

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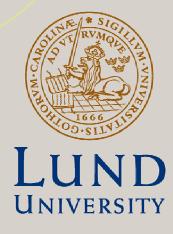
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Too Young to Die: Regression
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Too young to die: Regression discontinuity of a two-part minimum legal drinking age policy and the causal effect of alcohol on health*

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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 onpremise 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. 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

¹This 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

²Interviewees 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.

³Whilst 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.

⁴A 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.

⁵There 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	N	Mean	N
	,	(16 -	19)	(18 -	21)
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.

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

⁷At 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:

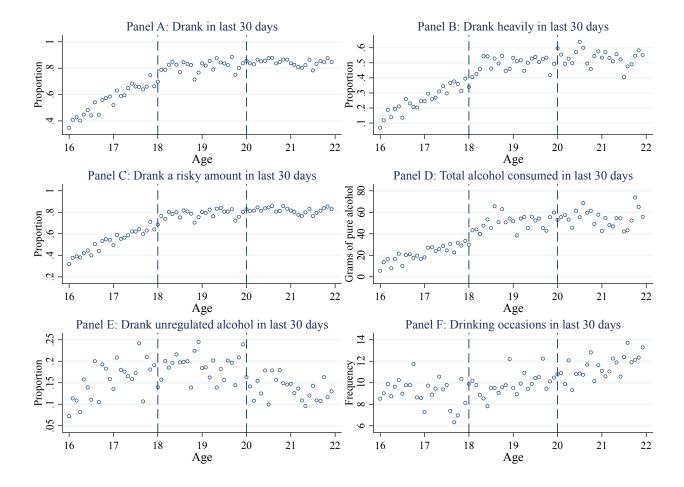


Fig. 1: Drinking behaviours by age

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

Source: Monitor Project Survey Data. Own calculations.

$$y_i = \alpha + \beta M L D A 18_i + f(age_i) + \varepsilon_i; \tag{1}$$

$$y_i = \alpha + \beta M L D A 20_i + f(age_i) + \varepsilon_i; \tag{2}$$

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 (aqe_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

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

⁹Individuals 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.

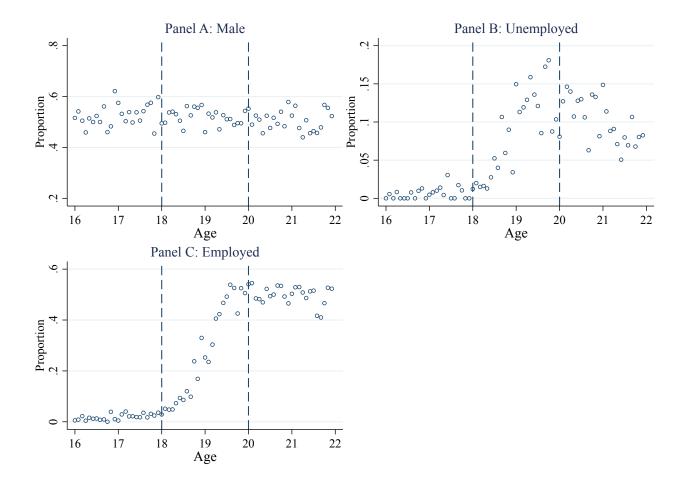


Fig. 2: Diagnostic tests: Covariates by age

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.

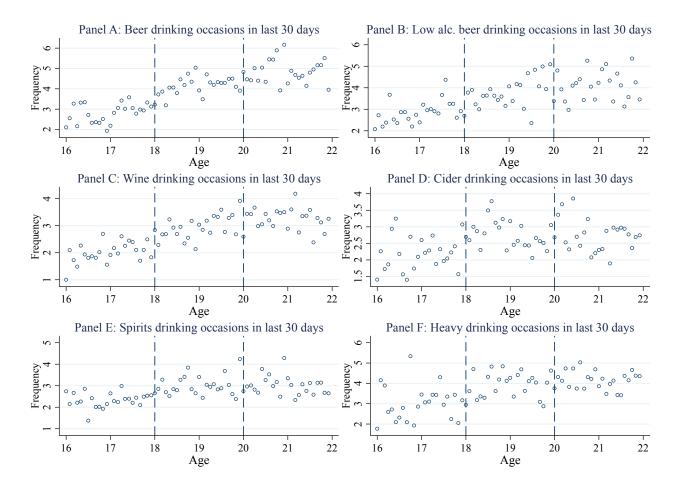


Fig. 3: Drinking frequency by age

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	$\stackrel{}{1.971}^{'}$	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				\checkmark

