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The Effect of Alcohol Consumption on Mortality: Regression Discontinuity Evidence from the Minimum Drinking Age[†]

By Christopher Carpenter and Carlos Dobkin[*]

We estimate the effect of alcohol consumption on mortality using the minimum drinking age in a regression discontinuity design. We find large and immediate increases in drinking at age 21, including a 21 percent increase in recent drinking days. We also find a discrete 9 percent increase in the mortality rate at age 21, primarily due to motor vehicle accidents, alcohol-related deaths, and suicides. We estimate a 10 percent increase in the number of drinking days for young adults results in a 4.3 percent increase in mortality. Our results suggest policies that reduce drinking among young adults can have substantial public health benefits. (*JEL* I12, I18)

Does alcohol consumption by young adults increase mortality? A role for alcohol-related impairment in motor vehicle fatalities among youths is widely acknowledged in the popular press and in academic literature in public health and economics. Research in biology, psychology, and medicine has identified several possible mechanisms. For example, in laboratory settings alcohol consumption has been shown to result in reduced inhibition, increased aggression, compromised motor skills, and blurred vision. Given these physical and behavioral consequences of alcohol consumption, it is not surprising that alcohol has been strongly associated not only with motor vehicle fatalities but also other types of mortality such as suicide, homicide, and deaths from falls, burns, and drowning.[1]

Understanding whether there is a causal link between youth alcohol consumption and mortality is especially relevant for public policy given that over half of young adults drink and about one-third drink heavily (i.e., five or more drinks at one time). But providing precise estimates of the strength of the causal relationship between alcohol consumption and mortality is complicated by the usual problem of unobserved heterogeneity among individuals that is likely related to alcohol consumption

* Carpenter: The Paul Merage School of Business, University of California, Irvine, 428 SB, Irvine, CA 92697-3125 (e-mail: kittc@uci.edu); Dobkin: Department of Economics, University of California Santa Cruz, 1156 High St., Santa Cruz, CA 95064 (e-mail: cdobkin@ucsc.edu). We thank Phil Cook, Tom Dee, Bob Kaestner, Matt Neidell, seminar participants at the 2008 Academy Health Annual Research Meeting in Washington, DC, and several anonymous referees for useful comments. We also thank Robert Krasowski and the rest of the staff of the Centers for Disease Control Research Data Center for their assistance. All analyses, interpretations, and conclusions are those of the authors only and do not reflect the views of the CDC or any other organization. We gratefully acknowledge grant funding from NIH-NIAAA R01 AA017302-01.

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[1] See Richard J. Bonnie and Mary E. O'Connell (2004) for a detailed review. Chronic consumption has also been medically linked to mortality caused by cirrhosis of the liver, pancreatitis, and hepatitis. Extreme acute consumption can also result in death attributable to alcohol poisoning or asphyxiation.

and to the determinants of mortality. Some of the strongest research to date has made use of changes in minimum legal drinking ages (MLDA) that occurred in the 1970s and 1980s. Specifically, the majority of US states experimented with drinking ages of 18, 19, and 20 before federal legislation in 1984 required that all states adopt an MLDA of 21 years old. Several studies that use these state policy changes as "natural experiments" find that a higher drinking age reduces alcohol consumption by young adults and fatalities from motor vehicle accidents (see Alexander C. Wagenaar and Traci L. Toomey 2002 for a detailed review).

There are, however, some important limitations of the existing research on state drinking age changes, youth alcohol consumption, and mortality. First, although the literature using state changes in the drinking age finds consistent evidence that the policies affected motor vehicle fatalities, there is far less research on the effects of the minimum legal drinking age on the other leading causes of death among young adults such as suicide, drug overdose, alcohol overdose, and homicide. And what research does exist reaches contradictory conclusions.[2] Second, there is not universal consensus regarding whether and how much the national movement toward an MLDA of 21 years old affected youth alcohol consumption.[3] Finally, and perhaps most importantly, one may be concerned that bias due to policy preferences is not eliminated even when using state changes in the MLDA that were "induced" by threatened loss of federal highway funding in the 1984 Minimum Drinking Age Law. For example, the states that initially reduced their MLDA down from 21 years old—the only states for whom federal compulsion creates meaningful variation—are arguably different from those states that never changed their MLDA in unobserved ways that may be related to the determinants of youth drinking and mortality (e.g., anti-drinking sentiment). Similarly, the speed with which states chose to comply with the federal mandate is also plausibly nonrandom.

In this paper, we estimate the causal effect of alcohol consumption on mortality using a regression discontinuity (RD) design. This RD design uses the fact that the MLDA produces sharp differences in alcohol access for young adults on either side of age 21. Since the observed and unobserved determinants of alcohol consumption and mortality are likely to trend smoothly across the age-21 threshold, we can use the

[2] A 2002 review of all published drinking age studies between 1960 and 1999, for example, identified only six studies that examined mortality outcomes other than traffic fatalities, each focusing on only one or two specific causes of death (Wagenaar and Toomey 2002). Among these studies, researchers have reached different conclusions. For example, some studies find a strong relationship between the MLDA and suicide (e.g., Johanna D. Birckmayer and David Hemenway 1999, Christopher S. Carpenter 2004) while other research with a similar design fails to find effects (Ralph W. Hingson, Daniel Merrigan, and Timothy Heeren 1985). Similar null findings with respect to the drinking age have been found for pedestrian fatalities, other injury-related fatalities, homicides, and drownings (see, for example, Nancy E. Jones, Carl F. Pieper, and Leon S. Robertson 1992; Hans C. Joksch and Ralph K. Jones 1993; and Jonathan Howland et al. 1998).

[3] Robert Kaestner (2000), for example, finds much weaker support for the idea that a higher drinking age reduces consumption after accounting for time-varying state effects in a triple difference framework that makes use of youths over and under the drinking age threshold. Wagenaar and Toomey (2002) show that only 11 of 33 studies meeting the criteria for "high quality" (i.e., those peer-reviewed studies with pre/post designs, treatment/control groups, and probability samples) found a significant inverse relationship between the legal drinking age and alcohol consumption. Moreover, the literature that does find significant effects of drinking ages on consumption produces a wide range of effect sizes. Philip J. Cook and Michael J. Moore (2001) find that a binding MLDA reduces drinking participation by 9 percent and binge drinking by about 17 percent. Thomas S. Dee (1999) finds smaller effects on heavy episodic drinking of about 8 percent among high school seniors, while Carpenter et al. (2007) find drinking age effects of about 4 percent.

estimates of discontinuous jumps in alcohol consumption and mortality at age 21 to identify the causal effect of alcohol consumption on mortality among young adults. The RD design also addresses concerns about policy endogeneity that have been raised as criticisms of the research designs used in much of the prior literature. By implementing an RD design using the MLDA, which has been set at 21 years old for two decades, we can be confident that our results are not biased by unobserved factors that determine policy.

