.00103	0.0116
	(0.0253)	(0.0414)	(0.0165)	(0.0247)
Bandwidth	1.104	0.552	2.208	1.104
Drank more than recommended weekly amount in last 30 days				
MLDA 18	0.0677***	0.0760**	0.0629***	0.0594**
	(0.0251)	(0.0380)	(0.0190)	(0.0252)
Bandwidth	1.505	0.753	3.011	1.505
MLDA 20	0.0295	0.0487	0.0128	0.0256
	(0.0292)	(0.0495)	(0.0182)	(0.0283)
Bandwidth	0.985	0.493	1.971	0.985
Had a heavy drinking episode in last 30 days				
MLDA 18	0.0649**	0.0224	0.0640***	0.0568*
	(0.0295)	(0.0471)	(0.0210)	(0.0291)
Bandwidth	1.488	0.744	2.977	1.488
MLDA 20	0.0348	0.0290	-0.0133	0.0628*
	(0.0354)	(0.0555)	(0.0229)	(0.0357)
Bandwidth	1.258	0.629	2.515	1.258
Quantity of pure alcohol consumed in last 30 days				
MLDA 18	12.09***	11.81***	15.44***	11.23***
	(3.047)	(4.329)	(2.578)	(2.839)
Bandwidth	2.579	1.289	5.158	2.579
MLDA 20	-4.130	6.234	-6.236**	-4.135
	(3.493)	(5.628)	(2.525)	(3.401)
Bandwidth	2.625	1.313	5.250	2.625
Drank unregulated alcohol in last 30 days				
MLDA 18	-0.00387	-0.00529	-0.0142	-0.00154
	(0.0197)	(0.0315)	(0.0152)	(0.0191)
Bandwidth	1.612	0.806	3.224	1.612
MLDA 20	-0.0466**	-0.0924**	-0.0493***	-0.0401*
	(0.0237)	(0.0381)	(0.0148)	(0.0238)
Bandwidth	1.301	0.650	2.602	1.301
Frequency of drinking occasions in last 30 days				
MLDA 18	1.085	2.805**	0.364	1.565**
	(0.745)	(1.107)	(0.538)	(0.769)
Bandwidth	1.371	0.685	2.741	1.371
MLDA 20	-0.223	-0.678	1.002***	-0.0886
	(0.532)	(0.850)	(0.377)	(0.540)
Bandwidth	1.920	0.960	3.841	1.920
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on alcohol consumption patterns from a LLR model using age in years and months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. All coefficients are from separate regressions. Columns 1 and 4 use the IK optimal bandwidth. Columns 2 and 3 use a bandwidth half and double the size of the IK optimal bandwidth. Column 4 includes covariates. Covariates include gender, economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor Project Survey Data. Own calculations.

using the optimal IK bandwidth (model 1) find that for 18 year olds there is a jump in frequency for all alcohol types and for heavy drinking but this is only statistically significant for beer, cider, spirits and heavy drinking episodes. The jump for frequency of beer consumption is 0.5 days/month, 0.6 days/ month for cider consumption, 0.5 days/month for frequency of spirits consumption, 0.6 days/month for frequency of heavy drinking episodes. Varying the bandwidth (models 2 and 3) does not lead to large changes in estimated impact of the MLDA on frequency of beer, cider and spirits consumption and heavy drinking for 18 year olds suggesting these are robust estimates. Overall the results suggest that frequency of beer, cider and spirits increased by about 0.4-0.6 occasions and heavy drinking episodes increased by about 0.6-1 occasions for 18 year olds. For 20 year olds, no significant jumps are observed except for frequency of cider consumption which jumps by about 0.6 occasions, but this is not robust to bandwidth selection. The coefficient estimate for frequency of low alcohol beer consumption at age 20 is fairly robust to bandwidth selection but is not precisely estimated.

In appendix D we investigate the relationship between age and alcohol consumption intensity. Eye balling the data in figure D.1 there appears to be little impact of the MLDA on intensity of consumption. The LLR results confirm this. Interestingly the MLDA appears to reduce intensity of consumption for all alcohol types but the only significant and robust results are found for beer and spirits consumption at 18 years (a reduction in the range of 0.8 to 1.7 grams of pure alcohol where 14 grams is equivalent to a "standard drink"). 20 year olds appear to switch away from low alcohol beer which would go hand in hand with now being able to shop at the off-licence. Together with the evidence on frequency of drinking episodes the results suggest that the increased ease of access to alcohol at age 18 does not change how individuals drink, rather it increases how often they drink and if anything intensity appears to fall after gaining easier access to alcohol but this fall is not economically meaningful.

The estimated impacts of the MLDA on frequency and intensity are affected by the fact that we only consider the sub-sample that drink and this sub-sample changes discontinuously at the cut-off. The estimates are therefore a combination of a change in the population that drink (population effect) and a change in the overall pattern of how people drink (distribution effect). We assess if there is a distribution effect for frequency and intensity in appendix E by plotting the distribution densities of frequency and intensity for ages 17 and 18, and 19 and 20. Whilst this analysis is not causal it indicates that there is a shift towards more frequent alcohol consumption for 18 year olds but not so much for 20 year olds. For intensity there appears to be a reduction in spirits intensity for both 18 and 20 year olds. Together this suggests that on top of greater participation after turning 18 individuals also drink more often and drink heavily more often but in proportion to how they drank before and potentially even in a more controlled manner. This is similar to the findings

Table 4: LLR regression estimates of MLDA impact on alcohol frequency

	(1)	(2)	(3)	(4)
Frequency of beer consumption in last 30 days				
MLDA 18	0.520*	0.429	0.638***	0.557**
	(0.282)	(0.425)	(0.232)	(0.278)
Bandwidth	1.713	0.857	3.426	1.713
MLDA 20	0.0347	0.424	0.0632	-0.0272
	(0.205)	(0.304)	(0.165)	(0.205)
Bandwidth	3.082	1.541	6.165	3.082
Frequency of low alcohol beer consumption in last 30 days				
MLDA 18	0.146	0.0810	0.432	-0.119
	(0.303)	(0.341)	(0.284)	(0.300)
Bandwidth	3.763	1.881	7.525	3.763
MLDA 20	-0.115	-0.250	-0.173	-0.140
	(0.301)	(0.460)	(0.235)	(0.294)
Bandwidth	3.235	1.617	6.470	3.235
Frequency of wine consumption in last 30 days				
MLDA 18	0.172	0.358	0.510**	0.242
	(0.227)	(0.315)	(0.207)	(0.239)
Bandwidth	2.259	1.129	4.518	2.259
MLDA 20	-0.151	-0.0533	-0.190	-0.332*
	(0.191)	(0.287)	(0.152)	(0.188)
Bandwidth	3.171	1.585	6.342	3.171
Frequency of cider consumption in last 30 days				
MLDA 18	0.596***	0.500	0.571***	0.512**
	(0.228)	(0.357)	(0.201)	(0.217)
Bandwidth	2.043	1.021	4.086	2.043
MLDA 20	0.587**	0.794**	0.00193	0.548**
	(0.268)	(0.392)	(0.184)	(0.264)
Bandwidth	1.895	0.948	3.790	1.895
Frequency of spirits consumption in last 30 days				
MLDA 18	0.481**	0.395	0.518**	0.437*
	(0.244)	(0.378)	(0.212)	(0.238)
Bandwidth	1.882	0.941	3.764	1.882
MLDA 20	-0.163	0.137	-0.193	-0.211
	(0.175)	(0.294)	(0.141)	(0.176)
Bandwidth	2.991	1.495	5.982	2.991
Frequency of heavy drinking episodes in last 30 days				
MLDA 18	0.599**	0.672*	0.835***	0.606**
	(0.285)	(0.369)	(0.262)	(0.286)
Bandwidth	2.719	1.359	5.438	2.719
MLDA 20	0.0517	0.584	0.0752	-0.0702
	(0.252)	(0.375)	(0.190)	(0.249)
Bandwidth	2.706	1.353	5.412	2.706
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on alcohol consumption patterns from a LLR model using age in years and months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. See notes for table 3. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor Project Survey Data. Own calculations.

in Carpenter and Dobkin (2009) and Yörük and Yörük (2012) for the United States MLDA at age 21.

In appendix F we present the results for alcohol consumption by gender. The estimates reveal that the participation impacts at 18 years of age are larger for males than for females where 8% more males drank after the cut-off vs 3% more for females, 10% more males consumed a risky amount of alcohol vs no impact for females and 8% more males participated in heavy drinking vs 5% for females. Similarly, males also saw the largest increase in quantity of alcohol consumed of 12g vs 8g for women. The increases in frequency at age 18 of beer, cider, spirits and heavy drinking observed in table 4 appear to be driven by a mix of both men or women depending on the alcohol type: both men and women see an increase in frequency of beer consumption, cider's increase appears to be driven by men, spirit's increase appears to be driven by women and heavy drinking appears to be driven by men. The drop in intensity of spirits consumption at age 18 is driven by both men and women but the effect is much larger for women.

Turning to the other covariates, we can see that including covariates in our LLR estimates (comparing model (1) to model (4) in tables 3, 4, D.1) has negligible effect on the estimates. This suggests that these covariates are a smooth function of age over the cut-offs of 18 and 20 years. As a further sensitivity test we also consider in appendix G the potential birthday effect. The concern is that the impacts we observe are not persistent jumps but just the very short-term impact of birthday celebrations. Comparing results that include the birthday month and exclude the birthday month finds no major difference, if anything the impacts are larger for those excluding the birthday month, suggesting that our results are not just birthday effects. Together with the diagnostics tests of section 4 this suggests that the covariates that we are unable to observe are also smooth across the cut-off and that the jumps we observe are due to the policy effects alone and not a discontinuity in some third unobserved factor.

In sum the MLDA at 18 that legalised on-premise alcohol consumption sees a jump in participation generally of 6%, participation in heavy drinking of 6%, proportion drinking a risky amount of 8% and a jump in quantity of alcohol consumed of about 12g of pure alcohol or near enough to a standard drink of 14g (a roughly 30% increase). These impacts are stronger for men than for women. Frequency of consumption also jumps for some alcohol types: beer, cider, spirits and also for heavy drinking episodes at age 18 all driven by men except for the jump in spirits frequency. Intensity of consumption episodes is unchanged at the MLDA at 18 except for spirits which drops and this is driven by women. The quantity impact observed therefore appears to be due to how often individuals choose to drink rather than in the way they drink. The MLDA at 20 has a negligible impact on alcohol consumption participation and on patterns of consumption with the notable exception of an increase in frequency of cider consumption. Unregulated alcohol is drunk

less often at the MLDA at 20 years of age implying a substitution away from these sources given no impacts are observed for quantity or participation at age 20.

5.2 Mortality

In this section we present the estimates of the effect of the MLDA on cause specific mortality. In figure 4 we plot the raw data for all deaths by age in Sweden for the years 1969-2015 and also split by external and internal causes.[10] The first thing to note is that even with a long time-span of 44 years of data, Sweden has not experienced a huge number of fatalities for this age range. It can be seen that there is an increasing death count with age and that the death count increases markedly after the age of 18. The age profile then appears to flatten out after the up-swing between 18 and 19 years. This pattern appears to be entirely driven by deaths due to external causes.

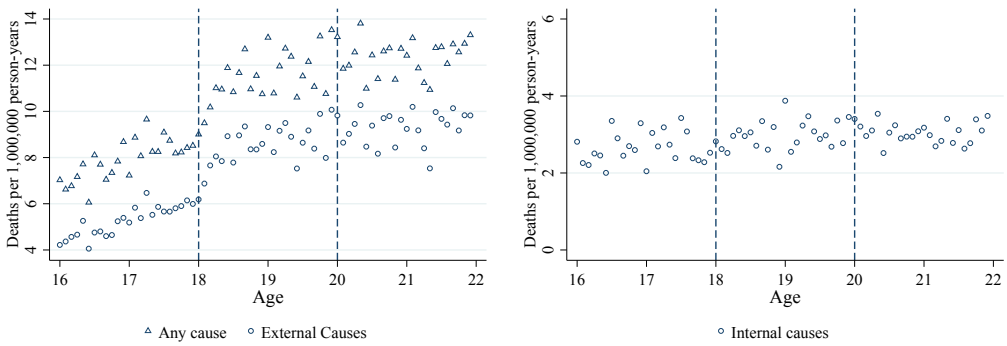

Fig. 4: Mortality by age

Notes: The scatter points are monthly age blocks of the mortality count for the years 1969-2015. External deaths are those defined as due to external causes plus alcohol and narcotics related causes. Internal causes are all remaining deaths not defined as due to external causes. The MLDA cut-offs at 18 years and 20 years are indicated by the dashed vertical lines.

Source: Death administrative data. Own calculations.

Regression analysis is of the log of the death count for each exact age at death measured in years and days for the period 1969-2015. The model is estimated over the age span 12-26 years of age inclusive. The coefficient estimates are interpreted as the percentage impact on the death count of the MLDA, so long as the effects are not too large. Models (1) through (4) are the same as the models used for the alcohol outcomes. The findings observed in the raw data in figure 4 are confirmed in table 5. Using the results from models (1-3) we find a jump in all cause mortality in the range of 5%-12% which is quite sensitive to bandwidth choice. We also find a jump for external causes in the range of 3% - 15% which is even more sensitive to bandwidth choice. For deaths due

[10] note: internal causes = all causes - external causes.

to internal causes there is no clear jump. The regression results appear to be picking up the marked increase in mortality after turning 18 but it is not clear that this is a jump and therefore a direct result of the MLDA.

Table 5: LLR regression estimates of MLDA impact on log mortality

	(1)	(2)	(3)	(4)
Any cause				
MLDA 18	0.119**	0.0522	0.106***	0.102**
	(0.0478)	(0.0727)	(0.0349)	(0.0449)
Bandwidth	1.040	0.520	2.080	1.040
MLDA 20	0.0226	0.0110	-0.0123	0.00637
	(0.0487)	(0.0665)	(0.0345)	(0.0450)
Bandwidth	0.968	0.484	1.935	0.968
External Causes				
MLDA 18	0.0929	0.0262	0.146***	0.0695
	(0.0680)	(0.0949)	(0.0497)	(0.0659)
Bandwidth	0.853	0.427	1.707	0.853
MLDA 20	0.00462	-0.00710	-0.0169	-0.0169
	(0.0606)	(0.0825)	(0.0430)	(0.0564)
Bandwidth	0.900	0.450	1.800	0.900
Internal causes				
MLDA 18	0.0699	0.129	-0.0124	0.0596
	(0.0883)	(0.133)	(0.0621)	(0.0884)
Bandwidth	1.024	0.512	2.049	1.024
MLDA 20	-0.0102	0.0219	-0.0173	-0.00432
	(0.0672)	(0.0910)	(0.0493)	(0.0675)
Bandwidth	1.567	0.784	3.134	1.567
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA for various causes of death. See notes for table 3. The dependent variable is the log of the death count that occurred at age x measured in years and days and bandwidth is measured in years and days. We add 0.5 to the count before taking logs to deal with zeros. Covariates include gender, county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Death administrative data. Own calculations.

