

# Breaking the Link Between Legal Access to Alcohol and Motor Vehicle Accidents: Evidence from New South Wales\*

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## Abstract

A large literature has documented significant public health benefits associated with the minimum legal drinking age in the United States, particularly because of the resulting effects on motor vehicle accidents. These benefits form the primary basis for continued efforts to restrict youth access to alcohol. It is important to keep in mind, though, that policymakers have a wide variety of alcohol-control options available to them, and understanding how these policies may complement or substitute for one another can improve policy making moving forward. Towards this end, we propose that investigating the causal effects of the minimum legal drinking age in New South Wales, Australia provides a particularly informative case study, because Australian states are among the world leaders in their efforts against drunk driving. Using an age-based regression-discontinuity design applied to restricted-use data from several sources, we find no evidence that legal access to alcohol has effects on motor vehicle accidents of any type in New South Wales, despite having large effects on drinking and on hospitalizations due to alcohol abuse.

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# 1 Introduction

The minimum legal drinking age (MLDA) has long been a subject of heated debate. Many U.S. states had a MLDA of 18 until the 1984 National Minimum Drinking Age Act threatened to take highway funding from states with MLDAs below 21. Though all U.S. states have since maintained a MLDA of 21, critics argue that this MLDA encourages more dangerous drinking among 18-20 year olds than would occur in a legal environment, that it is inconsistent with all of the other responsibilities bestowed upon 18 year olds, and that the volume and severity of alcohol-related health problems among youths suggests that the current policy is ineffective.<sup>1</sup> However, several studies have demonstrated that the current MLDA in the United States *does* have substantial public health benefits. In particular, Carpenter and Dobkin (2009, 2010, 2011) and Deza (2013) leverage a regression-discontinuity (RD) design to document the effects of legal access to alcohol, finding significant effects on alcohol consumption, binge drinking, crime, morbidity, and mortality.<sup>2</sup> While these studies indicate that the health benefits associated with the MLDA in the United States are broad, they also indicate that the reduction in motor vehicle accidents (MVs) is the most important component of these benefits. This also appears to be the case for Canada where the MLDA reduces youth mortality 6%, almost entirely due to its effects on motor vehicle accidents (Carpenter et al. 2013). With all of this said, in order to properly craft policy, we need to weigh the costs and benefits of the wide variety of alcohol-control options that are available and to determine how the effects of these policies may complement or substitute for one another. Towards this end, we propose that the MLDA in New South Wales, Australia (NSW) provides a particularly informative case study because Australian states are among the world leaders in their efforts against drunk driving: they pioneered the use of random breath tests and continue to use them extensively (today NSW drivers are stopped in a random

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<sup>1</sup>Though these sentiments have led to proposals to reduce the MLDA in several states, none have been adopted at this point.

<sup>2</sup>In similar studies, Crost and Guerrero (2012) and Deza (2013) find evidence that the additional consumption of alcohol has a displacement effect on the use marijuana and the use of harder drugs, respectively, whereas Crost and Rees (2013) find no evidence of effects on marijuana use. Also focusing on the United States, Carell et al. (2011) and Lindo et al. (2013) also find significant effects of the current MLDA on student achievement. Turning to the New Zealand context, Conover and Scrimgeour (2013) and Boes and Stillman (2013) find significant effects on alcohol-related hospitalizations though Boes and Stillman (2013) find no evidence of effects on alcohol consumption using survey data and no evidence of effects on motor vehicle accidents. There are also several studies that have exploited the state-level changes in that took place in the 1970s and 1980s in the United States, including Birckmayer and Hemenway (1999), Dee (1999), Wagenaar and Toomey (2002), Lovenheim and Slemrod (2010), Carpenter and Dobkin (2011). They find significant effects on suicides and motor vehicle accident fatalities, though Crost (2013) presents evidence that the effect on motor vehicle accidents dissipates as individuals become older. Fertig and Watson (2009) and Barreca and Page (2013) show that there are also effects on infant health outcomes.

breath test once a year on average, whereas 11 US states have prohibited these stops); they have lower blood alcohol concentration (BAC) limits for drivers; and their penalties for drunk driving are substantial.<sup>3</sup>

Two additional institutional features make NSW a particularly informative environment for considering the effects of minimum legal drinking ages. First, in contrast to the United States where BAC limits for drivers jump from zero to 0.08 at the MLDA, the BAC in limit for drivers remains at zero through the MLDA threshold in NSW.<sup>4</sup> As such, NSW arguably offers a cleaner natural experiment.<sup>5</sup> Second, and also in contrast to the United States, the MLDA in Australia is 18. Given that we tend to expect youths to become more responsible between the ages of 18 and 21, we might expect legal access to alcohol to have more severe effects at age 18. Moreover, this difference in laws might lead to different effects because 18 year olds are more likely to live at home and thus under the watchful eyes of their parents.<sup>6</sup>

This study uses a RD design that exploits the sharp change in legality at age 18 applied to data from several sources to provide a comprehensive analysis of the health effects of the MLDA in NSW. To estimate the effects on drinking behaviors, we use restricted-use data (including exact dates of birth and survey dates) from the 2001 to 2011 waves of the Household, Income, and Labour Dynamics in Australia (HILDA) Survey. To estimate the effects on per driver accident rates, we use data from 2000 to 2010 on individuals licensing dates and on the universe of MVAs involving an injury or a vehicle being towed, provided by NSW Roads and Maritime Services. To estimate the effects on especially severe MVAs that lead to hospitalization in addition to hospital admissions due to alcohol intoxication/poisoning, assault, and other injuries, we use restricted-use hospital admissions data from the National Hospital Morbidity Database (NMMD), which contains the universe of inpatient episodes from 2001 to 2010.<sup>7</sup>

This analysis reveals that drinking behavior increases substantially when NSW youths obtain

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<sup>3</sup>While not focusing on alcohol-related accidents, DeAngelo and Hansen (forthcoming) demonstrate that police enforcement has significant effects on overall traffic fatalities by exploiting variation generated by a mass layoff in Oregon. Additionally, Hansen (2013) shows that punishment for drunk driving has significant effects on recidivism using a BAC-based RD design.

<sup>4</sup>For unrestricted license holders, the BAC limit is 0.05. Drivers must be at least 20 years of age to obtain an unrestricted license.

<sup>5</sup>The evidence on the effects of zero-tolerance drunk driving laws (i.e. very low BAC limits, typically 0.01 or 0.02) on motor vehicle accident fatalities is mixed. In a review study, Zwerling and Jones (1999) highlight that all six previously published studies indicate these policies are effective (while acknowledging that three of the six yielded rather imprecise estimates). More recently, Dee and Evans (2001) and Carpenter (2006) find that these laws are not effective while Eisenberg (2003) and Voas et al. (2003) provide evidence that they are effective.

<sup>6</sup>It is not clear whether this feature of the context is likely to lead to larger or smaller effects. On one hand, it may lead to greater compliance with the MLDA and thus a larger effect on drinking-related behaviors. On the other hand, it may lead youths to drink more responsibly upon gaining legal access.

<sup>7</sup>The population of NSW is not large enough for a meaningful analysis of mortality using similar years of data.

legal access to alcohol. However, there is no corresponding increase in the probability of being in an accident involving an injury or a vehicle being towed, or of being injured in an accident, or of being in an accident with a high risk of death. These results are in stark contrast to what we know about the effects of the current U.S. MLDA on MVAs. Although we cannot determine precisely why these results diverge from what is observed in the United States, the fact that NSW has such strong policies to combat drunk driving seems like a likely candidate and highlights the importance of further research in this area. Our analysis of hospital admissions further corroborates our findings for MVAs, while also revealing that legal access to alcohol increases hospitalizations due to alcohol abuse and assault. Overall, this set of results indicates that legal access to alcohol need not result in additional MVAs but that a broad consideration of its public health costs should inform policy.

The remainder of this paper is structured as follows. In the next section, we provide background information on NSW to put our analysis into context. We then describe our empirical strategy and data. Next, we present our analysis of drinking behavior, MVAs, and hospital admissions before discussing our results and providing some concluding remarks.

## 2 NSW Institutions and Culture

The anti-drunk-driving policy in NSW has a long and rich history. Historically, it has been at the forefront of a number of policy initiatives designed to reduce the cost of alcohol-related MVAs, including statutory BAC limits, random breath testing, and graduated licensing systems with “zero tolerance” BAC limits for inexperienced drivers. While none of these policies are unique to NSW, their simultaneous use and scope distinguishes the state’s regulatory regime from that of most other jurisdictions around the world. Given the number and scale of anti-drunk-driving measures implemented in NSW beginning in the early 1980s, it is perhaps not surprising that it has experienced a dramatic decline in the fatality rate in road crashes (Figure 1A) and in alcohol related road crashes (Figure 1B). Moreover, while NSW had higher rates of MVA-related fatalities per person than the United States in the 1970s, the NSW rate is currently about half that of the United States; similarly, NSW has a fatality rate for alcohol-related crashes that is about half that of the United States. Nevertheless, alcohol remains a major contributing factor in MVA fatalities for NSW. In recent years, around 20% of MVA fatalities were in alcohol related crashes in NSW (Figure 1C). This section provides a brief

history and description of the anti-drunk-driving measures in NSW in a comparative context.

The history of anti-drunk-driving laws starts in early twentieth century. The state of New York was one of the first jurisdictions worldwide to outlaw drunk driving in 1910, with the United Kingdom following in 1925. In the 1930s various countries started to introduce laws imposing a limit on drivers' maximum allowable blood alcohol concentration (BAC). Particularly notable were the so-called *per se* laws pioneered by Norway and Sweden in the 1940s, under which drivers with a BAC level exceeding the legal limit are automatically deemed unfit for driving and typically would have drunk-driving charges brought against them. Scandinavian countries were also among the first to apply relatively low BAC limits of 0.05 and 0.08 (the BAC limit recommended by the American Medical Association at the time was 0.15). Following the lead of Norway and Sweden in the 1940s, Victoria introduced a statutory BAC limit of 0.05 in 1966. Within a short period of time, other Australian states adopted similar policies, with legal BAC limits set at 0.08 in Tasmania, NSW and South Australia and 0.10 in Queensland. These limits initially applied to all drivers, but stricter BAC limits for probationary drivers were introduced in the mid-1980s. This early adoption of the so-called "Scandinavian model" of *per se* anti-drunk-driving laws with low BAC limits put Australian states (along with the United Kingdom, Canada, New Zealand, and France) among the global leaders in this policy area.

Random breath testing (RBT) was introduced in Australia in Victoria in 1976, with NSW following with its own RBT trial in 1982. Only a few other jurisdictions (notably France and Norway) were experimenting with similar measures at that time. The distinguishing characteristics of the NSW policy were its scale and the publicity campaign that was conducted by the government around the time of its introduction. In the first year, NSW police conducted nearly one million random breath tests, or approximately one test per three licensed drivers. Homel (1988) points out that while the penalties for drunk driving in NSW in that period were reasonably harsh, they were not fully enforced for institutional and cultural reasons. Thus, the implicit policy goal behind the high level of intensity of the policy and the surrounding publicity campaign was to influence the perception of the likelihood of being caught while drunk driving, as well as to create a negative image of drunk drivers.

Survey data collected following the introduction of RBT in 1982 suggested that the policy had an effect on drinking behavior by forcing drinkers to be more aware of the amount of alcohol they have consumed. Moreover, some commentators credited the policy with the 36% reduction

in alcohol-related road fatalities over the period 1981–1986 (see Figure 1A) though other factors may have contributed to this sharp decline.<sup>8</sup> In any case, surveys indicate that public support for the policy grew to 90% by the end of 1980s and its use has only intensified since. By the late 1990s, NSW police were conducting between 1.8 and 2.3 million random tests per year, with about 0.5% of them leading to drunk-driving charges (RTA, 1997-1999). At present, about 4.5 million RBTs are conducted annually, or approximately one per licensed driver. This is substantially higher than in other jurisdictions with similar policies such as Finland, where one in three drivers is tested each year, and Sweden and France, where every fifth driver is tested (Laurell, 2004). The heavy use of RBT in NSW also contrasts sharply with the situation in the United States, where 11 states prohibit sobriety checkpoints altogether and those that allow them impose additional restrictions, such as mandatory warnings to the public about the police plans.

Another important component of the NSW regulatory regime is the graduated licensing system (GLS) which imposes various restrictions on novice drivers. The concept of GLS was developed by the U.S. National Highway Traffic Safety Administration and was first put in use in the states of New York and Maryland in the 1970s. Such systems typically envision a multi-stage process in which restrictions on drivers are gradually relaxed as they gain more experience and maintain a clean driving record. The first country to adopt a modern full scale GLS in 1987 was New Zealand (Langley, Wagenaar & Begg, 1996). The majority of other jurisdictions, including most of the United States and the Canadian states, implemented similar systems at the end of the 1990s.

The current NSW graduated licensing system was adopted in 2000. It has a three-stage structure, starting with the learner’s license that is available at age 16, followed by a first provisional license that is available at the age of 17, followed by a second provisional license available one year after holding a first provisional license, then an unrestricted license that is available after two years of holding a second provisional license. As such, youths can only obtain an unrestricted license in NSW turning 20 years old. In contrast, the intermediate stage lasts for 18 months in New Zealand and for two years in Northern Ireland and Norway, while in many U.S. states the probationary period is limited to one year or less. Another distinguishing feature of the NSW graduated licensing system is its tough stance on speed restrictions for drivers with

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<sup>8</sup>Notably, the reduction of the statutory BAC limit from 0.08 to 0.05 in December 1980 may have played an important role as well as the economic downturn experienced during this period.

learners' and provisional licenses, requiring them to drive at speeds below the nominal speed limits. In particular, learners cannot exceed the speed of 80 km/h; the first-stage provisional license allows for speeds of up to 90 km/h; and the second stage license has a limit of 100 km/h. These limits are stricter than in other jurisdictions with similar provisions in their GLS, including Canada and some European countries.

Finally, one feature of the NSW GLS that is especially relevant for this study is the BAC limit for both learners and drivers with provisional licenses: it was set at 0.02 initially and reduced to zero in 2004.<sup>9</sup> As a result, all drivers in NSW face a zero BAC limit for at least two years after they gain legal access to alcohol at age 18. This feature of the NSW system allows us to estimate the effect of MLDA using an age-based RD design without concerns that the estimates will be confounded by a simultaneous change in the BAC limit.

### 3 Methods and Data

#### 3.1 Empirical Strategy

Our empirical strategy exploits the sharp change in legal access to alcohol that occurs at the MLDA in a RD design to identify the causal effects of youths gaining legal access to alcohol. This strategy is motivated by the idea that characteristics related to behaviors and outcomes of interest are likely to vary smoothly through the MLDA threshold; thus, any discontinuity in behaviors or outcomes at this threshold can be reasonably attributed to the change in legal access. We operationalize this identification strategy by estimating

$$y_i = \beta_0 + \beta_1 MLDA + f(age_i) + \epsilon_i, \quad (1)$$

where  $y_i$  is a measure of drinking behavior or a health outcome for individual  $i$ ,  $f$  is some smooth function of age,  $MLDA$  is a binary indicator for whether the individual's age is 18+,  $\epsilon_i$  is a random error term, and  $\beta_1$  is the effect of legal access to alcohol. Following Imbens and Lemieux (2008), we adopt an interacted local linear specification for  $f(\cdot)$ , allowing the slope term to be flexible on each side of the MLDA threshold. We show estimates for a range of bandwidths while highlighting those estimates that use the Imbens and Kalyanaraman (2011)

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<sup>9</sup>Analyses focusing on the pre-2004 regime are imprecise but otherwise similar to our main results, offering no indication that there were any harmful effects of legal access to alcohol on MVAs in the pre-2004 regime. These results are available upon request.

(IK) optimal bandwidth. Following Lee and Card (2008), we present standard error estimates that are clustered on the running variable (age in days).

With any RD design, the identifying assumption is that characteristics related to outcomes of interest vary smoothly through the treatment threshold. The fact that our application uses age as the running variable (that is, the variable that determines treatment) helps to address concerns that this assumption may not hold. Because individuals have no control over their age, manipulation of the running variable (McCrary 2008) is not possible. That said, with an age-based RD design, it is important to consider whether there might be “additional treatments” related to outcomes that are coincident with the treatment of interest. Birthdays are one obvious example. In particular, outcomes may change around the time an individual’s age (in years) changes because of activities and behaviors related to celebrating one’s birthday. This particular confounder is typically addressed in the literature by estimating a “donut RD” in which the analysis omits observations that are in the immediate vicinity of the treatment threshold. The assumption underlying this approach is that the conditional expectation function estimated by the data outside the donut but within the chosen bandwidth correctly captures the expected outcomes that would be observed at the treatment threshold in the absence of behavioral changes associated with having a birthday. In short, this assumption implies that “birthday effects” do not exist outside of the donut and that the conditional expectation function is specified correctly. RD-based studies of the MLDA have typically excluded the birthday itself and the subsequent day after, or have included dummy variables for those days (e.g., Carpenter and Dobkin 2009, 2010, 2011). That said, a larger “donut” window may be appropriate. For instance, if a birthday falls on a weekday, it may conceivably induce celebratory behavior on the following weekend, perhaps also reducing similar behavior beforehand in anticipation of such celebrations. For this reason, we consider the sensitivity of the estimates to a range of alternative donut sizes.<sup>10</sup>

One additional issue that we must consider in our setting is that the MLDA in Australia corresponds to the age at which youths are no longer considered minors. Thus, we might be concerned that our estimates of the effect of legal access to alcohol could be confounded by “coming of age effects.” While we cannot rule out the existence of such effects, which might be related to the outcomes we consider, we find no evidence that the proportion of individuals living at home, being in school, or being employed changes discontinuously across the treatment threshold. Nor do we find any evidence that demographic characteristics, which could change

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<sup>10</sup>We apply the same donut exclusions around other birthdays as well.



as a result of coming-of-age-induced migration, are discontinuous across the threshold. Given these results, perhaps our main outstanding source of concern is that youths may be afforded more freedom from their parents after turning 18. For example, they may be able to go out more often and/or to stay out later at night. We note that this sort of phenomena, which seems plausible, would serve to bias our estimated effects on “negative outcomes” upwards (because youths are likely to engage in risky behaviors more often with less parental supervision) and thus imply that our estimates might be thought of as upper bounds. Because we find no evidence of any discontinuity in MVAs at age 18, we can then be especially confident in concluding that legal access to alcohol does not increase the incidence of MVAs among NSW’s youths.

These are the primary issues associated with estimating the effects of legal access to alcohol in the Australian context. That said, some additional issues are relevant to the outcomes that we consider and to the data that we use. We discuss these issues in greater detail below.

### 3.2 Data on Drinking Behavior

The source of the data on drinking behavior in NSW is the the Household, Income and Labour Dynamics Australia (HILDA) survey. HILDA is a nationally representative household panel survey, with data collected annually since 2001 for an initial sample of 19,914 persons in 7,682 households. We use an unconfidentialized version of the dataset, which includes respondents’ dates of birth and dates of interview, allowing for identification of exact age in days at the time each survey was completed. In each of the 11 currently available waves, all persons aged 15 and over within sampled households were asked to respond to the questionnaire.<sup>11</sup> Appendix Figure A1 shows the number of observations by age in 2-month bins, which appears to vary smoothly through the threshold.

In each wave, respondents were asked “Do you drink alcohol?” and asked to choose responses from which the frequency of drinking can be inferred.<sup>12</sup> In three waves (2007, 2009, and 2011), the respondents also were asked a gender-specific question about frequency of binge drinking.<sup>13</sup>

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<sup>11</sup>Parental consent was sought before interviewing persons aged under 18 years and living with their parents. Persons for whom parental consent was denied are not identifiable. However, the refusal rate (for any reason) does not vary through the threshold (9% for 16 year olds, 9% for 17 year olds, 9% for 18 year olds and 10% for 19 year olds).

<sup>12</sup>The response options are: No, I have never drunk alcohol; No, I no longer drink alcohol; Yes, I drink alcohol every day; Yes, I drink alcohol 5 or 6 days per week; Yes, I drink alcohol 3 or 4 days per week; Yes, I drink alcohol 1 or 2 days per week; Yes, I drink alcohol 2 or 3 days per month; Yes, but only rarely.

<sup>13</sup>Males (Female) were asked ‘How often do you have 7 (5) or more standard drinks on one occasion?’ Response options were: Not in the last year; Less than monthly but at least once a year; Once a month; 2 to 3 times a month; 1 to 2 times a week; 3 to 4 times a week; 5 or more times a week.

Using these data items, we construct five drinking behavior outcomes. Three of these are binary indicators for whether the individual (i) drinks at all; (ii) drinks at least once per week; or (iii) binge drinks at least once per week. The others indicate the implied proportions of days with (iv) any drinking and (v) binge drinking.<sup>14</sup>

In terms of our estimation strategy, the case for a larger “donut” for our RD approach is arguably stronger when we analyze drinking behavior using HILDA data, because the questions on drinking behavior do not specify an explicit reference period. The cognitive process of choosing a response to such a question is not clear. Consider a person who drinks more frequently after turning 18: it is not clear precisely when this person would update his or her perception of own-drinking frequency. The cognitive process of choosing “1 or 2 days per week” for example, as compared to the next response option of “2 or 3 days per month” is unclear. There may be a lag between an actual change in underlying drinking propensity and this self-evaluation. Conversely, this judgment may be overly influenced by recent celebratory behavior, which would be particularly problematic for observations just above the threshold.<sup>15</sup> For this reason, we focus on estimates that are based on a one-month donut (i.e., we exclude observations where the survey date is within 30 days of eighteenth birthday) when analyzing these data. In our other analyses, we instead focus on estimates that are based on a seven-day donut. In Appendix D, we also present estimates for a number of alternate donut sizes for each indicator. These results show that the size of the donut has little influence on the estimates.

Table 1 shows descriptive statistics from HILDA. The sample consists of 2,359 observations of persons living in NSW who were aged 16-19 at the time of data collection and not within 30 days of any birthday. This corresponds to the widest bandwidth we consider and the preferred donut width. The sample includes 711 observations from the waves that include binge drinking questions.

Since each individual in HILDA contributes up to 4 observations to a given regression (up to 2 observations for the binge drinking outcomes), we tested sensitivity of the results to clustering standard errors on the individual instead of on age. In those results (available upon request), the

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<sup>14</sup>The proportion of days drinking is set to: zero for those who do not drink or only drink ‘rarely’; 1 for those who drink alcohol every day; 5.5/7 for those who drink 5 or 6 days per week; 3.5/7 for those who drink 3 or 4 days per week; 1.5/7 for those who drink 1 or 2 days per week; 2.5/30 for those who drink 2 or 3 days per month. The proportion of days binge drinking variable was constructed similarly.

<sup>15</sup>In addition, in the first 8 waves of HILDA data, the exact date of the “self-completion questionnaire,” which contains the drinking questions, was not provided. For those years, we instead rely on the date of the interviewer-administered “person questionnaire.” We do not see this as a major issue. For those waves where we have exact dates of both questionnaires, these differ by less than 2 weeks in 91% of cases. Nevertheless, this measurement error may lead to attenuation, which is reduced if the dates immediately around 18th birthday are excluded.

standard errors are very similar to that of the main analysis, slightly larger in some instances, slightly smaller in others.

### 3.3 Driver and Motor Vehicle Accident Data

Our main results—focusing on MVAs—draw on CrashLink data and licensing data provided by the NSW Centre for Road Safety. The crash data include one record for each driver who was involved in each motor vehicle accident occurring on NSW roads in which at least one vehicle was towed away or one of the occupants was injured. These data provide the date of the accident and date of birth of the driver, and they are linked to additional information contained in the drivers’ license database. We analyze crash rates for drivers who first obtained a provisional license between their seventeenth and eighteenth birthdays, between July 2000 and December 2010. The analysis is conducted on a data set that has been collapsed to the day-of-age level where the outcome variables are rates of various types of accidents.<sup>16</sup>

License class progression presents a complication for the analysis. Since July 2000, NSW has had a graduated license scheme: individuals can apply to progress to a second provisional (P2) license after 12 months of holding a P1 license. This results in a small discontinuity in license class at the MLDA threshold (Figure A3). However, P1 and P2 licenses are quite similar—the major difference is the speed limit, which is capped at 100 km/h for P2 drivers compared with 90 km/h for P1 drivers.<sup>17</sup> More importantly, license progression is likely to increase MVAs if it has any effect at all, again leading us to believe that our estimates provide an upper bound for the true effect of legal access to alcohol on MVAs. And given that these estimates tend to be close to zero, we can be especially confident in inferring that—at least in our setting—legal access to alcohol does not increase MVAs in our setting.

