NBER WORKING PAPER SERIES

THE EFFECT OF ALCOHOL CONSUMPTION ON MORTALITY: REGRESSION DISCONTINUITY EVIDENCE FROM THE MINIMUM DRINKING AGE

Christopher Carpenter
Carlos Dobkin

Working Paper 13374
http://www.nber.org/papers/w13374

NATIONAL BUREAU OF ECONOMIC RESEARCH
1050 Massachusetts Avenue
Cambridge, MA 02138
September 2007

Carpenter: The Paul Merage School of Business, University of California Irvine, kittc@uci.edu. Dobkin: Department of Economics, University of California Santa Cruz, cdobkin@ucsc.edu. We thank Phil Cook, Tom Dee, and Bob Kaestner for useful comments. The views expressed herein are those of the author(s) and do not necessarily reflect the views of the National Bureau of Economic Research.

The Effect of Alcohol Consumption on Mortality: Regression Discontinuity Evidence from the Minimum Drinking Age
Christopher Carpenter and Carlos Dobkin
NBER Working Paper No. 13374
September 2007
JEL No. I1

ABSTRACT

This paper estimates the effect of alcohol consumption on mortality using the minimum drinking age in a regression discontinuity design. We find that granting legal access to alcohol at age 21 leads to large and immediate increases in several measures of alcohol consumption, including a 21 percent increase in the number of days on which people drink. This increase in alcohol consumption results in a discrete 9 percent increase in the mortality rate at age 21. The overall increase in deaths is due primarily to a 14 percent increase in deaths due to motor vehicle accidents, a 30 percent increase in alcohol overdoses and alcohol-related deaths, and a 15 percent increase in suicides. Combining the reduced-form estimates reveals that a 1 percent increase in the number of days a young adult drinks or drinks heavily results in a .4 percent increase in total mortality. Given that mortality due to external causes peaks at about age 21 and that young adults report very high levels of alcohol consumption, our results suggest that public policy interventions to reduce youth drinking can have substantial public health benefits.

Christopher Carpenter
University of California, Irvine
The Paul Merage School of Business
443 SB
Irvine, CA 92697-3125
and NBER
kittc@uci.edu

Carlos Dobkin
Engineering 2
UC, Santa Cruz Dept. of Economics
1156 High St.
Santa Cruz, CA 95064
cdobkin@ucsc.edu

Introduction

Does alcohol consumption by young adults increase mortality, and if so, by how much? These questions are highly relevant for public policy given that over half of young adults drink and about one-third drink heavily (i.e., 5 or more drinks at one time).[2] But providing credible answers to these questions is complicated by the usual problem of unobserved heterogeneity among individuals that is likely related both to alcohol consumption and to the determinants of mortality. Some of the most credible research to date has made use of the changes in the minimum legal drinking age (MLDA) that occurred in the 1970s and 1980s. Specifically, the majority of states experimented with drinking ages of 18, 19, and 20 before federal legislation required that all states adopt an MLDA of 21. Several studies that focus on the effect of these policy changes find that a higher drinking age reduces alcohol consumption by young adults and fatalities from motor vehicle accidents (see Wagenaar and Toomey 2002 for a review).[3] However, there is much less research on how the drinking age affects the other leading causes of death among young adults – suicide, drug overdose, alcohol overdose, and homicide – and what research does exist reaches contradictory conclusions. Moreover, the literature lacks credible estimates of the underlying structural relationship between alcohol consumption and mortality.[4] Estimates of how much a 1 percent reduction in alcohol consumption will reduce mortality are vital for policymakers evaluating the benefits of interventions intended to reduce alcohol consumption.

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

[2] Young adults are of particular policy interest with respect to alcohol consumption and its adverse effects. A 2004 Institute of Medicine report, for example, estimates that excessive alcohol consumption by young adults costs society billions of dollars each year in the form of traffic accidents, crime, and unintentional injury (Bonnie and O'Connell 2004).

[3] In addition to substantial changes in drinking behavior in the last 25 years, major technological innovations have made motor vehicles much safer. As such, it is not clear how relevant older estimates are for guiding current policy.

[4] Below, we briefly review other approaches that researchers have used to disentangle causality in the alcohol consumption and mortality relationship in the US. This includes research that relies on alcohol taxes and other alcohol prohibitions for identification.

mortality are likely to trend smoothly across the age-21 threshold, we can use the 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. To implement the design we estimate the reduced-form impact of the MLDA on alcohol consumption and mortality using both parametric and nonparametric regression discontinuity models. We then combine these estimates to obtain the implied instrumental variables estimate of the impact of alcohol consumption on mortality.

The RD design is very well suited for this research question. We are able to 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. 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. Specifically, it may be that places with strict alcohol control policies differ systematically in their unobserved determinants of alcohol consumption (e.g., anti-drinking sentiment) and mortality from places with weak alcohol control policies. Even research designs that leverage changes in state alcohol control policies are subject to the concern that the policy changes may coincide with changes in population preferences or state public health campaigns. By implementing an RD design we can be confident that our results are not biased by unobserved factors that determine policy.[5]

In addition to using a research design that we believe generates more credible estimates than the designs used in the prior literature, our paper makes several other important contributions.[6] First, we show that the MLDA laws reduce drinking by 16-21 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

[5] As we describe below, all states have had an MLDA of 21 since the mid-1980s. An additional benefit of using such a long-standing policy is that we largely avoid problems related to the substantial cross-state alcohol consumption by youths in the 1970s and 1980s that was related to differences in MLDAs across states at a point in time. Saffer and Grossman (1987) show that their estimates on motor vehicle mortality are sensitive to the drinking age in nearby border states. We also avoid the issue of how to deal with different state policies that were "grandfathered" for some youths when the state drinking age increased.