Figure 5 presents the raw data for alcohol related causes of mortality common amongst young adults where we would expect to be able to find an impact of the MLDA immediately at the cut-offs. The data shows a marked increase in motor vehicle related accidents at age 18. There is also a negative jump in deaths due to suicide at age 20. No clear jumps are observable from the raw data for homicides, alcohol and narcotics related deaths or other external causes. We test for jumps at the MLDA using LLR and the results are presented in table 6. We find a positive jump in motor vehicle related accidents at age 18 of between 10% and 36% and a drop in suicides at age

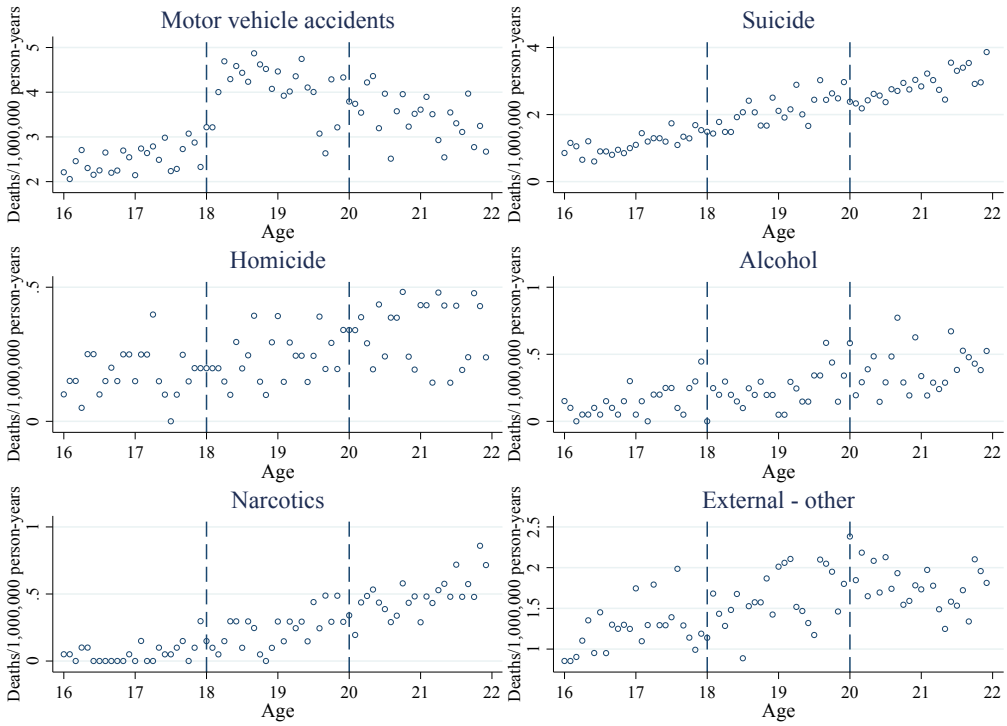

Fig. 5: Mortality due to external causes by age

Notes: The scatter points are monthly age blocks of the mortality count for the years 1969-2015. The causes are defined so they are non-overlapping using the following hierarchy: homicide, suicide, MVA, alcohol related, narcotics related, external other.

Source: Death administrative data. Own calculations.

20 of between -14% and -22% confirming what is shown in the raw data. We also find a small but statistically significant drop in alcohol related deaths at age 18 of between -8% and -12% (a drop of about 10 deaths over 44 years in absolute terms). In appendix H we present the results split by gender. The results in tables H.1 and H.2 show that it is males that are driving the drop in suicides at 20 years of age and the increase in motor vehicle related accidents at age 18. The reduction in alcohol related deaths at age 18 also appears to be driven by males but the estimates are very imprecise. In model (4) of tables 5 and 6 we include covariates for birthday and day after birthday to account for any birthday party effects. We also include dummy variables for counties who introduce OOP costs for medical care at the 18th or 20th birthday alongside controls for gender and whether foreign born. The inclusion of these covariates gives us very similar estimates.

In sum, we find evidence of a large relative jump in mortality at age 18, which is of similar size to that found for the United States (Carpenter and Dobkin, 2009). The impacts however are driven

entirely by motor vehicle related accidents and not by deaths due to any other alcohol related causes, whereas in the United States impacts were found for a number of alcohol related causes. Due to car driver licensing also beginning at age 18 we cannot disentangle if this jump in motor vehicle accidents is due entirely to the licensing regime or in some part due to alcohol. The fact that deaths specifically due to alcohol fall at age 18 suggests it is the licensing regime and not the MLDA at 18 that is driving the increase in motor vehicle related deaths. We also find a negative jump in suicides at age 20 when purchasing of off-licence alcohol is legalised. The relative impact found for suicides is much larger than that for alcohol related deaths and is even greater in absolute terms as there are that many more deaths due to suicide at age 20 than there are deaths due to alcohol related causes at age 18. These effects are all driven by males.

Table 6: LLR regression estimates of MLDA impact on external causes of log mortality

	(1)	(2)	(3)	(4)
Motor vehicle related accidents				
MLDA 18	0.239***	0.101	0.358***	0.215***
	(0.0847)	(0.119)	(0.0613)	(0.0818)
Bandwidth	1.000	0.500	2.001	1.000
MLDA 20	0.0759	-0.0832	0.0326	0.0469
	(0.0869)	(0.116)	(0.0621)	(0.0834)
Bandwidth	0.893	0.447	1.786	0.893
Suicide				
MLDA 18	0.00198	-0.0642	0.0461	0.0139
	(0.0861)	(0.126)	(0.0599)	(0.0881)
Bandwidth	1.098	0.549	2.196	1.098
MLDA 20	-0.216***	-0.149	-0.140**	-0.191**
	(0.0833)	(0.116)	(0.0601)	(0.0821)
Bandwidth	1.038	0.519	2.076	1.038
Homicide				
MLDA 18	-0.00313	-0.0420	-0.00666	-0.00500
	(0.0451)	(0.0633)	(0.0308)	(0.0451)
Bandwidth	1.054	0.527	2.107	1.054
MLDA 20	0.0494	0.0383	0.0163	0.0422
	(0.0446)	(0.0615)	(0.0319)	(0.0450)
Bandwidth	1.322	0.661	2.644	1.322
Alcohol related				
MLDA 18	-0.0817*	-0.125**	-0.0796**	-0.0835*
	(0.0458)	(0.0627)	(0.0342)	(0.0467)
Bandwidth	0.902	0.451	1.804	0.902
MLDA 20	0.00265	0.0248	0.0245	-0.00288
	(0.0528)	(0.0742)	(0.0369)	(0.0535)
Bandwidth	1.251	0.625	2.501	1.251
Narcotics related				
MLDA 18	0.0384	-0.0294	-0.00281	0.0372
	(0.0434)	(0.0697)	(0.0306)	(0.0440)
Bandwidth	0.910	0.455	1.820	0.910
MLDA 20	-0.0234	-0.0268	0.000495	-0.0161
	(0.0500)	(0.0668)	(0.0357)	(0.0509)
Bandwidth	1.382	0.691	2.764	1.382
Other external causes				
MLDA 18	-0.0419	0.0363	0.00540	-0.0402
	(0.0701)	(0.0967)	(0.0494)	(0.0695)
Bandwidth	1.642	0.821	3.284	1.642
MLDA 20	0.130	0.336**	0.113	0.0971
	(0.101)	(0.149)	(0.0706)	(0.100)
Bandwidth	0.771	0.385	1.542	0.771
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA for various external causes of death. See notes for table 5.
Source: Death administrative data. Own calculations.

5.3 Hospital admissions

In this section we present the estimates of the effect of the MLDA on cause specific hospital admissions. The structure and approach used in this section mirrors that of the mortality section. In figure 6 we plot the raw data for all hospital admissions in Sweden for the years 1969-2015 for inpatient care combined with the years 2001-2015 for outpatient care split by external and

internal causes (note that hospitalisations due to external causes are now the least common cause and therefore graphed on their own - right-hand side panel). The first thing to note is that the age profile of hospital admissions has a different age profile to that of mortality. Like mortality it increases from age 16 up to age 18, but where mortality increases after age 18 and then plateaus out after age 20, hospitalisations show a drop after age 18, plateau until age 20 after which they start to increase again. Another important difference is that internal causes are a more frequent reason for hospital admissions whereas external causes are more frequent reasons for mortality. As a consequence the age profile described for hospitalisations is driven by the age profile of internal causes - causes that are not directly attributable to alcohol. Hospital admissions due to external causes (right hand pane of figure 6) are much less frequent than internal cause related admissions and observe a large increase after the age of 18 before tailing off after age 20, mirroring much more closely the patterns observed for alcohol participation, quantity and frequency of alcohol consumption and mortality due to external causes.

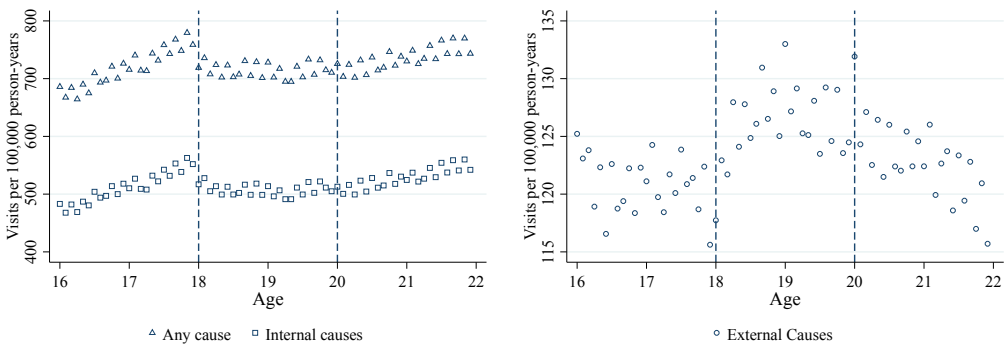

Fig. 6: Hospital admissions by age

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015. Internal causes and external causes do not sum to any cause due to missing information on diagnosis. The MLDA cut-offs at 18 years and 20 years are indicated by the dashed vertical lines.

Source: Patient administrative data. Own calculations.

In table 7 we present regression analysis of the log of the count of hospital admissions for each exact age at death measured in years and days. Similarly to mortality, the model is estimated over the age span 12-26 years of age and the coefficient estimates are of the impact of the MLDA and are interpreted as the percentage impact on hospital admissions, so long as the effects are not too large. Models (1) through (4) in table 7 are the same as the models used for the alcohol and mortality outcomes. In table 7 we find a robust and significant drop in overall hospital admissions at age 18 according to models (1-3) of between -4% to -5%, depending on model specification. This drop is driven by hospital admissions due to internal causes at age 18, causes not attributable to alcohol

Table 7: LLR regression estimates of MLDA impact on the log of hospital visits

	(1)	(2)	(3)	(4)
Any cause				
MLDA 18	-0.0540***	-0.0446**	-0.0541***	-0.0492***
	(0.0131)	(0.0226)	(0.00773)	(0.0126)
Bandwidth	0.217	0.108	0.433	0.217
MLDA 20	-0.0142	-0.00255	-0.00953	-0.00249
	(0.0266)	(0.0445)	(0.0153)	(0.0128)
Bandwidth	0.0664	0.0332	0.133	0.0664
External Causes				
MLDA 18	0.0722**	0.110**	0.0487**	0.0552*
	(0.0282)	(0.0432)	(0.0200)	(0.0314)
Bandwidth	0.0611	0.0305	0.122	0.0611
MLDA 20	0.0876**	0.140**	0.0487**	0.0545*
	(0.0404)	(0.0659)	(0.0248)	(0.0286)
Bandwidth	0.0540	0.0270	0.108	0.0540
Internal causes				
MLDA 18	-0.0647	-0.0635	-0.0651***	-0.0354
	(0.0399)	(0.0688)	(0.0233)	(0.0372)
Bandwidth	0.0701	0.0351	0.140	0.0701
MLDA 20	-0.0223	-0.0271	-0.0180	0.000980
	(0.0296)	(0.0484)	(0.0172)	(0.0137)
Bandwidth	0.0730	0.0365	0.146	0.0730
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA on hospital admissions. See notes for table 3. The dependent variable is the log of the count of hospital admissions that occurred at age x measured in years and days and bandwidth is measured in years and days. We add 0.5 to the count before taking logs to deal with zeros. Not all admissions have a recorded cause and therefore external and internal causes do not sum to the total. Covariates include gender, county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at admission measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

of which we a see a drop of about -6%. The drop in internal cause related hospital admissions is largely driven by the Swedish medical system's treatment of patients in need of psychiatric care.[11] Hospital admissions due to external causes and therefore attributable to alcohol see a jump at age 18 in the range of 5% - 11%. There is also an observed jump in hospital admissions due to external causes at age 20 in the region of 5% - 14%.

[11] At age 18 individuals no longer receive psychiatric care at the children and young persons psychiatric wards at hospitals. Instead provision of care moves to adult psychiatric care which is less generous, more patchily provided and often is dealt with by the GP or external providers and therefore not captured in the hospital patient database. This is therefore an independent event not related to MLDA impacts that does not impact our results on external causes. Unfortunately our register data does not allow us to show this as we do not have detailed ICD code information. However, in appendix I figure I.1 we present evidence using the household and living standards survey linked to patient register data and show that the drop occurs for internal causes related to mental disorders. No other drops are observed for any other internal cause.