We present the results for all MVAs, as well as for a range of accident types that are likely to be more serious or more suggestive of drunk driving (e.g., those in which the driver is injured, those that occur at night). While we can (and do) show estimates for motor vehicle accidents in which the driver died, in our setting fatal accidents are too infrequent to yield informative estimates. Therefore, our preferred strategy for identifying serious crashes is to weight them by

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<sup>16</sup>The denominator consists of counts of relevant drivers from the drivers’ license database and the numerator is a count of relevant crashes from the Crashlink database. Relevant drivers are those who first obtained a provisional license between their seventeenth and eighteenth birthdays and after June 2000. Each individual is considered a “driver” on all dates after obtaining a first license, through to the end of our crash data coverage (2010). Relevant crashes are those matched to relevant driver-days.

<sup>17</sup>From July 1, 2007, P1 drivers (but not P2 drivers) under the age of 25 are not permitted to drive a vehicle with more than one passenger under 21 years old between 11pm and 5am.

predicted fatality-risk. Three factors motivate this approach:

- (i) Our analysis of fatal MVAs does not have sufficient statistical power to be meaningful.
- (ii) There is a large element of chance in whether a given accident is fatal.
- (iii) Our data contain a large set of detailed crash characteristics with strong predictive power for fatalities.

This approach is a natural extension of selecting subsets of “serious” crashes based on a certain criterion (such as head-on crashes; crashes with an injury; night crashes; crashes occurring at high speed) that improves power. To operationalize this approach, we estimate fatality risk based on the predicted probability from a probit model in which fatality is a function of a rich set of driver and crash characteristics.<sup>18</sup> In order to estimate these probabilities with greater precision, we expand the sample to include all 17-21-year-old drivers involved in an accident. In addition to presenting estimates that focus on fatality-risk-weighted accidents, we show estimates for specifications focusing on accidents with a fatality risk that exceeds specific thresholds. Appendix B shows results from the fatality risk model. It shows the distribution of predicted fatality risk, and compares the observed characteristics of fatality-risk-weighted crashes to that of fatal crashes and to all (unweighted) crashes.

Table 1 shows descriptive statistics from the licensing and crash data, for relevant driver-days as described above where the driver is aged 17 or 18 and not within 7 days of any birthday.

### 3.4 Hospital inpatient data

To study morbidity, we draw on administrative data from the National Hospital Morbidity Database (NHMD), administered by the Australian Institute of Health and Welfare. The data contain the universe of inpatient episodes for admissions to NSW hospitals between July 2001 and June 2010. The file includes patients’ exact age in days at admission. It also contains ICD-10-AM clinical data, including principal diagnosis and a detailed classification of external causes.

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<sup>18</sup>These include driver’s gender and seatbelt usage, urbanization of crash location (6 indicators), time of crash (2 hour intervals = 12 indicators), indicators of estimated speed travelled (in 10 km/h intervals), ‘Road User Movement’ (RUM Code) (86 indicators), vehicle’s maneuver immediately prior to its involvement in the crash (46 indicators), vehicle’s role in the first impact (3 indicators), driver error factors (23 indicators) and ‘unusual’ factors (7 indicators). RUM code describes the first impact that occurred during the crash. Two examples of RUM codes are ‘Head on’ and ‘Reversing into object or parked vehicle’. Full details are in (provide reference). Two examples of maneuvers are ‘Cutting back after overtaking’ and ‘Stationary in Traffic’. An example of a driver error factor is ‘Controller turning right from wrong lane’. An example of an unusual factor is ‘This vehicle skidding, sliding or aquaplaning’.

We consider the following categories of acute care hospital episodes:

- Alcohol intoxication or poisoning. Episodes with a primary diagnosis of a “Mental and behavioral disorder due to use of alcohol” (ICD10 code F10) or “Toxic effect of alcohol” (ICD10 code T51).<sup>19</sup>
- Assaults. Episodes where assault is listed as an external cause of morbidity (ICD codes X85-Y09)
- Drivers injured in Motor Vehicle Accidents (excluding motor cycle riders) (ICD codes V30-V79; V86, with fourth character “0” or “5”)
- Motor cycle riders injured in transport accidents (ICD Codes V20-V29). These are distinguished from regular MVAs for two reasons, both related to licensing. First, approximately half of these episodes are for “non-traffic” accidents, i.e., accidents which did not occur on public roads. There is no requirement to hold a license to ride on private property and no risk of being caught riding under the influence of alcohol on private property. Second, until 2010, motor cycle riders could be eligible for unrestricted licenses from the age of eighteen. The transition to an unrestricted license includes a higher blood alcohol limit (0.05), representing a possible confounding factor in the relationship between the MLDA and the risk of accidents.
- Other external causes. All episodes with any other listed external cause (ICD codes V01-Y98). Most are accidents, self-harm, or events of undetermined intent.

Descriptive statistics from NHMD are shown in Table 1.

Like our analysis of MVAs, our analysis of hospital admissions is performed on data aggregated to the day-of-age level. However, in the case of hospital admissions, the relevant set of individuals is the entire population (not just drivers). As such, we rescale the discontinuity estimates to represent the number of additional episodes per 10,000 person-years that are attributable to legal access. Specifically, we multiply the counts of episodes for each day of age by  $365 \times 10,000$ , then divide by the estimated resident population of 18-year olds in NSW at June 2005 (ABS, 2013), and divide again by 9 (the number of years of hospital data).

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<sup>19</sup>About 84% of F10 episodes are for “acute intoxication.” A further 10% are due to a “dependency syndrome.”

## 4 Establishing the Validity of the Research Design

In this section, we consider the extent to which the potential confounders discussed in the previous sections are empirically relevant to our analyses. We begin by considering population characteristics using HILDA and then consider driver characteristics using our driver data.

As we alluded to above, most determinants (other than the MLDA) of health outcomes would seem likely to vary smoothly through the 18th birthday threshold. However, 18 is also the official age of adulthood in Australia in relation to voting, gambling, smoking, and culturally in relation to “coming of age,” broadly speaking. While we cannot rule out the possibility that these changes might have independent effects on health outcomes, we can investigate the extent to which turning 18 has other major changes on youths’ daily lives by looking at whether they live at home, whether they are enrolled in secondary school, and whether they are employed using HILDA data. In Figure 2, we show means (in two-month bins) for these characteristics in Panel A and estimated discontinuities across a range of bandwidths in Panel B.<sup>20</sup> The means reflect the changes we expect to see: as youths grow older they are less likely to live with their parents, less likely to be enrolled in school, and more likely to be employed. However, these major life changes events do not appear to be precipitated by turning 18, as these characteristics do not change discontinuously, which lends confidence to our research design.

While the results for “Enrolled in School” show no discontinuity at 18, the age pattern is worthy of discussion. Children in NSW typically begin school in late January or early February prior to their 6th birthday. Because most youths complete 13 years of schooling without repeating any grade, most finish school between the ages of approximately 17 years and 10 months and 18 years and 10 months. Thus, the kinks at these points in the means plot are to be expected. The fact there are no corresponding kinks in health outcomes (shown in the figures below) indicates that high school completion does not affect the health outcomes we consider.<sup>21</sup> Thus, the existence of these particular kinks does not pose a threat to the validity of our research design.

We have also confirmed that gender, being born in Australia, and being a native English speaker are smooth through the threshold (see Figure A2 in Appendix A).<sup>22</sup>

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<sup>20</sup>The discontinuity estimates in this figure are based on the one-month-donut RD approach described above in order to be consistent with our analysis of drinking behaviors which also uses HILDA data. Estimates that instead use a one-week donut are similar.

<sup>21</sup>See Card et al. (2012) for a formal discussion of causal inference based on kinks driven by pre-determined variables.

<sup>22</sup>These findings are not trivial since turning 18 could conceivably affect migration in a way that changes the

In Appendix Figure A3 we explore the extent to which driver characteristics might change through the treatment threshold (while presenting means in one-day bins, which can be estimated with great precision using these data). A priori, there is little reason to believe that most driver characteristics would change discontinuously at age 18 unless turning 18 has an effect on the probability that individuals hold licenses and that this effect is heterogeneous across individual characteristics. That said, license class is one characteristic that we do expect to be discontinuous at the threshold because youths who got their first provisional (P1) license on their 17th birthday become eligible for a second provisional (P2) license at age 18. The data confirm this intuition as the fraction of drivers with a P2 license jumps from zero to approximately 2% across the threshold. As discussed above, if anything, this imbalance is likely to bias our estimates of the effect of legal access to alcohol on MVAs upwards, which lends greater confidence to our results which indicate that legal access to alcohol does not have detrimental effects on MVAs in NSW. In the other panels of Figure A3, we demonstrate that the fraction of drivers who are female and the number of drivers are continuous across the threshold.<sup>23</sup>

## 5 Effects of Legal Access to Alcohol on Drinking Behavior

Figures 3 and 4 present discontinuity estimates for various measures of drinking behaviors. Each figure shows means plots using two-month bins (Panel A), as well as discontinuity estimates under a large range of bandwidths, with 95% confidence intervals (Panel B).

Figure 3 shows the results for three outcomes: ever drinks, drinks at least once per week and proportion of days drinks. For each outcome, the means plots in Panel A reveal an increase in drinking as youths grow older. More importantly, these mean plots also show discontinuities at the age of 18 for each outcome, indicating that legal access to alcohol has significant effects on drinking behavior in NSW. The point estimates in Panel B suggest a discontinuity in “ever drinks” of at least 16 percentage points across a wide range of bandwidths, including 18.6 percentage points at the IK-optimal bandwidth, which incorporates 312 days in either direction from the edges of the “donut.” For all but the smallest bandwidths, these estimates are highly

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characteristics of the underlying population. It’s also worth noting that the composition of HILDA respondents can change even if the underlying composition of the population does not, because HILDA respondents have some discretion over when and whether to complete the questionnaire. Thus, it is theoretically possible for heterogeneity in survey responses that interacts with turning age 18 to bias estimated effects that are based on these survey data. Nonetheless, there is no evidence that such heterogeneity exists.

<sup>23</sup>The number of drivers declines slightly after the MLDA threshold, because drivers are only kept in the sample for days covered by the crash database.

statistically significant. The estimated discontinuity in regular drinking (at least once per week) is similar, at around 20 percentage points under most reasonable bandwidths, including 17.9 at the IK-optimal bandwidth. The estimated discontinuity in “proportion of days drinks” is more sensitive to bandwidth, ranging from around 3 to 7 percentage points. At the IK-optimal bandwidth, the estimated discontinuity is 4.2 percentage points (and statistically significant at conventional levels).

Estimated discontinuities in binge drinking are shown in Figure 4. Only three of the eleven HILDA waves contain binge-drinking data, so these estimates are less precise than those in Figure 1. Nevertheless, the results provide consistent evidence of large discontinuities in binge drinking. The means plots are strongly suggestive of discontinuities in regular binge drinking and in the frequency of binge drinking. For each outcome, the estimates are large, insensitive to bandwidth, and at least marginally significant for reasonable bandwidths. The estimated discontinuity in “binge drinks at least once per week” is on the order of 20 percentage points for almost all bandwidths, 19.0 percentage points at the IK-optimal bandwidth. The estimated discontinuity in “proportion of days of days binge drinking” is around 4 percentage points for most bandwidths.

Although binge drinking data are not included in all waves, a comparison of the mean plots in Figures 3 and 4 is revealing. In, particular, it seems that almost all 18-year-old regular drinkers are also regular binge drinkers. This can be seen by comparing the mean plots for regular drinking on the right of the threshold in Panel A of Figures 3 and 4. This is indicative of Australia’s youth binge drinking culture.

The results in Figures 3 and 4 suggest that MLDA restrictions in NSW are quite effective in influencing youth drinking, especially regular drinking and regular binge drinking. The size of the discontinuities is best compared in relative terms, which we do using the IK-optimal bandwidth point estimates. The point estimates suggest that as youths cross the 18-year-old threshold and gain legal access, their propensity to identify as a drinker increases by around 25%. Their propensity to identify as a regular drinker increases much more, by around 100%, while their “proportion of days drinking” increases by around 70%. More strikingly, their propensity to identify as a regular binge drinker increases by around 150% and the “proportion of days binge drinking” increases by close to 100%. To put it differently, age-based prohibition reduces the frequency of drinking and binge drinking by roughly half at the MLDA threshold. In Section 6, we also show evidence of discontinuities in hospitalizations due to alcohol abuse.



Appendix E shows the results for all of these indicators again, this time by gender. These results are suggestive of some gender heterogeneity in the effects on drinking behavior. In particular, they suggest that the MLDA has a considerably larger effect on females on the “ever drinks” indicator. However, the discontinuities are larger for males on all of the other indicators. In particular, the IK-optimal point estimates are twice as large for males on “proportion of days drinking” and “proportion of days binge drinking,” and more than twice as large for “regular binge drinking.” Overall, there is evidence of large MLDA discontinuities in drinking behavior for both genders.

## 6 Effects of Legal Access to Alcohol on Motor Vehicle Accidents

This section presents estimated discontinuities in rates of “serious” motor vehicle crashes—that is, crashes in which a person was injured and/or a vehicle was towed away. We present results pooling both genders in the body of the text, and results stratified by gender in Appendix E, just as we have for other outcomes. In NSW, males account for over 80% of drunk driving offenses, and more than 80% of crashes in which the driver was recorded to have non-zero BAC. Nevertheless, the results are remarkably similar across gender; they show no discontinuities for any crash types, as will be shown.

Figure 5 shows the results for males for an initial set of three outcomes. The first of these is the rate of all serious crashes. The means-plot shows a crash rate decreasing with age (from left to right). At the left extremity, the crash rate exceeds 10 per 100 driver years, three times higher than at the right extremity. This suggests that driver experience is a major factor in the crash rate. At the same time, this is also consistent with a possible selection effect—only people who obtained their license shortly after age-eligibility are included in the left (young) extremity of the sample, and such drivers may have higher crash rates. In any case, the crash rate varies smoothly through the MLDA threshold. The point estimates are essentially zero at small bandwidths where the linear specification is valid. At the IK-optimal bandwidth, the 95% confidence interval (CI) excludes effects as small as 0.34 crashes per 100 driver-years, or 7% relative to the mean crash rate.

Figure 5 also shows corresponding results for crashes in which the driver was injured and killed, respectively. The driver-injured crash rates are around one-fifth that of the overall crash rates, but show a similar pattern. They follow a smooth downward sloping shape throughout,

with no evidence of discontinuity at age 18. The 95% CI rules out reasonably small discontinuities in driver-injured crashes. The driver-killed rates are much more variable, because such crashes are around 100 times less frequent than driver-injured crashes. The mean plot for fatalities (Panel C) again shows no evidence of a discontinuity, but the estimates are imprecise and cannot rule out even large effects. In Appendix C, we show results for MVA fatalities from an alternate data set which covers a longer period (1994–2000). Even with the additional coverage, those results are still imprecise.

For this reason, we turn in Figure 6 to fatality-risk weighted crashes. For these estimates, we include all crashes, weighted by the predicted probability of driver-fatality, based on a rich set of driver and crash characteristics as discussed in Section 3. The pattern again resembles those in Figure 5: the estimated discontinuity is small and statistically insignificant for all bandwidths, and is negative for many bandwidths. The standard errors are much smaller than for the driver-killed estimates.

Figure 6 also considers crashes with high predicted fatality risks, i.e., those exceeding 0.05 and 0.1, respectively. Unlike the (preferred) approach of fatality-risk weighting, for this analysis each included crash satisfying this condition is weighted equally. To give a sense of where these crashes lie in the distribution of fatality risk, 1% of serious crashes are deemed high-fatality risk ( $p > 0.05$ ), and around 0.3% are treated as very high-fatality risk ( $p > 0.1$ ). These analyses also show no visual discontinuity at the MLDA, which is confirmed by small and statistically insignificant estimates at all bandwidths.

Because most alcohol related crashes occur at night, we repeat the analysis considering only night crashes.<sup>24</sup> These results are shown in Figures 7 and 8. The means plots again shows no visible evidence of discontinuities. For the set of all night crashes, the discontinuity estimates are precise and are close to zero at all reasonable bandwidths. The IK-optimal estimate is almost exactly zero. For night-injury crashes, the point-estimates are small and positive but are not close to being statistically significant, except at large bandwidths where the linear specification is not valid. The estimated discontinuities for night-killed crashes are all negative, but are very imprecise. Figure 6 shows the estimates for night-time fatality-risk-weighted crashes, and for high-fatality-risk crashes. Once again, we see no visual evidence of discontinuities in any of these indicators and find no indication of RD estimates that approach statistical significance.

In results not shown, we have considered many other (more restricted) sets of crashes (in-

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<sup>24</sup>This excludes around three quarters of crashes.

cluding weekend crashes and non-urban crashes) in similar analyses. We also tried interactions of such crash characteristics (e.g. night time non-urban crashes). Consistently, we found no evidence of discontinuities.

In Appendix E we show estimates by gender for all of the crash indicators discussed above. In most categories, crash rates are higher for males than for females. However, without exception, the discontinuity estimates are not statistically significant for either gender for any MVA outcomes.

## 7 Effects of Legal Access to Alcohol on Hospitalizations

This section presents discontinuity estimates for various types of inpatient episodes. In particular, we focus inpatient episodes involving alcohol poisoning, motor vehicle accidents, motor cycle accidents, assaults, and other external causes.<sup>25</sup>

Figure 9 shows the results for two key outcomes, providing further support for the findings shown in earlier sections. There is a clear discontinuity in hospitalizations for alcohol intoxication or poisoning: the estimate is statistically significant and insensitive to bandwidth, with a magnitude of approximately 4 episodes per 10,000 person years, representing a 30% increase as youths gain legal access to alcohol. It is possible that this estimate is at least partly caused by underage drinkers being less likely to go to a hospital for fear of punishment. However, the estimate is consistent with the finding from HILDA that legal access restrictions have a substantial impact on youth binge drinking.<sup>26</sup>

In contrast, we find no evidence of a discontinuity in hospitalizations of drivers injured in MVAs (Figure 9). Indeed, the point estimates are negative for almost all bandwidths.<sup>27</sup>

<sup>25</sup>We initially considered hospitalization rates overall and found evidence of a positive discontinuity across the age-18 threshold driven by episodes in which the principal diagnosis is in the category of “Diseases of oral cavity, salivary glands and jaws,” the majority of which are “impacted teeth.” For obvious reasons, it is highly unlikely that legal access to alcohol is responsible for this discontinuity. Rather, it is more likely to be driven by conscious decisions (by doctors, dentists, and/or patients) to schedule such procedures after the patient reaches adulthood (i.e. turns 18).

<sup>26</sup>While it would be desirable to consider hospitalizations due to other types of substance abuse (following up on Deza (2013) who examines effects on hard drug use), our hospital data do not allow for a convincing analysis along these lines. A significant component of episodes where the primary diagnosis related to psychoactive substance use are “due to multiple drug use and use of other psychoactive substances.” The ICD10 guidelines state that these codes “should also be used when the exact identity of some or even all the psychoactive substances being used is uncertain or unknown.” For these cases, we cannot determine whether alcohol was a contributing (or even the only) factor. Our estimates for non-alcohol substance abuse were sensitive to the treatment of such cases. We only found significant discontinuities when these cases were included.

<sup>27</sup>The left extremity of the means plot shows relatively low hospitalization rates, because eligibility for a provisional license begins at age 17. Drivers injured at age 16 were either unlicensed, or holding a learner’s permit. For this reason, only estimates with bandwidths less than 365 days are meaningful for this indicator.

However, there is some suggestive evidence of a discontinuity in hospitalizations of riders injured in motor cycle accidents (Figure 10). The magnitude of the discontinuity is around 2 episodes per 10,000 person years, an increase of about 13% across the threshold. The size and significance of the estimate is somewhat sensitive to bandwidth. However, it is at least marginally significant at bandwidths exceeding 270 days. Two other aspects of the results for motorcycle accidents are noteworthy. First, the hospitalization rate for injuries due to riding motorcycles is just as high as it is for injuries due to driving other motor vehicles (shown in Figure 9) for individuals in this age range. This is despite the much smaller number of motor cycle riders. Second, there is a relatively high rate of hospitalization for 16-year olds, suggesting that licensing restrictions may not be a major factor in motor cycle accidents. As mentioned previously, only half of these episodes are for accidents occurring on public roads. Thus, the broad set of alcohol-related policies that are used to prevent drunk driving on public NSW roads are less relevant for motor cycle accidents. In addition, for almost all of the time spanned by the data, motorcycle riders could receive an unrestricted license at age 18.

Figure 10 also shows the results for hospitalizations caused by assault. It shows a clear upward trend in assaults for 16- and 17-year olds. It also shows a clear discontinuity at the MLDA threshold, which is statistically significant and insensitive to bandwidth. At least visually, this discontinuity appears to be driven by high assault rates in the first four months after turning 18. If so, this discontinuity may be attributable to a process of transition, or learning to negotiate the freedom associated with gaining legal access to alcohol. Further exploration of this possibility would require a departure from the RD design, which we leave for future work.

Figure 10 also shows the estimates for episodes with any other external cause, i.e. all external causes, excluding assaults and drivers/riders injured in MVAs. This encompasses a broad range of causes, including pedestrians, pedal cycle riders and passengers injured in transport accidents, other accidents, self-harm, and events of undetermined intent. The results show no evidence of a discontinuity at any bandwidth. Similarly, there are no discontinuities when episodes with each of these causes are analyzed separately (results not shown).

Appendix E shows corresponding discontinuity estimates by gender. With the exceptions of assault and motorcycle accidents, there is little heterogeneity by gender. For hospitalizations due to alcohol intoxication/poisoning, the discontinuity is a bit clearer for females, resulting in significant estimates for a larger range of bandwidths. Nevertheless, the effect does not appear to differ greatly by gender. The discontinuity in assaults is much larger for males,

as is their baseline rate of hospitalizations due to assault. Finally, hospitalizations due to motorcycle accidents almost exclusively involve males. Thus, the male plots look almost exactly like the pooled plots, but with rates that are roughly twice as high. Moreover, young males are considerably more likely to be hospitalized following a motor cycle accident than as a driver in a motor vehicle accident.

## 8 Discussion and Conclusion

As discussed in the introduction, a large literature on the effects of legal access to alcohol precedes this study. While this literature documents public health costs that are broad in scope, researchers have emphasized that the most important component of these costs is driven by MVAs. This study’s primary contribution is in presenting evidence that suggests that legal access to alcohol need not be accompanied by these large public health costs. We acknowledge, of course, that there might be effects that are too small to be detected during the time period spanned by our data. However, the estimated effects on MVAs are routinely close to zero, if not negative, whether the focus is on serious accidents defined broadly or narrowly, on all accidents or nighttime accidents, on males or females, and on accidents reported in data provided by the NSW’s Centre for Road Safety or accidents identified in the National Hospital Morbidity Database. Expressed relative to the crash rate of 17-18 year-old drivers, we can rule out effects on “serious motor vehicle accidents” above 7% at the five percent level, effects on being injured in an accident above 14%, and fatality-risk weighted accidents above 27%. Given that the MVA fatality rate in NSW is low (around half that of the US), these are reasonably precise zeros. We have also argued that these estimates are likely biased upwards, primarily due to license class progression.

Although we do not find any evidence that legal access to alcohol affects MVAs, we do find some evidence of public health costs associated with youths gaining legal access to alcohol: there are significant effects on hospitalizations due to alcohol poisoning and assault victimization. Based on the average hospital costs associated with intoxication episodes (\$2,313) and assaults (\$3,730), our preferred discontinuity estimates imply that a year of legal access per NSW youth imposes additional hospital costs of \$1 for intoxication and \$3 for assault.<sup>28</sup>

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<sup>28</sup>Cost estimates were derived using 2009-10 National Public Cost Weight Tables. These tables show the estimated cost of treatment for episodes in each Diagnosis Related Group (DRG). Each DRG category represents episodes with similar conditions and similar usage of hospital resources. However, DRGs do not concord with the classification we have used to group episodes (ICD-10). Our calculation is based on the average cost from these

This study shows that there need not be a link between legal access to alcohol and MVAs, but more work is needed to understand precisely how any such link can be broken. We view the relative seriousness with which NSW enforces and punishes drunk driving as the leading explanation for why our results differ from what has been found in the U.S. context, but we cannot rule out the importance of other cultural and institutional factors. Thus, we think of our results as highlighting the potential importance of the interplay between drunk driving policies and youth access to alcohol and view this as an important area for future research, because optimal policy design requires that we understand the degree to which different alcohol control policies can substitute and complement one another.