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

Table 4: LLR regression estimates of MLDA	*			· ·
	(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	3.1,1	1.000	0.012	0.1.1
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**
111111111111111111111111111111111111111	(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	1.000	0.010	0.150	1.000
MLDA 18	0.481**	0.395	0.518**	0.437^{*}
MEDIT 10	(0.244)	(0.378)	(0.212)	(0.238)
Bandwidth	1.882	0.941	3.764	1.882
MLDA 20	-0.163	0.941 0.137	-0.193	-0.211
MIDDIT 20	(0.175)	(0.294)	(0.141)	(0.176)
Bandwidth	(0.173) 2.991	(0.294) 1.495	5.982	(0.176) 2.991
Frequency of heavy drinking episodes in last 30 days	4.331	1.430	0.304	4.331
MLDA 18	0.599**	0.672^{*}	0.835***	0.606**
MILDA 10		(0.369)		
Dandwidth	(0.285) 2.719	. ,	(0.262)	(0.286) 2.719
Bandwidth MLDA 20		1.359	5.438	
MLDA 20	0.0517	0.584	0.0752	-0.0702
Dandwidth	(0.252)	(0.375)	(0.190)	(0.249)
Bandwidth IK optimal bandwidth	2.706	1.353	5.412	2.706
IK ontimal handwidth	\checkmark	0.5x	2x	\checkmark
Covariates	•			\checkmark

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.

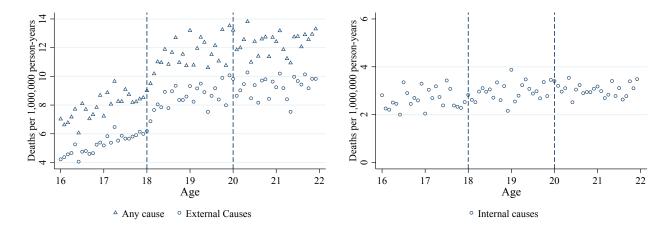


Fig. 4: Mortality by age

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

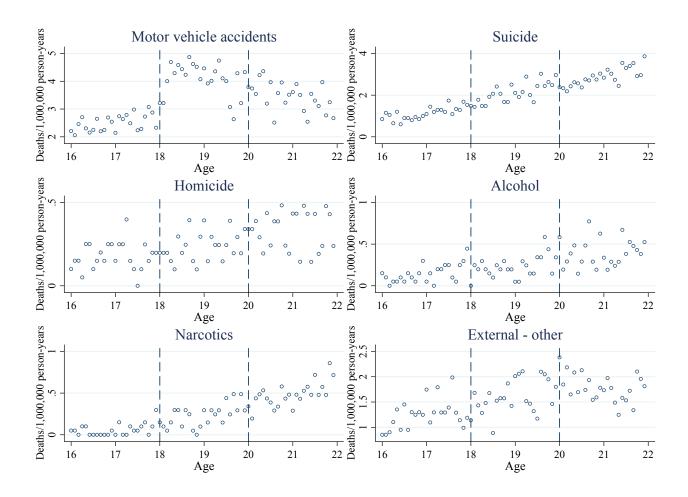


Fig. 5: Mortality due to external causes by age

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
MI DA OO	0.130	0.336**	0.113	0.0971
MLDA 20				
MLDA 20	(0.101)	(0.149)	(0.0706)	(0.100)
Bandwidth	$(0.101) \\ 0.771$	$(0.149) \\ 0.385$	(0.0706) 1.542	(0.100) 0.771
	,	,	,	

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.