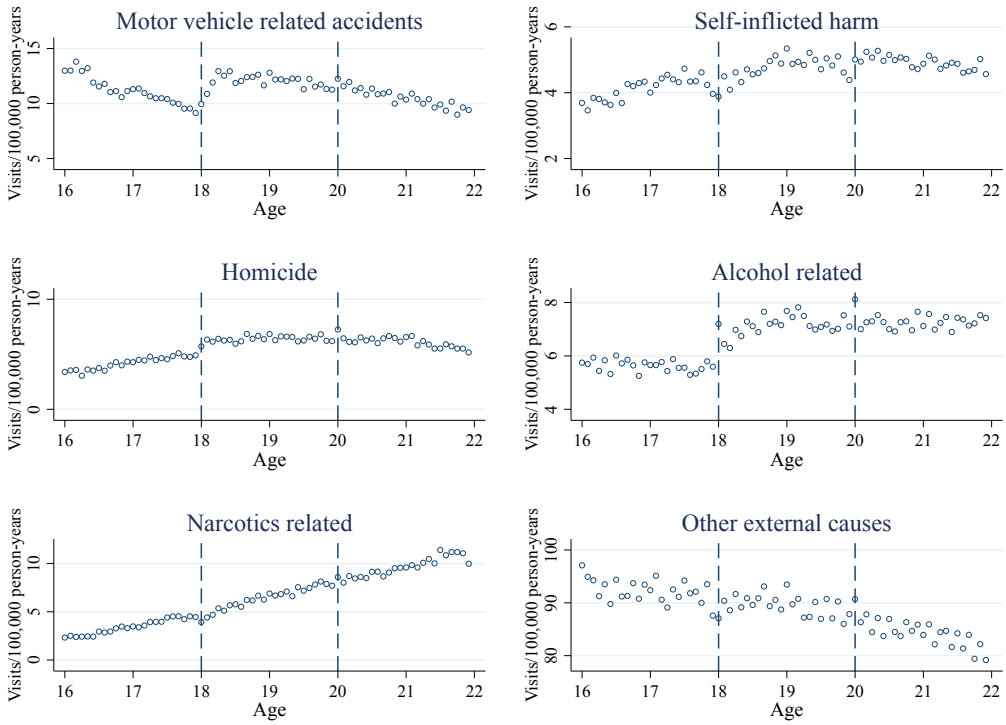

Fig. 7: Hospital admissions due to external causes by age

Notes: The scatter points are monthly age blocks of count of hospital visits for the years 1969-2015.

Source: Patient administrative data. Own calculations.

In figure 7 we present the raw data of the age profile of hospital admissions due to external causes. Alcohol related causes of hospital admissions show a clear jump at age 18 and also the clear impact of celebrating one's own birthday at ages 18 and 20. This highlights that there is an immediate birthday impact on hospitalisations that is not a long-term impact. Other jumps we can see in the raw data occur for homicides at age 18 and suicides at age 20. There is also a clear birthday effect at age 20 for homicides. We test for jumps at the MLDA on specific external causes of hospital admission in table 8. Robust and statistically significant results across models (1-3) are found at age 18 years for alcohol related admissions in the range of 29% - 68% and homicide related admissions in the range of 12% - 49% and at age 20 years for self-inflicted harm in the range of 15% - 33%. Smaller impacts are also observed at age 20 for homicides in the range of 23% - 29% and alcohol related causes in the range of 14% - 38%.

Clear birthday effects are seen in the raw data for hospital admissions and we control for these in model (4) using a dummy for birthday and a dummy for the day after. We also control for counties

that have OOP payments that start at either 18 or 20 years of age, gender and whether foreign born. The impact of the covariates on overall admissions is negligible but has a larger impact on external causes. The largest impact of including covariates is on alcohol related external causes and highlights the importance of removing the birthday impact in order to get a handle on the more long lasting impact of the MLDA on morbidity. For alcohol related admissions the impact of the MLDA at age 18 drops from 45% to 26% when controlling for covariates. The impact of the MLDA on homicide related admissions at age 18 falls from 33% to 25% and at age 20 falls from 29% to 18% when controlling for covariates. Self-inflicted harm related admissions see a fall in MLDA impact at age 20 from 24% to 22% when controlling for covariates.

In appendix J we present the raw data (figures J.1, J.3, J.2, J.4) for counties that do not implement changes in OOP costs at the same time as the MLDA in order to assess whether changes in OOP costs specifically are impacting our results. The jumps in figures J.1, J.3, J.2 and J.4 are very similar to the the jumps found in figures 6 and 7. We test the impact of OOP costs in tables J.1 and J.2 where estimates are calculated for counties that do not implement changes in OOP costs at the same time as the MLDA. The sample sizes for the counties that do not implement an OOP cost change simultaneously with the MLDA 20 is quite small and therefore the standard errors are very large for these estimates. For the MLDA at 18 however we can conclude that the estimates are not impacted by any changes in OOP cost at 18 and that the impact of the covariates shown in Model (4) in tables 7 and 8 is driven by the birthday dummies and not other simultaneous rule changes.

Table 8: LLR regression estimates of MLDA impact on the log of hospital visits due to external causes

	(1)	(2)	(3)	(4)
Motor vehicle related accidents				
MLDA 18	0.00731	-0.143	0.0237	0.0956
	(0.0849)	(0.113)	(0.0695)	(0.114)
Bandwidth	0.0433	0.0217	0.0866	0.0433
MLDA 20	0.0777	0.0131	0.0810**	0.114*
	(0.0539)	(0.0779)	(0.0410)	(0.0624)
Bandwidth	0.0652	0.0326	0.130	0.0652
Self-inflicted harm				
MLDA 18	0.0292	0.143	-0.0409	0.0126
	(0.107)	(0.154)	(0.0755)	(0.117)
Bandwidth	0.0907	0.0453	0.181	0.0907
MLDA 20	0.235**	0.331***	0.148**	0.217**
	(0.0916)	(0.111)	(0.0658)	(0.102)
Bandwidth	0.0825	0.0413	0.165	0.0825
Homicide				
MLDA 18	0.333***	0.485***	0.122	0.245**
	(0.0953)	(0.119)	(0.0744)	(0.105)
Bandwidth	0.0817	0.0409	0.163	0.0817
MLDA 20	0.286**	0.267	0.229**	0.180
	(0.130)	(0.164)	(0.0900)	(0.125)
Bandwidth	0.0553	0.0276	0.111	0.0553
Alcohol related				
MLDA 18	0.445***	0.676***	0.288***	0.254**
	(0.122)	(0.162)	(0.0841)	(0.101)
Bandwidth	0.0932	0.0466	0.186	0.0932
MLDA 20	0.188*	0.378***	0.140**	0.0999
	(0.101)	(0.143)	(0.0675)	(0.0877)
Bandwidth	0.0915	0.0458	0.183	0.0915
Narcotics related				
MLDA 18	-0.135	-0.0869	-0.185***	-0.113
	(0.101)	(0.152)	(0.0640)	(0.0960)
Bandwidth	0.118	0.0591	0.236	0.118
MLDA 20	0.0555	0.262*	0.134**	0.0394
	(0.0962)	(0.142)	(0.0622)	(0.0910)
Bandwidth	0.0537	0.0269	0.107	0.0537
Other external causes				
MLDA 18	0.0102	0.0216	0.0208	0.0108
	(0.0347)	(0.0523)	(0.0221)	(0.0350)
Bandwidth	0.0578	0.0289	0.116	0.0578
MLDA 20	0.00525	0.0282	0.0110	-0.0107
	(0.0208)	(0.0332)	(0.0132)	(0.0172)
Bandwidth	0.121	0.0603	0.241	0.121
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA on the count of hospitalisations by various causes. See notes for table 7. Standard errors clustered by age at admission measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

6 Discussion

We have considered the impact of a unique alcohol control policy - Sweden's two-part MLDA. The results have found that the first part of the MLDA at age 18 when alcohol consumption is legalised on premise leads to a 6% jump in participation, a 6% jump in heavy drinking participation and

an 8% jump in risky drinking among those who have just turned 18 years compared to those who are not quite 18 years old. We also find a jump in the number of days reported when beer, cider, spirits and heavy drinking occurred by those who have just turned 18 of the order of between 16% and 22% and that by and large these impacts are driven by males. The impact on intensity was negative but small and insignificant. The impact of the second part of the MLDA that legalised purchase of alcohol off-premise at age 20 was negligible on participation, frequency and intensity and primarily led to a substitution away from low alcohol beer available at all shops and home brew and illegally imported alcohol.

The impacts on the pattern of alcohol consumption of the MLDA at age 18 are similar in pattern but smaller in scale compared to the findings of Carpenter and Dobkin (2009) who find that the United States' MLDA at age 21 leads to a 21% increase in the number of drinking days and 7% increase in the number who drank in the last 12 months but not an increase in first-time use of alcohol. Similarly to Carpenter and Dobkin (2009) we also find in general no impact on intensity for those who just turned 18 with the exception of a small negative impact on beer and spirits intensity suggesting a slight moderation in drinking behaviour. The fact that Sweden's two-part MLDA influences similar alcohol drinking patterns but by lesser degree compared to the United States is suggestive evidence that Sweden's two-part MLDA leads to a greater level of control in drinking relative to a single MLDA at age 18.

But what is the total effect of the MLDA on health? The answer to this is complicated by two factors. First, young adults can also apply for a driving licence after turning 18 and that motor vehicle related accidents may jump at 18 as a consequence of this policy in combination with the MLDA at 18. Second, we have shown there is also a discontinuity in hospital admissions due to internal causes at age 18, which is due to an administrative issue to do with how mental health patients are treated before and after turning 18. We argue that internal causes are causes not impacted by the MLDA for the age groups we consider, so in order to summarise we focus on external causes. In table 9 we present the results for mortality and hospital admissions due to external causes excluding motor vehicle related accidents. The results suggest a negative impact of the MLDA at 18 on mortality of between -3% and -10% but these are imprecisely estimated. For hospital admissions the results suggest a positive jump at 18 of between 6% and 14% but the results are not significant when controlling for birthday and other covariate effects. These results present a potentially downward biased estimate of the total impact at 18 of the MLDA on health because we are missing the impact of the MLDA that goes through motor vehicle related accidents not due to the licensing laws. However, we found no statistically significant impact of the MLDA at 18 on hospital admissions due to motor vehicle related accidents which suggests the summary results for hospital admissions presented in table 9 are in fact not downward biased. For mortality we found

a statistically significant positive impact of the MLDA at 18 on motor vehicle related accidents. However, the mortality results rely on a much smaller sample with a much larger corresponding bandwidth compared to the hospital admission results. The raw mortality data shown in figure 5 combined with the hospital admissions results suggest that the positive LLR result shown in table 6 for motor vehicle related accidents is rather just a trend break. We also found no positive jump in alcohol related deaths after turning 18 which would suggest it is the driving licence regime that is driving the increase in motor vehicle related deaths and not the MLDA at 18. Together, this would suggest that the results for mortality in table 9 are also a fair representation of the true impact of the MLDA on overall mortality. The overall impact at age 20 is an insignificant jump in mortality due to external causes of between -0.7% and 0.5% and a significant jump in hospital admissions due to external causes of between 5% and 14%.

Table 9: LLR regression estimates of MLDA impact on mortality and hospital admissions due to external causes excluding motor vehicle accidents

	(1)	(2)	(3)	(4)
Deaths due to non-motor vehicle related external causes				
MLDA 18	-0.0952	-0.0821	-0.0293	-0.0986
	(0.0718)	(0.101)	(0.0515)	(0.0726)
Bandwidth	1.312	0.656	2.624	1.312
Hospital admissions due to non-motor vehicle related external causes				
MLDA 18	0.0828***	0.140***	0.0615***	0.0573
	(0.0311)	(0.0490)	(0.0230)	(0.0360)
Bandwidth	0.0507	0.0254	0.101	0.0507
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA on the count of hospitalisations by various causes. See notes for table 7. Standard errors clustered by age at admission measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Death and patient administrative data. Own calculations.

We summarise the overall impact on mortality of the increase in alcohol consumption at age 18 as small and insignificant. We observe no clear jump in alcohol consumption at age 20 and find no positive jump in overall mortality either. Conversely, there appears to be a fall in suicides at age 20. The changes in alcohol behaviour at age 20 showed a shift away from home brew, smuggled alcohol and low alcohol beer and there appears to be a moderation in intensity but these do not seem to be enough to explain a drop in suicides. We find no impacts on deaths due to homicide, drugs related causes or other external causes of mortality (accidents due to fire, falling or drowning). We find a positive jump in hospital admissions due to external causes at both cut-offs of 18 and 20 but these are not very precisely estimated when aggregated. For specific causes, the results show a 26% increase in alcohol related admissions and a 25% increase in homicide related admissions after turning 18. We also find an 18% increase in homicide related admissions and a 22% jump in

self-harm related admissions after turning 20. The jumps at 18 in hospital admissions coincide with clear jumps in alcohol consumption, but the jumps at 20 are harder to explain using the available data we have on consumption patterns.

We have found that Sweden's two-part MLDA influences similar alcohol drinking patterns but by a lesser degree compared to the United States. Evidence from the United States has found large impacts on mortality due to the changes in alcohol at the MLDA of 21 years (Carpenter and Dobkin, 2009), but we find no corresponding positive jump at 18 or 20 in mortality (and this isn't because mortality is already very high beforehand). However, we find large relative jumps for hospital admissions at 18. Impacts on hospital admissions have been found in Canada for the MLDA at 18 where alcohol related hospital admissions jumped 17% for those just turned 18 (Callaghan et al., 2013). For Sweden we find a much larger relative impact of 26% (using our most conservative estimate) for alcohol related hospital admissions. Perhaps Sweden's health care system is also helping protect Sweden's young adults from the most extreme health impacts of increased alcohol consumption?

To summarise, let us assume that the impacts on health at age 18 are driven by the number of days of heavy drinking (increase of 16%). The results then imply an elasticity between heavy alcohol consumption frequency and hospital admissions due to external causes of about 31% (0.05/0.16) and that this impact would be predominantly driven by males. The results suggest that changes to young adults alcohol consumption can have substantial impacts on the societal costs of alcohol by reducing immediate hospital care costs. The second part of Sweden's MLDA at 20 years of age has had little impact on drinking behaviour beyond an apparent shift away from home brew and low alcohol beer. The fact that we find no clear impact of the MLDA at 20 years of age is potentially due to the fact it is quite easy to circumvent the restriction by asking friends who are old enough to buy alcohol for you whereas at 18 you need to go to the pub yourself. This suggests the MLDA at 20 has more of a progressive impact rather than an abrupt impact precisely after turning 20, because the nearer to 20 you are the more likely you are to know someone who is already 20 and therefore have progressively easier access to alcohol. This potentially explains away the insignificant findings for alcohol consumption at age 20 but we are unable to provide an explanation for the decrease in male suicides or the increase in hospitalisations at age 20 using the data we have at our disposal. Perhaps further research can tease out what behaviour changes happen at age 20 that lead to this reduction in mortality and increases in morbidity.