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cost weights across intoxication and assault episodes, where the patient was aged between 16 and 19, inclusive.

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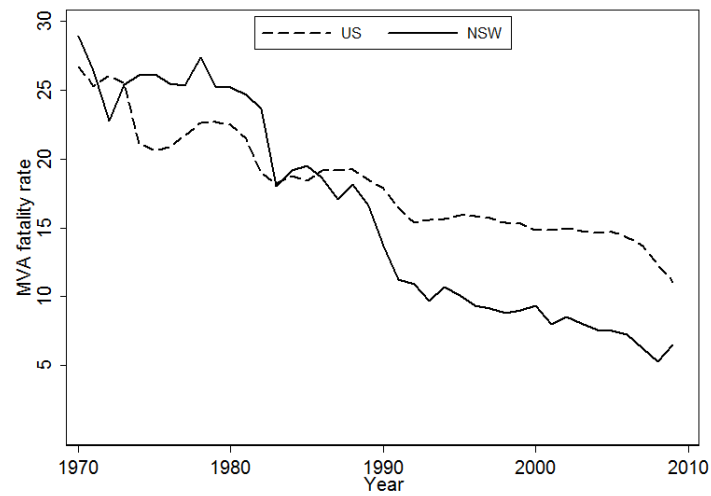
Table 1  
Summary Statistics

<b>Data on Drinking Behavior (HILDA)</b>	
<i>Means of Key Variables:</i>	
Lives with parents	0.861
At School	0.496
Employed	0.551
Female	0.515
Ever drinks	0.685
Drinks regularly	0.228
Proportion of days drinks	0.075
Binge drinks regularly	0.200
Proportion of days binge drinks	0.055
<i>Sample Size:</i>	
Number of observations	2359
Number of observations - binge drinking	711
<b>Data on Drivers and Motor Vehicle Accidents</b>	
<i>Number of Drivers:</i>	
All	733954
Male	420797
Female	313157
Number of relevant MVAs	37978
<i>MVAs Per 100 Driver Years:</i>	
MVAs Involving a Vehicle Being Towed, Injury, or Death	5.1743
Injuries in MVAs	0.9751
Deaths in MVAs	0.0109
Mortality-Risk-Weighted MVAs	0.0138
MVAs with Mortality Risk > 0.025	0.1273
MVAs with Mortality Risk > 0.05	0.0523
MVAs with Mortality Risk > 0.075	0.0286
MVAs with Mortality Risk > 0.010	0.0189
<i>Nighttime MVAs Per 100 Driver Years:</i>	
MVAs Involving a Vehicle Being Towed, Injury, or Death	1.3175
Injuries in MVAs	0.2797
Deaths in MVAs	0.0041
Mortality-Risk-Weighted MVAs	0.0059
MVAs with Mortality Risk > 0.025	0.0612
MVAs with Mortality Risk > 0.05	0.0228
MVAs with Mortality Risk > 0.075	0.0132
MVAs with Mortality Risk > 0.010	0.0087
<b>Data on Hospitalizations</b>	
Number of relevant inpatient episodes	99989
<i>Inpatient Episodes Per 10,000 Person Years:</i>	
Alcohol Intoxication/Poisoning	16.662
Assault	26.098
Driver in Motor Vehicle Accident	14.078
Rider in Motor Cycle Accident	15.798
Any Other External Cause	250.133

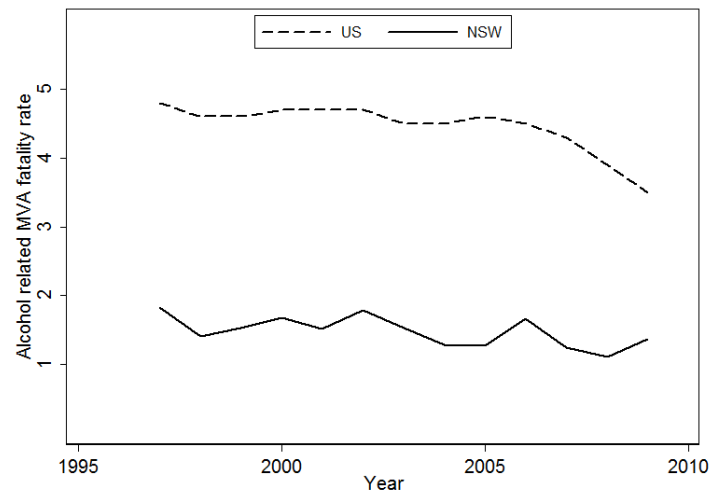
Notes: Data on drinking behavior are from are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey (2001–2011). Data on drivers and motor vehicle accidents are from the NSW Center for Road Safety. The sample includes all drivers who are 17- or 18-years old first obtaining a license after July 1, 2000 in NSW. Driver years are calculated using individual licensing dates. Mortality risk is predicted based on a probit model for drivers age 17–21 (inclusive) with regressors for the type of accident, general location of the accident, speed, use of seatbelt, role in first impact, primary cause of accident, and gender. Inpatient hospital data are from the AIHW National Hospital Morbidity Database (July 2001–June 2010).

Figure 1  
Fatal Motor Vehicle Accidents in New South Wales and United States

Panel A: Fatality rate (per 100,000) in all motor vehicle accidents, 1970-2009



Panel B: Fatality rate (per 100,000) in alcohol related motor vehicle accidents, 1997-2009



Panel C: Share of MVA fatalities that are alcohol related, 1997-2009

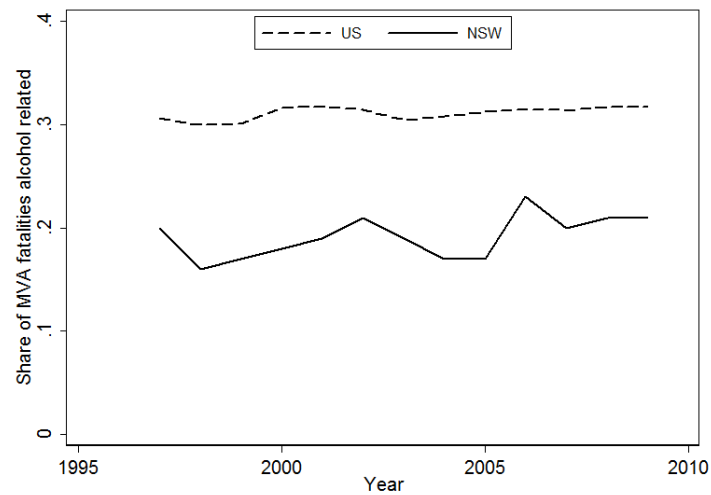
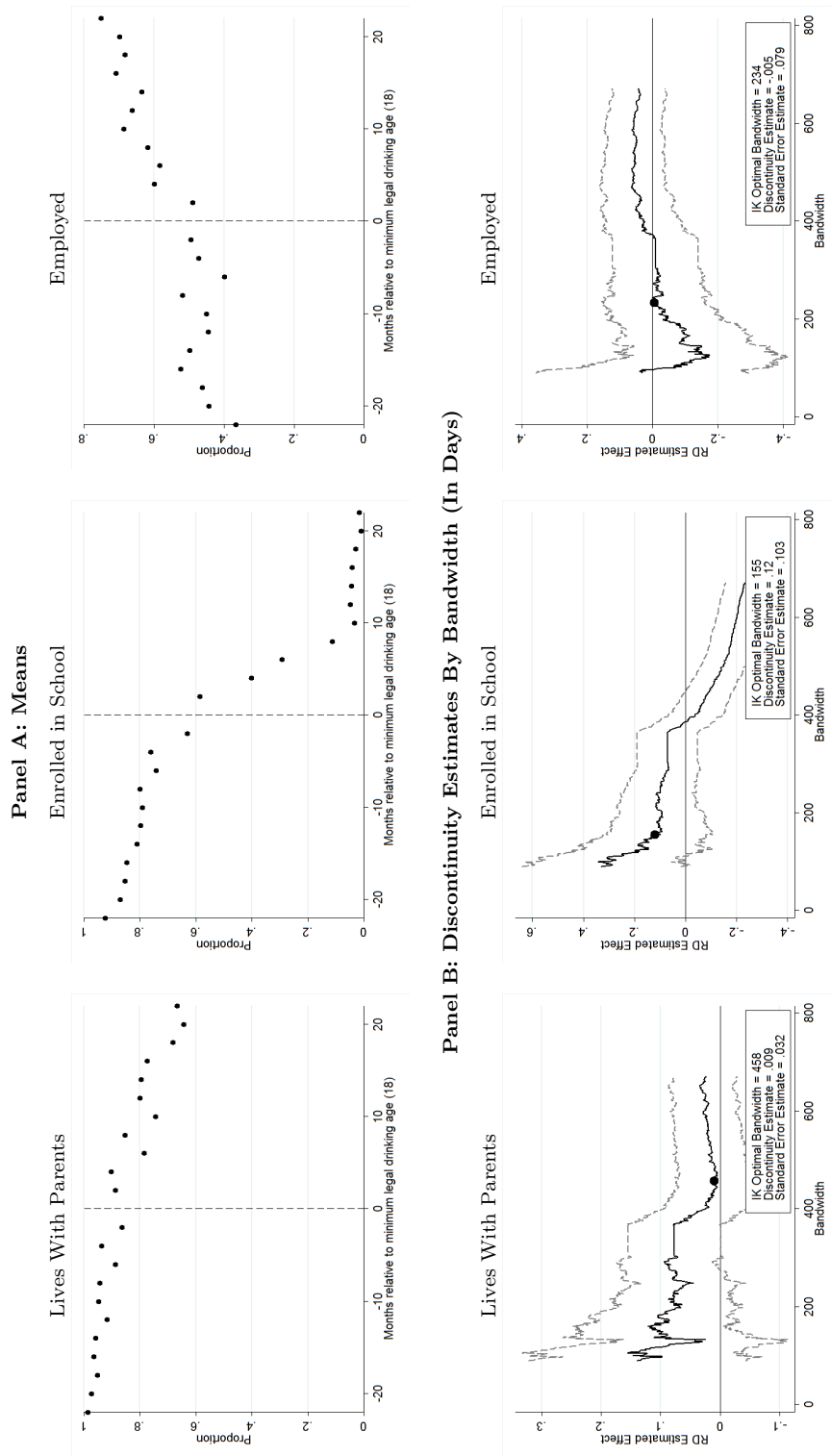
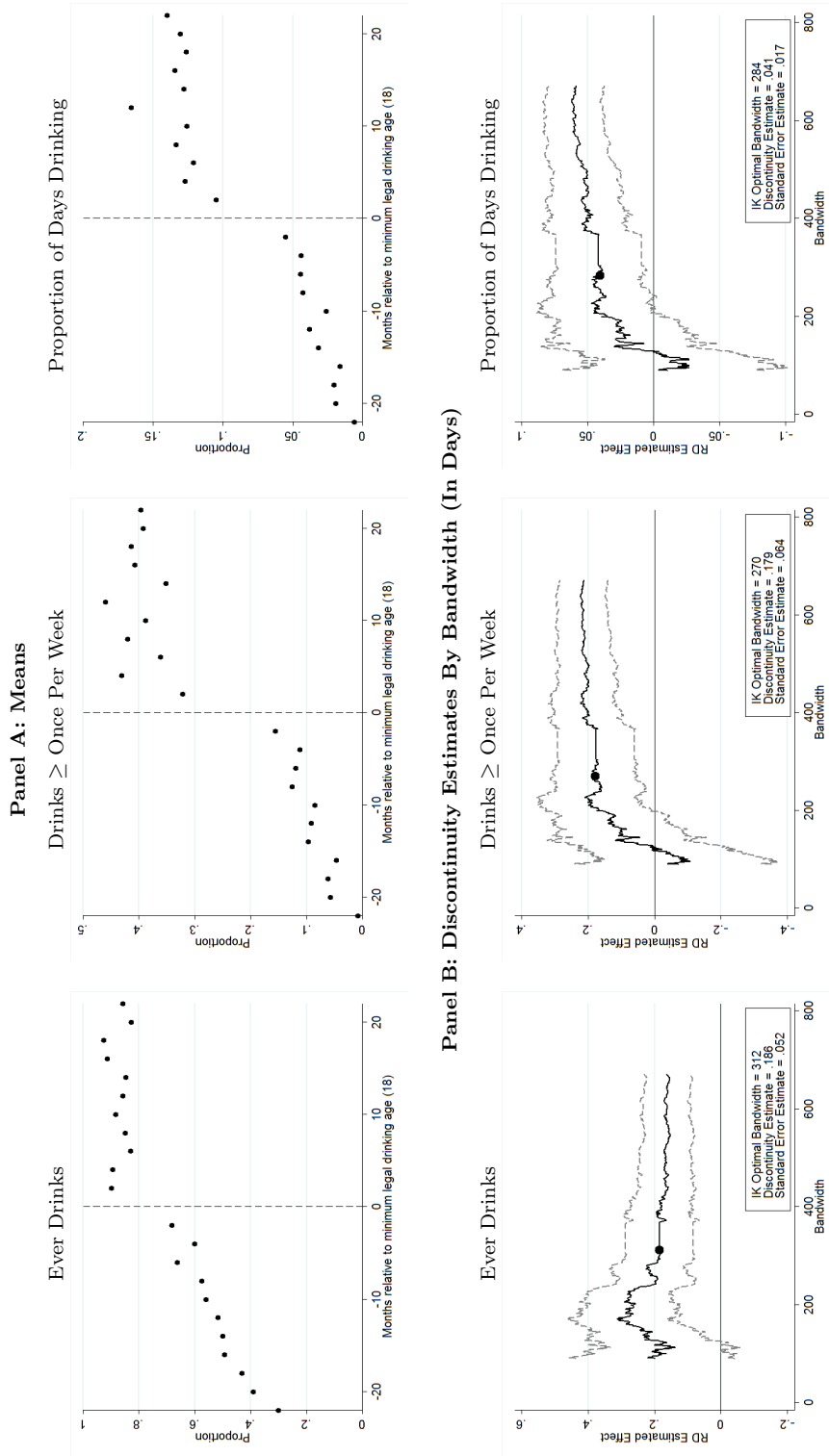


Figure 2  
Estimated Discontinuities in Population Characteristics  
Based on HILDA Survey Data



Notes: Data are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey (2001-2011). Panel A reports means in two-month bins. Panel B reports estimates from local linear regressions, using rectangular kernel weights and allowing the slopes to vary on each side of the threshold, for a range of different bandwidths. The textbox within the figure shows the optimal bandwidth selected by the procedure described in Imbens and Kalyanaraman (2012) along with the corresponding point estimate and standard error estimate.

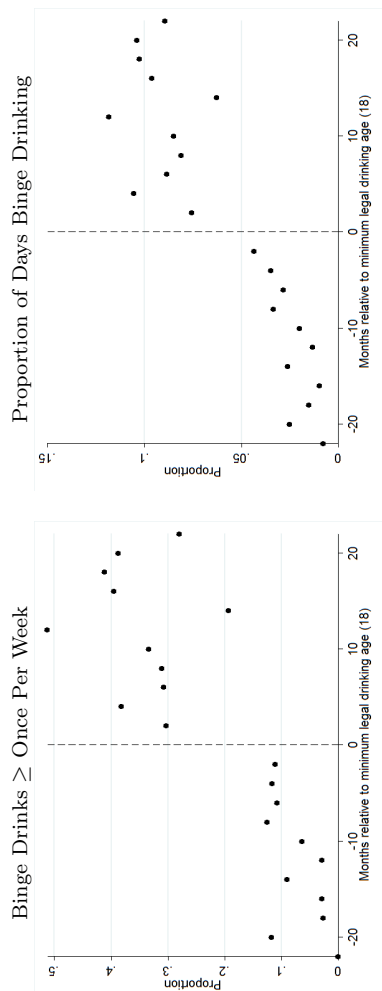
Figure 3  
Estimated Discontinuities in Reported Drinking Behavior



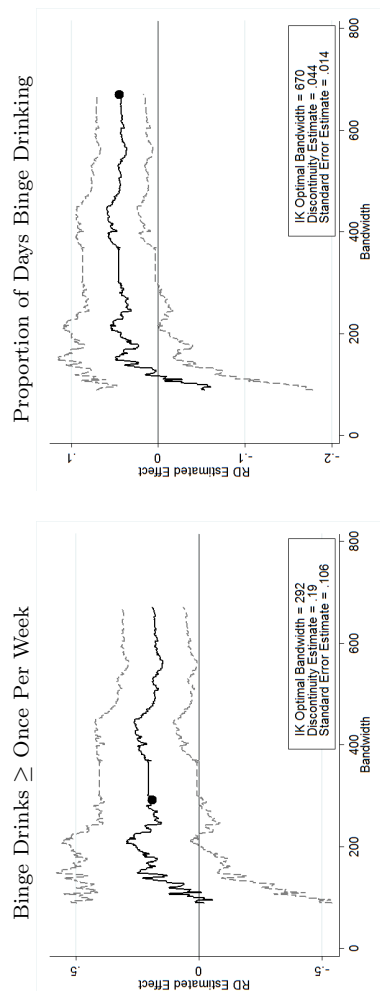
Notes: Data are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey (2001–2011). Panel A reports means in two-month bins. Panel B reports estimates from local linear regressions, using rectangular kernel weights and allowing the slopes to vary on each side of the threshold, for a range of different bandwidths. The textbox within the figure shows the optimal bandwidth selected by the procedure described in Imbens and Kalyanaraman (2012) along with the corresponding point estimate and standard error estimate. Drinking frequency is based on responses to the question “do you drink alcohol?” (in surveys spanning 2001 to 2011) to which individuals can select that they never drink, rarely drink, drink 2 or 3 days per month, drink 1 or 2 days per week, drink 3 or 4 days per week, 5 or 6 days per week, or every day. The survey does not provide respondents a time frame of reference, e.g., “during the past 30 days.” In order to mitigate this problem, we omit from the analysis observations in which a respondent is within 30 days of obtaining or having obtained legal access to alcohol, or within 30 days of any other birthday (to account for possible celebration effects). Appendix D shows results for alternative “donut” widths.

Figure 4  
Estimated Discontinuities in Reported Binge Drinking Behavior

Panel A: Means

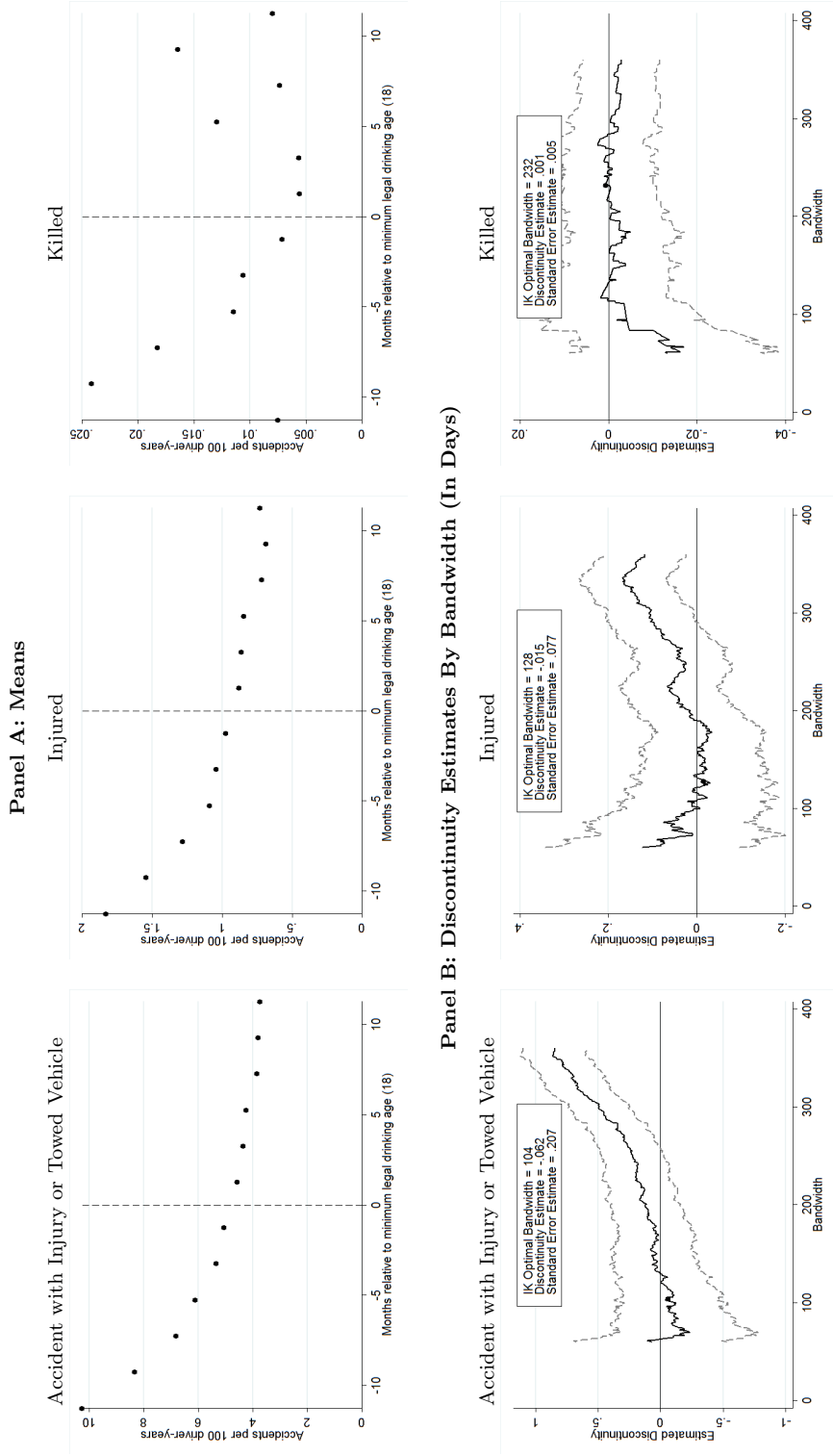


Panel B: Discontinuity Estimates By Bandwidth (In Days)



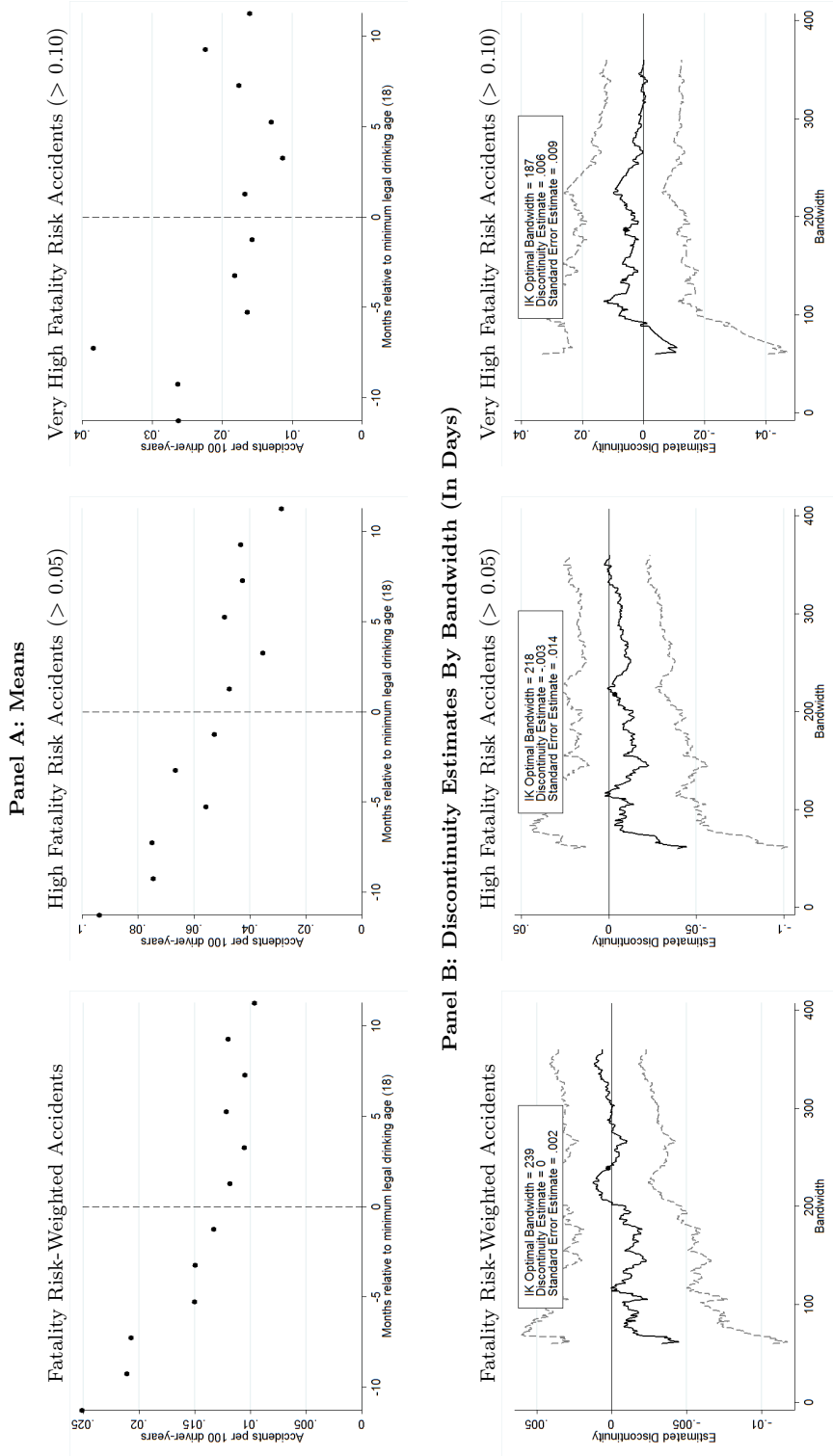
Notes: Data are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey. Binge drinking questions were only asked in three HILDA waves (2007, 2009 and 2011). Binge drinking is defined as five or more standard drinks on one occasion for females and seven or more standard drinks on one occasion for males. See also Figure 3 notes.