We use detailed survey data on alcohol consumption and a census of deaths in the United States to provide compelling graphical and regression-based evidence that the MLDA laws result in sharp differences in alcohol consumption and mortality for youths on either side of the age-21 threshold.[4] To implement the RD design, we estimate the reduced-form impact of the MLDA on alcohol consumption and mortality using both parametric and nonparametric regression discontinuity models. We show that the MLDA laws reduce drinking by 11–21 percent depending on the consumption measure being examined. These estimates are new and interesting in their own right and are substantially larger than most of the existing estimates from the literature on drinking ages. We then show that the increase in alcohol consumption that occurs at age 21 results in an immediate 9 percent increase in mortality, with the largest effects due to motor vehicle accidents, suicides, and deaths with an explicit mention of alcohol. We then combine these estimates to obtain the implied instrumental variables estimate of the impact of alcohol consumption on mortality. Specifically, we estimate that a 1 percent increase in the number of days on which 21-year-olds drink or drink heavily is associated with a 0.4 percent increase in mortality. This is the first direct estimate of this elasticity in the large literature on drinking ages, alcohol consumption, and mortality that we are aware of, and it is of significant value to policymakers designing interventions intended to reduce alcohol consumption and its adverse effects, particularly among young adults.

The remainder of the paper proceeds as follows. Section I describes the National Health Interview Survey alcohol consumption data and the National Center for Health Statistics mortality detail data that we use for this study. Section II provides a detailed description of the empirical methods. Section III presents the main results, and Section IV offers a discussion and concludes.

I. Data

This project uses two main data sources. For the consumption analyses, we use survey data on alcohol consumption from the National Health Interview Survey (NHIS) for the period of 1997–2005. To evaluate the impact of alcohol consumption on mortality, we use the National Center for Health Statistics' mortality detail files for the 1997 to 2004 period, and we use population estimates from the census to convert mortality counts into rates. For both parts of the analysis, we focus on young adults age 19–22, inclusive.

[4] The RD design has the advantage over the cross section and panel approaches typically used in this literature in that it is possible to indirectly check the assumptions under which the model is identified (that potentially confounding variables evolve smoothly through the discontinuity) and visually check the fit of the regression model. We revisit these issues below.

The NHIS is a survey of a stratified random sample of the US population that asks respondents about a variety of health outcomes and behaviors including alcohol consumption. The questions about alcohol consumption were included as part of the sample adult survey from 1997 to 2005. We have obtained access to a confidential version of these data with information on the respondents' exact date of birth and exact interview date, which we use to compute the exact age at which an individual was surveyed. Each NHIS survey asked a variety of questions about alcohol consumption.[5] We consider five main outcomes using responses to these questions. The first three outcomes are measures of drinking participation: whether the respondent reported having consumed 12 or more drinks in her lifetime, whether the respondent reported having consumed 12 or more drinks in any one year, and whether the respondent reported engaging in any "heavy" drinking in the previous year.[6] We also examine two measures of drinking frequency and intensity: the percentage of days on which an individual drinks and the percentage of days on which an individual engages in "heavy" drinking.

There are two potential problems with using the NHIS to estimate the change in alcohol consumption behavior that occurs at age 21. The first is that the questions on alcohol consumption typically refer to the prior 12 months. This feature implies that in the first months after turning 21, the reference period is largely composed of the person's twentieth year. However, despite being asked about their behavior over the past year, respondents can choose to report on their alcohol consumption over the past year, the past month, or the past week. Fortunately, when asked how many days they drank in the past year, 71 percent of the respondents interviewed in the month after they turned 21 answered using a reference period of either the past week or the past month.[7] The other potential problem with our alcohol consumption analysis is the

[5] Two screening questions were asked of all respondents: "In any one year, have you had at least 12 drinks of any type of alcoholic beverage?" and "In your entire life, have you had at least 12 drinks of any type of alcoholic beverage?" Individuals who had at least 12 drinks in their lifetime were then asked about the frequency and intensity of their alcohol consumption over the past year. Specifically, respondents were asked, "In the past year, how often did you drink any type of alcoholic beverage?" Here, the respondents could report their past year's consumption in terms of the number of drinks over the past year, the past month, or the past week. Finally, individuals were asked, "In the past year, on how many days did you have five or more drinks of any alcoholic beverage?" We use these responses to construct the measures of drinking frequency and intensity used in the paper (the percentage of days on which an individual drinks and the percentage of days on which an individual engages in "heavy" drinking). These outcomes are constructed logically as follows: individuals who reported that they consumed alcohol on one day in the past week are assigned a value of $1/7$, individuals who reported that they consumed alcohol on three days in the past month are assigned a value of $3/30$, and so forth. Note that we include lifetime abstainers in all models since we are interested in estimating effects on population alcohol consumption.

[6] A common variable in alcohol research is "binge" or "heavy episodic" drinking, which measures the consumption of five or more drinks at one sitting. The NHIS question asks respondents, "In the past year, on how many days did you have five or more drinks of any alcoholic beverage?" It is possible, though unlikely, that individuals could have consumed their five drinks evenly spaced across the day. For this reason, we refer to this behavior as heavy drinking rather than binge drinking.

[7] For the question, "In the past year, on how many days did you have five or more drinks of any alcoholic beverage?," 37 percent of respondents used a reference period of one week or one month rather than one year. Below we show that the reference period creates a significant measurement problem only for people just over 21 years of age. For all the drinking outcomes we examine, people interviewed in the first month after they turn 21 years old reported rates of alcohol consumption very similar to people interviewed just before they turned 21 years old and substantially lower than other 21-year-olds. Moreoever, people interviewed in the second month after turning 21 years old reported alcohol consumption levels very similar to their 21-year-old peers. This suggests that the "reference period effect" does not persist past the first month. To adjust for this problem we include a dummy variable in our regression models for individuals surveyed in the first month after they turned 21 years old.

possibility that drinking is underreported due to desirability bias. This is a particular problem for this research design because there may be a discontinuous change in the desirability bias at age 21 since the behavior in question is illegal for people under 21 years old. This could generate a discrete increase in the reported level of alcohol consumption at age 21 even if there is no true change in behavior. Fortunately, there are three compelling pieces of evidence that the increase in alcohol consumption we document in this paper is not due to desirability bias. First, as we show below, there is a large discrete increase in alcohol-related deaths (e.g., alcohol overdoses) at age 21. This is indirect but compelling evidence of a change in alcohol consumption. Second, we show below that a majority of young adults under age 21 reports that they have consumed at least 12 drinks in their lifetime and that there is essentially no change in reported lifetime drinking participation rates at age 21. Both of these facts are inconsistent with desirability bias driving our consumption results. Finally, as documented above, individuals interviewed in the month just after their twenty-first birthday reported alcohol consumption behavior that is much more similar to that of 20-year-olds than of 21-year-olds. If the increase in reported alcohol consumption we document were due to desirability bias, the reported consumption would change immediately at age 21. Though there may be some desirability bias, the pattern we observe is most consistent with honest responses to a retrospective question.

The mortality data come from the NCHS confidential national mortality detail files and include the decedent's date of birth and date of death. These data are derived from death certificates and include the universe of all deaths in the United States during the period of 1997–2004.[8] We use information on the cause of death as reported on the death certificate to create two categories: deaths due to internal causes and deaths due to external causes.[9] We then split the deaths due to external causes into the following mutually exclusive subcategories: homicides, suicides, motor vehicle accidents, deaths with a mention of alcohol, deaths with a mention of drug use, and deaths due to other external causes.[10] To account for a pronounced twenty-first-birthday mortality effect (see the day-specific death rates in Web Appendix A), for all outcomes we report estimates from regression models that include dummy variables

[8] Estimates using data from 1990–1996 are very similar to estimates using the 1997–2004 data (see Web Appendix B, http://www.aeaweb.org/articles.php?doi=10.1257/aejapplied.1.1.), suggesting that including the 2005 data, so as to match the data available from the NHIS exactly, would not significantly affect our results.