Young Swedes largely deal with the transition to easier access to alcohol during young adulthood without the large negative repercussions that have been observed for the United States and Canada. Young adulthood is too young to die and perhaps a two-part MLDA offers a promising alternative approach to reduce the heightened mortality and morbidity that coincides with easier access to

alcohol observed in other countries.

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A Variable definitions

Table A.1: ICD code definitions of causes of death/hospitalisation

Diagnosis	*ICD 10 code*	*ICD 9 code*	*ICD 8 code*
External causes	S,T,V,W,X,Y,E24.4 F10-F19 (excl .6, F17.1,F17.2),F55 G31.2,G62.1,G72.1 G72.0,I42.6-7, K29.2,K70,K85.2-3 K86.0,R78.0	8,9,E8,E9 291,292,303-305 425F,571A-D,790D	8,9,E8,E9 291,292,304-305 571.0-.98
Motor vehicle related	*	E810-E828	E810-E827
Suicide/Self-inflicted harm	X60-X84,Y8	E950-E959	E950-E959
Homicide	X85-Y09, Y87.1	E960-E969	E960-E969
Alcohol related	E24.4,F10 G31.2,G62.1,G72.1 I42.6,K29.2, K70.0-K70.9,K85.2 K86.0,O35.4,P04.3 Q86.0,T51.0-T51.9 Y90.1-Y90.9, Y91.1-Y91.9,Z50.2 Z71.4,Z72.1	291,303,305A 357F,425F,535D 571A-D, E860,E980	291,303,305.0 357.5,425.5, 535.3,571.0-.3, E860,E980
Narcotics related	F11-F16,F18,F19, O35.5,P04.4,T40 T43.6,Z50.3,Z71.5 Z72.2	304A-X,305X 965A,968E,969G, 969H	304.0-304.9 305.2-305.7, 305.9,965.0, 968.5,969.6, 969.7
Other external causes	*= 1 if external cause but not sub-external cause above*		
Internal causes	*= 1 if not external cause*		

* ICD 10 codes for motor vehicle related accidents: V02-04, V09.2, V09.3, V12.3-V12.9, V13.3-V13.9, V14.3-V14.9, V19.4-V19.6, V20.3-V20.9, V21.3-V21.9, V22.3-V22.9, V23.3-V23.9, V24.3-V24.9, V25.3-V25.9, V26.3-V26.9, V27.3-V27.9, V28.3-V28.9, V29.4-V29.9, V30.4-V30.9, V31.4-V31.9, V32.4-V32.9, V33.4-V33.9, V34.4-V34.9, V35.4-V35.9, V36.4-V36.9, V37.4- V37.9, V38.4-V38.9, V39.4-V39.9, V40.4-V40.9, V41.4-V41.9, V42.4-V42.9, V43.4-V43.9, V44.4-V44.9, V45.4-V45.9, V46.4-V46.9, V47.4-V47.9, V48.4-V48.9, V49.4-V49.9, V50.4-V50.9, V51.4-V51.9, V52.4-V52.9, V53.4-V53.9, V54.4- V54.9, V55.4-V55.9, V56.4-V56.9, V57.4-V57.9, V58.4-V58.9, V59.4-V59.9, V60.4-V60.9, V61.4-V61.9, V62.4-V62.9, V63.4-V63.9, V64.4-V64.9, V65.4-V65.9, V66.4-V66.9, V67.4-V67.9, V68.4-V68.9, V69.4-V69.9, V70.4-V70.9, V71.4- V71.9, V72.4-V72.9, V73.4-V73.9, V74.4-V74.9, V75.4-V75.9, V76.4-V76.9, V77.4-V77.9, V78.4-V78.9, V79.4-V79.9, V80.3-V80.5, V81.1, V82.1, V83.0-V83.3, V84.0-V84.3, V85.0-V85.3, V86.0-V86.3, V87.0-V87.8, V89.2

B Tobacco MLSA impact on smoking behaviour

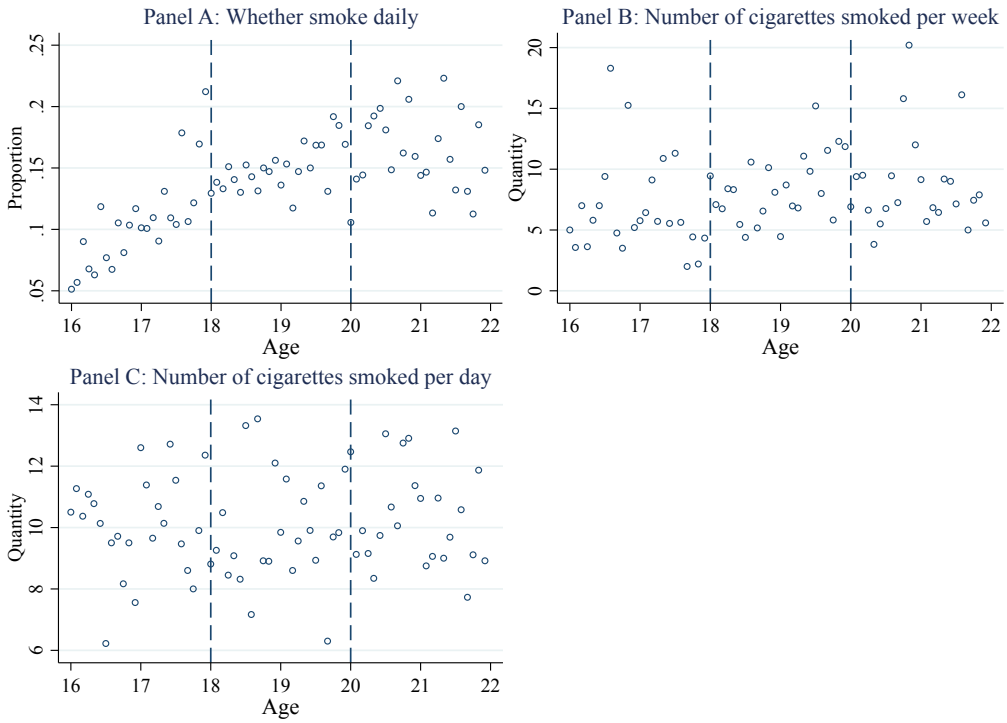

Fig. B.1: Tobacco consumption by age

Notes: This figure plots the scatter points of mean smoking behaviour by monthly age blocks. The MLDA cut-offs at 18 years and 20 years are indicated by the dashed vertical lines.

Source: Monitor project survey data. Own calculations.

Table B.1: LLR regression estimates of MLSA impact on tobacco consumption

	(1)	(2)	(3)	(4)
Smoked in the last 30 days				
MLDA 18	-0.0131	-0.0376	-0.0124	-0.00732
	(0.0199)	(0.0322)	(0.0158)	(0.0195)
Bandwidth	1.679	0.840	3.359	1.679
MLDA 20	-0.0121	-0.0467	0.0173	0.00613
	(0.0296)	(0.0488)	(0.0192)	(0.0286)
Bandwidth	1.106	0.553	2.211	1.106
Quantity smoked daily in last 30 days				
MLDA 18	-0.596	-1.070	-0.978	-0.461
	(0.942)	(1.154)	(0.884)	(0.933)
Bandwidth	2.535	1.268	5.071	2.535
MLDA 20	-0.259	-0.120	0.116	0.0691
	(0.767)	(1.010)	(0.668)	(0.792)
Bandwidth	3.458	1.729	6.915	3.458
Quantity smoked weekly in the last 30 days				
MLDA 18	2.089	5.026***	-0.343	2.228
	(2.065)	(1.916)	(1.531)	(2.189)
Bandwidth	1.203	0.602	2.406	1.203
MLDA 20	-0.699	-3.202	-0.122	-1.386
	(1.402)	(2.359)	(1.104)	(1.390)
Bandwidth	2.949	1.474	5.898	2.949
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on smoking patterns from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Daily is equal to one if individual smoked daily in last 30 days, zero otherwise. Quantity smoked is equal quantity an individual smoked per day in last 30 days. Weekly quantity is equal to the quantity an individual smoked per week in last 30 days. Each estimate is from a separate regression. Columns 1 and 4 use the IK optimal bandwidth. Columns 2 and 3 use a bandwidth half and double the size of the IK optimal bandwidth. Column 4 includes covariates. Covariates include gender, economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor project survey data. Own calculations.

C Diagnostic tests

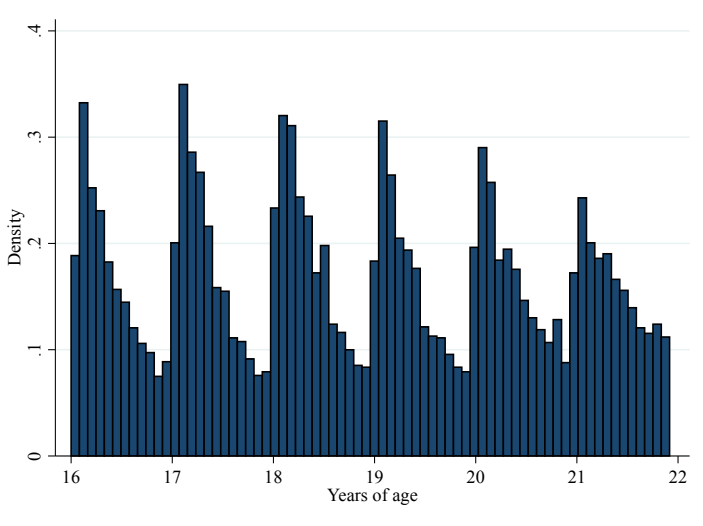

Fig. C.1: Diagnostic test: manipulation of the cut-off

Notes: This figure plots the population density by monthly age blocks. The interview sampling frame asks who in the household most recently had a birthday to answer the questionnaire, hence the decreasing density within each year.

Source: Monitor project survey data. Own calculations.

Table C.1: Diagnostic tests: LLR regression estimates for jump in covariates at the cut-off

	(1)	(2)	(3)	(4)
Proportion who are male				
MLDA 18	-0.0188	-0.0424	-0.0218	-0.0232
	(0.0264)	(0.0400)	(0.0200)	(0.0185)
Bandwidth	1.512	0.756	3.025	6.050
MLDA 20	0.0155	-0.0187	-0.00996	-0.0135
	(0.0281)	(0.0440)	(0.0182)	(0.0140)
Bandwidth	1.443	0.721	2.886	5.771
Proportion employed				
MLDA 18	-0.0253**	-0.00377	-0.0159*	0.137***
	(0.0125)	(0.0186)	(0.00949)	(0.00907)
Bandwidth	1.190	0.595	2.380	4.759
MLDA 20	-0.0761*	0.0483	-0.0927***	0.0519***
	(0.0402)	(0.0642)	(0.0252)	(0.0165)
Bandwidth	0.868	0.434	1.736	3.472
Proportion unemployed				
MLDA 18	-0.0124*	0.0113	0.0171***	0.0681***
	(0.00693)	(0.00981)	(0.00595)	(0.00555)
Bandwidth	1.261	0.631	2.523	5.046
MLDA 20	0.00467	0.0208	-0.0407***	-0.00178
	(0.0250)	(0.0401)	(0.0153)	(0.00975)
Bandwidth	0.999	0.500	1.998	3.997
IK optimal bandwidth	✓	0.5x	2x	4x
Covariates				

Notes: This table shows the impact of the MLDA at 18 and 20 years on covariates from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Male is 1 if male, 0 otherwise. Employed is 1 if employed, 0 otherwise. Unemployed is 1 if unemployed, 0 otherwise. Column 1 uses the IK optimal bandwidth. Columns 2, 3, 4 use a bandwidth half, double and four times the size of the IK optimal bandwidth. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Source: Monitor project survey data. Own calculations.

D Alcohol intensity results

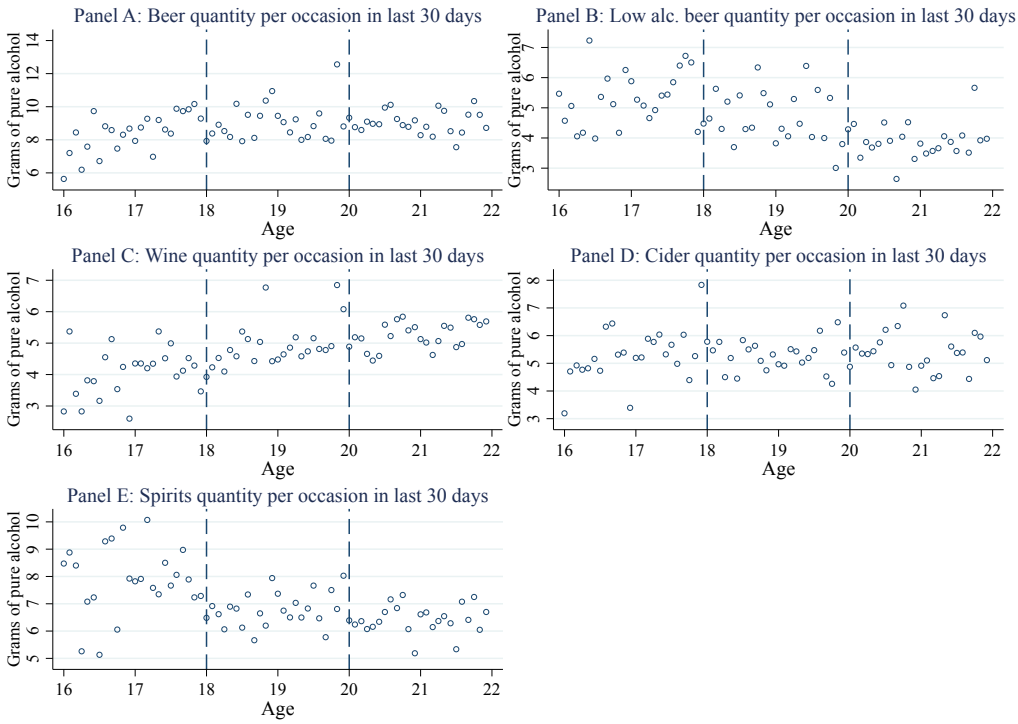

Fig. D.1: Average intensity per drinking occasion by age

Notes: Monitor project data sample 2001-2011. The scatter points are monthly age blocks representing the proportion reporting the drinking behaviour. Regression lines are LLRs using a uniform kernel and 1 year bandwidth estimated separately either side of the MLDA cut-offs at ages 18 and 20.