Figure 5  
Estimated Discontinuities in Serious Motor Vehicle Accidents



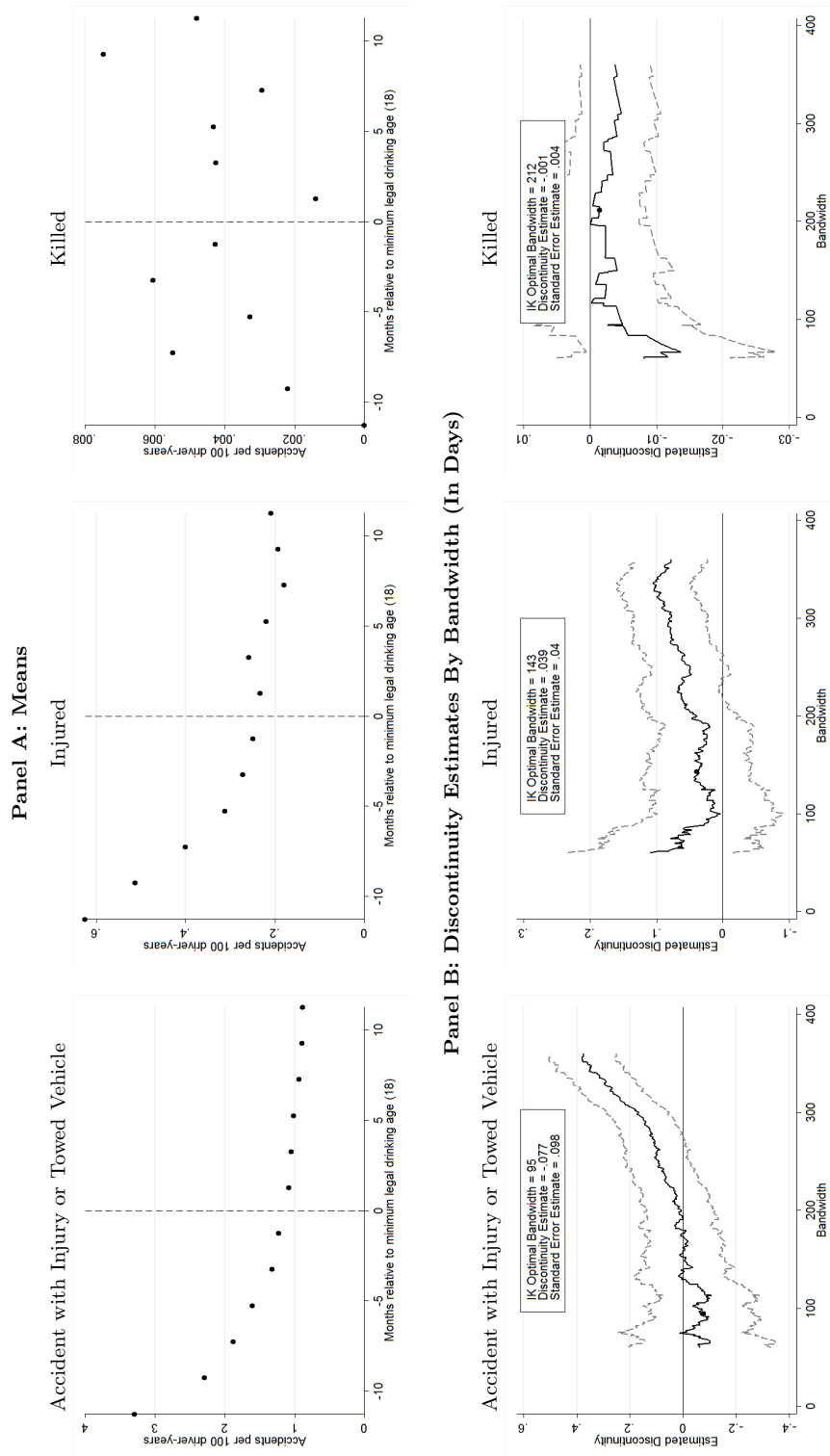
Notes: Data are from the NSW Center for Road Safety (2001-2010). Panel A reports means in two-month bins. Panel B reports estimates from local linear regressions, using rectangular kernel weights and allowing the slopes to vary on each side of the threshold, for a range of different bandwidths. The textbox within the figure shows the optimal bandwidth selected by the procedure described in Imbens and Kalyanaraman (2012) along with the corresponding point estimate and standard error estimate.

Figure 6  
Estimated Discontinuities in Motor Vehicle Accidents Weighted by Fatality Risk



Notes: Each crash in the database was assigned a fatality-risk probability, equal to the predicted value from a probit model of fatality, using a large number of crash characteristics as explanatory variables. The fatality risk model is described in Section 3.3 and Appendix B. See also Figure 5 notes.

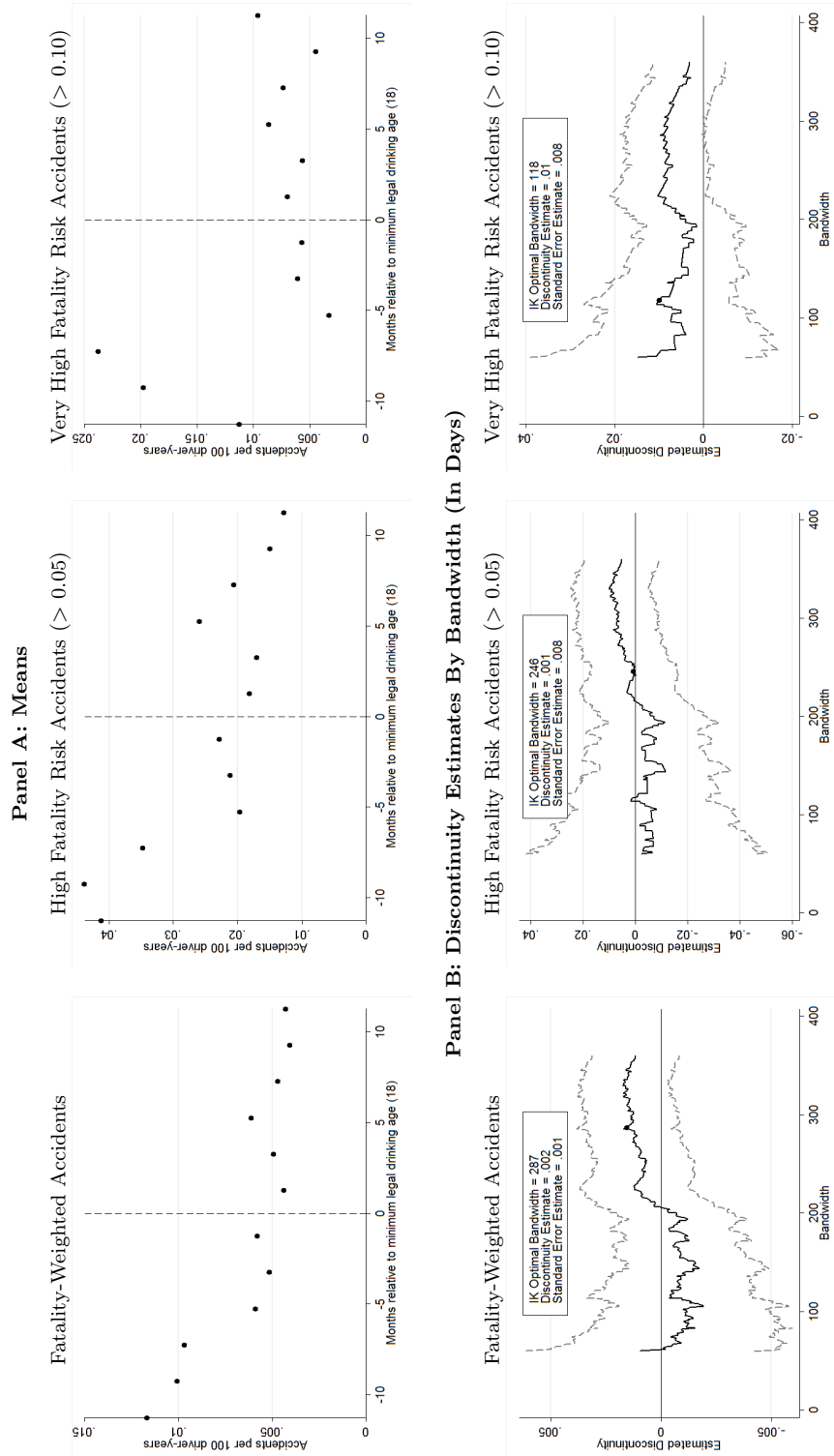
Figure 7  
Estimated Discontinuities in Serious Motor Vehicle Accidents at Night



Notes: See Figure 5 notes

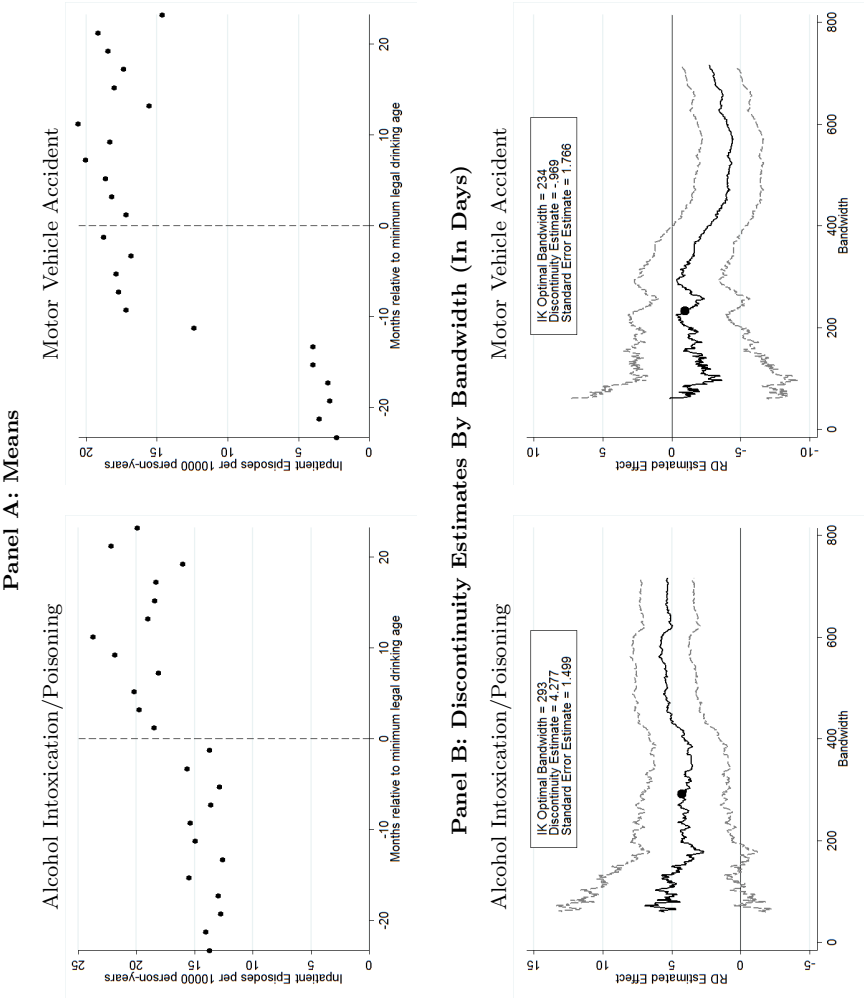


Figure 8  
Estimated Discontinuities in Motor Vehicle Accidents at Night Weighted by Fatality Risk



Notes: See Figure 5 notes and Figure 6 notes

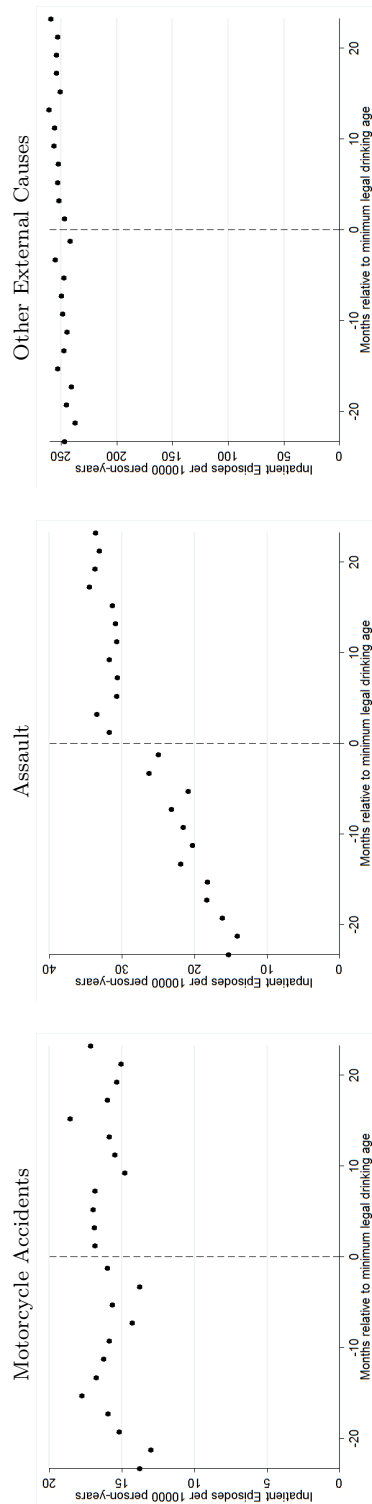
Figure 9  
Estimated Discontinuities in Hospitalizations for Alcohol Poisoning and Motor Vehicle Accidents



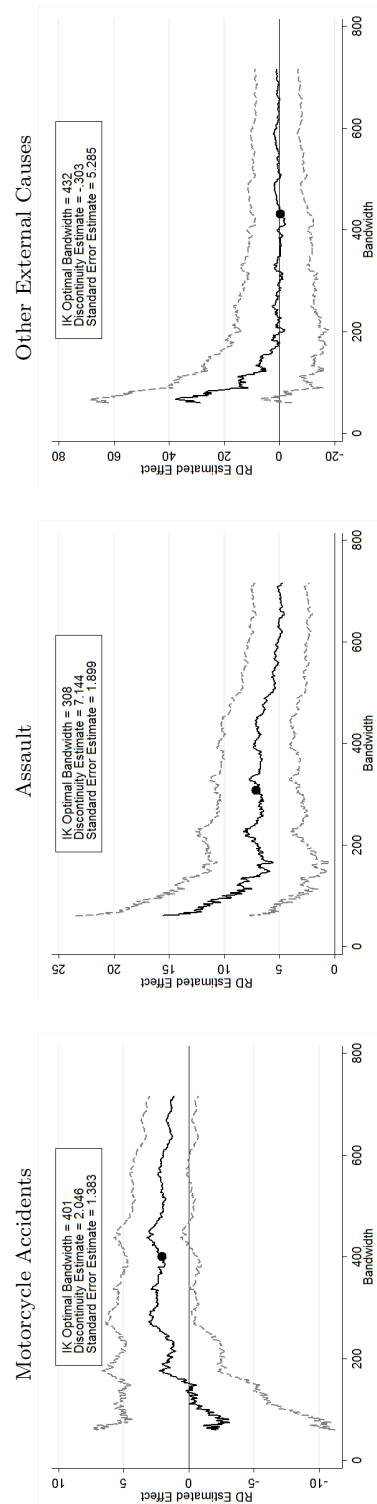
Notes: Data are from the AIHW National Hospital Morbidity Database (July 2001-June 2010). Panel A reports means in two-month bins. Panel B reports estimates from local linear regressions, using rectangular kernel weights and allowing the slopes to vary on each side of the threshold, for a range of different bandwidths. The textbox within the figure shows the optimal bandwidth selected by the procedure described in Imbens and Kalyanaraman (2012) along with the corresponding point estimate and standard error estimate. In order to mitigate the problem of birthday “celebration effects”, we omit from the analysis observations within 7 days of any birthday. Appendix D shows results for alternative “donut” widths.

Figure 10  
Estimated Discontinuities in Hospitalizations for Other Causes

Panel A: Means



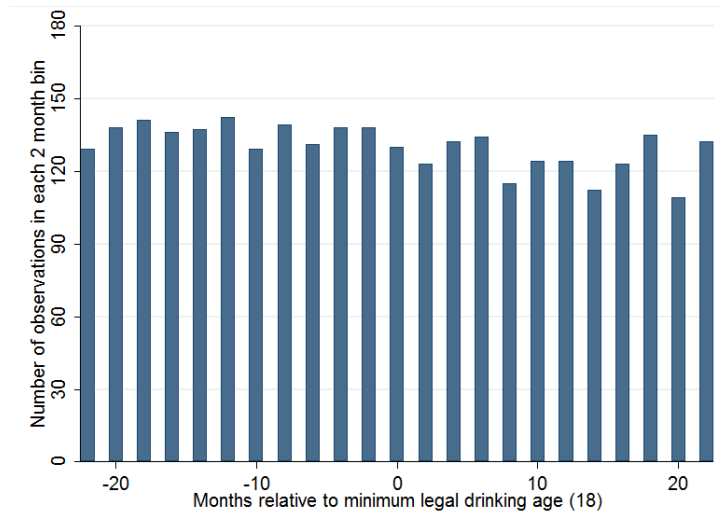
Panel B: Discontinuity Estimates By Bandwidth (In Days)



Notes: See Figure 9 notes

## Appendix A: Additional Balance Figures

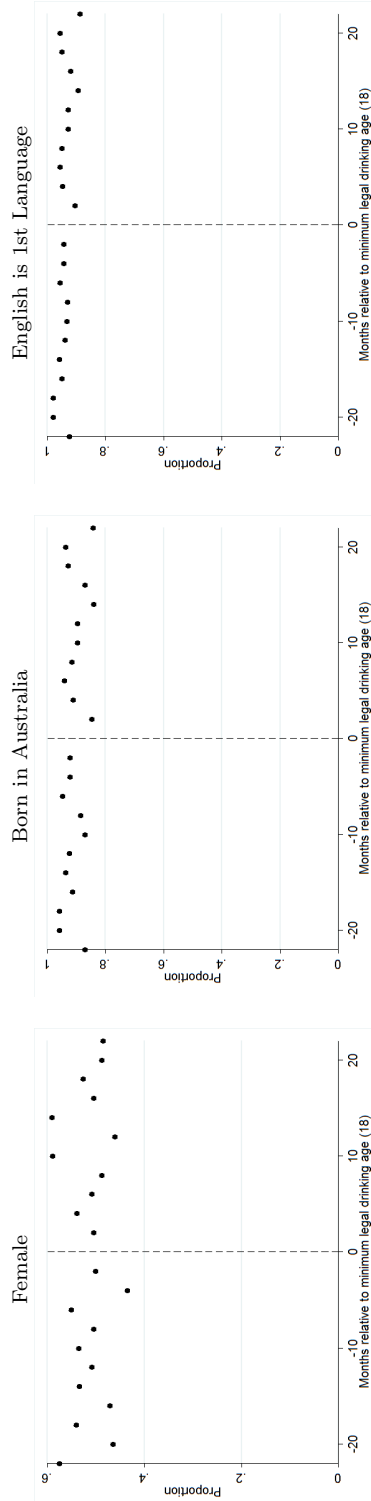
Figure A1  
Number of Observations in HILDA by Age in Months relative to MLDA



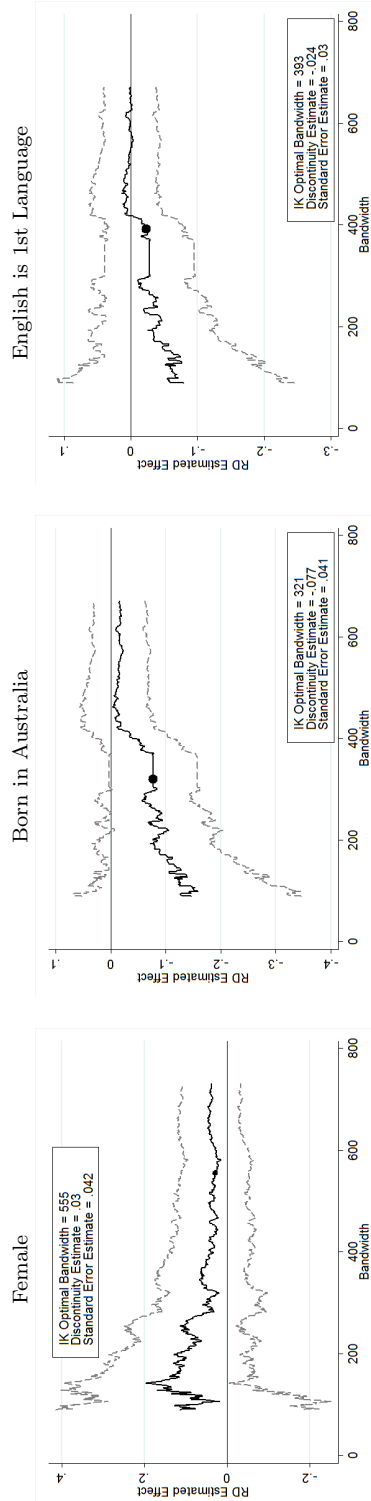
Notes: Data are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey (2001-2011). Frequency counts are shown by age in two-month bins. The column centered around zero corresponds with observations that are not used in our preferred “donut RD” analysis.