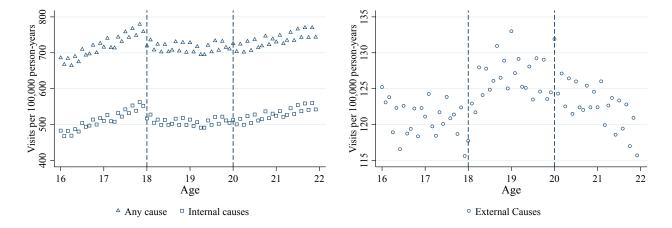


Fig. 6: Hospital admissions by age

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				\checkmark

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. 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%.

¹¹At 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.

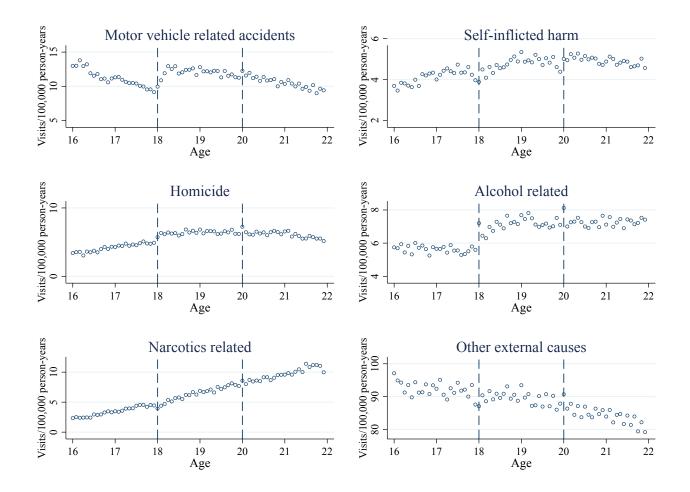


Fig. 7: Hospital admissions due to external causes by age Notes: The scatter points are monthly age blocks of count of hospital visits for the years 1969-2015. Source: Patient administrative data. Own calculations.

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			
	(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	0.0000	0.0210	0.111	0.0000
MLDA 18	0.445***	0.676***	0.288***	0.254**
MIBBIT TO	(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
WIDDII 20	(0.101)	(0.143)	(0.0675)	(0.0877)
Bandwidth	0.0915	0.0458	0.183	0.0915
Narcotics related	0.0310	0.0400	0.103	0.0310
MLDA 18	-0.135	-0.0869	-0.185***	-0.113
WIBBIT TO	(0.101)	(0.152)	(0.0640)	(0.0960)
Bandwidth	0.118	0.0591	0.236	0.118
MLDA 20	0.0555	0.0591 0.262^*	0.230	0.110
MLDA 20	(0.0962)	(0.142)	(0.0622)	(0.0394)
Bandwidth	0.0537	0.0269	0.107	0.0537
Other external causes	0.0551	0.0209	0.107	0.0551
	0.0109	0.0016	0.0000	0.0100
MLDA 18	0.0102	0.0216	0.0208	0.0108
Dandari dth	(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
D 1:141	(0.0208)	(0.0332)	(0.0132)	(0.0172)
Bandwidth	0.121	0.0603	0.241	0.121
IK optimal bandwidth	\checkmark	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.

Discussion 6

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	or vehicle re	lated extern	al 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	e to non-mot	tor vehicle i	related extern	nal causes 0.0573
MLDA 16	(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