Source: Monitor project survey data. Own calculations.

Table D.1: LLR regression estimates of MLDA impact on average intensity per drinking occasion

	(1)	(2)	(3)	(4)
Average intensity of beer consumption in last 30 days				
MLDA 18	-0.869*	-1.463**	-0.800*	-0.619
	(0.464)	(0.689)	(0.433)	(0.456)
Bandwidth	2.240	1.120	4.480	2.240
MLDA 20	-0.00306	0.229	-0.0406	0.0877
	(0.319)	(0.481)	(0.262)	(0.306)
Bandwidth	3.173	1.587	6.346	3.173
Average intensity of low alcohol beer consumption in last 30 days				
MLDA 18	-0.593	-0.676	-0.817*	-0.594
	(0.477)	(0.639)	(0.461)	(0.466)
Bandwidth	2.954	1.477	5.908	2.954
MLDA 20	-0.482*	-0.659	-0.584**	-0.179
	(0.292)	(0.421)	(0.243)	(0.306)
Bandwidth	3.298	1.649	6.595	3.298
Average intensity of wine consumption in last 30 days				
MLDA 18	-0.0313	0.0897	0.283	-0.0939
	(0.357)	(0.402)	(0.338)	(0.371)
Bandwidth	2.938	1.469	5.876	2.938
MLDA 20	-0.303	-0.821	-0.00269	-0.232
	(0.315)	(0.556)	(0.219)	(0.316)
Bandwidth	2.130	1.065	4.261	2.130
Average intensity of cider consumption in last 30 days				
MLDA 18	-0.728*	-0.607	-0.865**	-0.221
	(0.417)	(0.669)	(0.379)	(0.393)
Bandwidth	1.972	0.986	3.945	1.972
MLDA 20	0.448	0.115	0.107	0.524
	(0.353)	(0.559)	(0.245)	(0.362)
Bandwidth	2.312	1.156	4.625	2.312
Average intensity of spirits consumption in last 30 days				
MLDA 18	-1.553***	-1.136	-1.348***	-1.117**
	(0.530)	(0.810)	(0.491)	(0.535)
Bandwidth	2.005	1.003	4.011	2.005
MLDA 20	-0.614	-0.770	0.134	-0.360
	(0.379)	(0.590)	(0.272)	(0.370)
Bandwidth	2.099	1.049	4.198	2.099
IK optimal bandwidth	✓	0.5x	2x	✓
Covariates				✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on alcohol consumption patterns from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Each estimate is from a separate regression. Columns 1 and 4 use the IK optimal bandwidth. Columns 2 and 3 use a bandwidth half and double the size of the IK optimal bandwidth. Column 4 includes covariates. Covariates include gender, economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor project survey data. Own calculations.

E Densities of frequency and intensity for ages 17, 18, 19 and 20 years

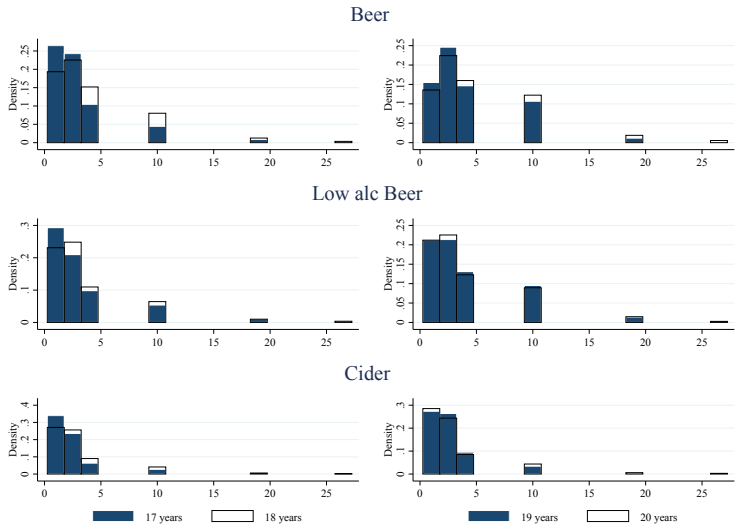

Fig. E.1: Density plot of frequency of alcohol consumption 12 months before and after the MLDA cut-offs (Beer, low alcohol beer and cider)

Notes: Histograms are of individuals 12 months of age either side of the cut-off. Bin widths are 1 day. X-axis is frequency of drinking episodes in last 30 days.

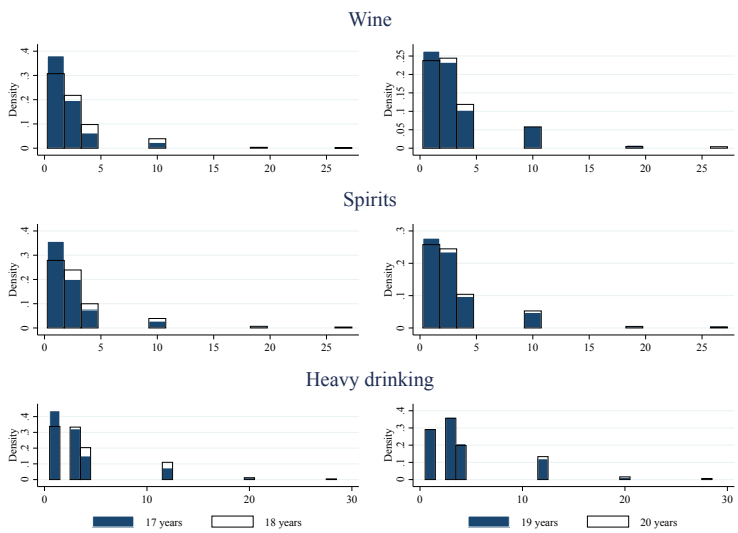

Fig. E.2: Density plot of frequency of alcohol consumption 12 months before and after the MLDA cut-offs (wine, spirits and heavy drinking)

Notes: Histograms are of individuals 12 months of age either side of the cut-off. Bin widths are 1 day. X-axis is frequency of drinking episodes in last 30 days.

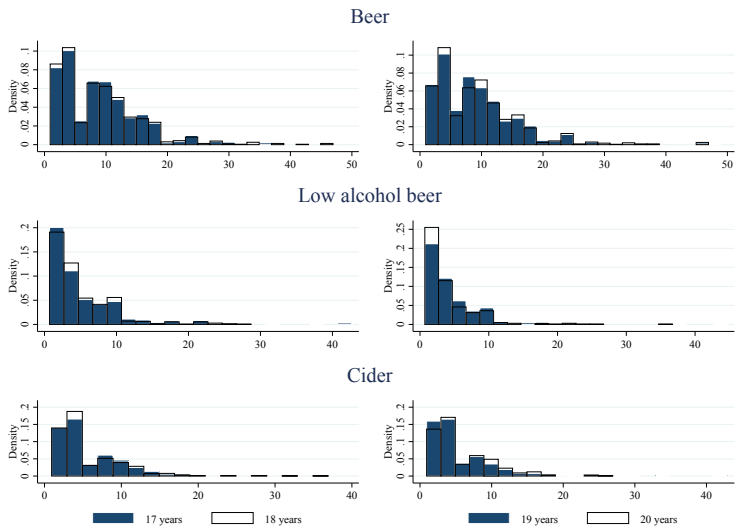

Fig. E.3: Density plot of intensity of alcohol consumption 12 months before and after the MLDA cut-off (Beer, low alcohol beer and cider)

Notes: Histograms are of individuals 12 months of age either side of the cut-off. Bin widths are 2 grams of pure alcohol. X-axis is frequency of drinking episodes in last 30 days.

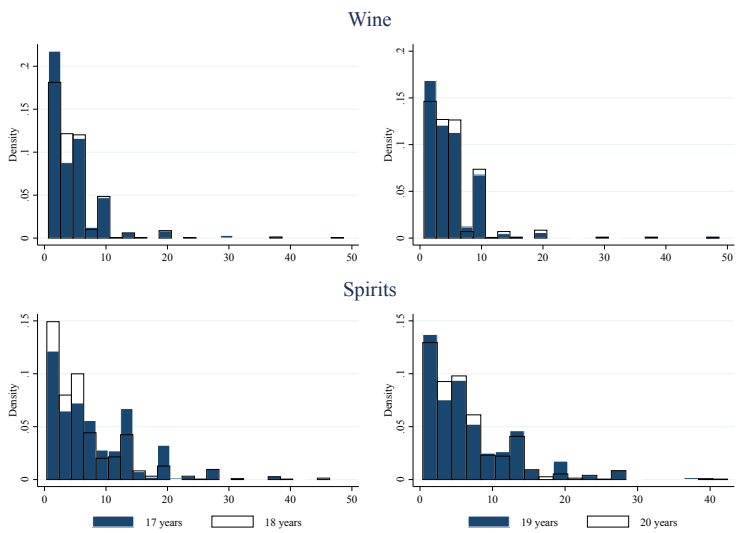

Fig. E.4: Density plot of intensity of alcohol consumption 12 months before and after the MLDA cut-off (wine and spirits)

Notes: Histograms are of individuals 12 months of age either side of the cut-off. Bin widths are 2 grams of pure alcohol. X-axis is frequency of drinking episodes in last 30 days.

F MLDA impact on alcohol consumption, split by gender

Table F.1: Alcohol participation LLR results by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Drank alcohol in last 30 days						
MLDA 18	0.0601*	0.0484*	0.0690**	0.0310	0.0479*	0.0413
	(0.0314)	(0.0248)	(0.0322)	(0.0339)	(0.0269)	(0.0346)
Bandwidth	1.590	3.181	1.590	1.590	3.181	1.590
MLDA 20	0.0120	0.00603	0.0119	0.0283	-0.0205	0.0128
	(0.0310)	(0.0209)	(0.0315)	(0.0379)	(0.0239)	(0.0394)
Bandwidth	1.104	2.209	1.104	1.104	2.209	1.104
Had a heavy drinking episode in last 30 days						
MLDA 18	0.0951**	0.0730**	0.119***	-0.00824	0.0266	-0.0130
	(0.0399)	(0.0287)	(0.0412)	(0.0400)	(0.0287)	(0.0410)
Bandwidth	1.485	2.970	1.485	1.485	2.970	1.485
MLDA 20	0.111**	0.0246	0.123**	-0.0241	-0.0460	-0.0000406
	(0.0474)	(0.0306)	(0.0491)	(0.0485)	(0.0313)	(0.0522)
Bandwidth	1.257	2.514	1.257	1.257	2.514	1.257
Drank more than recommended weekly amount in last 30 days						
MLDA 18	0.0790**	0.0463*	0.0872**	0.0517	0.0589**	0.0466
	(0.0352)	(0.0256)	(0.0362)	(0.0379)	(0.0277)	(0.0387)
Bandwidth	1.498	2.997	1.498	1.498	2.997	1.498
MLDA 20	0.0113	0.0132	0.00495	0.0552	0.0139	0.0483
	(0.0358)	(0.0232)	(0.0367)	(0.0429)	(0.0264)	(0.0447)
Bandwidth	0.984	1.968	0.984	0.984	1.968	0.984
Quantity of pure alcohol consumed in last 30 days						
MLDA 18	11.72***	18.26***	12.60***	7.843**	10.54***	8.337**
	(4.281)	(3.867)	(4.323)	(3.537)	(2.928)	(3.506)
Bandwidth	2.586	5.172	2.586	2.586	5.172	2.586
MLDA 20	-8.359	-5.576	-9.084	-0.770	-6.524***	0.360
	(5.663)	(4.127)	(5.767)	(3.195)	(2.461)	(3.260)
Bandwidth	2.622	5.243	2.622	2.622	5.243	2.622
Drank unregulated alcohol in last 30 days						
MLDA 18	-0.00639	-0.0117	0.00663	-0.0163	-0.0260	-0.0114
	(0.0286)	(0.0223)	(0.0291)	(0.0236)	(0.0190)	(0.0243)
Bandwidth	1.612	3.224	1.612	1.612	3.224	1.612
MLDA 20	-0.0527	-0.0717***	-0.0413	-0.0356	-0.0227	-0.0370
	(0.0360)	(0.0228)	(0.0369)	(0.0274)	(0.0166)	(0.0290)
Bandwidth	1.298	2.597	1.298	1.298	2.597	1.298
Frequency of drinking occasions in last 30 days						
MLDA 18	0.479	0.592	0.653	2.081*	0.174	2.634**
	(0.992)	(0.724)	(1.046)	(1.080)	(0.778)	(1.143)
Bandwidth	1.376	2.752	1.376	1.376	2.752	1.376
MLDA 20	0.0516	1.329***	0.0395	-0.328	0.578	-0.297
	(0.697)	(0.503)	(0.725)	(0.757)	(0.541)	(0.812)
Bandwidth	1.921	3.842	1.921	1.921	3.842	1.921
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on covariates from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor project survey data. Own calculations.