[9] The one exception to this coding rule is that we code deaths with an explicit mention of alcohol on the death certificate as "alcohol-related" deaths regardless of whether the originating cause was internal or external. For example, we code a death due to alcoholic fatty liver—an internal cause—as an external death with a mention of alcohol. We use this terminology because "alcohol-related" causes of death are fairly standard in the literature (Luis G. Escobedo and Melchor Ortiz 2002, and others).

[10] Throughout, we use the International Classification of Diseases, Ninth and Tenth revisions. A full list of the cause-of-death codes is provided in Web Appendix C. We code "alcohol-related" deaths to include: alcoholic psychoses, alcohol dependence syndrome, nondependent abuse of alcohol, alcoholic neuropathy, alcoholic cardiomyopathy, alcoholic gastritis, alcoholic fatty liver, acute alcoholic hepatitis, alcoholic cirrhosis of the liver, other alcoholic-related liver toxicity, and overdose by ethyl alcohol. The use of "alcohol-related" therefore refers to a death for which there was a strong likelihood that alcohol played an important and direct role in the outcome. When more than one cause of death was included on the death certificate, we created mutually exclusive categories in the following order: homicide, suicide, MVA, mention of alcohol, mention of drug use, and other external causes. Deaths due to "other external causes" include mortality from falls, burns, and drownings, all of which are strongly associated with alcohol consumption.

for the twenty-first birthday and the day after.[11] In nearly all cases the inclusion of these birthday dummies does not change the main results.

II. Methods

Consider a simple reduced-form model:

$$y_{ai} = X_{ai}\,\beta^{y} + g^{y}(a) + D_{ai}\,\pi^{y} + v^{y}_{ai}, \tag{1}$$

where y_{ai} is an outcome measure for individual i at age a; X_{ai} represents a set of measured characteristics of individual i at age a; $g^{y}(a)$ is a smooth function representing the age profile of the outcome y (e.g., a low-order polynomial); D_{ai} is an indicator for being 21 years old or older; and v^{y}_{ai} is an unobserved error component. The parameter π^{y} measures any discrete change in the expectation of y_{ai} that occurs precisely at age 21. Since the age profile and the dummy D_{ai} are the same for all individuals with the same age, identification of π^{y} arises from variation across age cells. Let y denote the population mean of the outcome variable y in age cell a. Ignoring within-age cell variation in the X's or assuming the data have been adjusted for such variation, equation (1) implies

$$y_{a} = E\left[y_{ai} \mid \text{age}_{i} = a\right] = X_{a}\,\beta^{y} + g^{y}(a) + D_{a}\,\pi^{y} + v^{y}_{a}. \tag{2}$$

We estimate the model above by modeling $g^{y}(a)$ as a low-order polynomial.

In addition to estimating the discontinuity parametrically as described above, we also estimate it nonparametrically using local linear regression as detailed in Jinyong Hahn, Petra Todd, and Wilbert van der Klaauw (2001). To estimate the discontinuity nonparametrically we implement a local linear regression procedure using the bandwidth selection procedure suggested in Jianqing Fan and Irene Gijbels (1996). We then use this bandwidth and a triangular kernel to fit local linear regressions on each side of age 21, and we estimate the limit of the expectation function from the left and the right of age 21. We compare the nonparametric estimates with the polynomial estimates to ensure that the results are robust to specification.

The reduced-form approach of equation (2) will let us estimate the impact of the MLDA laws on alcohol consumption and mortality. For other purposes it is helpful to interpret the discontinuity in y in the context of an underlying causal structure. We can obtain the implied instrumental variables estimate of the causal effect of alcohol consumption on mortality by forming the ratio of the estimated discontinuity in mortality at age 21 to the estimated discontinuity in alcohol consumption.

[11] The means in the figures include these observations, though their effect is not particularly discernible because averaging over 30 days largely masks the birthday effect. For the local linear regressions (described below), we drop deaths occurring on the twenty-first birthday and the day immediately after the twenty-first birthday from the analysis.

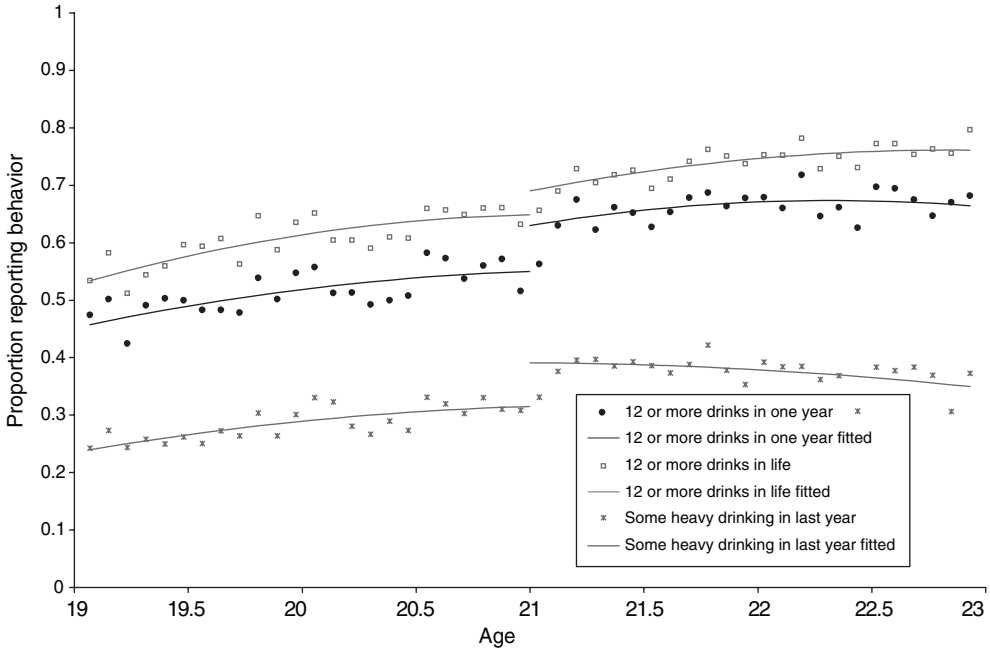

FIGURE 1. AGE PROFILE OF DRINKING PARTICIPATION

Notes: NHIS Sample Adult 1997–2005. Cells are the proportion of people in a 30-day block that report the behavior. The regression line is a second-order polynomial fitted on unweighted individual observations on either side of the age 21 cutoff.

III. Results

This section is divided into two subsections. In the first subsection, we document the substantial increase in alcohol consumption that occurs when people turn 21. In the second subsection, we estimate how much the age-specific mortality rate increases after age 21 and determine what particular causes of death are driving the increase. We also examine how the increase in mortality is distributed across gender, race, and educational attainment.