	LCD 40	, -	
Diagnosis	ICD 10 code	ICD 9 code	ICD 8 code
External causes	S,T,V,W,X,Y,E24.4	8,9,E8,E9	8,9,E8,E9
	F10-F19 (excl .6,	291,292,303-305	291,292,304-305
	F17.1,F17.2),F55	425F,571A-D,790D	571.098
	G31.2,G62.1,G72.1		
	G72.0,I42.6-7,		
	K29.2,K70,K85.2-3		
	K86.0,R78.0		
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	291,303,305A	291,303,305.0
	G31.2,G62.1,G72.1	357F,425F,535D	357.5,425.5,
	I42.6,K29.2,	571A-D,	535.3,571.03,
	K70.0-K70.9,K85.2	E860,E980	E860,E980
	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		
Narcotics related	F11-F16,F18,F19,	304A-X,305X	304.0-304.9
	O35.5,P04.4,T40	965A,968E,969G,	305.2-305.7,
	T43.6,Z50.3,Z71.5	969H	305.9,965.0,
	Z72.2		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 c		
	- ij 1000 00001 1000 01		

^{*} 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

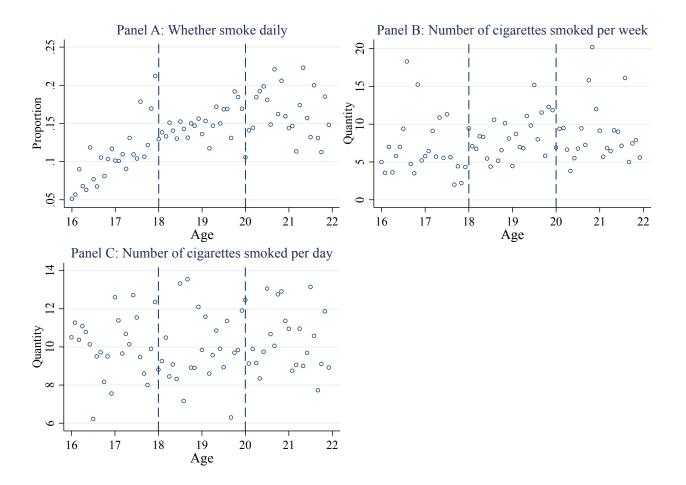


Fig. B.1: Tobacco consumption by age

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.

Table B.1: LLR regression estimates of MLSA impact on tobacco consumption

	(1)	(2)	(3)	(4)
Smoked in the last 30 de	ays			
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 i	n last 30 d	ays		
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	t 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

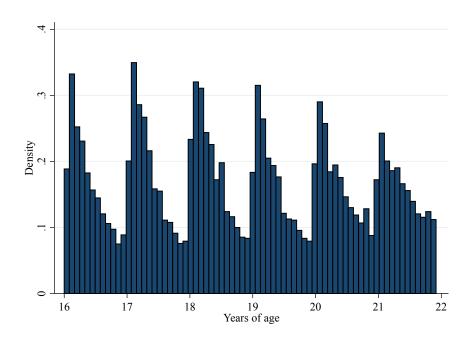


Fig. C.1: Diagnostic test: manipulation of the cut-off

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 made	le			
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.

D Alcohol intensity results

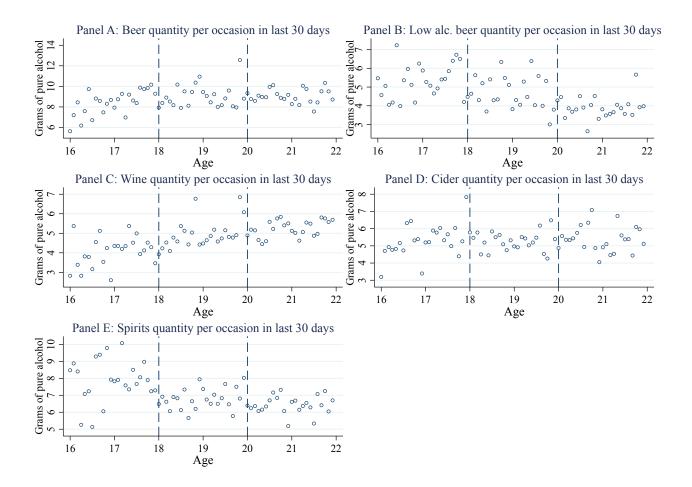


Fig. D.1: Average intensity per drinking occasion by age

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.