Figure A2  
Estimated Discontinuities in Population Characteristics  
Based on HILDA Survey Data

Panel A: Means



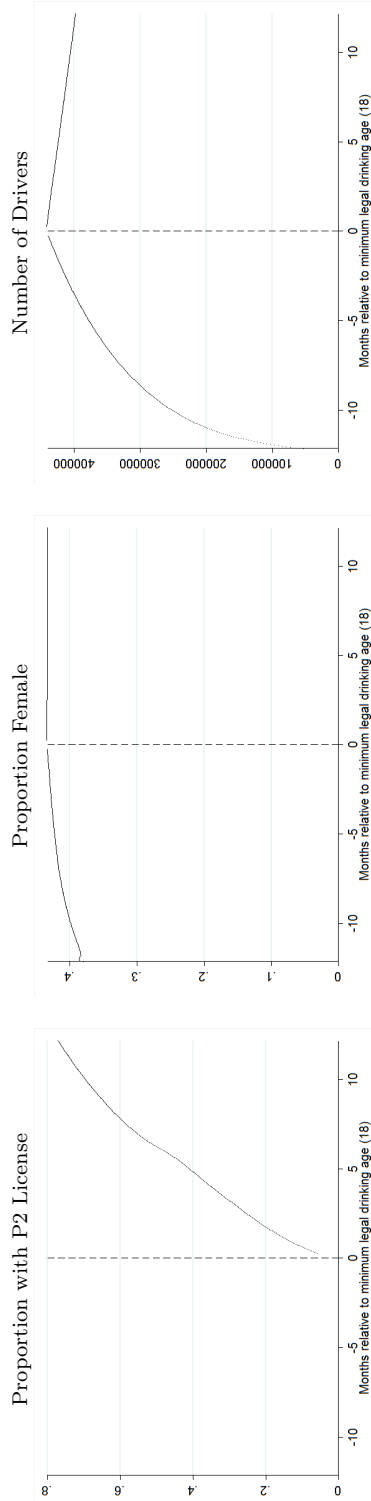
Panel B: Discontinuity Estimates By Bandwidth (In Days)



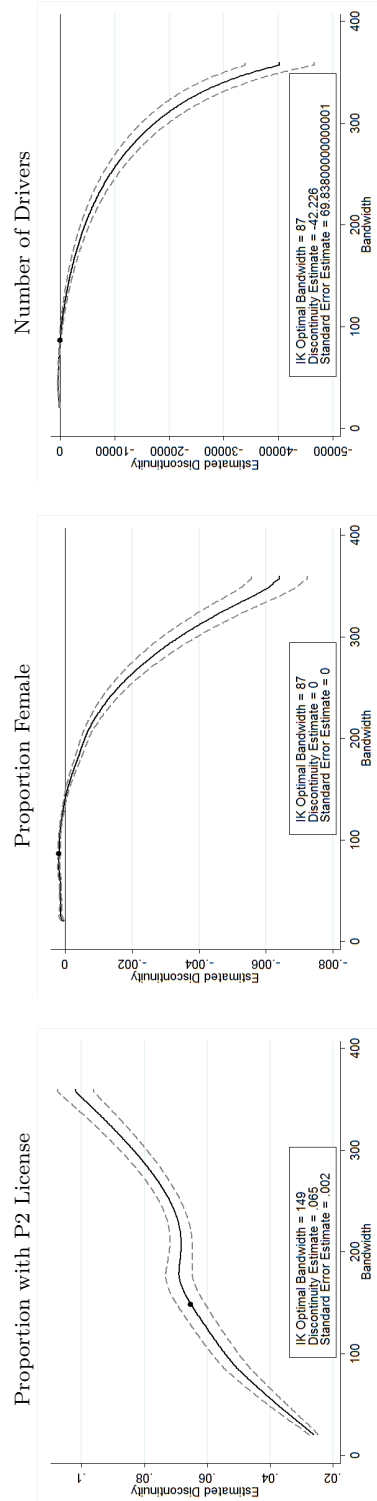
Notes: Data are from the Household, Income and Labour Dynamics in Australia (HILDA) Survey (2001-2011).

Figure A3  
 Estimated Discontinuities in Driver Characteristics  
 Based on Licensing Data

Panel A: Proportions and Counts



Panel B: Discontinuity Estimates By Bandwidth (In Days)



Notes: Data are from the NSW Center for Road Safety (2001-2010).

## Appendix B: Results of Fatality Risk Model

The distribution of predicted fatality risk is highly skewed (Table B1). It is zero or close to zero for the majority of crashes. It exceeds 1% for over 2,000 crashes, or 5.7% of crashes.

Table B2 shows key characteristics of fatality-risk weighted crashes (column 2), in comparison with all (unweighted) crashes (1), as well as the set of fatal crashes (3). As intended, fatality-risk weighted crashes resemble fatal crashes on these characteristics. The small discrepancies between fatality-risk weighted crashes and driver-killed crashes are due to the larger sample of crashes included in the fatality risk model. Unsurprisingly, fatal crashes are very different from the full set of crashes. For example, the average speed travelled is more than twice as high in fatal crashes. The driver was travelling at high speed in less than 5% of crashes overall, compared to 57% of fatal crashes. Likewise, fatal crashes are far more likely to not involve seatbelt use, to occur at night and to occur in non-urban areas. Further, 93% of fatal crashes are either head-on collisions or “off-path” crashes. In contrast, only one third of all crashes fall into this category, with head on crashes making up just 4% of them. Finally the driver is far more likely to be travelling on the wrong side of the road in fatal crashes.

Table B1  
Distribution of Predicted Fatality-Risk For Relevant Crashes

Fatality-Risk	Number of Crashes	Share of Crashes
Zero	4716	0.124
0 to 0.01%	17250	0.454
0.01% to 0.1%	8533	0.225
0.1% to 1%	5307	0.140
1% to 5%	1788	0.047
5% to 10%	245	0.006
10% +	139	0.004
All relevant crashes	37978	1.000

Notes: Data are from the NSW Center for Road Safety (2001-2010). This table shows the distribution of predicted fatality risk within the estimation sample from the main analysis. For greater precision, the fatality risk probit model was estimated using an expanded sample including all drivers aged 17-21 years.

Table B2  
Key Crash Characteristics: Fatality-Risk Weighted versus Unweighted Crashes

	All crashes		Driver-killed Crashes (3)
	Unweighted (1)	Fatality-Risk Weighted (2)	
Mean speed (km/h)	38	87	88
Fast (90 km/h+)	0.053	0.562	0.571
No seatbelt	0.003	0.081	0.075
Night	0.255	0.429	0.375
Non-urban	0.086	0.379	0.388
Head on crash	0.037	0.286	0.375
Off-path crash	0.311	0.579	0.550
All other Road User Movements	0.653	0.135	0.075
Incorrect side	0.029	0.266	0.375
Proceeding in lane	0.687	0.628	0.575
All Other Manoeuvres	0.285	0.107	0.050

Notes: Data are from the NSW Center for Road Safety (2001-2010). This table compares the observed characteristics of fatality risk-weighted crashes to that of unweighted crashes and driver-killed crashes, respectively.



## Appendix C: Effect of MLDA on Mortality from External Causes

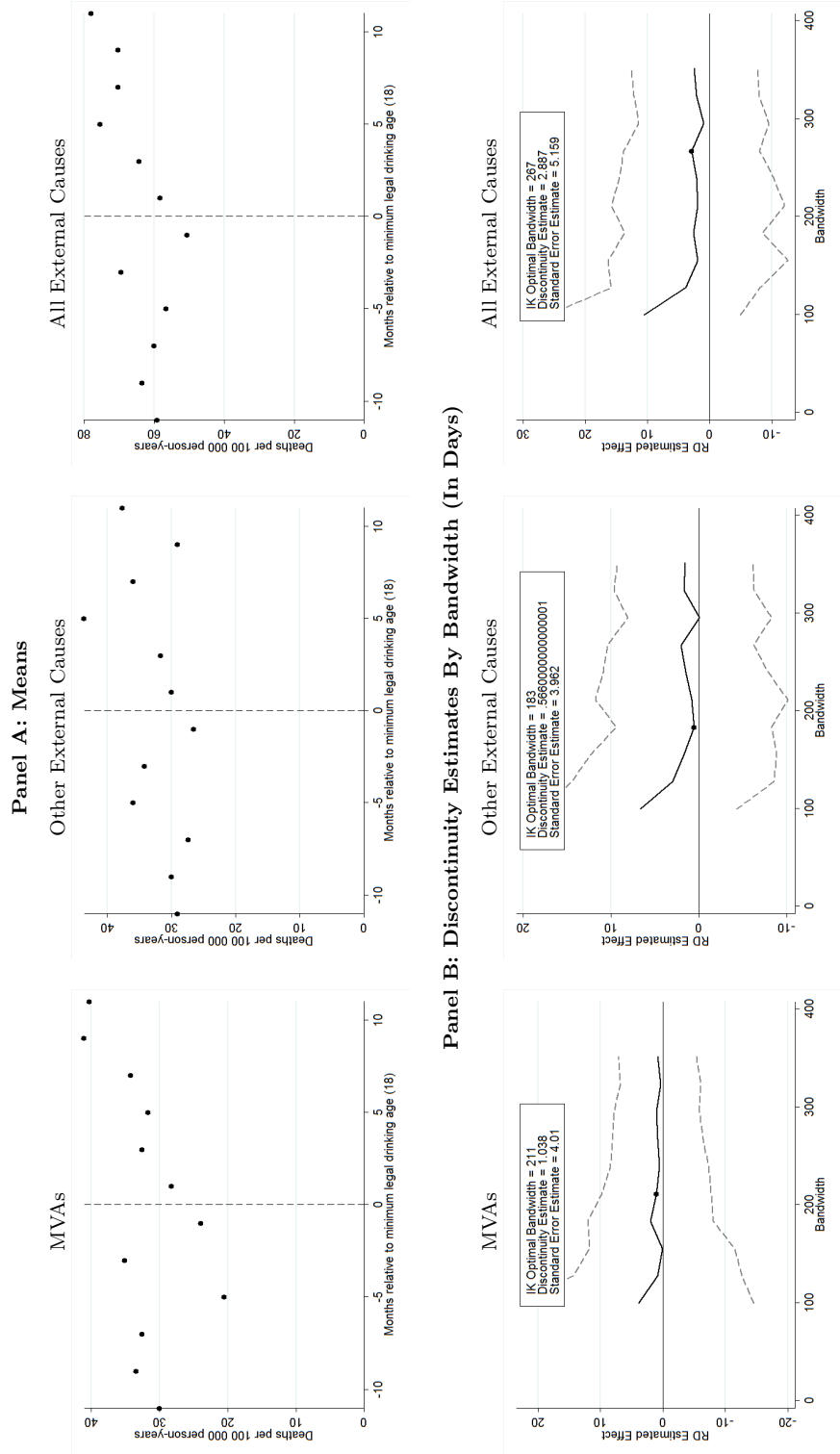
This appendix shows estimated discontinuities in mortality from external causes in NSW, following the approach used throughout the paper. Data were obtained from the AIHW National Mortality Database for deaths occurring between 1994 and 2010. Due to privacy regulations, the data were provided as counts by age at death relative to 18th birthday, in 28-day bins, and causes are classified in only two categories (MVA and other external). Deaths on 18th birthday and one day either side were excluded.

The results (Figure C1) show no evidence of discontinuities in MVA fatalities or in deaths from other external causes. The point-estimates are close to zero at all bandwidths, and the confidence intervals are reasonably tight. Using the optimal bandwidth, the 95% CIs rule out a 25% increase in MVA fatalities and a 20% increase in all externally caused mortality.

However, we do not emphasize these results for a number of reasons. The analysis shown in the body of the text has emphasized several nuances in studying MLDA effects in NSW. These include the role of motor vehicle licensing, including discrepancies between licensing for motorcycles and for other motor vehicles, as well as gender differences in binge drinking and drunk driving. Due to the paucity of mortality data in NSW (because of its relatively small population), combined with data access restrictions due to privacy, we are unable to explore those nuances using mortality data.

Nevertheless, these results are reassuring. They are consistent with those found using hospital data and with MVA crash data.

Figure C1  
Estimated Discontinuities in Mortality Due to External Causes



Notes: Data are from the AIHW National Mortality Database (1994-2010). Only deaths in NSW are included. Panel A reports means in two-month bins. Panel B reports estimates from local linear regressions, using rectangular kernel weights and allowing the slopes to vary on each side of the threshold, for a range of different bandwidths. The text box within the figure shows the optimal bandwidth selected by the procedure described in Imbens and Kalyanaraman (2012) along with the corresponding point estimate and standard error estimate. In order to mitigate the problem of birthday “celebration effects”, we omit from the analysis observations within 1 day of any birthday. Ideally, we would use a “donut” width that is consistent with the main analysis, but we are restricted by the categories in which the frequency tabulations were provided by the data custodian.

## Appendix D: Sensitivity of Results to ‘Donut’ Size

Table D1  
Sensitivity of Key Estimates to ‘Donut’ Size

Outcomes	Donut Size in Days from Each Birthday			
	1	7	14	30
<b><u>Drinking Behavior</u></b>				
Ever drinks	0.213*** (0.051)	0.165*** (0.039)	0.150*** (0.049)	0.186*** (0.052)
bandwidth	302	498	373	312
Drinks regularly	0.226*** (0.036)	0.200*** (0.048)	0.182*** (0.052)	0.179*** (0.064)
bandwidth	637	377	344	270
Proportion of days drinks	0.054*** (0.012)	0.045*** (0.014)	0.040*** (0.015)	0.041** (0.017)
bandwidth	449	312	301	284
Binge drinks regularly	0.186** (0.073)	0.124 (0.082)	0.203*** (0.078)	0.190* (0.106)
bandwidth	444	388	438	292
Proportion of days binge drinks	0.033** (0.017)	0.034* (0.018)	0.037** (0.019)	0.044*** (0.014)
bandwidth	392	368	359	670
<b><u>Motor Vehicle Accidents</u></b>				
MVAs Involving a Vehicle Being Towed, Injury, or Death	-0.0529 (0.1823)	-0.0625 (0.2067)	-0.0070 (0.2000)	0.1184 (0.2702)
bandwidth	118	104	110	100
Injuries in MVAs	0.0003 (0.0585)	-0.0154 (0.0771)	-0.0415 (0.0655)	-0.0313 (0.1077)
bandwidth	182	128	181	117
Deaths in MVAs	0.0003 (0.0055)	0.0007 (0.0054)	-0.0005 (0.0062)	0.0010 (0.0083)
bandwidth	224	232	193	152
Mortality-Risk-Weighted MVAs	-0.0009 (0.0017)	0.0002 (0.0018)	-0.0003 (0.0022)	0.0023 (0.0027)
bandwidth	207	239	181	184
MVAs with Mortality Risk > 0.05	-0.0102 (0.0139)	-0.0034 (0.0143)	-0.0106 (0.0164)	-0.0038 (0.0145)
bandwidth	209	218	191	245
MVAs with Mortality Risk > 0.010	0.0041 (0.0077)	0.0057 (0.0087)	0.0030 (0.0089)	0.0112 (0.0094)
bandwidth	192	187	172	195
MVAs Involving a Vehicle Being Towed, Injury, or Death	-0.1163 (0.0945)	-0.0765 (0.0983)	0.0215 (0.0923)	-0.0014 (0.1489)
bandwidth	117	95	113	77
Injuries in MVAs	0.0357 (0.0345)	0.0388 (0.0400)	0.0849*** (0.0304)	0.0336 (0.0559)
bandwidth	156	143	288	127
Deaths in MVAs	-0.0038 (0.0035)	-0.0014 (0.0035)	-0.0028 (0.0041)	0.0006 (0.0055)
bandwidth	197	212	181	134
Mortality-Risk-Weighted MVAs	0.0005 (0.0012)	0.0015 (0.0011)	-0.0024 (0.0027)	-0.0017 (0.0023)
bandwidth	223	287	85	103
MVAs with Mortality Risk > 0.05	-0.0025 (0.0088)	0.0007 (0.0085)	-0.0072 (0.0145)	0.0084 (0.0084)
bandwidth	225	246	148	260
MVAs with Mortality Risk > 0.010	0.0029 (0.0059)	0.0100 (0.0080)	0.0009 (0.0064)	-0.0020 (0.0071)
bandwidth	152	118	182	122
<b><u>Hospitalizations</u></b>				
Alcohol Intoxication/Poisoning	5.339*** (1.513)	4.277*** (1.499)	4.051** (1.726)	2.390 (2.035)
bandwidth	305	293	253	203
Driver in Motor Vehicle Accident	-1.212 (1.753)	-0.969 (1.766)	-2.587 (1.752)	-4.299*** (1.433)
bandwidth	209	234	255	431
Rider in Motor Cycle Accident	2.928** (1.380)	2.046 (1.383)	3.640** (1.538)	2.924* (1.605)
bandwidth	363	401	342	313
Assault	7.357*** (1.899)	7.144*** (1.899)	6.173*** (1.620)	0.979 (3.804)
bandwidth	288	308	458	148
Any Other External Cause	3.603 (5.894)	-0.303 (5.285)	-0.735 (4.899)	-6.220 (7.677)
bandwidth	339	432	488	254

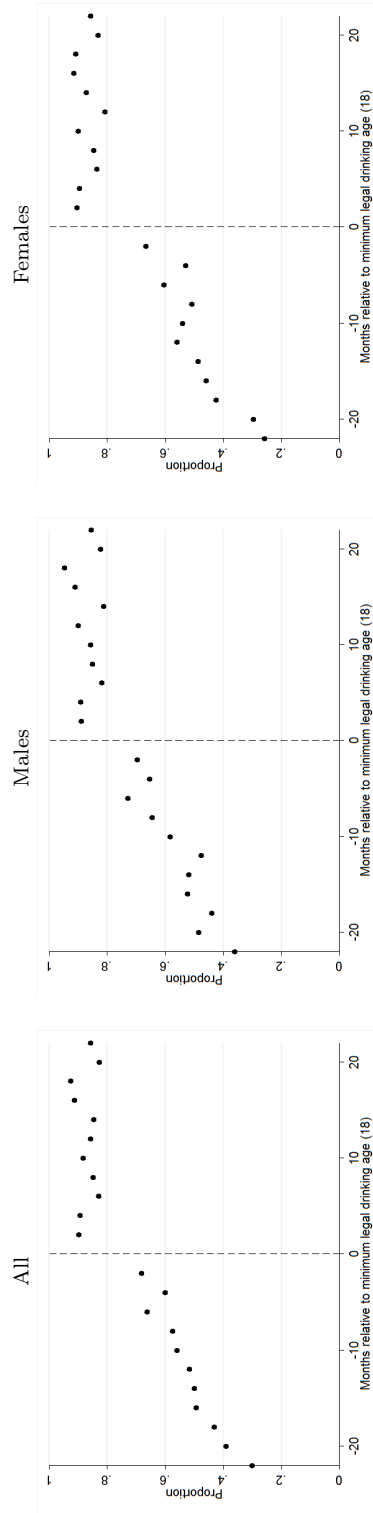
Notes: This Table considers sensitivity of the key estimates to alternate “donut” sizes. For each indicator and donut size, it shows the estimated discontinuity, robust standard error, and the bandwidth used, selected by the procedure described in Imbens and Kalyanaraman (2012).

## **Appendix E: Estimated Effects on Across Gender**

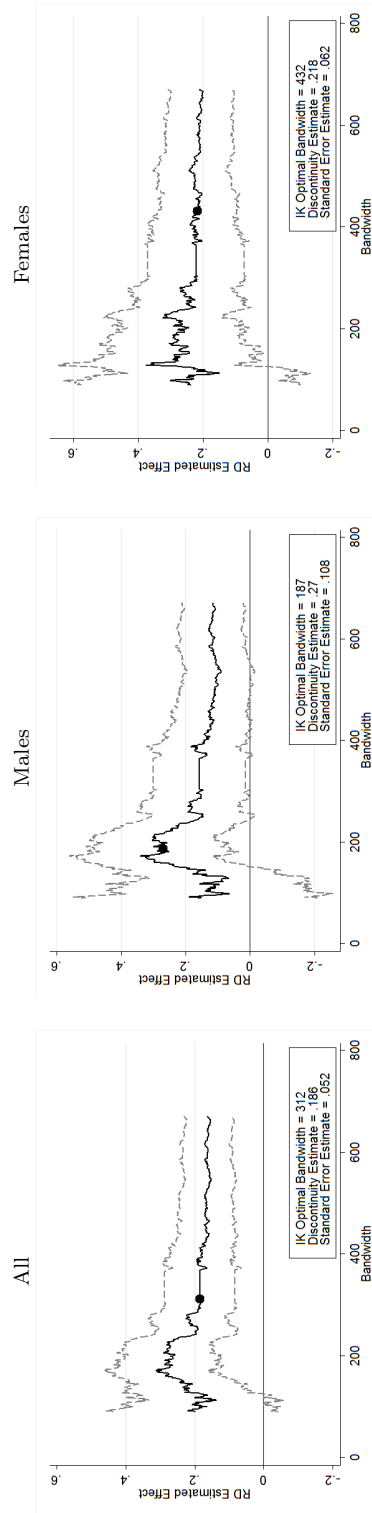
This appendix shows estimated effects across gender for all of the outcomes considered in the main text.

Figure E1  
Ever Drinks

Panel A: Means



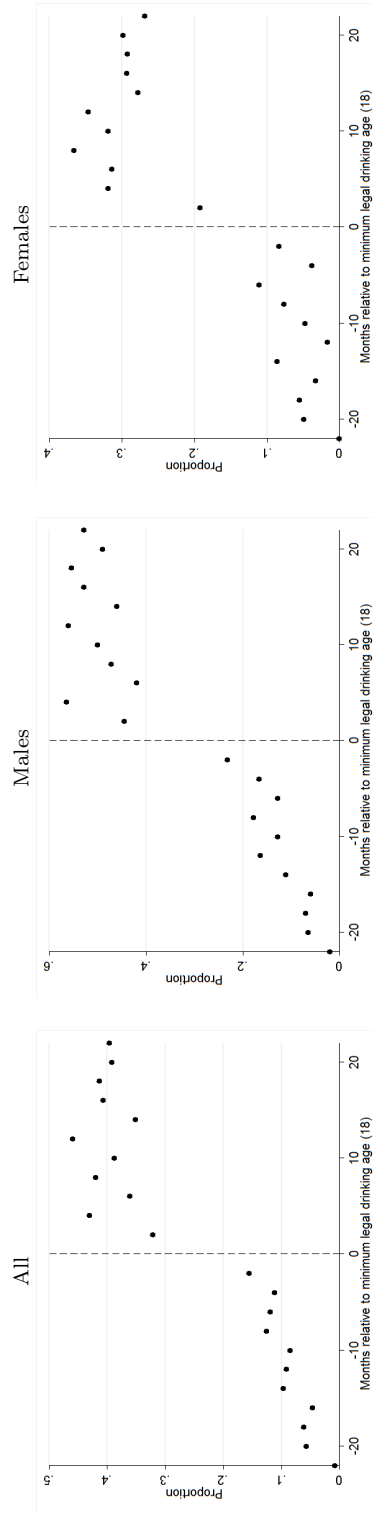
Panel B: Discontinuity Estimates By Bandwidth (In Days)



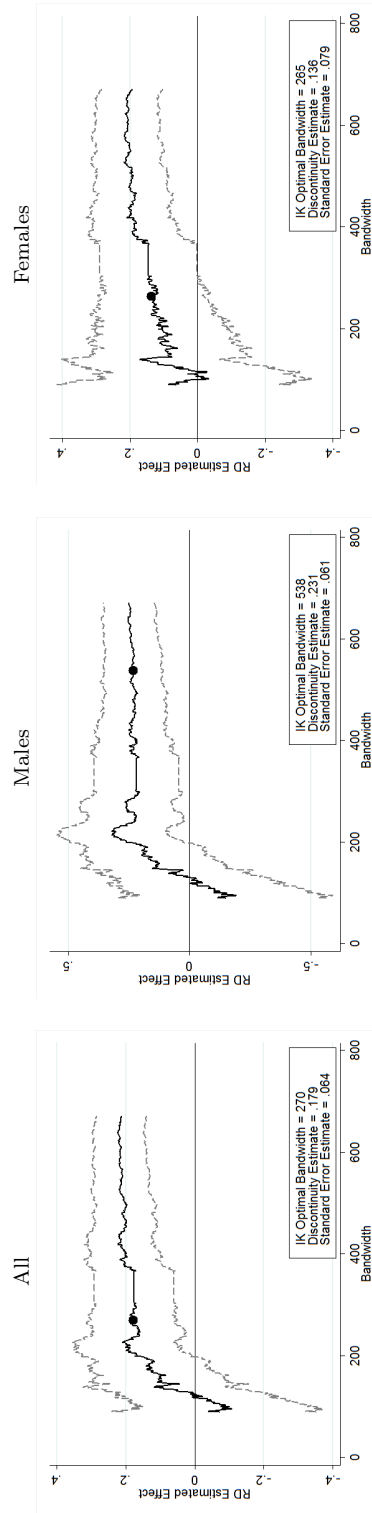
Notes: See Figure 3 notes.