A. *Alcohol Consumption*

In this subsection, we document how the frequency and intensity of alcohol consumption changes when people turn 21 years old.[12] In Figure 1 we present the age profiles for the proportion of respondents who reported consuming 12 or more alcoholic drinks in any one year, those who reported consuming 12 or more drinks during their entire lifetime, and those who reported any heavy drinking in the past year. To make the age profile less noisy, the proportions have been calculated for 30-day blocks of age rather than individual days. We have superimposed the fitted lines from

[12] We note that estimating the effect of the current minimum drinking age (21) on alcohol consumption is an independently interesting exercise, since much previous research has used data on high school youths to evaluate historical changes in drinking ages. Since only a handful of high school youths are age 20 or 21, these samples cannot be used to identify the effects of an age-21 drinking age.

TABLE 1—ALCOHOL CONSUMPTION: PARTICIPATION

	(1)	(2)	(3)	(4)	(5)
12 or more drinks in lifetime					
Over 21	0.0418	0.0316	0.0268	0.0198	0.0199
	(0.0242)	(0.0301)	(0.0292)	(0.0423)	(0.0179)
Observations	16,107	16,107	16,107	16,107	
R^2	0.02	0.03	0.10	0.10	
Prob > Chi-Squared			0.00	0.61	
12 or more drinks in one year					
Over 21	0.0796	0.0657	0.0611	0.0603	0.0461
	(0.0254)	(0.0313)	(0.0301)	(0.0438)	(0.0218)
Observations	16,107	16,107	16,107	16,107	
R^2	0.02	0.03	0.11	0.11	
Prob > Chi-Squared			0.00	0.56	
Any heavy drinking in last year					
Over 21	0.0761	0.0527	0.0492	0.0262	0.0398
	(0.0248)	(0.0304)	(0.0291)	(0.0430)	(0.0201)
Observations	16,107	16,107	16,107	16,107	
R^2	0.01	0.01	0.10	0.10	
Prob > Chi-Squared			0.00	0.67	
Covariates	N	N	Y	Y	N
Weights	N	Y	Y	Y	N
Quadratic terms	Y	Y	Y	Y	N
Cubic terms	N	N	N	Y	N
LLR	N	N	N	N	Y

Notes: The first column of each panel contains the regression from the corresponding figure. Robust standard errors are in parentheses. Covariates include dummies for census region, race, gender, health insurance, employment status, twenty-first birthday, twenty-first birthday + 1 day, and looking for work. Weights are the NHIS adult sample weights and reduce the precision of the regressions significantly as the weights vary substantially across observations. People reporting five or more drinks on one day (Nt necessarily in one sitting) are coded as heavy drinkers. The first four columns give the estimates from polyNmial regressions on age interacted with a dummy for being over 21. The age variable is centered on 21, so the Over 21 variable gives us an estimate of the discontinuous increase at age 21. In the fifth column, we present the results of a local linear regression procedure with a rule-of-thumb bandwidth. For this procedure, we follow Fan and Gijbels (1996) and fit a fourth order polyNmial separately on each side of the age-21 cutoff. We use the fit of this regression to estimate the average second derivative of the expectation function (D) and the mean squared error of this function (σ^2). The rule-of-thumb bandwidth is $h = c(\sigma^2 R/D)$, where c is a constant that depends on the kernel ($c = 3.44$ for a triangular kernel), and R is the range of the running variable (i.e., the range of ages used to estimate the polyNmial on each side). We then use this bandwidth, and a triangular kernel, to fit local linear regressions on each side of age 21, and estimate the limit of the expectation function from the left and the right of age 21. The local linear regressions have two fewer observations because the twenty-first birthday and the day after the twenty-first birthday have been dropped.

regressions on the underlying micro data over these proportions.[13] The figure shows that about 65 percent of 20-year-olds in the NHIS reported having consumed 12 or more drinks in their lifetime, and there is not much increase in drinking at age 21.[14]

[13] The regression is a quadratic polynomial in age fully interacted with a dummy for over 21 years old. In addition there is a dummy for people interviewed in the month after their birthday. An examination of the figure suggests that the quadratic polynomial fits the data well.

[14] The means in all the figures derived from the NHIS are unweighted. Adjusting for sample weights increases the variance of the estimate and increases the variation visible in the figure. The fitted regression line is from a quadratic polynomial in age fully interacted with a dummy for over 21 years old. In addition there is a dummy for people interviewed in the month after their birthday (as is the case in all our consumption models). This accounts for the retrospective nature of the question, which can be seen in the fact that people interviewed in the first month after their twenty-first birthday report drinking behavior very similar to that reported by 20-year-olds. Note that this reference window problem is not an issue for the mortality analysis. We therefore do not include this dummy variable in the mortality regressions.

In the top panel of Table 1, we present the regression estimates of the increase in the proportion of people who reported they had consumed 12 or more drinks in their lifetime. The regression in the first column of the panel is the one that generated the fitted line in Figure 1. This regression confirms that the increase in the figure is about 4 percentage points and is statistically insignificant.[15] In the second column of the top panel, we present the same regression using the NHIS sample weights to obtain the predicted jump at age 21 for the entire US population. Adjusting for the sampling weights modestly decreases the point estimate of the age-21 discontinuity and increases the standard errors because some individuals have very large weights. In the third specification, we include dummy variables for people interviewed on their twenty-first birthday or the day immediately after. We also add dummy variables for the four census regions, male, black, Hispanic, no high school diploma, employment status, and health insurance coverage. Though these covariates are correlated with alcohol consumption, their inclusion has little impact on the estimated size of the discontinuous jump at age 21. In the fourth specification, we add a cubic term to the polynomial model, which substantially reduces the size of the estimated jump. The *p*-values from the Wald statistic (bottom row) suggest that the third specification is the preferred model of the four parametric models, a pattern that holds for all the measures of alcohol consumption we examine. Finally, in column 5, we present the results from a local linear regression with the rule-of-thumb bandwidth for each side of the age-21 cutoff (Fan and Gijbels 1996). This estimate also indicates a small and statistically insignificant discontinuity at age 21 in the likelihood of ever having consumed 12 alcoholic drinks. We focus on the results from the parametric models because they are less sensitive to the retrospective nature of the questions than the nonparametric models. That the discontinuity in lifetime drinking participation at age 21 is very small and statistically insignificant is important because it suggests that the abrupt increase in mortality at age 21 that we document below is not due to people having their first experience with alcohol.

Figure 1 also plots the age profile of the proportion of people who reported consuming 12 or more drinks in one year (represented by the dark circles) and 5 or more drinks on a single day at least once in the previous year (represented by the crosses). The regression line superimposed on the dark circles in Figure 1 and the corresponding regression results in the second panel of Table 1 indicate that the increase in the probability of drinking 12 or more drinks in one year is about 6 percentage points and is statistically significant in the preferred quadratic specification with covariates and the twenty-first birthday dummies (column 3). Relative to the one-year drinking participation rate of young adults just under age 21 of about 55 percent, our preferred estimate suggests an increase of about 11 percent. Our preferred estimate in the bottom panel of Table 1 also indicates a sharp and statistically significant increase in the probability of any heavy drinking in the past year of about 5 percentage points at age 21. This represents an increase of approximately 16 percent relative to the heavy drinking participation rate of people just under 21, though these estimates

[15] Since the age variable in the regression is the number of days until (or since) the person's twenty-first birthday at the time of the interview, the "Over 21" dummy gives the estimate of the discrete increase in the outcome that occurs at age 21. Full sets of estimates on the age profile of the drinking outcomes and the coefficients on the "twenty-first birthday" dummies are available online in Appendices D and E.