Table D.1: LLR regression estimates of MLDA impact on average intensity per drinking occasion

Stession estimates of				
	(1)	(2)	(3)	(4)
Average intensity of bee			-	0.010
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 bee	er consump	tion 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 win	ne consumpt	tion 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 cide	er consump	tion 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 spir	rits consum	ption in las	st 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	<u> </u>
Covariates	•	0.012		↓
				•

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

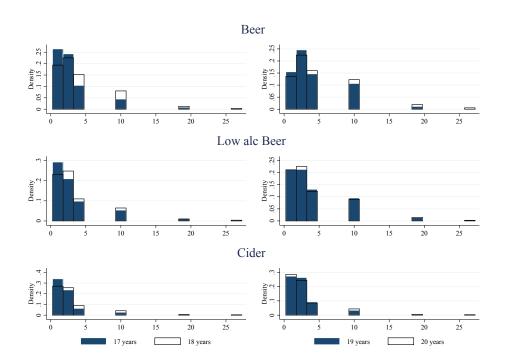


Fig. E.1: Density plot of frequency of alcohol consumption 12 months before and after the MLDA cut-offs (Beer, low alcohol beer and cider)

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.

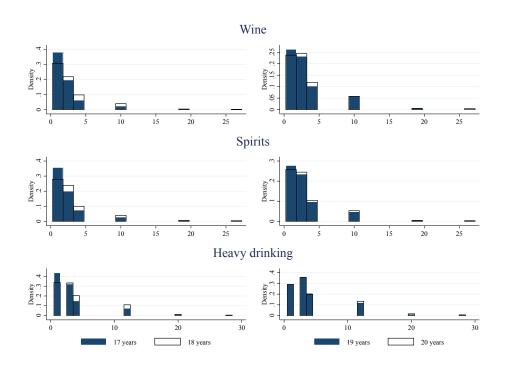


Fig. E.2: Density plot of frequency of alcohol consumption 12 months before and after the MLDA cut-offs (wine, spirits and heavy drinking)

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.

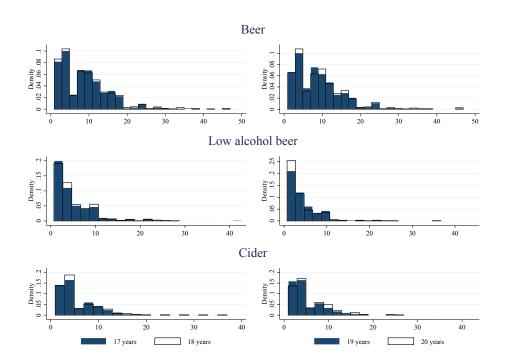


Fig. E.3: Density plot of intensity of alcohol consumption 12 months before and after the MLDA cut-off (Beer, low alcohol beer and cider)

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.

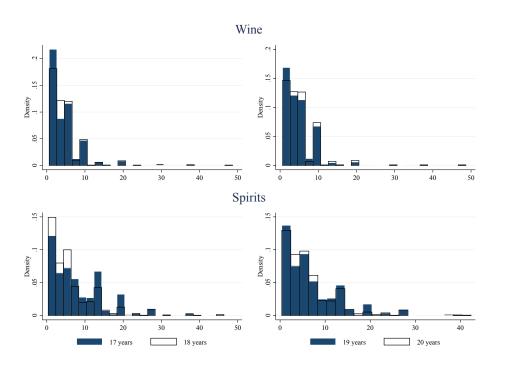


Fig. E.4: Density plot of intensity of alcohol consumption 12 months before and after the MLDA cut-off (wine and spirits)

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		results by	Females	
	(1)	(2)	(3)	(4)	(5)	(6)
Drank alcohol in last 30		()	(-)	()	(-)	(-)
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 e	pisode in la	st 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 recon		ekly amount				
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 alcoho						
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 alco						
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 of		_				
MLDA 18	0.479	0.592	0.653	2.081*	0.174	2.634**
D 1 111	(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
D 1 : 141	(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	,	9	\checkmark	,	0	√
IN Optimal bandwidth	\checkmark	2x	\checkmark	\checkmark	2x	\checkmark