Figure E2  
Drinks  $\geq$  Once Per Week

Panel A: Means



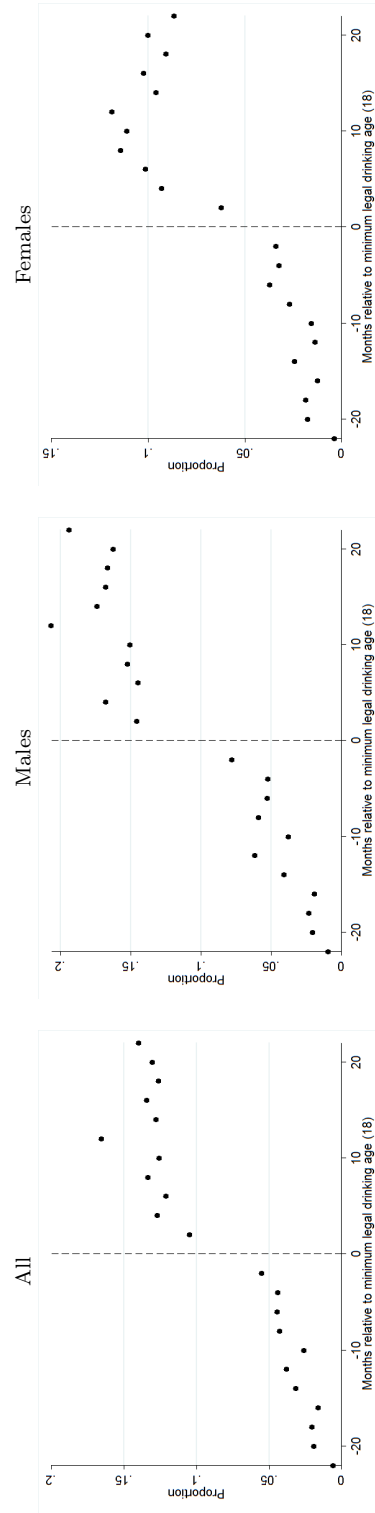
Panel B: Discontinuity Estimates By Bandwidth (In Days)



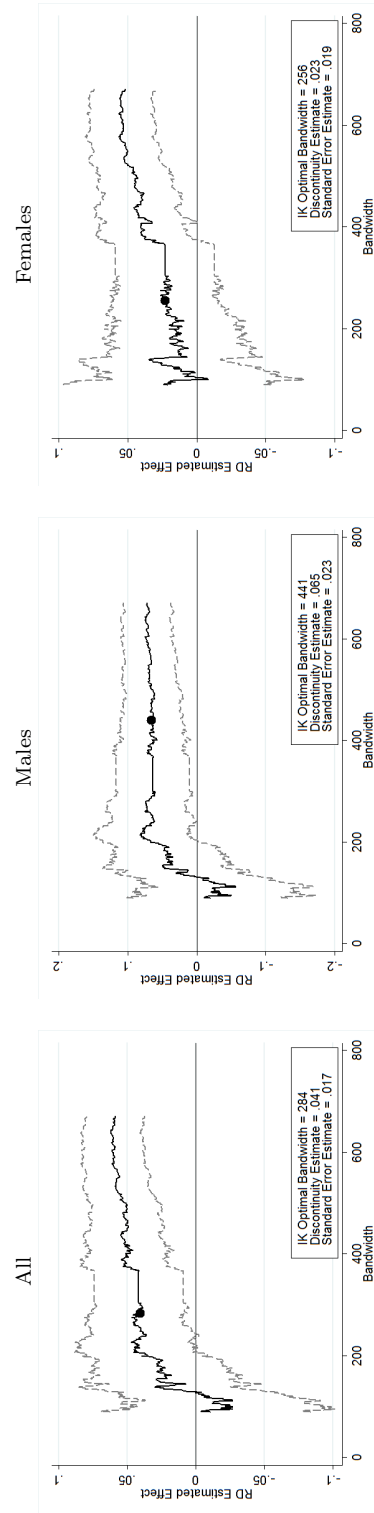
Notes: See Figure 3 notes.

Figure E3  
Proportion of Days Drinks

Panel A: Means



Panel B: Discontinuity Estimates By Bandwidth (In Days)

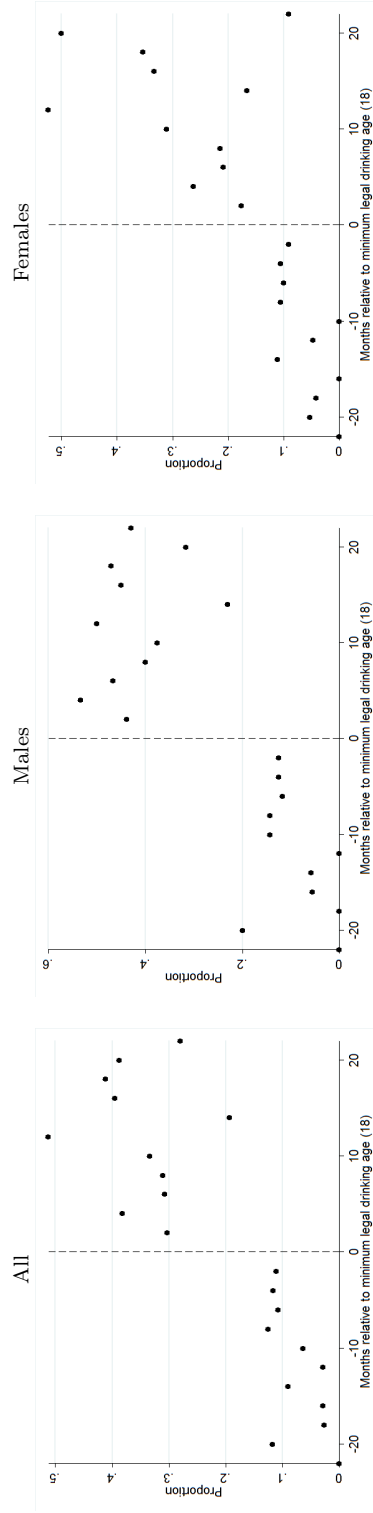


Notes: See Figure 3 notes.

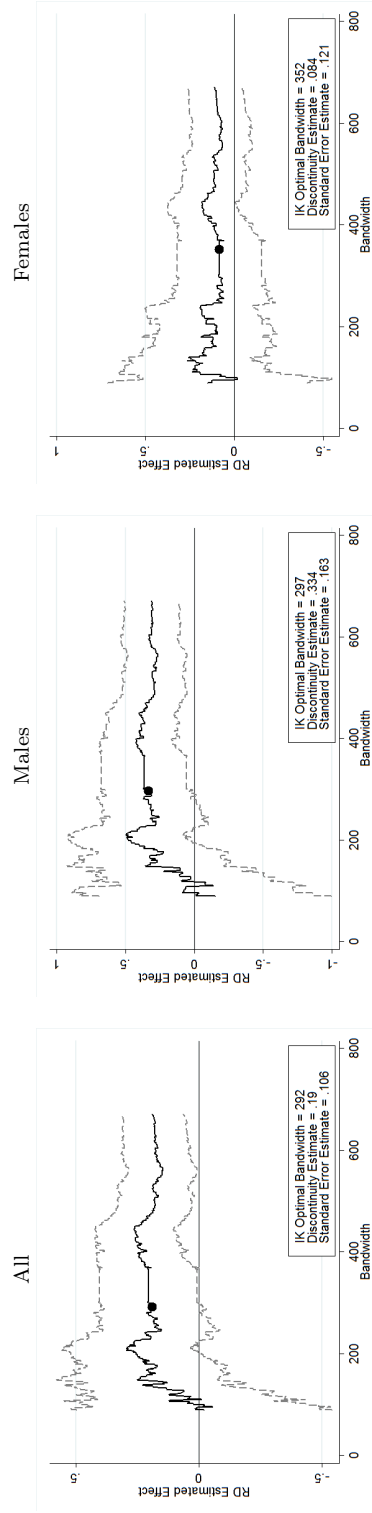


Figure E4  
Binge Drinks  $\geq$  Once Per Week

Panel A: Means



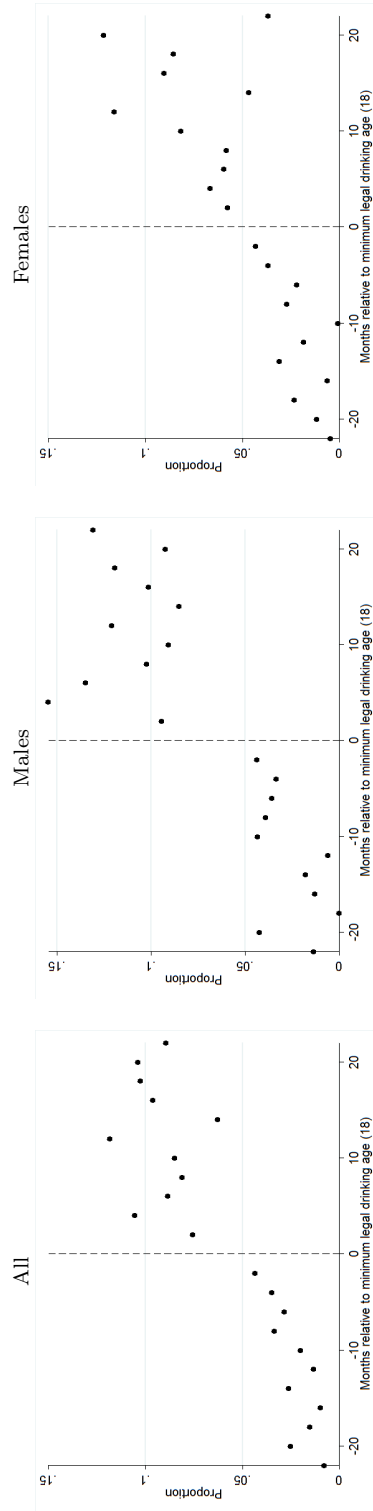
Panel B: Discontinuity Estimates By Bandwidth (In Days)



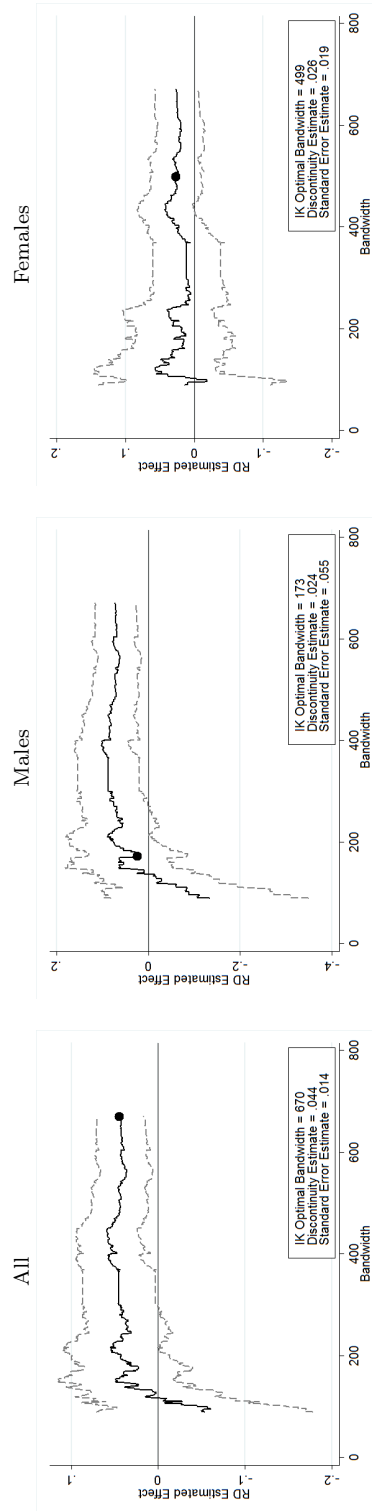
Notes: See Figure 3 notes and Figure 4 notes.

Figure E5  
Proportion of Days Binge Drinks

Panel A: Means



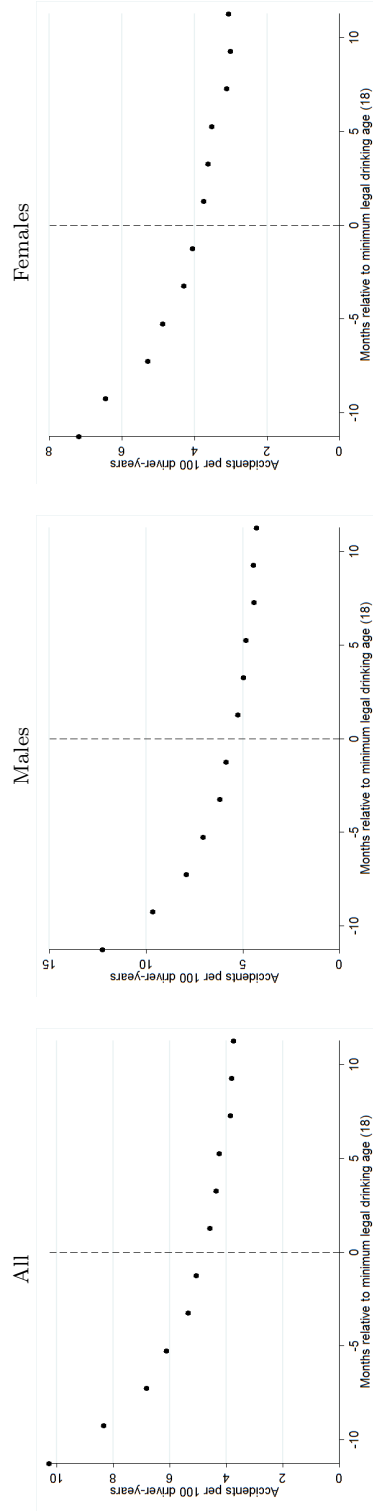
Panel B: Discontinuity Estimates By Bandwidth (In Days)



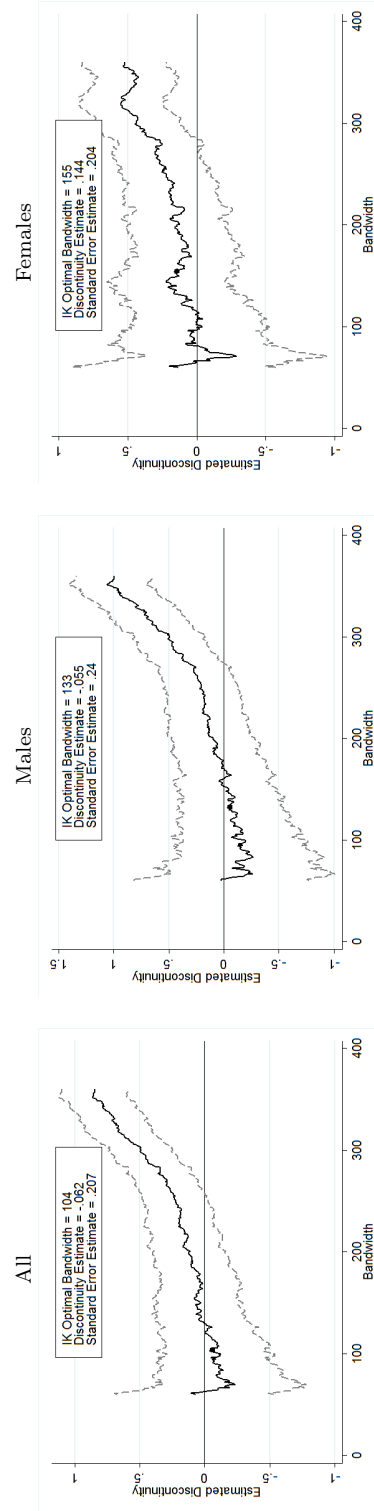
Notes: See Figure 3 notes and Figure 4 notes.

Figure E6  
All Serious Motor Vehicle Accidents

Panel A: Means



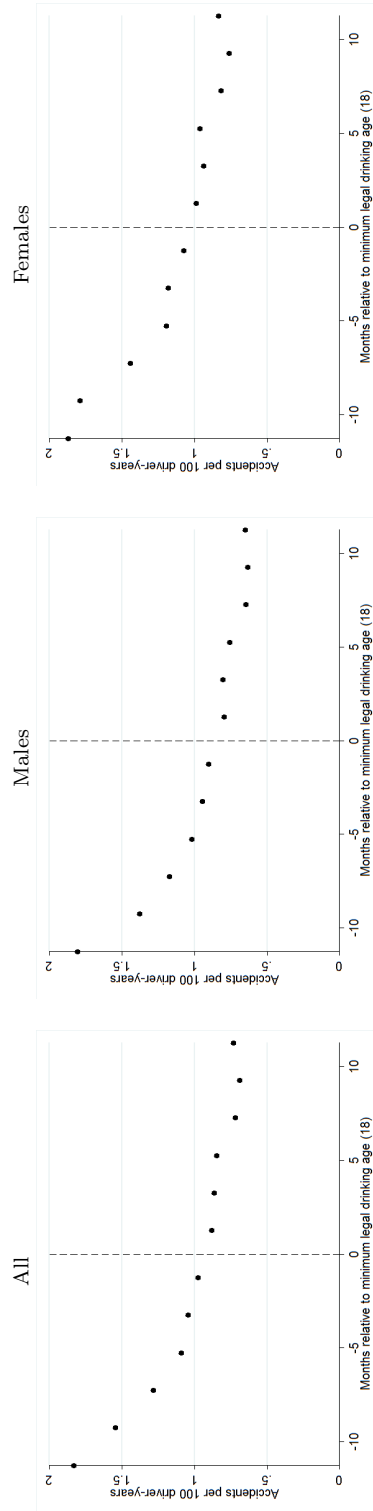
Panel B: Discontinuity Estimates By Bandwidth (In Days)



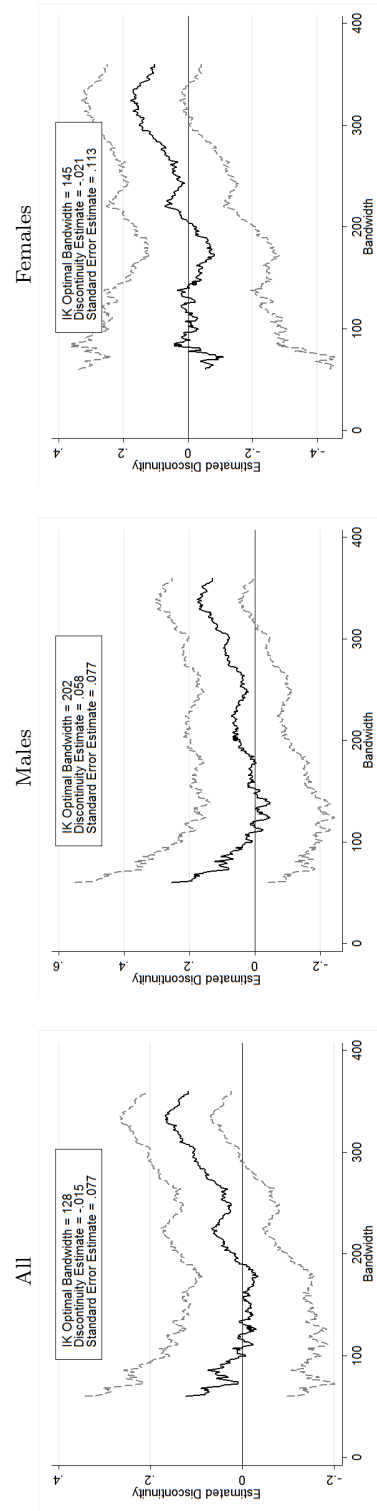
Notes: See Figure 5 notes.

Figure E7  
Injured in a Motor Vehicle Accident

Panel A: Means



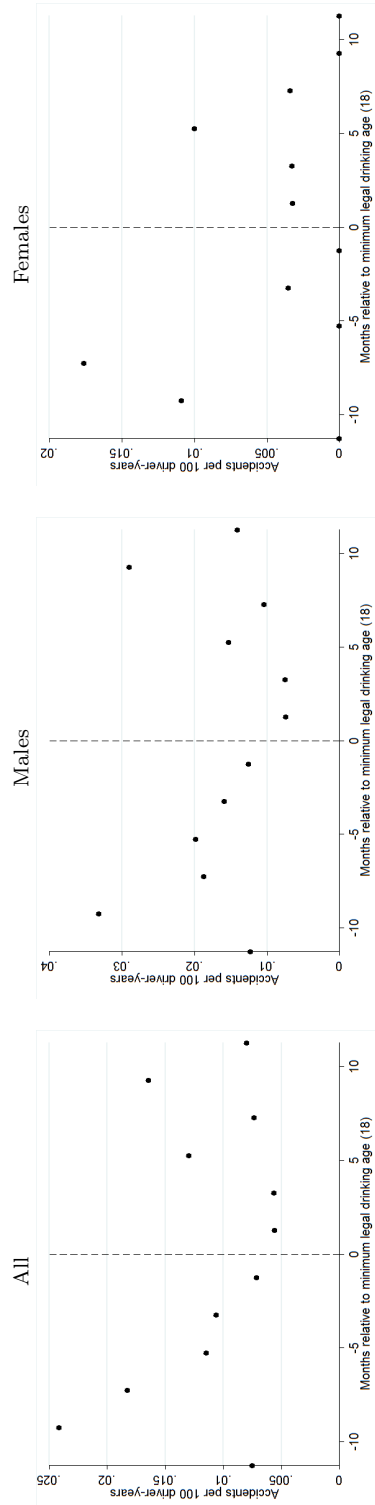
Panel B: Discontinuity Estimates By Bandwidth (In Days)



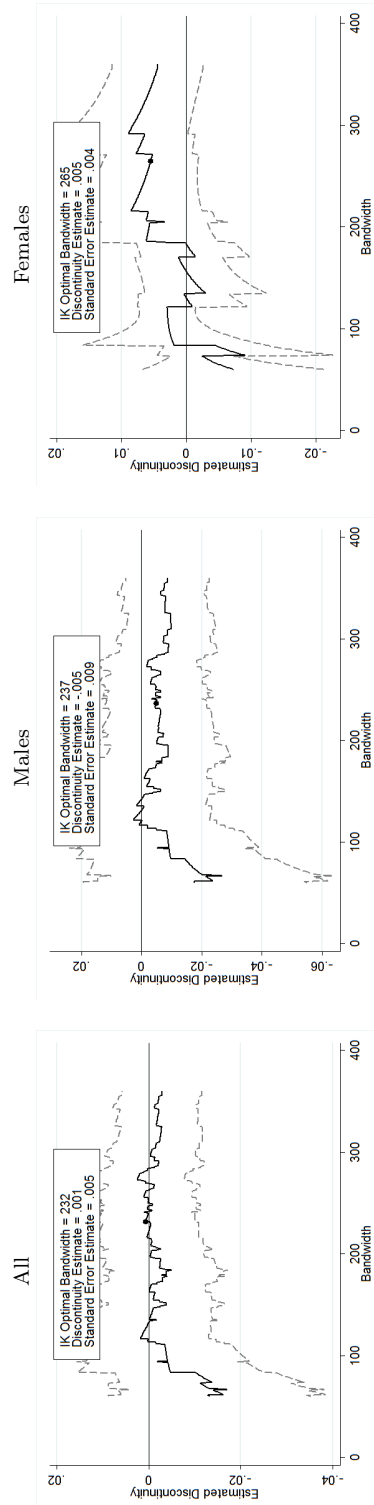
Notes: See Figure 5 notes.

Figure E8  
Killed in a Motor Vehicle Accident

Panel A: Means



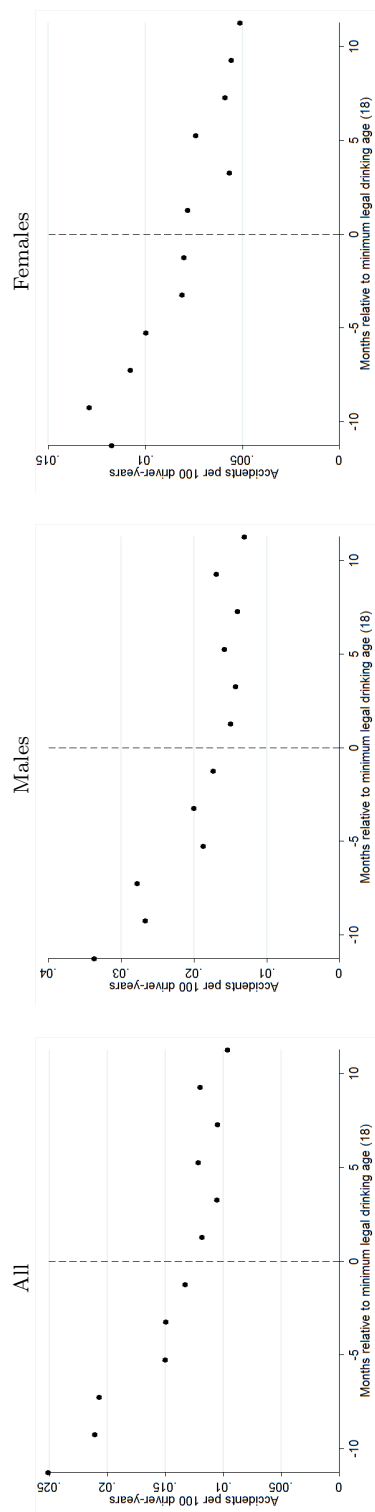
Panel B: Discontinuity Estimates By Bandwidth (In Days)



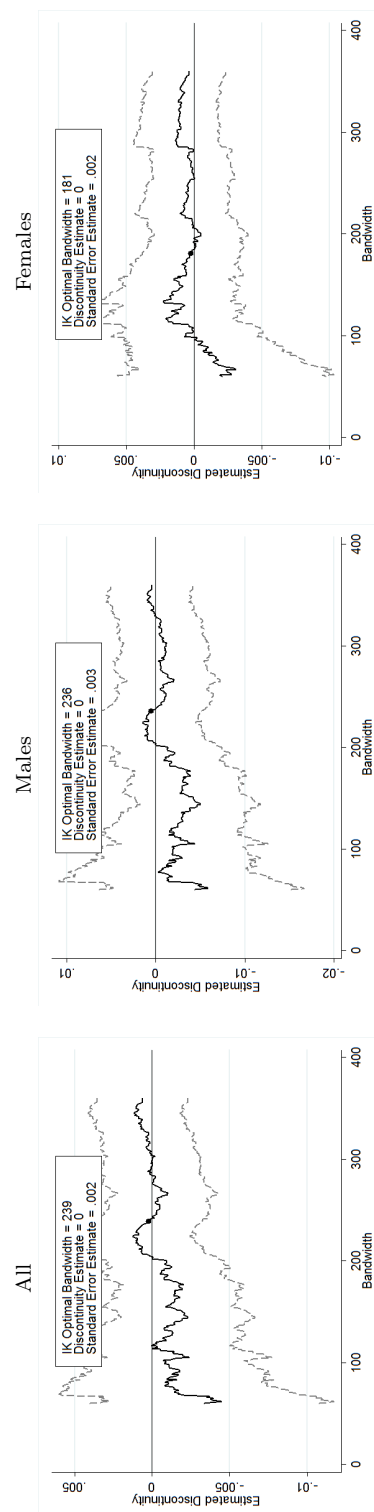
Notes: See Figure 5 notes.