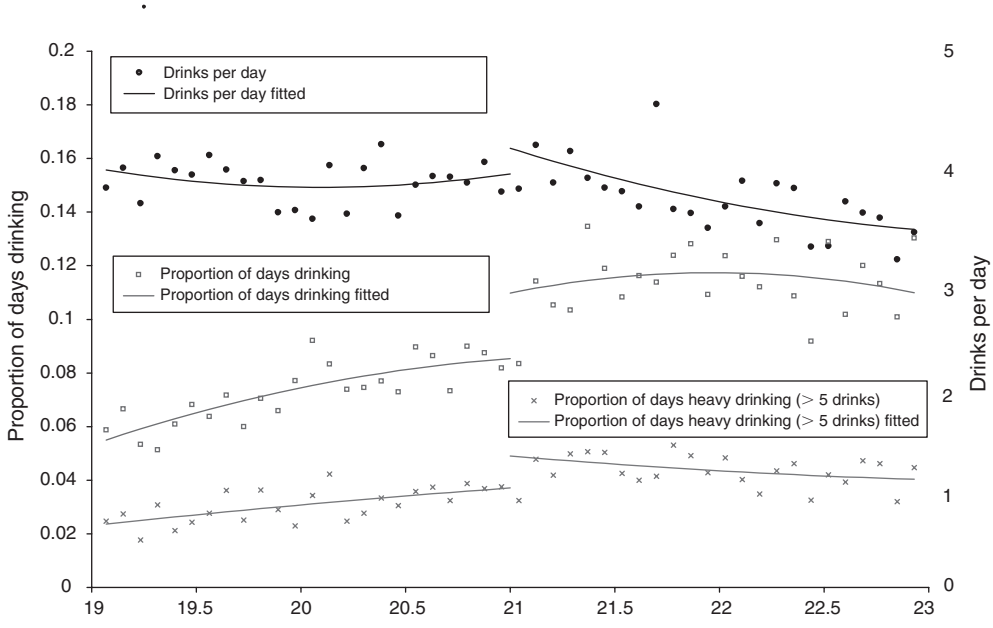

FIGURE 2. AGE PROFILE OF DRINKING INTENSITY

Notes: People can report their drinking for the last week, month, or year; 71 percent of respondents used a reference period of one week or one month. Average number of drinks per day is for people who reported some drinking.

are somewhat sensitive to the inclusion of the cubic term in age. For all outcomes in Figure 1, it is likely that the specification with the cubic term in age produces smaller estimated discontinuities because it is more sensitive to the retrospective nature of the question.

In Figure 2, we present the age profiles of various measures of drinking intensity. The figure reveals a discrete jump of about 2 percentage points in the proportion of days drinking any alcohol (represented by the hollow squares). In the top panel of Table 2, we present the corresponding regression estimates for proportion of days drinking, which confirm that the increase we observe in the figure is about 2–2.5 percentage points across all specifications with a quadratic in age. This is substantial given that the average 20-year-old reported drinking on about 8.5 percent of days (i.e., a 21 percent increase in drinking days relative to people just under 21 shown in column 3). As with the drinking participation outcomes in Table 1, we find that the addition of a cubic term substantially reduces the estimate of the jump at age 21, though Figure 2 shows that this likely is due to the low level of reported drinking among people interviewed just after their twenty-first birthday. Importantly, the model with the cubic in age is not supported by the data. Figure 2 also shows evidence of an increase in the proportion of days of heavy drinking (represented by crosses). Though the corresponding regressions presented in the middle panel of Table 2 reveal that the 20 percent increase in heavy drinking days at age 21 in the preferred specification is not statistically significant, the estimated proportional increase is remarkably similar to the increase in regular drinking days. Together with the evidence that the probability of any heavy drinking days increases significantly at

Table 2—Alcohol Consumption: Intensity

	(1)	(2)	(3)	(4)	(5)
Proportion of days drinking					
Over 21	0.0245	0.0180	0.0182	0.0119	0.0107
	(0.0086)	(0.0097)	(0.0095)	(0.0135)	(0.0072)
Observations	16,107	16,107	16,107	16,107	
R^2	0.02	0.02	0.07	0.07	
Prob > Chi-Squared			0.00	0.56	
Proportion of days heavy drinking					
Over 21	0.0120	0.0075	0.0075	0.0021	0.0026
	(0.0061)	(0.0063)	(0.0062)	(0.0091)	(0.0048)
Observations	15,825	15,825	15,825	15,825	
R^2	0.00	0.01	0.05	0.05	
Prob > Chi-Squared			0.00	0.72	
Drinks per day on days drinking					
Over 21	0.2387	0.2068	0.2465	0.2806	0.1886
	(0.2810)	(0.3403)	(0.3291)	(0.4782)	(0.2024)
Observations	9,906	9,906	9,906	9,906	
R^2	0.00	0.00	0.07	0.07	
Prob > Chi-Squared			0.00	0.92	
Covariates	N	N	Y	Y	N
Weights	N	Y	Y	Y	N
Quadratic terms	Y	Y	Y	Y	N
Cubic terms	N	N	N	Y	N
LLR	N	N	N	N	Y

Notes: See Notes from Table 1. People can report their drinking for the last week, month, or year. For people who reported any drinking, 71 percent of those interviewed in the month after they turned 21 reported about their drinking in the last week or month rather than the last year. The dependent variable "Drinks per Day" is the number of drinks the respondent reported drinking on a typical day in which they consumed some alcohol.

age 21, these results suggest that the amount of heavy drinking increases in response to the easier alcohol availability that results from turning 21.[16]

Finally, before examining the increase in mortality rates that results from the increase in alcohol consumption documented above, we examine the possibility that there are other changes occurring at age 21 that could confound our analysis. That the regression estimates are robust to the inclusion of covariates (i.e., moving from specification 2 to 3 in Tables 1 and 2) suggests that the observable characteristics are smoothly distributed across the discontinuity. In Table 3, we provide a more direct test that demographic characteristics, employment status, insurance status, and

[16] We also examined the number of drinks consumed on the days the individual drank. There was no visual evidence of a discrete break in the average number of drinks that people consume after they turn 21 (see Figure 2). Moreover, all of the discontinuity estimates for the "drinks on days drinking" variable were smaller as a proportion of the age-20 mean than the previous outcomes (with our preferred specification returning an effect size of about 6.4 percent), and none was statistically significant (see the estimates in the bottom panel of Table 2). A problem with this analysis, however, is that it may confound compositional changes in the group of drinkers at age 21, since only people who reported drinking are included and the number of people who reported drinking changes discretely at age 21. For this reason, we also created a histogram of the number of average drinks per day for 20-year-olds and 21-year-olds in the NHIS sample (also available in the Web Appendix F). We found that, apart from an almost 10 percentage point difference in the likelihood of being a drinker, the distribution of average drinks per day for 21-year-olds is similar to the distribution for 20-year-olds. This is consistent with the similar size, in percentage terms, of increases in the number of days of drinking and days of heavy drinking documented above. Taken together, the evidence suggests that after turning 21 people drink on more days, and there is an increase in heavy drinking, but that the increase in heavy drinking it is not disproportionate.