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

	11100110	Males	<u> </u>	results by	Females	
	(1)	(2)	(3)	(4)	(5)	(6)
Frequency of beer const	umption in	last 30 da	ys			
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 alcoho	ol beer con	sumption i	n last 30 a	lays		
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 cons	umption in	ı last 30 da	ys			
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 cons	$\overline{sumption}$ i	n last 30 de	\overline{ays}			
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 con	$\overline{nsumption}$	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 driv	iking episa	des 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	\checkmark	2x	\checkmark	\checkmark	2x	\checkmark

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: $^*p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table F.3: Alcohol intensity LLR results by gender

Table F.5	o. Alcone	or intensity	у ппите	suits by a		
		Males			Females	
	(1)	(2)	(3)	(4)	(5)	(6)
Average intensity of bee						
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 lov	v alcohol b	eer consum	ption in le	ast 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 wir	ne consum	ption in las	t 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 cia	ler consum	nption in la	st 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 spi	irits consu	mption in l	ast 30 day	S		
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	\checkmark	2x	✓	\checkmark	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 < 0.05, p < 0.01.

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

Table G.1: Alcohol	participa	Baseline	CBG105 W101		birthday mo	
	(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 en	pisode in la	st 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 recom	mended we	ekly amount	in last 30 de	ays		
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 alcoho	$\overline{l\ consumed}$	in last 30 de	ıys			
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 alcoh	hol in last 3	0 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 of	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	\checkmark	2x	\checkmark	\checkmark	2x	\checkmark

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.

Table G.2: Alcohol frequency LLR results with and without birthday month

		Baseline		No i	birthday mo	onth
	(1)	(2)	(3)	(4)	(5)	(6)
Frequency of beer consu						
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 alcoho		sumption i		lays		
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 cons	umption in	n last 30 de	iys			
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 cons	umption i		ays			
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 con	a sumption	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 drin	king episc	odes 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			✓			√

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: $^*p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table G.3: Alcohol intensity LLR results with and without birthday month

Table G.s. Alcohol		Baseline			birthday mo	
	(1)	(2)	(3)	(4)	(5)	(6)
Average intensity of bed			0 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 lov	v alcohol bee	er consumpt	ion 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 wir	ne consumpt	tion in last 3	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 cid	ler consump	tion 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 spi			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.

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	\checkmark	2x	\checkmark	\checkmark	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 ac						
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	- /		<u>√</u>			<u>√</u>
IK optimal bandwidth	\checkmark	2x	√	\checkmark	2x	√
optimica bandwidth	•	-22	•	•	-22	•

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

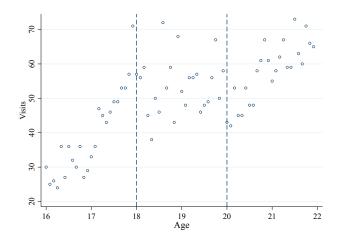


Fig. I.1: Age profile of hospital admissions due to mental disorders (Survey sample) Notes: The scatter points are monthly age blocks of count of hospital admissions for mental disorders (ICD grouping F). Source: Patient administrative data, merged to Swedish survey of household living standards (ULF). Own calculations.

J Sensitivity analysis - Hospital admissions

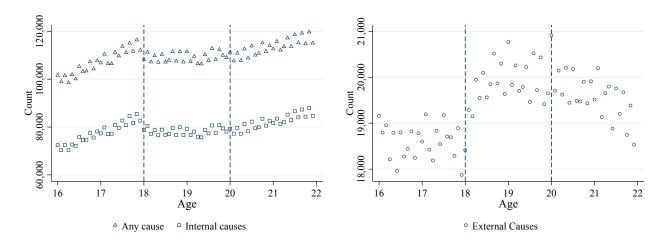


Fig. J.1: The effects of MLDA on hospital admissions (excluding counties with OOP costs at 18th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

 $Source\colon \textsc{Patient}$ administrative data. Own calculations.