Figure E9  
Fatality-Risk-Weighted Motor Vehicle Accidents

Panel A: Means



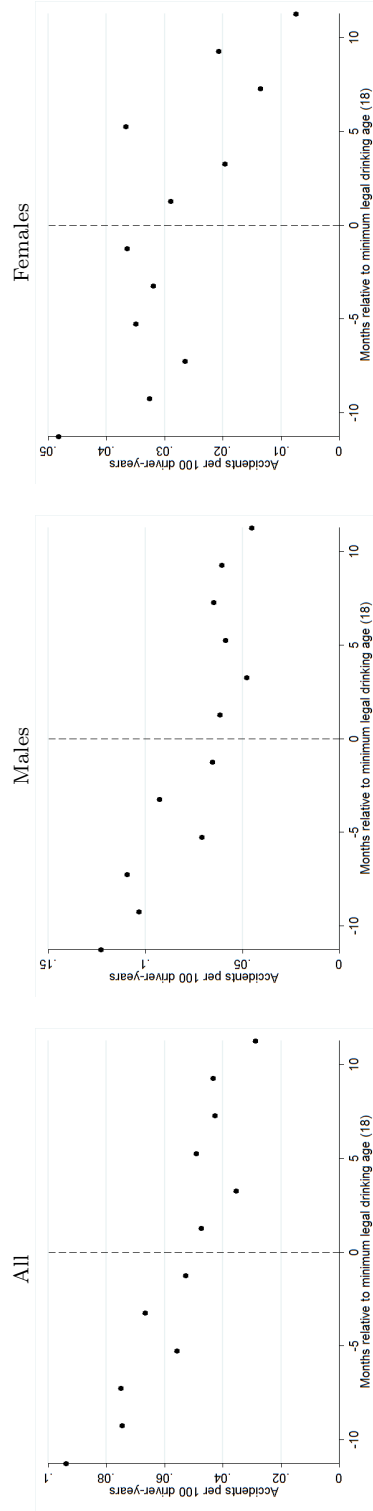
Panel B: Discontinuity Estimates By Bandwidth (In Days)



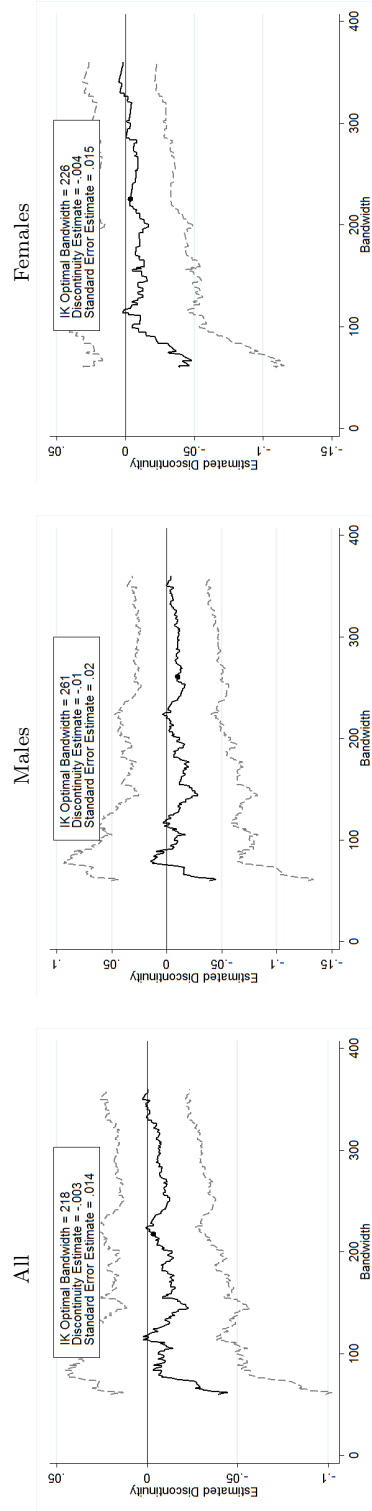
Notes: See Figure 5 notes and Figure 6 notes.

Figure E10  
High Fatality Risk Accidents ( $> 0.05$ )

Panel A: Means



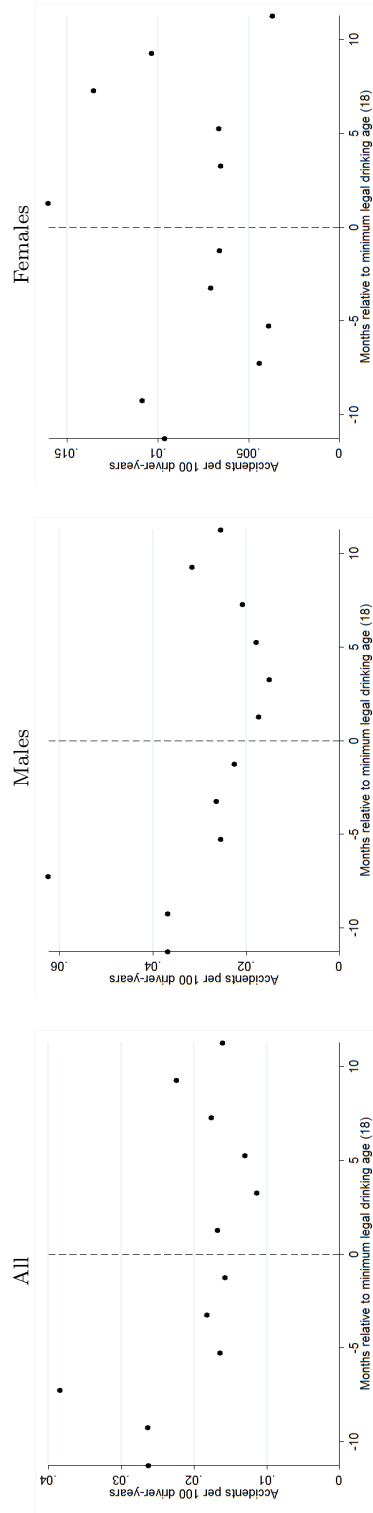
Panel B: Discontinuity Estimates By Bandwidth (In Days)



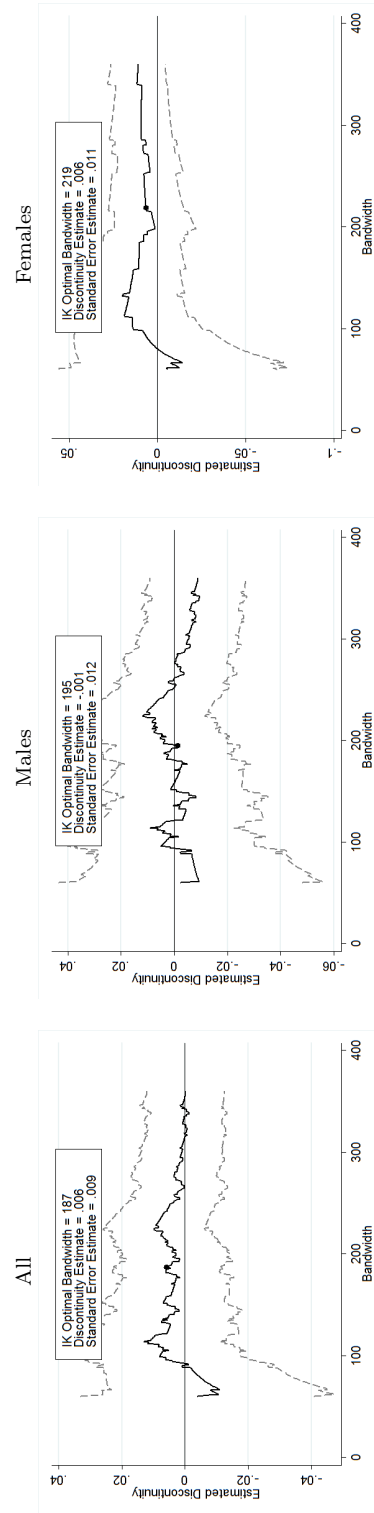
Notes: See Figure 5 notes and Figure 6 notes.

Figure E11  
Very High Fatality Risk Accidents ( $> 0.10$ )

Panel A: Means



Panel B: Discontinuity Estimates By Bandwidth (In Days)

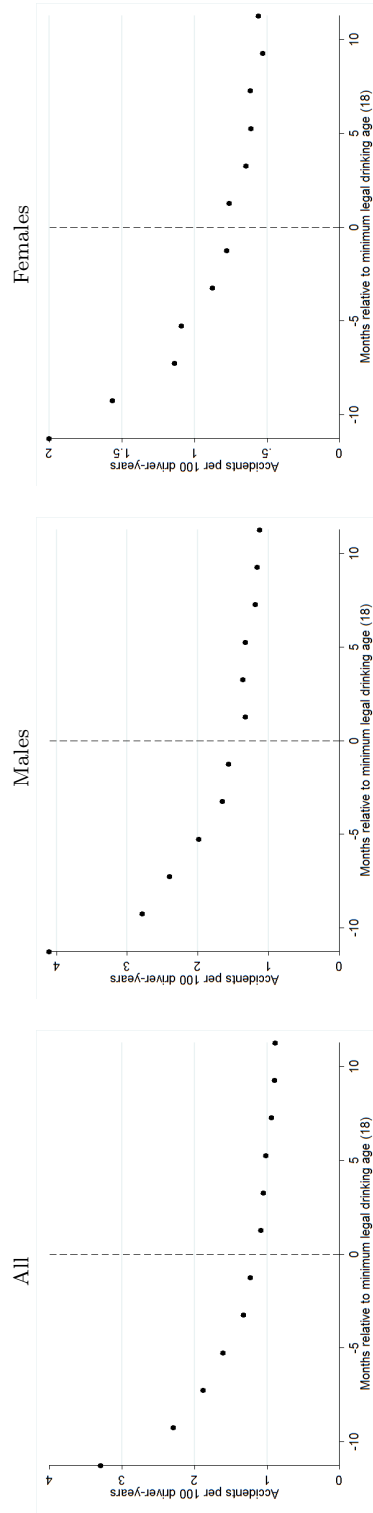


Notes: See Figure 5 notes and Figure 6 notes.

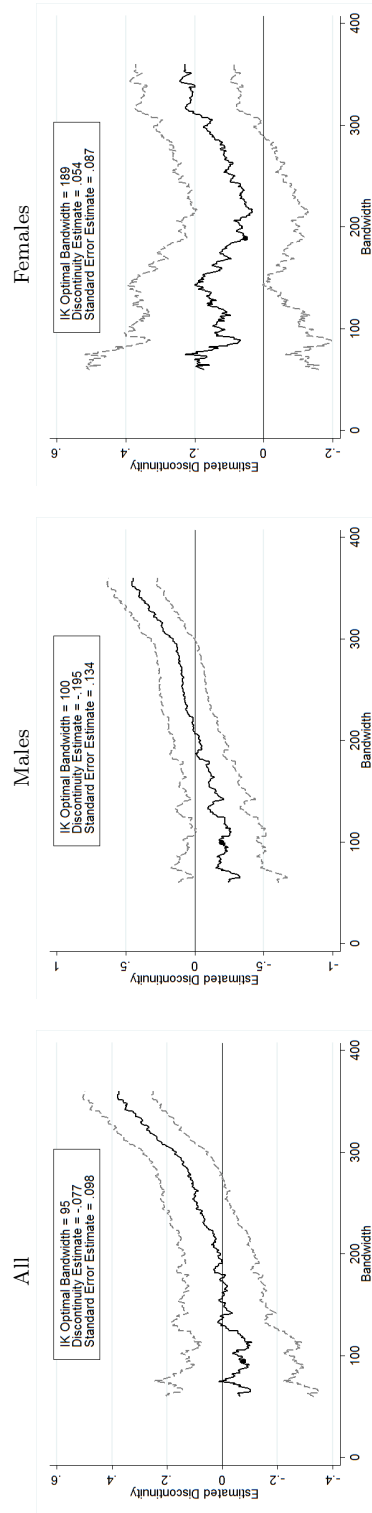


Figure E12  
All Serious Motor Vehicle Accidents at Night

Panel A: Means



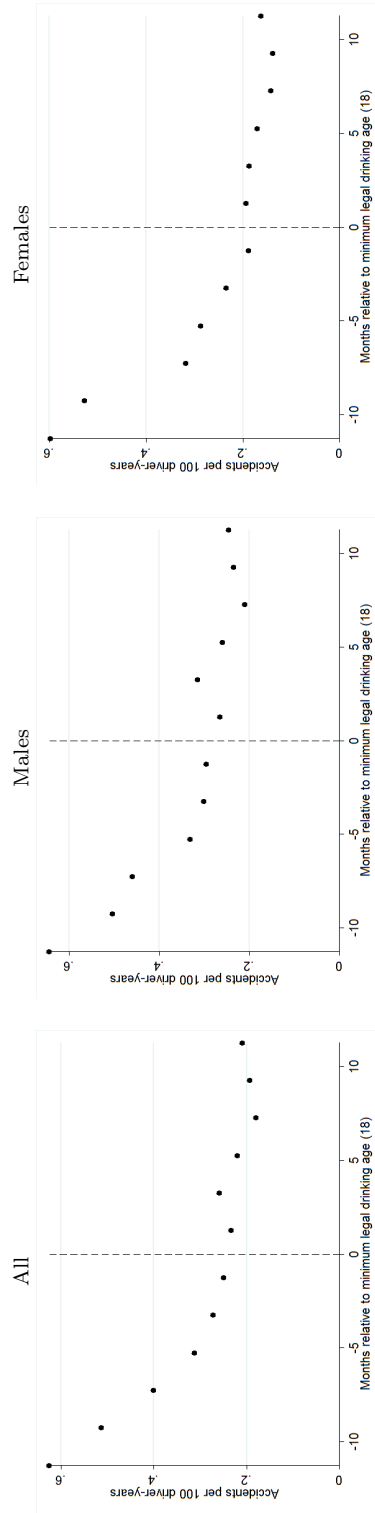
Panel B: Discontinuity Estimates By Bandwidth (In Days)



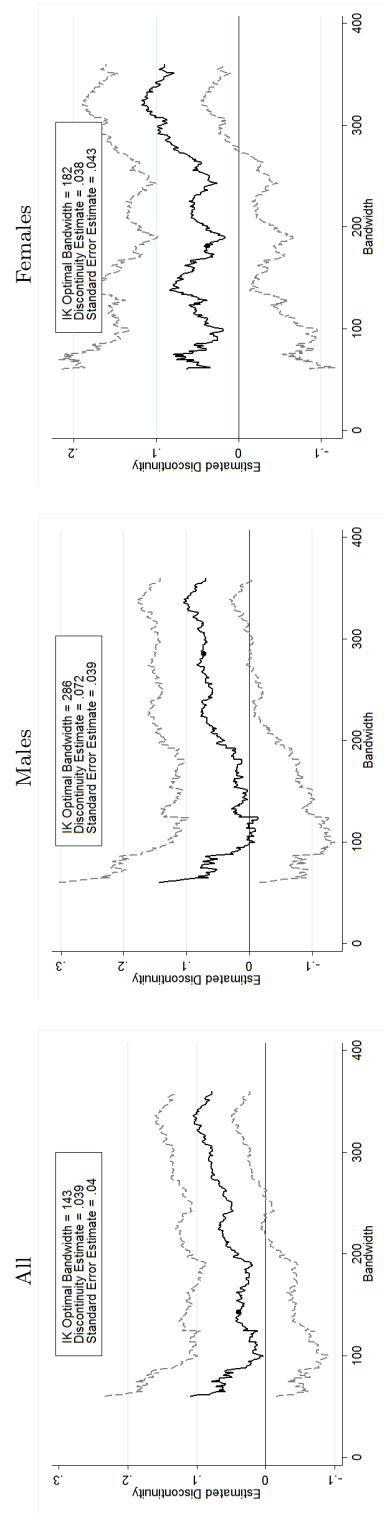
Notes: See Figure 5 notes.

Figure E13  
Injured in a Motor Vehicle Accident at Night

Panel A: Means



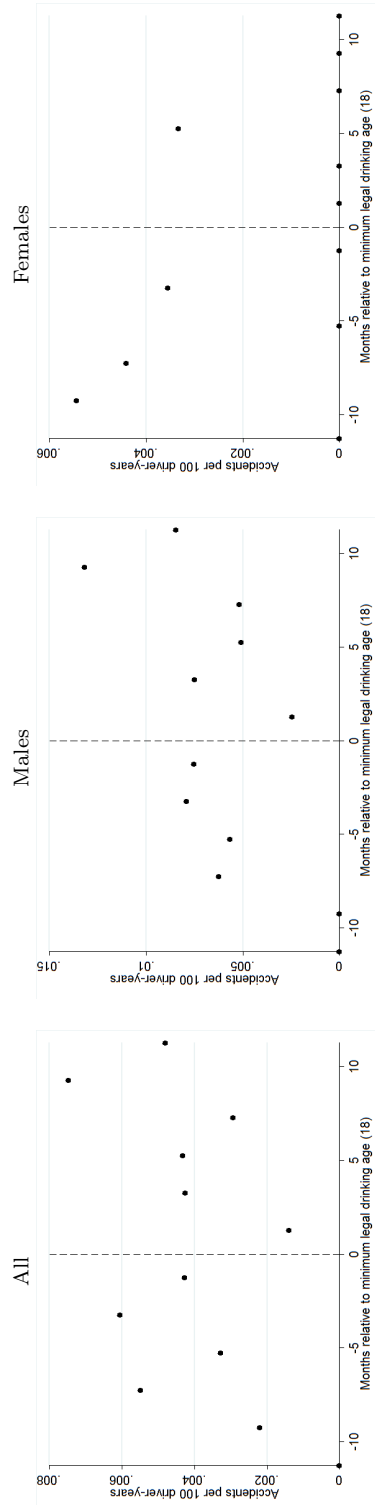
Panel B: Discontinuity Estimates By Bandwidth (In Days)



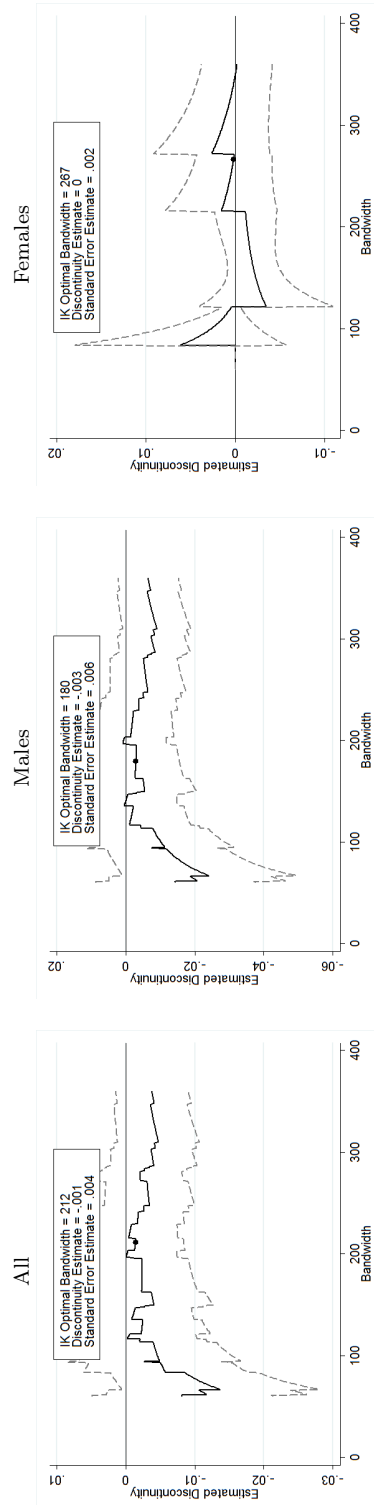
Notes: See Figure 5 notes.