TABLE 3—AGE PROFILE OF DEMOGRAPHIC CHARACTERISTICS FROM THE NHIS

	Male (1)	White (2)	Black (3)	Hispanic (4)	No HS Diploma (5)	HS Diploma (6)	Employed (7)	Looking for work (8)	No health insurance (9)
Over 21	0.0156	0.0172	−0.0250	0.0095	−0.0108	0.0040	0.0019	−0.0061	0.0043
	(0.0260)	(0.0293)	(0.0211)	(0.0203)	(0.0230)	(0.0291)	(0.0302)	(0.0171)	(0.0293)
Constant	0.4405	0.6440	0.1415	0.1638	0.1810	0.2941	0.6453	0.0849	0.2970
	(0.0168)	(0.0195)	(0.0137)	(0.0129)	(0.0154)	(0.0194)	(0.0203)	(0.0114)	(0.0191)
Observations	16,107	16,107	16,107	16,107	16,107	16,107	16,107	16,107	16,107
R^2	0.00	0.00	0.00	0.00	0.01	0.00	0.01	0.00	0.00

Notes: Sample from NHIS Sample Adult File (1997–2005). Standard errors in parentheses. The regressions do not include covariates other than a second order polynomial in age interacted with the over 21 dummy. Since Age = persons age − 21, the constant is the predicted value for people about to turn 21.

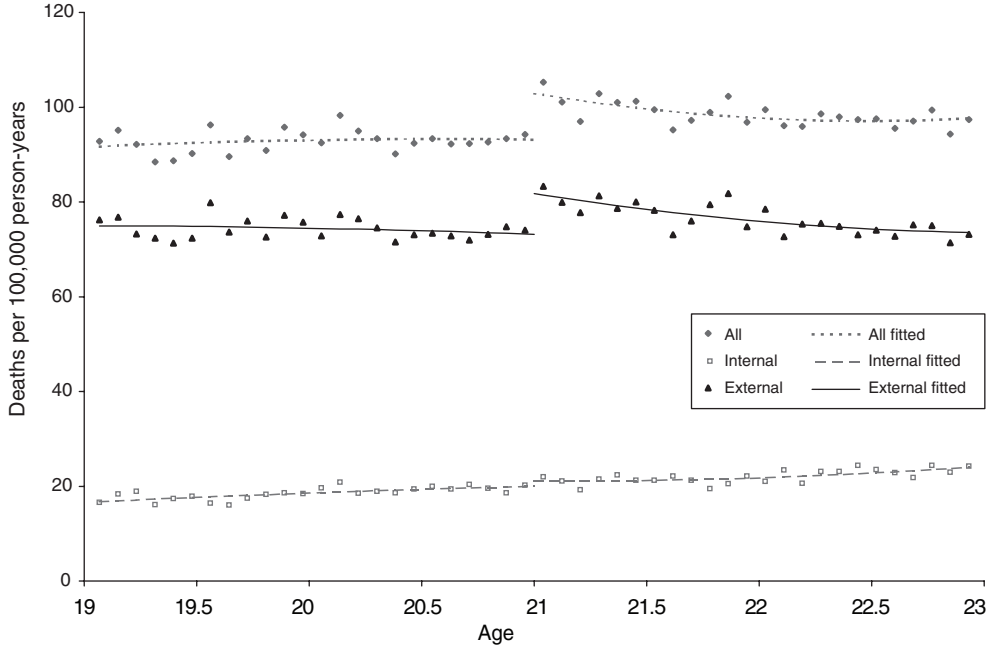

FIGURE 3. AGE PROFILE FOR DEATH RATES

Notes: Deaths from the National Vital Statistics Records. Includes all deaths that occurred in the United States between 1997–2003. The population denominators are derived from the census. See online Appendix C for a list of causes of death.

educational attainment all evolve smoothly through the age 21 threshold. We find no evidence of a discrete change at age 21 in any of these characteristics, and this is confirmed visually in Web Appendix G.[17] That these observable characteristics are

[17] Although when we examine the NHIS survey data, and we do not find discrete changes in health insurance status at age 21, there is an important set of age-based insurance laws that are worth noting. There are 19 states that extend public health insurance eligibility to older adolescents through age 21 who meet income guidelines using the "Ribicoff" and "medically needy" options to state Medicaid programs (federal Medicaid guidelines cover children through age 18) (Fox et al. 2007). However, there are a couple of reasons that it is likely that the

TABLE 4—DISCONTINUITY IN LOG DEATHS AT AGE 21

	(1)	(2)	(3)	(4)
Deaths due to all causes				
Over 21	0.096	0.087	0.091	0.074
	(0.018)	(0.017)	(0.023)	(0.016)
Observations	1,460	1,460	1,460	1,458
R^2	0.04	0.05	0.05	
Prob > Chi-Squared		0.000	0.735	
Deaths due to external causes				
Over 21	0.110	0.100	0.096	0.082
	(0.022)	(0.021)	(0.028)	(0.021)
Observations	1,460	1,460	1,460	1,458
R^2	0.06	0.08	0.08	
Prob > Chi-Squared		0.000	0.788	
Deaths due to internal causes				
Over 21	0.063	0.054	0.094	0.066
	(0.040)	(0.040)	(0.053)	(0.031)
Observations	1,460	1,460	1,460	1,458
R^2	0.10	0.10	0.10	
Prob > Chi-Squared		0.000	0.525	
Covariates	N	Y	Y	N
Quadratic terms	Y	Y	Y	N
Cubic terms	N	N	Y	N
LLR	N	N	N	Y

Notes: See Notes from Table 1. The dependent variable is the log of the number of deaths that occurred x days from the person's twenty-first birthday. External deaths include all deaths with mention of an injury, alcohol use, or drug use. The Internal Death category includes all deaths Nt coded as external. Please see Web Appendix C for the ICD codes for each of the categories above. The first three columns give the estimates from polyNmial regressions on age interacted with a dummy for being over 21.

smoothly distributed across the discontinuity suggests that the unobservable characteristics are also distributed smoothly across the discontinuity and reduces our concerns about omitted variables bias.

B. *Mortality*

In this section, we evaluate whether the increase in alcohol consumption documented above results in an increase in mortality rates. In Figure 3, we present the overall age profile of deaths per 100,000 person years, and we also show the age profiles separately for deaths due to internal and external causes. We computed deaths per 100,000 person years by dividing the total number of deaths at a particular age by the total number of years lived at that age by everyone in the cohort. External causes of death include motor vehicle accidents, suicide, homicide, deaths with a mention of alcohol use, deaths with a mention of drug use, and other deaths due to

laws are at most leading to modest bias in our estimates. First, federal law requires hospitals to stabilize patients needing care without regard to ability to pay. Second, because the states that do extend public health insurance eligibility to older children generally impose much stricter income requirements (generally less than 75 percent of the federal poverty line) than for regular Medicaid eligibility for children, our mortality findings—which are largely driven by white college-educated individuals—are unlikely to be driven by a change in insurance status.