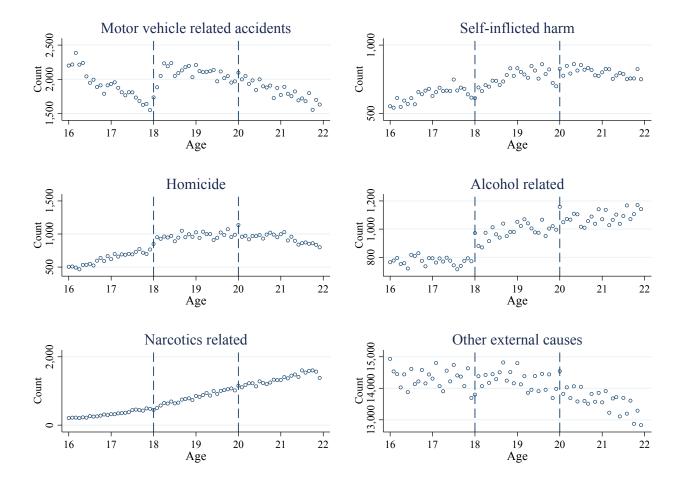


Fig. J.2: The effects of MLDA on hospital admissions due to external causes (excluding counties with OOP costs at 18th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

Source: Patient administrative data. Own calculations.

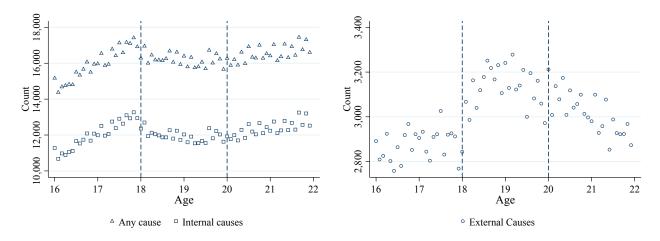


Fig. J.3: The effects of MLDA on hospital admissions (excluding counties with OOP costs at 20th birthday)

Notes: The scatter points are monthly age blocks of count of hospital admissions for the years 1969-2015.

Source: Patient administrative data. Own calculations.

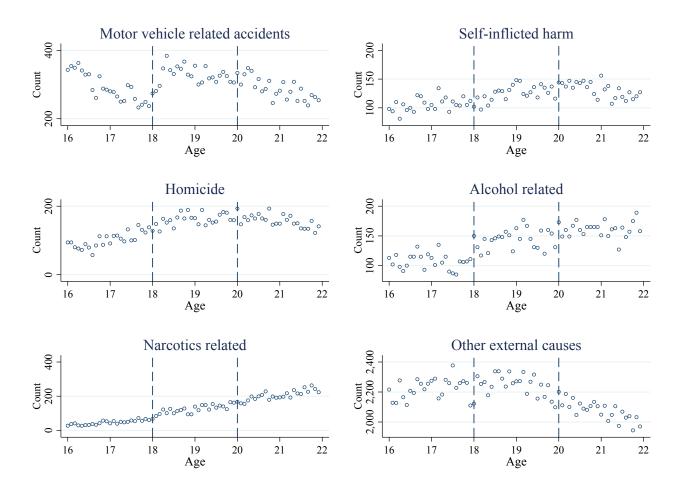


Fig. J.4: The effects of MLDA on hospital admissions due to external causes (excluding counties with OOP costs at 20th birthday)

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

arresnoid	No OOP at 18			No OOP at 20			
	(1)	(2)	(3)	(4)	(5)	(6)	
Motor vehicle related ac	ccidents						
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	√ √	\checkmark	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	\checkmark	2x	\checkmark	\checkmark	2x	\checkmark	

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 as	ccidents						
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			<u>√</u>			<u>√</u>	
IK optimal bandwidth	\checkmark	2x	√	\checkmark	2x	√	
pulled build with	•		•	<u> </u>		-	

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.