Table F.2: Alcohol frequency LLR results by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Frequency of beer consumption in last 30 days						
MLDA 18	0.327	0.649**	0.493	0.745*	0.555	0.743
	(0.339)	(0.292)	(0.348)	(0.438)	(0.342)	(0.459)
Bandwidth	1.710	3.419	1.710	1.710	3.419	1.710
MLDA 20	0.166	0.167	0.119	0.00440	-0.172	-0.0456
	(0.263)	(0.211)	(0.269)	(0.289)	(0.239)	(0.303)
Bandwidth	3.077	6.154	3.077	3.077	6.154	3.077
Frequency of low alcohol beer consumption in last 30 days						
MLDA 18	0.152	0.446	-0.0207	-0.0672	0.0553	-0.276
	(0.385)	(0.367)	(0.387)	(0.426)	(0.407)	(0.448)
Bandwidth	3.746	7.492	3.746	3.746	7.492	3.746
MLDA 20	-0.386	-0.319	-0.375	0.276	0.0824	0.354
	(0.364)	(0.295)	(0.377)	(0.414)	(0.315)	(0.455)
Bandwidth	3.241	6.482	3.241	3.241	6.482	3.241
Frequency of wine consumption in last 30 days						
MLDA 18	-0.180	0.308	-0.141	0.483	0.599**	0.405
	(0.314)	(0.284)	(0.345)	(0.295)	(0.271)	(0.317)
Bandwidth	2.253	4.507	2.253	2.253	4.507	2.253
MLDA 20	0.0799	0.0985	-0.138	-0.396*	-0.429**	-0.445*
	(0.321)	(0.250)	(0.319)	(0.222)	(0.186)	(0.231)
Bandwidth	3.172	6.345	3.172	3.172	6.345	3.172
Frequency of cider consumption in last 30 days						
MLDA 18	0.808**	0.871***	0.639*	0.395	0.334	0.438
	(0.362)	(0.330)	(0.372)	(0.271)	(0.248)	(0.267)
Bandwidth	2.016	4.033	2.016	2.016	4.033	2.016
MLDA 20	0.205	0.0277	0.240	0.762***	-0.0377	0.729***
	(0.475)	(0.339)	(0.499)	(0.263)	(0.189)	(0.282)
Bandwidth	1.891	3.783	1.891	1.891	3.783	1.891
Frequency of spirits consumption in last 30 days						
MLDA 18	0.301	0.498	0.254	0.614**	0.510**	0.682**
	(0.350)	(0.327)	(0.353)	(0.305)	(0.254)	(0.304)
Bandwidth	1.871	3.742	1.871	1.871	3.742	1.871
MLDA 20	-0.155	-0.120	-0.210	-0.267	-0.349**	-0.216
	(0.251)	(0.207)	(0.258)	(0.212)	(0.172)	(0.222)
Bandwidth	2.992	5.983	2.992	2.992	5.983	2.992
Frequency of heavy drinking episodes in last 30 days						
MLDA 18	0.630*	1.011***	0.685*	0.384	0.393	0.495
	(0.357)	(0.330)	(0.367)	(0.436)	(0.415)	(0.444)
Bandwidth	2.861	5.722	2.861	2.861	5.722	2.861
MLDA 20	0.134	0.283	0.0811	-0.339	-0.358	-0.326
	(0.338)	(0.259)	(0.348)	(0.322)	(0.253)	(0.343)
Bandwidth	2.703	5.406	2.703	2.703	5.406	2.703
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on frequency of alcohol consumption from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Source: Monitor project survey data. Own calculations.

Table F.3: Alcohol intensity LLR results by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Average intensity of beer consumption in last 30 days						
MLDA 18	-0.784	-0.602	-0.582	-1.003*	-0.912*	-0.814
	(0.586)	(0.553)	(0.610)	(0.571)	(0.538)	(0.587)
Bandwidth	2.205	4.411	2.205	2.205	4.411	2.205
MLDA 20	-0.169	-0.0679	-0.104	0.0746	-0.214	0.467
	(0.405)	(0.335)	(0.416)	(0.398)	(0.330)	(0.391)
Bandwidth	3.183	6.366	3.183	3.183	6.366	3.183
Average intensity of low alcohol beer consumption in last 30 days						
MLDA 18	-0.388	-0.633	-0.335	-1.187	-1.201	-1.362
	(0.564)	(0.546)	(0.543)	(0.829)	(0.813)	(0.894)
Bandwidth	3.042	6.083	3.042	3.042	6.083	3.042
MLDA 20	-0.531	-0.668**	-0.340	-0.0547	-0.190	0.146
	(0.395)	(0.320)	(0.413)	(0.321)	(0.298)	(0.354)
Bandwidth	3.305	6.610	3.305	3.305	6.610	3.305
Average intensity of wine consumption in last 30 days						
MLDA 18	-0.326	-0.298	-0.429	-0.0183	0.440	-0.0522
	(0.670)	(0.644)	(0.725)	(0.411)	(0.390)	(0.428)
Bandwidth	2.892	5.783	2.892	2.892	5.783	2.892
MLDA 20	-0.894	-0.665	-0.710	-0.0424	0.361	0.0325
	(0.619)	(0.407)	(0.621)	(0.314)	(0.246)	(0.336)
Bandwidth	2.132	4.265	2.132	2.132	4.265	2.132
Average intensity of cider consumption in last 30 days						
MLDA 18	-0.570	-0.797	0.508	-0.693	-0.864**	-0.633
	(0.808)	(0.745)	(0.714)	(0.436)	(0.398)	(0.455)
Bandwidth	1.952	3.904	1.952	1.952	3.904	1.952
MLDA 20	-0.0769	-0.00500	0.0613	0.739**	0.106	0.762*
	(0.647)	(0.456)	(0.667)	(0.366)	(0.259)	(0.402)
Bandwidth	2.312	4.623	2.312	2.312	4.623	2.312
Average intensity of spirits consumption in last 30 days						
MLDA 18	-1.316*	-0.947	-1.123	-1.737**	-1.658**	-1.360*
	(0.724)	(0.690)	(0.752)	(0.730)	(0.680)	(0.768)
Bandwidth	1.981	3.963	1.981	1.981	3.963	1.981
MLDA 20	-1.003*	-0.0224	-0.810	-0.103	0.342	0.0827
	(0.524)	(0.394)	(0.541)	(0.434)	(0.320)	(0.459)
Bandwidth	2.099	4.198	2.099	2.099	4.198	2.099
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on intensity of alcohol consumption from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor project survey data. Own calculations.

G Sensitivity analysis - Alcohol results with and without birthday month

There is a potential cause for concern that the observed increase in alcohol participation and consumption is purely a birthday party effect and not a structural jump. This affects the interpretation of the results. We provide estimates including and excluding the first month after turning 18 and turning 20 to assess the sensitivity of the results to the "birthday effect". This is a fairly crude method as we would prefer to look at the birthday effect, but the data does not allow this. We find no significant impact of removing the birthday month suggesting the main results are not birthday effects but structural jumps in consumption patterns as a result of the MLDA.

Table G.1: Alcohol participation LLR results with and without birthday month

	Baseline			*No birthday month*		
	(1)	(2)	(3)	(4)	(5)	(6)
Drank alcohol in last 30 days						
MLDA 18	0.0456**	0.0477***	0.0567**	0.0643***	0.0598***	0.0775***
	(0.0230)	(0.0182)	(0.0236)	(0.0234)	(0.0184)	(0.0240)
Bandwidth	1.590	3.181	1.590	1.590	3.181	1.590
MLDA 20	0.0193	-0.00709	0.0116	0.0180	0.00101	0.00921
	(0.0245)	(0.0159)	(0.0248)	(0.0254)	(0.0165)	(0.0257)
Bandwidth	1.104	2.209	1.104	1.104	2.209	1.104
Had a heavy drinking episode in last 30 days						
MLDA 18	0.0429	0.0488**	0.0566*	0.0647**	0.0639***	0.0793***
	(0.0285)	(0.0205)	(0.0291)	(0.0295)	(0.0210)	(0.0301)
Bandwidth	1.485	2.970	1.485	1.485	2.970	1.485
MLDA 20	0.0476	-0.0113	0.0628*	0.0348	-0.0133	0.0501
	(0.0342)	(0.0222)	(0.0357)	(0.0354)	(0.0229)	(0.0369)
Bandwidth	1.257	2.514	1.257	1.257	2.514	1.257
Drank more than recommended weekly amount in last 30 days						
MLDA 18	0.0650**	0.0514***	0.0690***	0.0796***	0.0614***	0.0857***
	(0.0258)	(0.0188)	(0.0265)	(0.0263)	(0.0190)	(0.0270)
Bandwidth	1.498	2.997	1.498	1.498	2.997	1.498
MLDA 20	0.0330	0.0133	0.0258	0.0297	0.0129	0.0221
	(0.0279)	(0.0176)	(0.0284)	(0.0292)	(0.0182)	(0.0298)
Bandwidth	0.984	1.968	0.984	0.984	1.968	0.984
Quantity of pure alcohol consumed in last 30 days						
MLDA 18	9.677***	14.31***	10.74***	11.61***	15.69***	12.46***
	(2.831)	(2.487)	(2.828)	(3.029)	(2.563)	(3.023)
Bandwidth	2.586	5.172	2.586	2.586	5.172	2.586
MLDA 20	-4.748	-6.337**	-4.134	-4.124	-6.390**	-3.929
	(3.368)	(2.477)	(3.400)	(3.493)	(2.537)	(3.510)
Bandwidth	2.622	5.243	2.622	2.622	5.243	2.622
Drank unregulated alcohol in last 30 days						
MLDA 18	-0.0137	-0.0209	-0.00178	-0.00407	-0.0143	0.00836
	(0.0190)	(0.0149)	(0.0191)	(0.0197)	(0.0152)	(0.0199)
Bandwidth	1.612	3.224	1.612	1.612	3.224	1.612
MLDA 20	-0.0419*	-0.0479***	-0.0400*	-0.0465**	-0.0492***	-0.0434*
	(0.0231)	(0.0144)	(0.0238)	(0.0237)	(0.0148)	(0.0244)
Bandwidth	1.298	2.597	1.298	1.298	2.597	1.298
Frequency of drinking occasions in last 30 days						
MLDA 18	1.198	0.394	1.558**	1.078	0.301	1.448*
	(0.731)	(0.530)	(0.769)	(0.745)	(0.537)	(0.783)
Bandwidth	1.376	2.752	1.376	1.376	2.752	1.376
MLDA 20	-0.135	0.975***	-0.0890	-0.222	1.003***	-0.186
	(0.513)	(0.369)	(0.540)	(0.533)	(0.377)	(0.561)
Bandwidth	1.921	3.842	1.921	1.921	3.842	1.921
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on drinking patterns from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Source: Monitor project survey data. Own calculations.

Table G.2: Alcohol frequency LLR results with and without birthday month

	Baseline			*No birthday month*		
	(1)	(2)	(3)	(4)	(5)	(6)
Frequency of beer consumption in last 30 days						
MLDA 18	0.449*	0.595***	0.559**	0.522*	0.639***	0.635**
	(0.271)	(0.228)	(0.278)	(0.282)	(0.232)	(0.290)
Bandwidth	1.710	3.419	1.710	1.710	3.419	1.710
MLDA 20	0.139	0.0769	0.0757	0.140	0.0635	0.0579
	(0.202)	(0.163)	(0.207)	(0.207)	(0.165)	(0.211)
Bandwidth	3.077	6.154	3.077	3.077	6.154	3.077
Frequency of low alcohol beer consumption in last 30 days						
MLDA 18	0.0718	0.327	-0.103	0.173	0.409	-0.0104
	(0.295)	(0.281)	(0.299)	(0.303)	(0.285)	(0.307)
Bandwidth	3.746	7.492	3.746	3.746	7.492	3.746
MLDA 20	-0.193	-0.189	-0.139	-0.114	-0.172	-0.0768
	(0.287)	(0.229)	(0.294)	(0.301)	(0.235)	(0.308)
Bandwidth	3.241	6.482	3.241	3.241	6.482	3.241
Frequency of wine consumption in last 30 days						
MLDA 18	0.243	0.490**	0.241	0.171	0.509**	0.162
	(0.225)	(0.207)	(0.239)	(0.226)	(0.207)	(0.239)
Bandwidth	2.253	4.507	2.253	2.253	4.507	2.253
MLDA 20	-0.219	-0.228	-0.332*	-0.150	-0.190	-0.280
	(0.184)	(0.150)	(0.188)	(0.191)	(0.152)	(0.195)
Bandwidth	3.172	6.345	3.172	3.172	6.345	3.172
Frequency of cider consumption in last 30 days						
MLDA 18	0.553**	0.540***	0.513**	0.598***	0.558***	0.554**
	(0.218)	(0.199)	(0.217)	(0.228)	(0.202)	(0.229)
Bandwidth	2.016	4.033	2.016	2.016	4.033	2.016
MLDA 20	0.528**	-0.0194	0.549**	0.589**	0.00262	0.593**
	(0.248)	(0.177)	(0.264)	(0.269)	(0.184)	(0.283)
Bandwidth	1.891	3.783	1.891	1.891	3.783	1.891
Frequency of spirits consumption in last 30 days						
MLDA 18	0.437*	0.492**	0.437*	0.482**	0.523**	0.504**
	(0.236)	(0.210)	(0.238)	(0.244)	(0.213)	(0.246)
Bandwidth	1.871	3.742	1.871	1.871	3.742	1.871
MLDA 20	-0.203	-0.210	-0.211	-0.162	-0.192	-0.172
	(0.171)	(0.139)	(0.176)	(0.175)	(0.141)	(0.180)
Bandwidth	2.992	5.983	2.992	2.992	5.983	2.992
Frequency of heavy drinking episodes in last 30 days						
MLDA 18	0.530*	0.754***	0.582**	0.604**	0.815***	0.643**
	(0.277)	(0.259)	(0.285)	(0.282)	(0.261)	(0.290)
Bandwidth	2.861	5.722	2.861	2.861	5.722	2.861
MLDA 20	-0.0338	0.0434	-0.0702	0.0520	0.0755	-0.0259
	(0.242)	(0.186)	(0.249)	(0.252)	(0.190)	(0.259)
Bandwidth	2.703	5.406	2.703	2.703	5.406	2.703
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on frequency of alcohol consumption from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Source: Monitor project survey data. Own calculations.