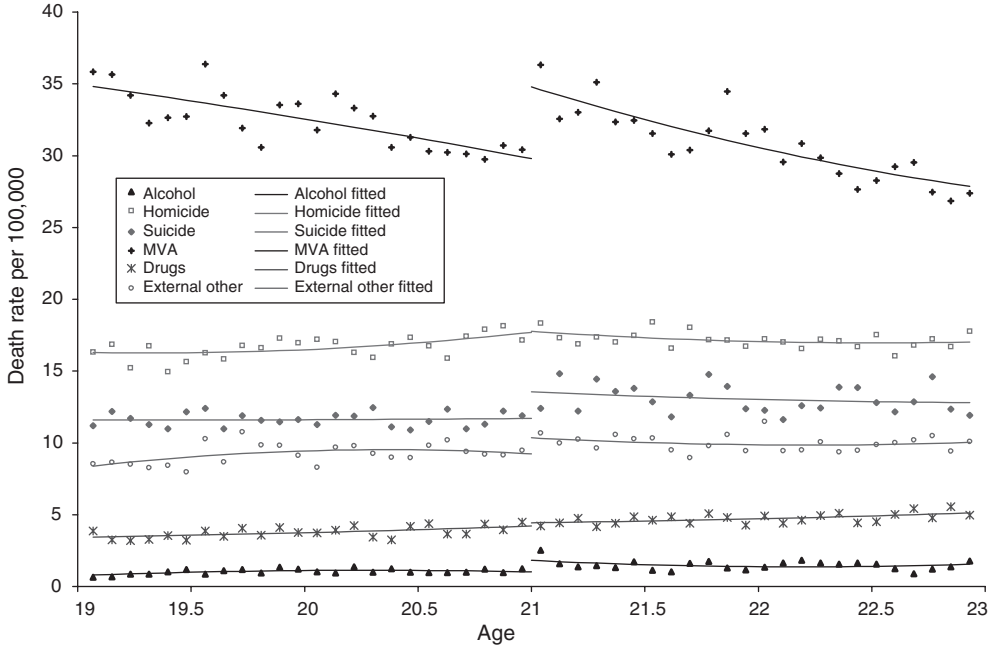

FIGURE 4. AGE PROFILES FOR DEATH RATES BY EXTERNAL CAUSE

Notes: See notes to Figure 3. The categories are mutally exclusive. The order of precedence is homicide, suicide, MVA, deaths with a mention of alcohol, and deaths with a mention of drugs. The ICD-9 and ICD-10 Codes are in Appendix C.

external injuries. Figure 3 shows a sharp increase in overall mortality at age 21 of about 10 deaths per 100,000 person years. Grouping the deaths by cause reveals that for this age group the majority of deaths are due to external causes and the increase in deaths at age 21 is attributable largely to deaths due to external causes.

Table 4 presents the regression estimates corresponding to Figure 3. The dependent variable in the regression is the log of the total number of people who died at an exact age (in years and days) during the 1997–2004 period. We estimate the model over the 1,460 days between ages 19 and 22, inclusive.[18] The coefficient of interest on the over 21 indicator can, for small changes, be interpreted as the percentage change in deaths at age 21. In the first column of each panel, we present the estimates from fitting a quadratic polynomial to the age profile of deaths. In the second column, we add a dummy for the twenty-first birthday and a dummy for the day immediately after. In the third specification of each panel, we add a cubic term to the polynomial. In the fourth column, we present the estimate from a local linear regression with a rule-of-thumb bandwidth for each side of the age-21 cutoff

[18] We did not use the death rates as the dependent variable because measurement error in the denominator is likely to reduce the precision of the estimates. Rates are unnecessary for the regressions because combining the cohorts smoothes out most of the bumps in the age profile and the polynomial in age absorbs the remaining variation.

Table 5—Discontinuity in Log Deaths at Age 21 Due to External Causes

	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
	Alcohol				Homicide			
Over 21	0.388	0.346	0.406	0.441	0.009	0.002	−0.003	−0.014
	(0.119)	(0.116)	(0.156)	(0.117)	(0.045)	(0.045)	(0.061)	(0.041)
Observations	1,460	1,460	1,460	1,458	1,460	1,460	1,460	1,458
R^2	0.03	0.04	0.04			0.01	0.01	0.01
Prob > Chi-Squared		0.000	0.228			0.000	0.495	
	Suicide				Motor vehicle accidents			
Over 21	0.160	0.154	0.135	0.105	0.158	0.143	0.145	0.139
	(0.059)	(0.059)	(0.086)	(0.045)	(0.033)	(0.032)	(0.044)	(0.032)
Observations	1,460	1,460	1,460	1,458	1,460	1,460	1,460	1,458
R^2	0.02	0.02	0.02	0.15	0.16	0.16		
Prob > Chi-Squared		0.000	0.892	0.000	0.666			
	Drugs				Other external causes			
Over 21	0.070	0.067	0.004	−0.016	0.087	0.098	0.098	0.074
	(0.081)	(0.082)	(0.107)	(0.078)	(0.060)	(0.059)	(0.075)	(0.043)
Observations	1,460	1,460	1,460	1,458	1,460	1,460	1,460	1,458
R^2	0.04	0.04	0.04	0.01	0.01	0.01		
Prob > Chi-Squared		0.000	0.643			0.000	0.877	
Covariates	N	Y	Y	N	N	Y	Y	N
Quadratic terms	Y	Y	Y	N	Y	Y	Y	N
Cubic terms	N	N	Y	N	N	N	Y	N
LLR	N	N	N	Y	N	N	N	Y

Notes: See Notes from Table 4. There are 276 observations where there are N deaths coded as due to alcohol; for this variable 0.5 was added to the dependent variable before taking the log. There are 15 observations where there are N deaths coded as due to drug use; for this variable 0.5 was added to the count before taking the log.

(Fan and Gijbels 1996).[19] The p-values from the Wald statistic (bottom row) suggest that the second specification is the preferred model of the three parametric models. However, all four specifications give us very similar estimates and confirm what we observed in Figure 3, that there is a statistically significant 9 percent increase in overall mortality when people turn 21 that is mostly due to a 10 percent increase in deaths due to external causes.[20]

Figure 4 plots the age profile of external deaths separately by cause. The figure shows a large and noticeable increase in motor vehicle accidents at age 21, with

[19] In Web Appendix H, we show the mortality results are not sensitive to the parameterization of the age profile. The same is true for the alcohol consumption results in Web Appendix I. In Web Appendix J, we present the sensitivity of the mortality results to varying the bandwidth and show that the estimates are only sensitive to reducing the bandwidth well below that suggested by the Fan and Gijbels (1996) procedure described in the footnotes to Table 1. The same is true for the alcohol consumption results in Web Appendix K. Full sets of estimates on the age profile of the mortality and the coefficients on the "twenty-first birthday" dummies are available online in Appendices L, M, and N.