Figure E14  
Killed in a Motor Vehicle Accident at Night

Panel A: Means

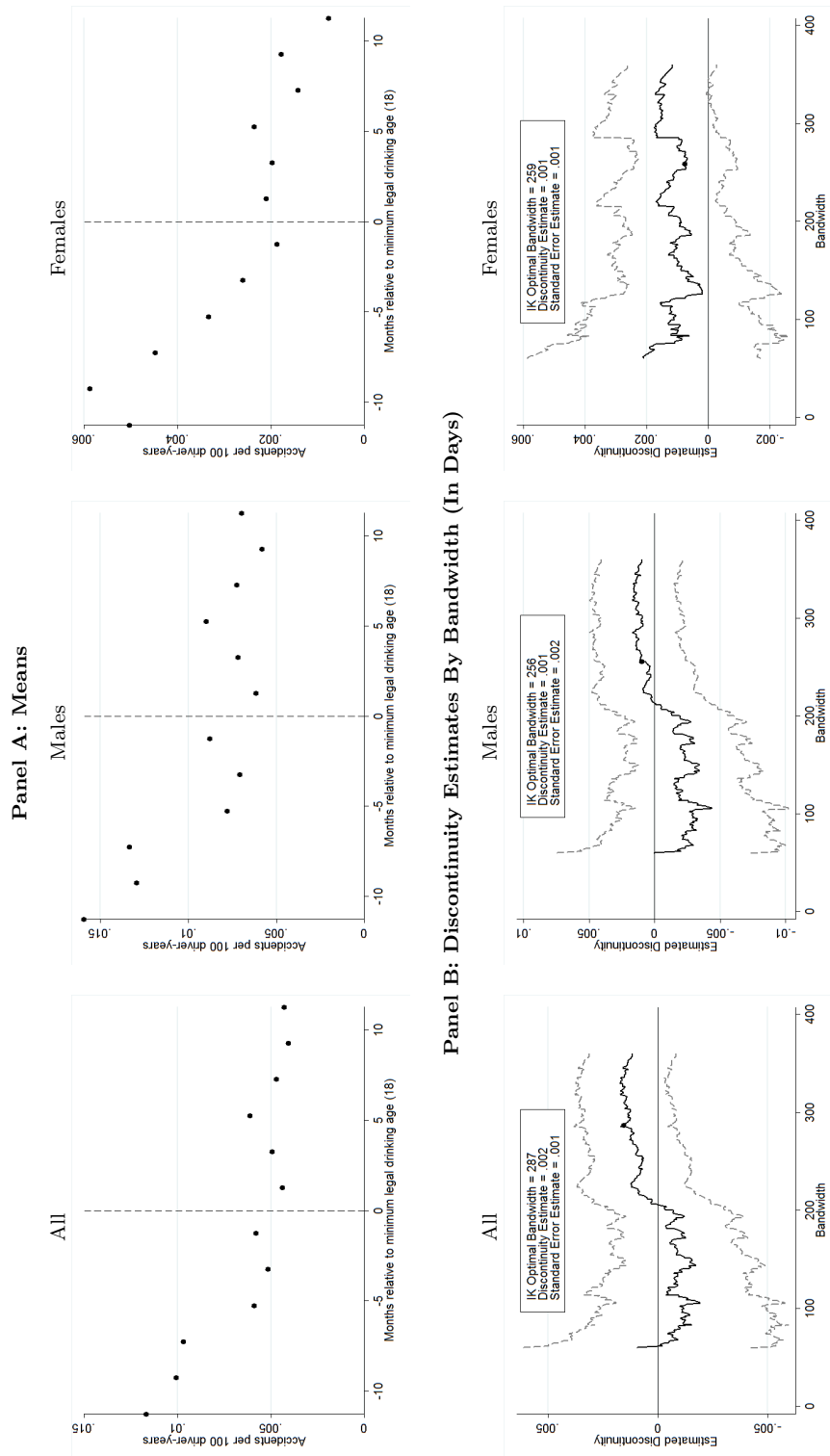


Panel B: Discontinuity Estimates By Bandwidth (In Days)



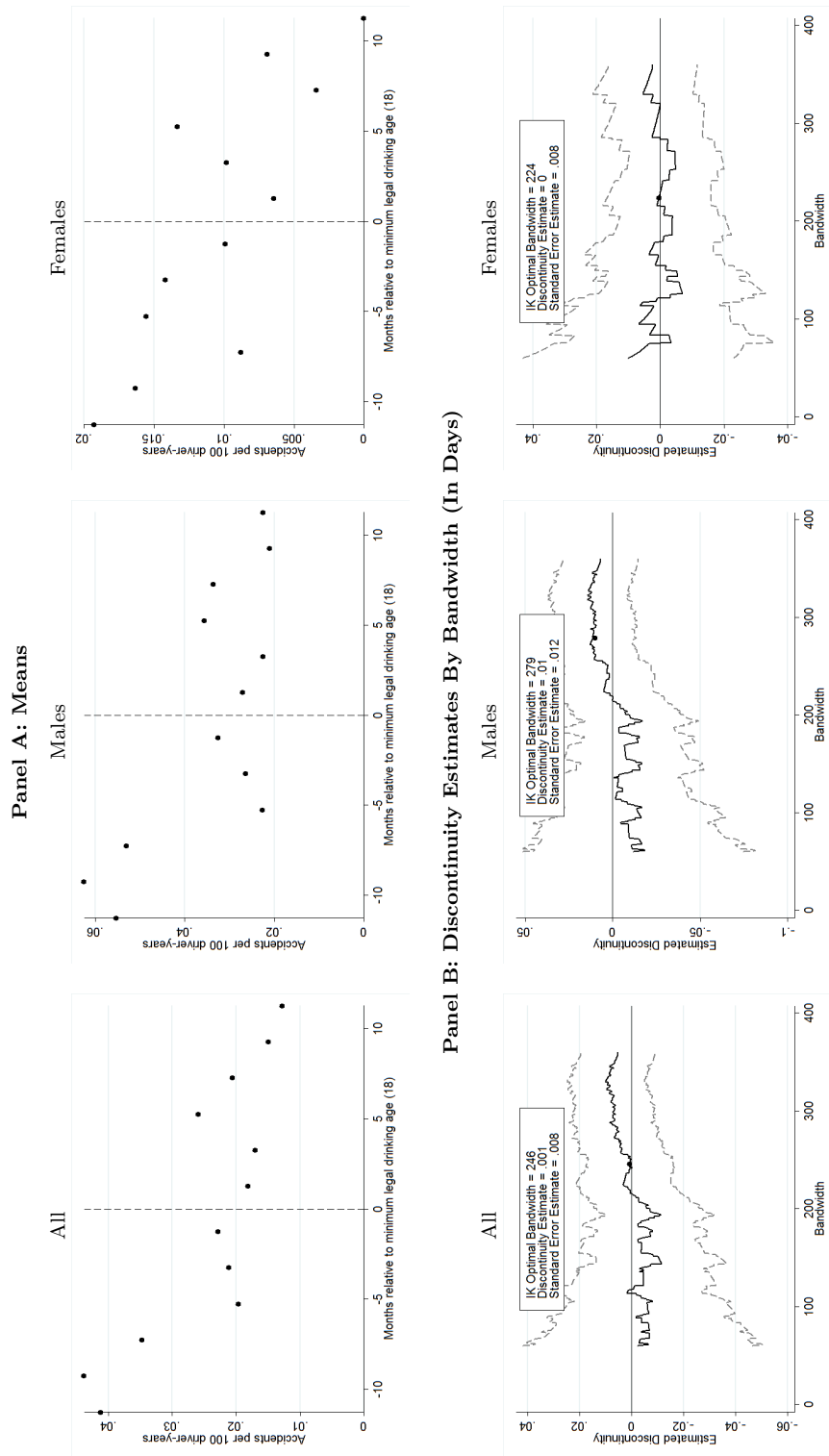
Notes: See Figure 5 notes.

Figure E15  
 Fatality-Risk-Weighted Motor Vehicle Accidents at Night



Notes: See Figure 5 notes and Figure 6 notes.

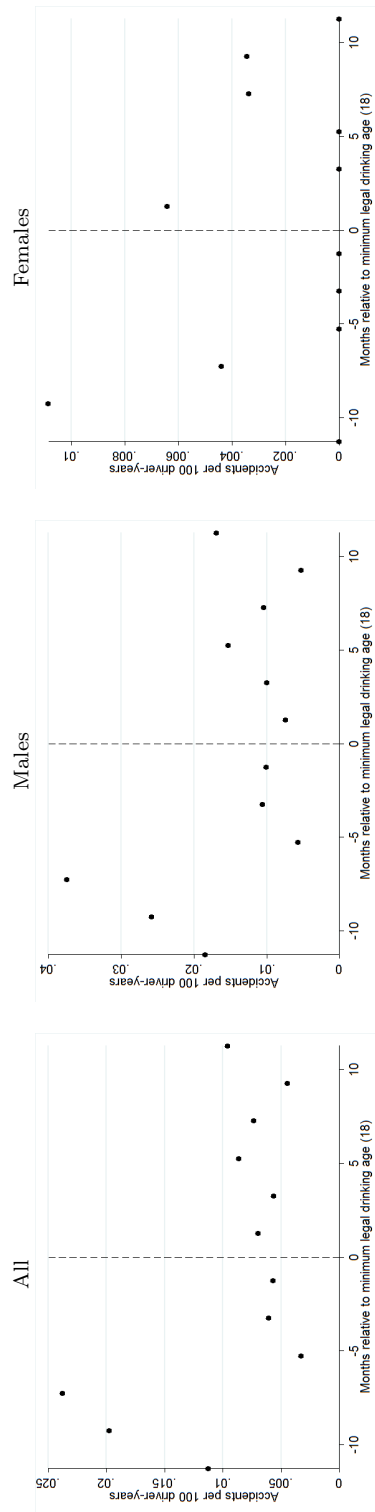
Figure E16  
High Fatality Risk Accidents ( $> 0.05$ ) at Night



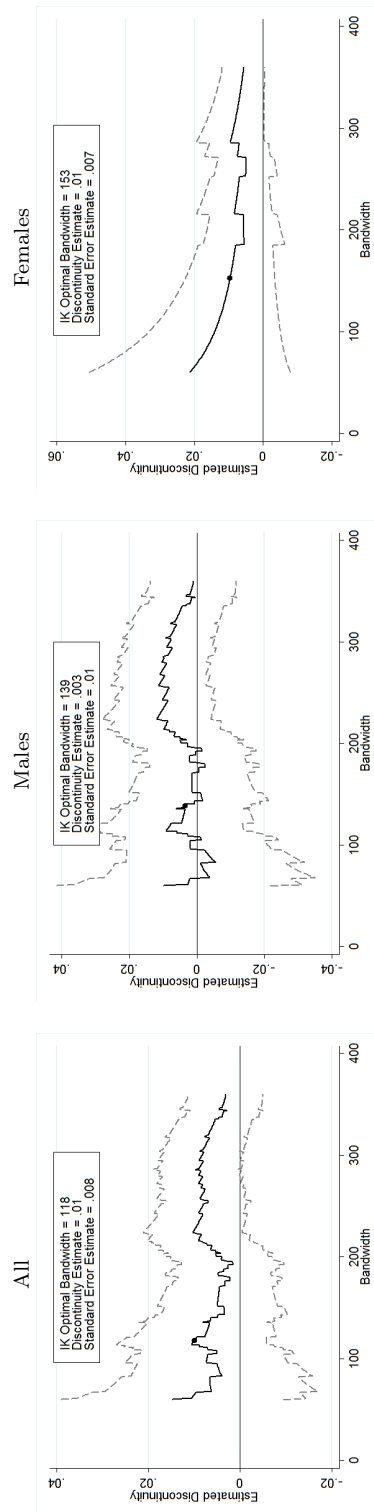
Notes: See Figure 5 notes and Figure 6 notes.

Figure E17  
Very High Fatality Risk Accidents (> 0.10) at Night

Panel A: Means



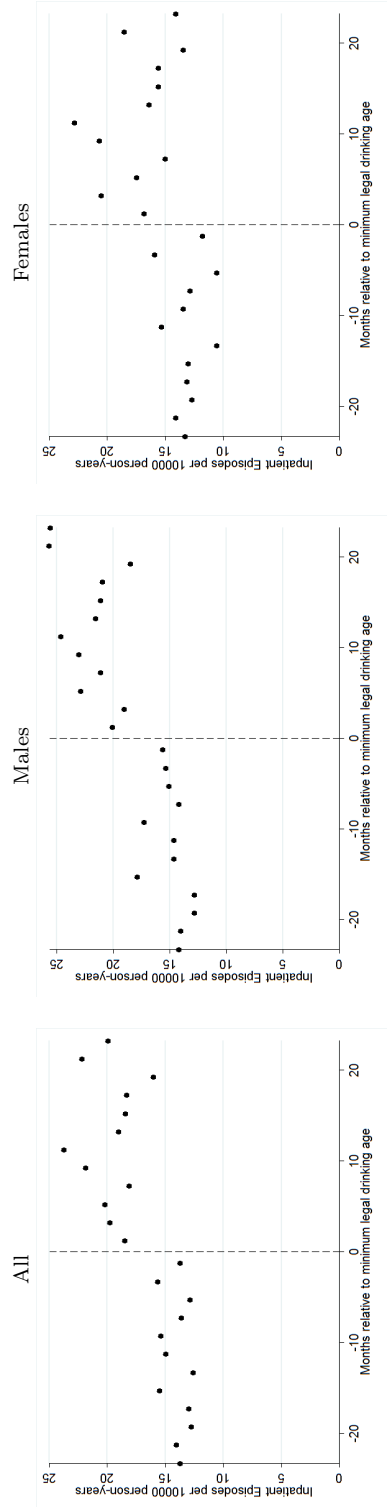
Panel B: Discontinuity Estimates By Bandwidth (In Days)



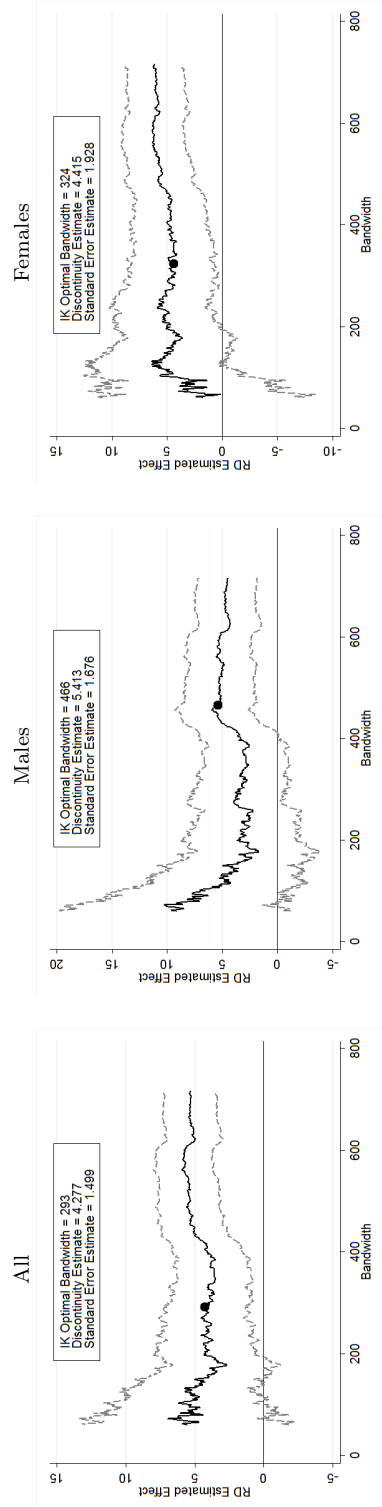
Notes: See Figure 5 notes and Figure 6 notes.

Figure E18  
Hospitalizations for Alcohol Intoxication/Poisoning

Panel A: Means



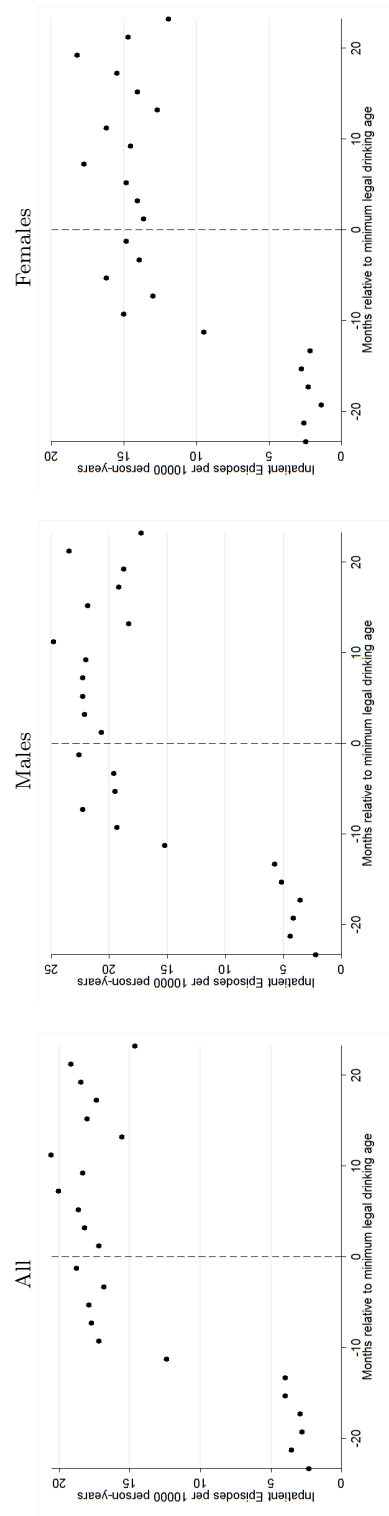
Panel B: Discontinuity Estimates By Bandwidth (In Days)



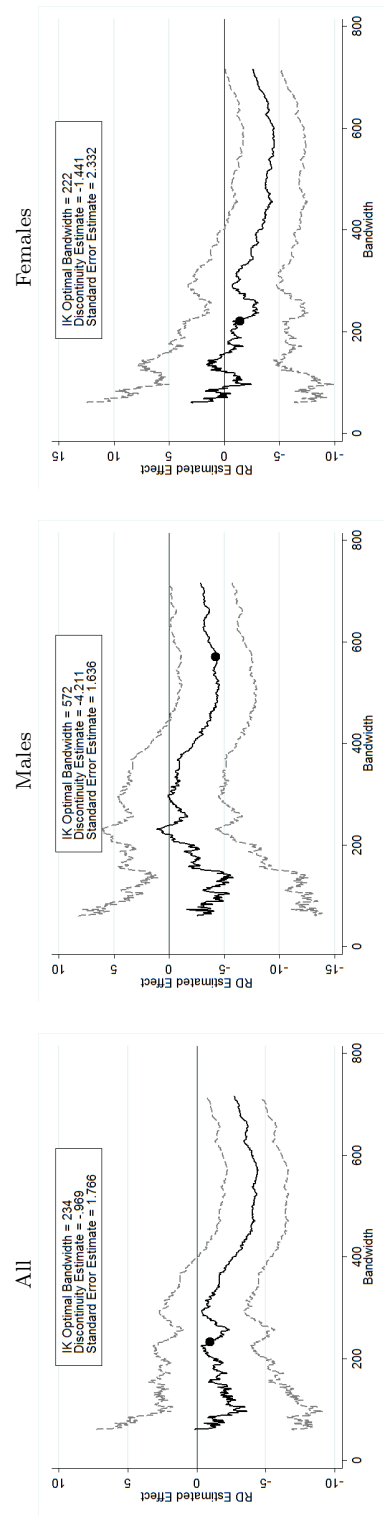
Notes: See Figure 9 notes.

Figure E19  
Hospitalizations for Motor Vehicle Accidents

Panel A: Means



Panel B: Discontinuity Estimates By Bandwidth (In Days)



Notes: See Figure 9 notes.



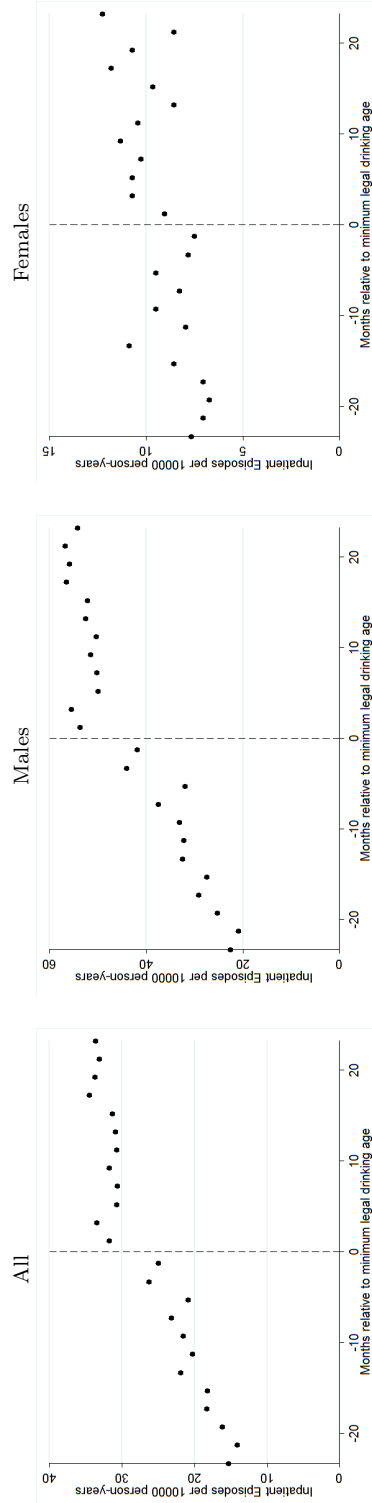
Figure E20



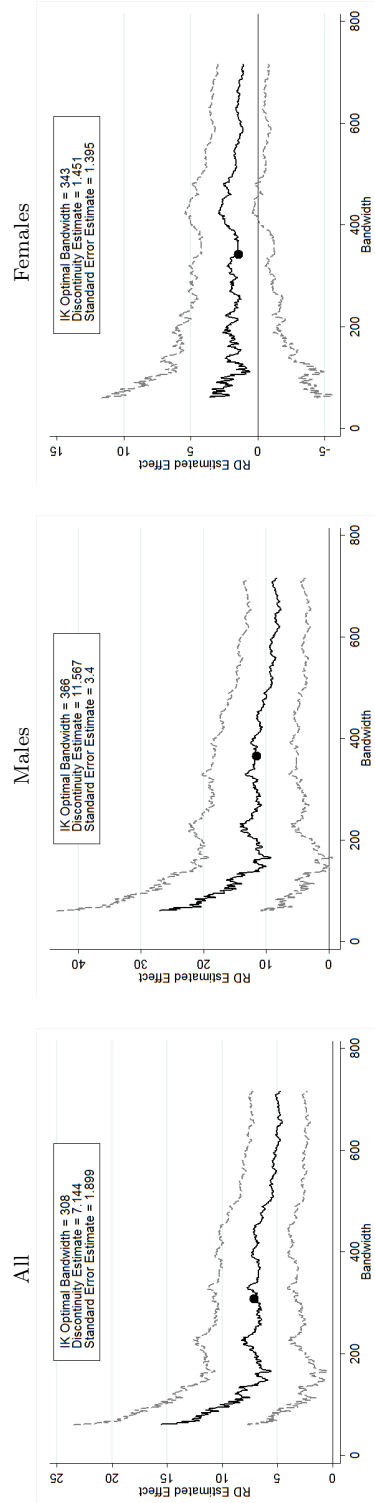
Notes: See Figure 9 notes.

Figure E21  
Hospitalizations for Assaults

Panel A: Means



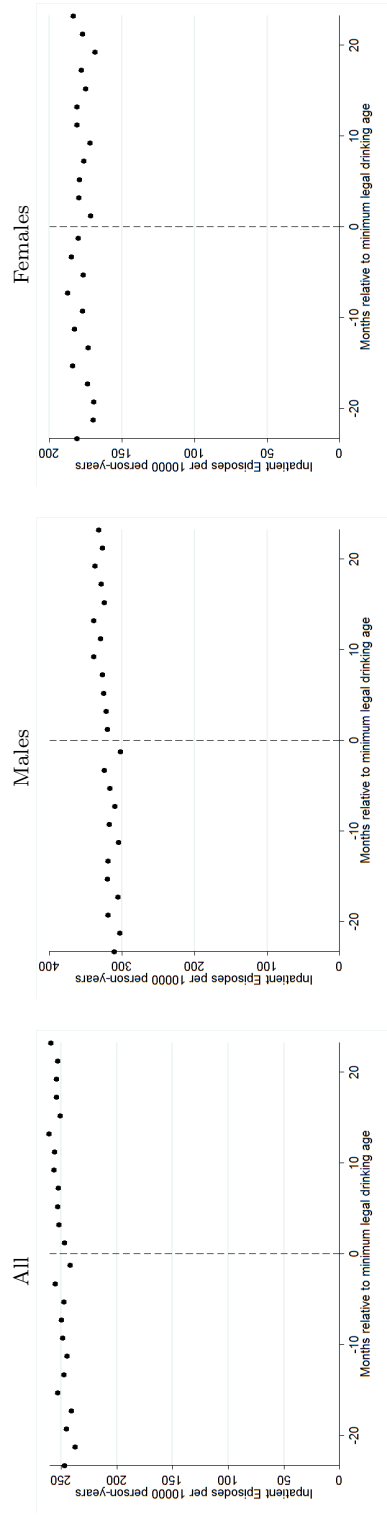
Panel B: Discontinuity Estimates By Bandwidth (In Days)



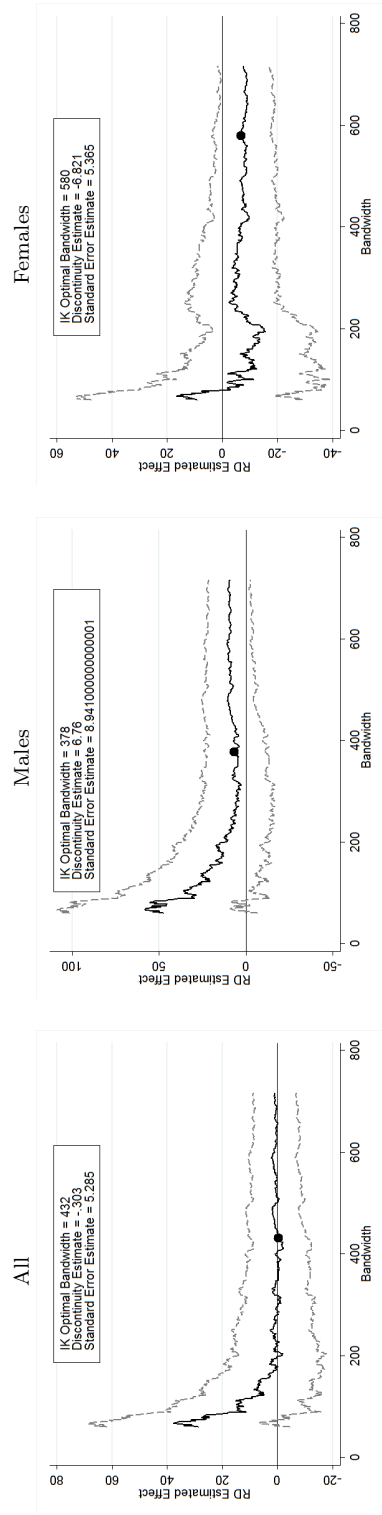
Notes: See Figure 9 notes.

Figure E22  
Hospitalizations for Other External Causes

Panel A: Means



Panel B: Discontinuity Estimates By Bandwidth (In Days)



Notes: See Figure 9 notes.