Table G.3: Alcohol intensity LLR results with and without birthday month

	Baseline			*No birthday month*		
	(1)	(2)	(3)	(4)	(5)	(6)
Average intensity of beer consumption in last 30 days						
MLDA 18	-0.991**	-0.844*	-0.619	-0.869*	-0.792*	-0.495
	(0.455)	(0.431)	(0.456)	(0.464)	(0.433)	(0.466)
Bandwidth	2.205	4.411	2.205	2.205	4.411	2.205
MLDA 20	0.0232	-0.0236	0.0864	-0.00442	-0.0410	0.0188
	(0.314)	(0.260)	(0.306)	(0.320)	(0.262)	(0.312)
Bandwidth	3.183	6.366	3.183	3.183	6.366	3.183
Average intensity of low alcohol beer consumption in last 30 days						
MLDA 18	-0.669	-0.823*	-0.601	-0.596	-0.795*	-0.548
	(0.472)	(0.458)	(0.466)	(0.476)	(0.460)	(0.471)
Bandwidth	3.042	6.083	3.042	3.042	6.083	3.042
MLDA 20	-0.430	-0.550**	-0.179	-0.482*	-0.584**	-0.244
	(0.294)	(0.243)	(0.306)	(0.292)	(0.243)	(0.302)
Bandwidth	3.305	6.610	3.305	3.305	6.610	3.305
Average intensity of wine consumption in last 30 days						
MLDA 18	-0.0856	0.225	-0.0827	-0.0281	0.272	-0.0404
	(0.353)	(0.336)	(0.372)	(0.358)	(0.338)	(0.376)
Bandwidth	2.892	5.783	2.892	2.892	5.783	2.892
MLDA 20	-0.362	-0.0163	-0.232	-0.303	-0.00339	-0.184
	(0.299)	(0.215)	(0.316)	(0.315)	(0.219)	(0.332)
Bandwidth	2.132	4.265	2.132	2.132	4.265	2.132
Average intensity of cider consumption in last 30 days						
MLDA 18	-0.648	-0.831**	-0.218	-0.726*	-0.872**	-0.316
	(0.410)	(0.377)	(0.393)	(0.417)	(0.379)	(0.402)
Bandwidth	1.952	3.904	1.952	1.952	3.904	1.952
MLDA 20	0.386	0.0461	0.525	0.449	0.107	0.535
	(0.341)	(0.241)	(0.362)	(0.353)	(0.245)	(0.373)
Bandwidth	2.312	4.623	2.312	2.312	4.623	2.312
Average intensity of spirits consumption in last 30 days						
MLDA 18	-1.608***	-1.364***	-1.147**	-1.598***	-1.337***	-1.101**
	(0.519)	(0.488)	(0.538)	(0.530)	(0.491)	(0.548)
Bandwidth	1.981	3.963	1.981	1.981	3.963	1.981
MLDA 20	-0.620*	0.133	-0.361	-0.615	0.134	-0.322
	(0.362)	(0.267)	(0.370)	(0.378)	(0.272)	(0.386)
Bandwidth	2.099	4.198	2.099	2.099	4.198	2.099
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA at 18 and 20 years on alcohol intensity of consumption patterns from a LLR model using age in months as the running variable and bandwidth chosen as per the IK optimal bandwidth selection. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include economic status and county of residence. Standard errors are shown in parenthesis and are clustered at the age in years and months level. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Monitor project survey data. Own calculations.

H Heterogeneity analysis - Mortality results by gender

Table H.1: LLR results of MLDA on mortality by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Any cause						
MLDA 18	0.784**	0.667***	0.801**	0.0770	0.231	0.106
	(0.317)	(0.247)	(0.322)	(0.289)	(0.212)	(0.290)
Bandwidth	1.040	2.080	1.040	1.040	2.080	1.040
MLDA 20	0.0630	-0.125	0.0660	-0.00433	-0.0755	-0.0415
	(0.430)	(0.304)	(0.434)	(0.269)	(0.205)	(0.270)
Bandwidth	0.968	1.935	0.968	0.968	1.935	0.968
External Causes						
MLDA 18	0.298	0.669***	0.294	0.126	0.274	0.171
	(0.326)	(0.244)	(0.334)	(0.284)	(0.200)	(0.279)
Bandwidth	0.853	1.707	0.853	0.853	1.707	0.853
MLDA 20	0.00820	-0.0166	-0.0121	0.0495	-0.188	-0.00169
	(0.409)	(0.287)	(0.414)	(0.286)	(0.207)	(0.281)
Bandwidth	0.900	1.800	0.900	0.900	1.800	0.900
Internal causes						
MLDA 18	0.276	-0.0179	0.279	-0.141	-0.0381	-0.127
	(0.208)	(0.141)	(0.207)	(0.176)	(0.134)	(0.180)
Bandwidth	1.024	2.049	1.024	1.024	2.049	1.024
MLDA 20	-0.0665	-0.0337	-0.0419	0.0849	0.0486	0.0862
	(0.159)	(0.118)	(0.162)	(0.148)	(0.109)	(0.148)
Bandwidth	1.567	3.134	1.567	1.567	3.134	1.567
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of death, split by gender. The dependent variable is the log of the death count that occurred at age x measured in years and days. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Death administrative data. Own calculations.

Table H.2: LLR results of MLDA on external causes of mortality by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Motor vehicle related accidents						
MLDA 18	0.698***	0.974***	0.686***	0.0954	0.233*	0.0962
	(0.222)	(0.171)	(0.227)	(0.169)	(0.121)	(0.166)
Bandwidth	1.000	2.001	1.000	1.000	2.001	1.000
MLDA 20	0.222	0.130	0.170	0.0240	-0.129	0.0537
	(0.260)	(0.182)	(0.260)	(0.185)	(0.130)	(0.188)
Bandwidth	0.893	1.786	0.893	0.893	1.786	0.893
Suicide						
MLDA 18	-0.120	0.0352	-0.120	0.158	0.0920	0.209
	(0.149)	(0.109)	(0.153)	(0.133)	(0.0914)	(0.135)
Bandwidth	1.098	2.196	1.098	1.098	2.196	1.098
MLDA 20	-0.531***	-0.320**	-0.483***	0.0569	0.0202	0.0472
	(0.180)	(0.133)	(0.181)	(0.130)	(0.101)	(0.131)
Bandwidth	1.038	2.076	1.038	1.038	2.076	1.038
Homicide						
MLDA 18	-0.0132	-0.0311	-0.00815	0.0451	0.0599	0.0326
	(0.0575)	(0.0379)	(0.0581)	(0.0546)	(0.0406)	(0.0562)
Bandwidth	1.054	2.107	1.054	1.054	2.107	1.054
MLDA 20	0.0611	0.0372	0.0592	0.0167	-0.0225	-0.00685
	(0.0537)	(0.0390)	(0.0532)	(0.0570)	(0.0396)	(0.0562)
Bandwidth	1.322	2.644	1.322	1.322	2.644	1.322
Alcohol related						
MLDA 18	-0.0904*	-0.0703	-0.0879	-0.0242	-0.0683	-0.0301
	(0.0526)	(0.0432)	(0.0543)	(0.0728)	(0.0467)	(0.0723)
Bandwidth	0.902	1.804	0.902	0.902	1.804	0.902
MLDA 20	-0.0124	0.0120	-0.0206	-0.0226	0.00279	-0.0234
	(0.0686)	(0.0479)	(0.0695)	(0.0460)	(0.0346)	(0.0473)
Bandwidth	1.251	2.501	1.251	1.251	2.501	1.251
Narcotics related						
MLDA 18	0.0368	-0.0181	0.0351	0.00193	0.00382	0.00286
	(0.0619)	(0.0419)	(0.0635)	(0.0321)	(0.0305)	(0.0343)
Bandwidth	0.910	1.820	0.910	0.910	1.820	0.910
MLDA 20	-0.0162	0.0136	-0.00699	-0.0287	-0.0487	-0.0255
	(0.0692)	(0.0501)	(0.0704)	(0.0450)	(0.0308)	(0.0448)
Bandwidth	1.382	2.764	1.382	1.382	2.764	1.382
Other external causes						
MLDA 18	-0.0815	-0.00260	-0.0660	-0.0388	-0.0340	-0.0483
	(0.128)	(0.0921)	(0.129)	(0.0879)	(0.0633)	(0.0876)
Bandwidth	1.642	3.284	1.642	1.642	3.284	1.642
MLDA 20	0.230	0.218	0.200	-0.0436	-0.0425	-0.0942
	(0.207)	(0.138)	(0.210)	(0.146)	(0.0937)	(0.140)
Bandwidth	0.771	1.542	0.771	0.771	1.542	0.771
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of death, split by gender. The dependent variable is the log of the death count that occurred at age x measured in years and days. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Death administrative data. Own calculations.

I Diagnostics - Hospital admissions due to mental disorders

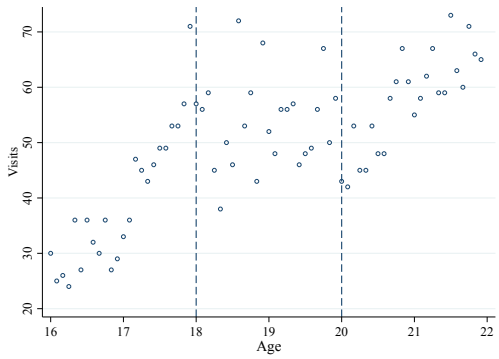

Fig. I.1: Age profile of hospital admissions due to mental disorders (Survey sample)
Notes: The scatter points are monthly age blocks of count of hospital admissions for mental disorders (ICD grouping F).
Source: Patient administrative data, merged to Swedish survey of household living standards (ULF). Own calculations.

J Sensitivity analysis - Hospital admissions

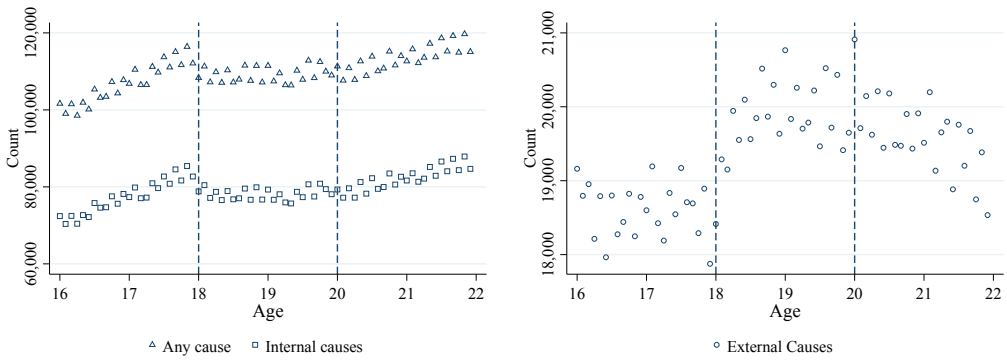

Fig. J.1: The effects of MLDA on hospital admissions (excluding counties with OOP costs at 18th birthday)
Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.
Source: Patient administrative data. Own calculations.

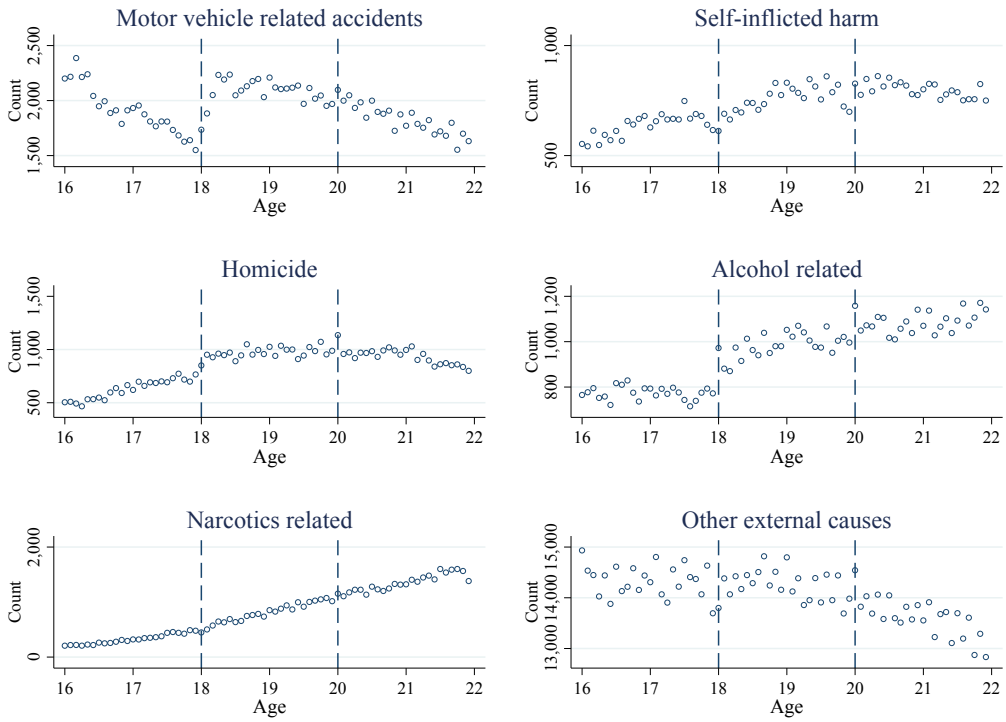

Fig. J.2: The effects of MLDA on hospital admissions due to external causes (excluding counties with OOP costs at 18th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

Source: Patient administrative data. Own calculations.

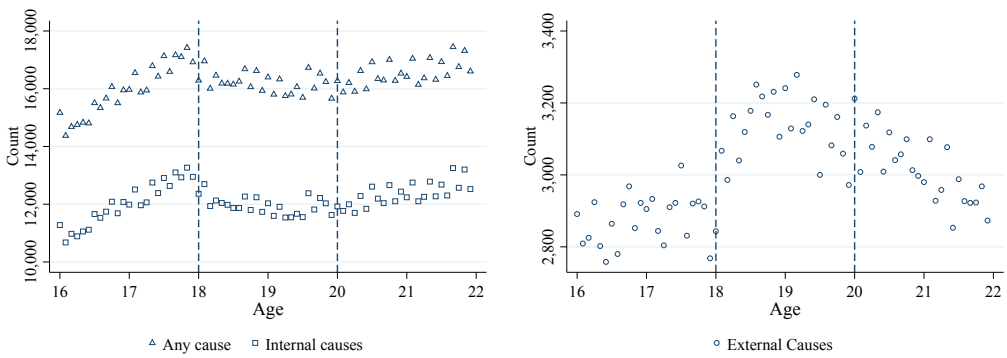

Fig. J.3: The effects of MLDA on hospital admissions (excluding counties with OOP costs at 20th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

Source: Patient administrative data. Own calculations.