[20] Note that the estimated discontinuity for internal causes of death is statistically indistinguishable from 0 but economically significant (a 5.4 percent increase in mortality). An observed discontinuity at age 21 in internal causes of death could arise for several reasons. First, alcohol may worsen or exacerbate preexisting medical conditions that result in internal causes of death. Second, it is likely that some alcohol poisoning/alcohol overdose deaths are not reported accurately on the death certificate. Since we consider any alcohol poisoning deaths to be external causes, this type of miscoding will appear as an increase in internal mortality at age 21.

smaller but noticeable discontinuities in alcohol-related deaths (e.g., alcohol overdoses) and suicides. We find no visual evidence of increases in deaths attributable to drugs or homicide at age 21. Table 5 presents the corresponding regression results and confirms what we saw in Figure 4, that deaths with an explicit mention of alcohol increase by about 35 percent at age 21, homicides exhibit essentially no change, and the suicide rate increases by over 16 percent. Table 5 also shows that deaths due to motor vehicle accidents increase by about 15 percent at age 21.[21] Estimates of external deaths attributable to drugs are not estimated precisely, while deaths due to "other external causes" (i.e., all deaths due to external causes not included elsewhere in Table 5) show evidence of a discrete increase of about 10 percent, an estimate that nears statistical significance in some of the models.[22] This result for "other external deaths" provides evidence of a causal relationship between alcohol consumption and deaths due to fires, falls, and drownings.

In Web Appendix O, we present the estimates of how the increase in total mortality varies by gender, race, and education. The regressions stratified by gender reveal that men experience a 10 percent increase in mortality and account for the majority of the overall increase in mortality. The regressions stratified by race reveal that the increase in deaths is largely driven by the 15 percent increase in deaths among whites, despite the fact that they have considerably lower death rates than either blacks or Hispanics. In the final panel, we present the result by educational attainment. These regressions reveal that the overall increase in deaths is driven largely by a 9 percent increase in deaths among high school graduates and a 17 percent increase in deaths among people attending college. In summary, the overall increase in mortality rates we observe at age 21 is due to large increases in mortality among white males who are high school graduates or are attending college.[23]

[21] The estimated discontinuity in motor vehicle accidents at age 21 understates the true effects of alcohol consumption. To see this, note that this will only capture the true mortality effect of the drinking age if there are no other individuals involved in the accident (e.g., a one-car crash fatality with one occupant in the car). To the extent that there are other occupants in the driver's car or occupants in a different car (in a multi-car crash), these other fatalities should more properly be attributed to the effects of the drinking age and alcohol consumption. We revisit this issue below.

[22] For deaths with a mention of alcohol and deaths with a mention of drugs, there are some observations with zero deaths. In the regressions for these outcomes in Table 5, we have added 0.5 to the dependent variable before taking the log. Web Appendix P shows that for these two outcomes the results are robust to two alternative approaches: adding 1 (instead of 0.5) to the dependent variable before taking the log (column 2 under each outcome) and estimating the model in levels instead of logs (column 3 under each outcome).

[23] For compactness, we only report the results from our preferred specification for the mortality results by group in Web Appendix O. The regressions are robust to specification, and the full set of regressions and figures is available on request. Unfortunately, because of the much smaller sample sizes in the NHIS, the estimates of the changes in alcohol consumption that occur at age 21 are too imprecise to make strong statements about the differences in alcohol consumption across subgroups. For completeness, however, we present the RDD drinking estimates in Web Appendix Q. Note that there may be heterogeneity in the structural relationships between alcohol consumption and mortality by subgroup. Moreover, the results in Web Appendix Q confirm the well-documented structural differences in youth drinking patterns and mortality (both overall and cause-specific) by sex and race. Since drinking ages affect very different parts of the race and sex-specific alcohol consumption and mortality distributions, it is not obvious that comparisons of the implied elasticities of mortality with respect to consumption by group are instructive.

IV. Discussion and Conclusion

In this paper, we document that age-based restrictions on access to alcohol have a substantial effect on both alcohol consumption and mortality. Individuals just over age 21 report drinking on 21 percent more days than individuals just under 21 years old, and we estimate a similar proportional increase for heavy drinking days. We find no evidence of an increase in the average number of drinks people consumed on days of drinking, nor do we find a shift in the distribution of drinking intensity. We find that this increase in alcohol consumption results in a 9 percent increase in mortality at age 21. When we disaggregate by cause of death, we find particularly large increases for alcohol-related deaths, motor vehicle fatalities, and suicides.

Our results provide new evidence on some commonly held beliefs about how the increase in drinking causes an increase in mortality. First, the mortality effects we observe are not due entirely to "new" drinkers, those without previous exposure to alcohol. This is borne out by the very small increases (in both absolute and proportional terms) in first-time use of alcohol at age 21. Second, the mortality effects are also unlikely to be driven by people's first experiences with heavy drinking. Figure 1 shows that people do experiment with heavy drinking immediately after they turn 21. But if the increase in mortality were caused by experimentation with heavy drinking, then mortality rates should rise discretely at age 21 and return rapidly to the pre-21 level. Figures 3 and 4 show that this is not the case. As noted above, we do not find any evidence of a disproportionate increase in drinking intensity, which suggests that the increase in mortality rates is due to an increase in the number of days on which people drink or drink heavily.[24] If the increase in mortality is due to the increase in the number of days on which people drink or drink heavily, then the implied elasticity is 0.43 for individuals who change their drinking behavior because of the law (i.e., 0.091/0.21). This elasticity suggests that a substantial proportion of deaths among 21-year-olds are due directly to alcohol consumption. It also suggests that the cost of an increase in alcohol consumption is potentially quite high.[25] Our estimates suggest that reducing the drinking age nationally to age 20 would result in approximately 408 additional deaths among 20-year-olds.[26] Given a value of a

[24] One margin that we do not observe in the data is the travel distance to the location at which people drink. However, the fact that suicides increase by about the same percentage as MVA deaths suggests that it is not an increase in driving exposure per drink that is responsible for the overall increase in mortality documented above.

[25] An important question that relates to the interpretation of our results is: how persistent are the effects of easing access to alcohol at age 21? Some researchers have suggested that the MLDA laws simply shift the timing of deaths instead of reducing the number of deaths (Dee and William N. Evans 2001 and Mike A. Males 1986), while other research does not find strong evidence of shifting (Henry Saffer and Michael Grossman 1987 and Cook and George Tauchen 1984). The age profiles in Figures 3 and 4 are more consistent with a lasting change in mortality than a change in the timing of deaths.

[26] To get an estimate of the number of additional deaths that would result from a national reduction in the drinking age by one year we took the expected number of deaths among 20-year-olds in 2007 (4,027) based on a simple linear projection from 1997 to 2005 mortality figures and applied the percent increase implied by the RDD estimates. The RDD estimate of the increase in MVA deaths was increased by 21 percent to account for the fact that, according to our analysis of data from the Fatality Analysis Reporting System, the typical 21- to 22- year-old killed while driving drunk killed an additional 0.21 people.

statistical life of $8.4 million (in 2007 US dollars) this gives a total cost of about $3.4 billion per year.[27]

Finally, our finding that policies that reduce alcohol consumption among young adults substantially reduce mortality is extremely relevant for current and ongoing public policy debates about stricter alcohol control targeted at youths. Given that over half of 18-to-20-year-olds report recent alcohol consumption, and about one-third report heavy episodic or "binge" drinking, there is wide latitude to affect alcohol consumption in this age group. Moreover the majority of acute alcohol-related deaths occur among 18- to 24-year-olds. Our results therefore suggest that stricter alcohol control targeted toward young adults could result in meaningful reductions in mortality, substantially reducing the number of life years lost to alcohol.

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