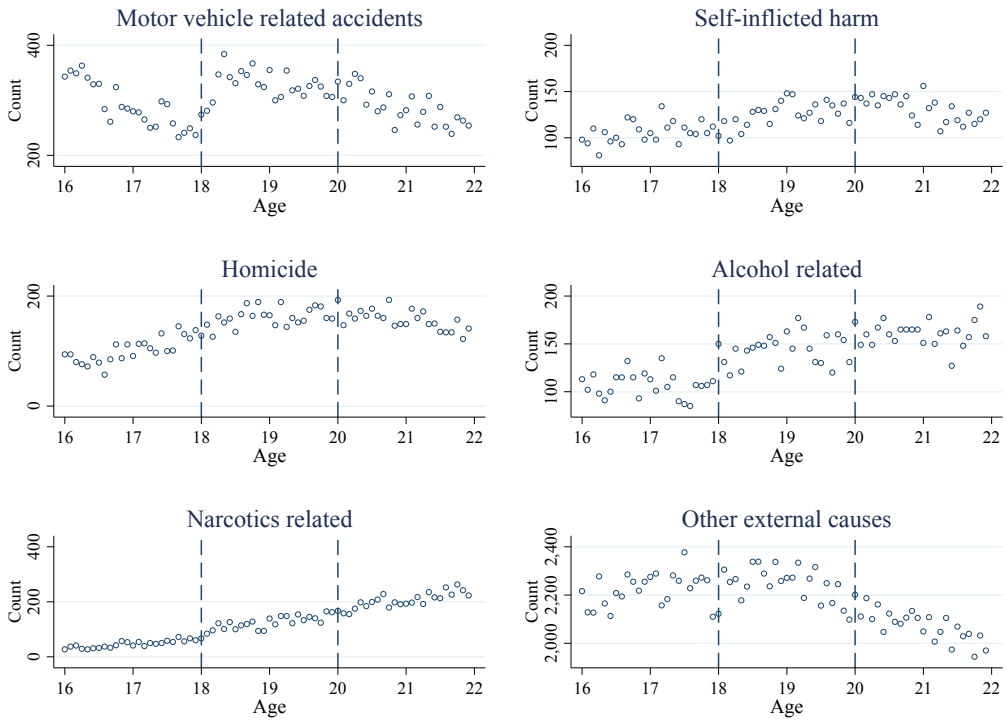

Fig. J.4: The effects of MLDA on hospital admissions due to external causes (excluding counties with OOP costs at 20th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

Source: Patient administrative data. Own calculations.

Table J.1: LLR results of MLDA on hospital admissions, counties without OOP costs at MLDA threshold

	No OOP at 18			*No OOP at 20*		
	(1)	(2)	(3)	(4)	(5)	(6)
Any cause						
MLDA 18	-0.0355***	-0.0395***	-0.0309**	-0.0272	-0.0431***	-0.0291*
	(0.0129)	(0.00777)	(0.0121)	(0.0171)	(0.0116)	(0.0171)
Bandwidth	0.217	0.433	0.217	0.217	0.433	0.217
MLDA 20	-0.0108	-0.00749	0.00139	0.0351	0.0233	0.0418*
	(0.0276)	(0.0158)	(0.0128)	(0.0273)	(0.0173)	(0.0223)
Bandwidth	0.0664	0.133	0.0664	0.0664	0.133	0.0664
External Causes						
MLDA 18	0.0702***	0.0536***	0.0640**	0.0768	0.0293	0.0291
	(0.0268)	(0.0191)	(0.0277)	(0.0631)	(0.0433)	(0.0595)
Bandwidth	0.0611	0.122	0.0611	0.0611	0.122	0.0611
MLDA 20	0.0885*	0.0513*	0.0491*	0.173**	0.118**	0.114
	(0.0456)	(0.0273)	(0.0275)	(0.0794)	(0.0531)	(0.0722)
Bandwidth	0.0540	0.108	0.0540	0.0540	0.108	0.0540
Internal causes						
MLDA 18	-0.0374	-0.0396*	-0.00909	-0.0318	-0.0379	-0.0129
	(0.0386)	(0.0227)	(0.0350)	(0.0375)	(0.0246)	(0.0376)
Bandwidth	0.0701	0.140	0.0701	0.0701	0.140	0.0701
MLDA 20	-0.0160	-0.0129	0.00722	-0.00947	0.00519	0.00823
	(0.0299)	(0.0174)	(0.0137)	(0.0269)	(0.0187)	(0.0279)
Bandwidth	0.0730	0.146	0.0730	0.0730	0.146	0.0730
Male	✓	✓	✓			
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of hospital admission, split by OOP cost regime. The dependent variable is the log hospital admissions. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

Table J.2: LLR results of MLDA on hospital admissions, specific causes, counties without OOP costs at MLDA threshold

	No OOP at 18			*No OOP at 20*		
	(1)	(2)	(3)	(4)	(5)	(6)
Motor vehicle related accidents						
MLDA 18	0.000893	0.0440	0.0892	-0.194	0.0712	-0.0711
	(0.103)	(0.0784)	(0.135)	(0.228)	(0.194)	(0.292)
Bandwidth	0.0433	0.0866	0.0433	0.0433	0.0866	0.0433
MLDA 20	0.0285	0.0547	0.0433	0.446**	0.193	0.404*
	(0.0652)	(0.0478)	(0.0778)	(0.174)	(0.129)	(0.222)
Bandwidth	0.0652	0.130	0.0652	0.0652	0.130	0.0652
Self-inflicted harm						
MLDA 18	0.0634	-0.0569	0.0168	0.103	-0.0992	-0.0278
	(0.118)	(0.0872)	(0.133)	(0.251)	(0.199)	(0.303)
Bandwidth	0.0907	0.181	0.0907	0.0907	0.181	0.0907
MLDA 20	0.255**	0.199***	0.216**	-0.0661	0.175	0.0274
	(0.101)	(0.0722)	(0.103)	(0.269)	(0.177)	(0.289)
Bandwidth	0.0825	0.165	0.0825	0.0825	0.165	0.0825
Homicide						
MLDA 18	0.204**	0.0382	0.139	0.203	-0.0451	0.257
	(0.101)	(0.0768)	(0.118)	(0.228)	(0.185)	(0.254)
Bandwidth	0.0817	0.163	0.0817	0.0817	0.163	0.0817
MLDA 20	0.317**	0.216**	0.176	0.243	0.0501	-0.00530
	(0.141)	(0.0938)	(0.126)	(0.218)	(0.176)	(0.193)
Bandwidth	0.0553	0.111	0.0553	0.0553	0.111	0.0553
Alcohol related						
MLDA 18	0.468***	0.282***	0.283**	0.544*	0.329	0.243
	(0.123)	(0.0933)	(0.120)	(0.312)	(0.221)	(0.313)
Bandwidth	0.0932	0.186	0.0932	0.0932	0.186	0.0932
MLDA 20	0.125	0.136*	0.0297	0.0105	0.242	-0.251
	(0.111)	(0.0718)	(0.0869)	(0.301)	(0.194)	(0.370)
Bandwidth	0.0915	0.183	0.0915	0.0915	0.183	0.0915
Narcotics related						
MLDA 18	-0.0822	-0.194**	-0.108	0.266	0.249	0.185
	(0.142)	(0.0963)	(0.163)	(0.345)	(0.242)	(0.385)
Bandwidth	0.118	0.236	0.118	0.118	0.236	0.118
MLDA 20	0.0575	0.165*	0.0322	-0.0681	0.0331	-0.194
	(0.128)	(0.0875)	(0.123)	(0.353)	(0.244)	(0.350)
Bandwidth	0.0537	0.107	0.0537	0.0537	0.107	0.0537
Other external causes						
MLDA 18	0.00804	0.0290	0.0144	0.0118	-0.00431	-0.0438
	(0.0319)	(0.0214)	(0.0289)	(0.0678)	(0.0472)	(0.0652)
Bandwidth	0.0578	0.116	0.0578	0.0578	0.116	0.0578
MLDA 20	0.00711	0.0237	-0.0101	0.0712	0.0276	0.0556
	(0.0241)	(0.0152)	(0.0204)	(0.0576)	(0.0410)	(0.0608)
Bandwidth	0.121	0.241	0.121	0.121	0.241	0.121
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of hospital admission, split by OOP cost regime. The dependent variable is the log hospital admissions. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

K Heterogeneity analysis - Hospital visits results by gender

Table K.1: LLR results of MLDA on hospital admissions by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Any cause						
MLDA 18	-0.0471***	-0.0493***	-0.0467***	-0.0593***	-0.0577***	-0.0511***
	(0.0123)	(0.00762)	(0.0122)	(0.0155)	(0.00916)	(0.0147)
Bandwidth	0.217	0.433	0.217	0.217	0.433	0.217
MLDA 20	-0.000781	-0.000866	0.00542	-0.0239	-0.0156	-0.00810
	(0.0223)	(0.0138)	(0.0153)	(0.0318)	(0.0184)	(0.0166)
Bandwidth	0.0664	0.133	0.0664	0.0664	0.133	0.0664
External Causes						
MLDA 18	0.0571**	0.0328	0.0418	0.0956**	0.0734**	0.0748*
	(0.0280)	(0.0201)	(0.0353)	(0.0375)	(0.0286)	(0.0416)
Bandwidth	0.0611	0.122	0.0611	0.0611	0.122	0.0611
MLDA 20	0.0731*	0.0328	0.0424	0.114**	0.0765**	0.0753
	(0.0429)	(0.0276)	(0.0323)	(0.0499)	(0.0335)	(0.0472)
Bandwidth	0.0540	0.108	0.0540	0.0540	0.108	0.0540
Internal causes						
MLDA 18	-0.0566	-0.0570**	-0.0344	-0.0705*	-0.0707***	-0.0361
	(0.0388)	(0.0231)	(0.0365)	(0.0425)	(0.0253)	(0.0399)
Bandwidth	0.0701	0.140	0.0701	0.0701	0.140	0.0701
MLDA 20	-0.0105	-0.00790	0.0118	-0.0287	-0.0234	-0.00512
	(0.0251)	(0.0156)	(0.0156)	(0.0337)	(0.0195)	(0.0172)
Bandwidth	0.0730	0.146	0.0730	0.0730	0.146	0.0730
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of hospital admission, split by gender. The dependent variable is the log hospital admissions. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

Table K.2: LLR results of MLDA on hospital admissions, specific causes by gender

	Males			*Females*		
	(1)	(2)	(3)	(4)	(5)	(6)
Motor vehicle related accidents						
MLDA 18	0.0134	0.0243	0.0574	0.00381	0.0253	0.160
	(0.105)	(0.0824)	(0.132)	(0.153)	(0.117)	(0.236)
Bandwidth	0.0433	0.0866	0.0433	0.0433	0.0866	0.0433
MLDA 20	0.0780	0.0604	0.0498	0.0587	0.127	0.241**
	(0.0713)	(0.0515)	(0.0813)	(0.111)	(0.0817)	(0.108)
Bandwidth	0.0652	0.130	0.0652	0.0652	0.130	0.0652
Self-inflicted harm						
MLDA 18	0.570***	0.187	0.473*	-0.151	-0.145*	-0.126
	(0.208)	(0.166)	(0.254)	(0.118)	(0.0814)	(0.118)
Bandwidth	0.0907	0.181	0.0907	0.0907	0.181	0.0907
MLDA 20	-0.0272	-0.0540	-0.0436	0.324***	0.219***	0.303***
	(0.165)	(0.124)	(0.211)	(0.106)	(0.0756)	(0.108)
Bandwidth	0.0825	0.165	0.0825	0.0825	0.165	0.0825
Homicide						
MLDA 18	0.406***	0.162*	0.302**	0.106	-0.00854	0.0983
	(0.130)	(0.0949)	(0.137)	(0.145)	(0.0991)	(0.153)
Bandwidth	0.0817	0.163	0.0817	0.0817	0.163	0.0817
MLDA 20	0.332**	0.231**	0.282**	0.143	0.274*	-0.129
	(0.133)	(0.0928)	(0.136)	(0.219)	(0.157)	(0.219)
Bandwidth	0.0553	0.111	0.0553	0.0553	0.111	0.0553
Alcohol related						
MLDA 18	0.442***	0.283***	0.268*	0.470***	0.302***	0.265**
	(0.153)	(0.100)	(0.140)	(0.138)	(0.0980)	(0.126)
Bandwidth	0.0932	0.186	0.0932	0.0932	0.186	0.0932
MLDA 20	0.153	0.156*	0.0584	0.214*	0.128	0.132
	(0.121)	(0.0870)	(0.124)	(0.118)	(0.0781)	(0.104)
Bandwidth	0.0915	0.183	0.0915	0.0915	0.183	0.0915
Narcotics related						
MLDA 18	-0.0993	-0.214***	-0.122	-0.241	-0.191*	-0.103
	(0.105)	(0.0752)	(0.118)	(0.192)	(0.114)	(0.129)
Bandwidth	0.118	0.236	0.118	0.118	0.236	0.118
MLDA 20	-0.167	-0.00704	-0.190*	0.417***	0.358***	0.393**
	(0.111)	(0.0774)	(0.105)	(0.127)	(0.0958)	(0.168)
Bandwidth	0.0537	0.107	0.0537	0.0537	0.107	0.0537
Other external causes						
MLDA 18	-0.0193	-0.000975	-0.00923	0.0572	0.0553*	0.0416
	(0.0363)	(0.0237)	(0.0436)	(0.0427)	(0.0306)	(0.0449)
Bandwidth	0.0578	0.116	0.0578	0.0578	0.116	0.0578
MLDA 20	0.00934	0.0106	-0.00629	-0.00545	0.0110	-0.0220
	(0.0250)	(0.0166)	(0.0227)	(0.0338)	(0.0226)	(0.0338)
Bandwidth	0.121	0.241	0.121	0.121	0.241	0.121
Covariates			✓			✓
IK optimal bandwidth	✓	2x	✓	✓	2x	✓

Notes: This table shows the impact of the MLDA for various causes of hospital admission, split by gender. The dependent variable is the log hospital admissions. We add 0.5 to the count before taking logs to deal with zeros. All estimates are from separate regressions. Columns 1,3,4 and 6 use the IK optimal bandwidth. Columns 2 and 5 use double the size of the IK optimal bandwidth. Columns 3 and 6 include covariates. Covariates include county, whether foreign born and dummies for birthday at 18 and 20 and the day after birthday at 18 and 20 and for whether county started charging out of pocket payments at 18th or 20th birthday. Standard errors clustered by age at death measured in days are shown in parenthesis. Testing the null of the coefficient: * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.
Source: Patient administrative data. Own calculations.

Health, inequality and the impact of public policy

Gawain has a Masters in Economics from University College London. He has worked as a labour economist for the Civil Service in London, UK, with a focus on poverty policy. This thesis is an empirical investigation of two important public policies and their impact on health and income related health inequality: education and drinking age laws. The thesis contributes to the literature by developing a new health inequality decomposition method. It also uses a number of quasi-experiments to identify the impact of education and drinking age laws on health. The results find no support for a health or health inequality improving impact of increased years of education, but do find support for Sweden's particular design of minimum legal drinking age.

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