[6] 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.

existing estimates from the literature on drinking ages. Second, we show that the increase in alcohol consumption that occurs at age 21 results in an immediate 9 percent increase in mortality. Unlike the prior literature, we examine all the major causes of death and find that the overall increase is due to increases in deaths due to motor vehicle accidents, suicides, and deaths with an explicit mention of alcohol (such as overdoses and alcohol poisoning). Third, we use the large samples of deaths to stratify the mortality analysis by sex, race, and education group and find that the increase in mortality at age 21 is largest for males, whites, and high school graduates. Finally, we combine our reduced-form estimates of the effect of the MLDA laws on alcohol consumption with the estimates of their impact on mortality to obtain the implied instrumental variables estimate of the impact of alcohol consumption on the probability of dying. Specifically, we find that a 1 percent increase in the number of days on which 21-year-olds drink or drink heavily is associated with a .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 1 provides a brief review of the relevant literature that has used state drinking age policy experiments to evaluate the effects of alcohol access on alcohol consumption and mortality. Section 2 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 3 provides a detailed description of the empirical methods, Section 4 presents the main results, and Section 5 offers a discussion and concludes.

1. Literature Review

There is a large literature on alcohol consumption and its effect on mortality, particularly from alcohol-related traffic accidents. Many of the papers in this literature leverage changes in state alcohol control policies. For example, a large number of studies have used state variation in alcohol excise taxes to identify the effects of alcohol

consumption on mortality rates.[7] Others have used the variation in the availability of alcohol generated by liquor outlet density or county-level prohibitions on alcohol sales.[8] A full review of the literature is beyond the scope of this paper; instead, we focus on a class of studies that – like ours – relies on MLDA laws to analyze the effects of drinking on mortality. The intuition behind the literature reviewed here is that reductions in the total cost of drinking will increase alcohol consumption and alcohol-related harms (to the extent that such harms arise directly from alcohol consumption).

A large literature has studied the effects of minimum drinking ages, though no previous work uses an RD framework. Instead, the most credible existing work has used state policy experiments with changes in the MLDA. These historical policy changes carry a few advantages in the context of identifying causal effects of alcohol access on consumption and mortality. First, there is substantial variation in state policies over time. Specifically, a majority of states experimented with reducing their MLDA to 18 or 19 (down from 21) in the 1970s, although all states eventually reinstated the age-21 MLDA by the late 1980s. Second, unlike beer taxes or wet/dry prohibitions, the use of MLDA laws creates natural age-based predictions about which groups should and should not have been affected by the minimum drinking age. Third, using changes in the MLDA laws alleviates some concerns about policy endogeneity because the federal government threatened states with the loss of federal highway funds if they did not adopt an MLDA of 21. As such, researchers have argued that states were "compelled" against their will to

[7] See, for example, Grossman et al. (1994), Kenkel (1993), Ruhm (1996), and Cook and Moore (2001). These studies generally show that higher beer taxes are associated with lower alcohol consumption by youths and young adults, though the magnitude of the price/consumption relationship varies across studies. With respect to mortality, studies have found negative relationships between alcohol taxes and cirrhosis mortality (Cook and Tauchen 1982), traffic fatalities (Saffer and Grossman 1987), and all-cause mortality (Cook et al. 2005). There has been some disagreement, however, regarding the ability of panel data evaluations to credibly estimate effects of state beer taxes on consumption and mortality (see Dee 1999 and others).

[8] Baughman et al. (2001), for example, consider numerous county-level changes in "wet" and "dry" status in Texas from the 1970s to the 1990s. They find that the sale of alcohol for on-premises consumption at bars and restaurants does increase traffic fatalities, though off-premises consumption availability appears to *reduce* traffic fatalities. Research has also used variations in the physical access to alcohol associated with differences in the density of alcohol and liquor outlets. Using data on liquor outlet density in communities in New Mexico, Escobedo and Ortiz (2002) found statistically significant associations with suicide, alcohol-related crashes, and alcohol-related fatalities. They did not, however, find significant relationships between liquor outlet density and other adverse outcomes such as homicide, drug-related mortality, or alcohol-related mortality (e.g., overdoses).

impose stricter alcohol control policies.[9] Finally, there is credible evidence that MLDA policies induced changes in alcohol use. Cook and Moore (2001) used the National Longitudinal Surveys of Youth (NLSY) to show that changes in the MLDA were associated with sharp changes in alcohol consumption, and Dee (1999) and Carpenter et al. (2007) find a similar result using the Monitoring the Future (MTF) study.

There are, however, at least three remaining criticisms of the work on drinking ages and alcohol consumption. First, one may still be concerned that bias due to policy preferences is not completely eliminated even when using state changes in the MLDA that were "induced" by the federal compulsion. For example, the states that initially reduced their MLDA down from 21 – 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 drinking (e.g., anti-drinking sentiment). Similarly, the speed with which states chose to comply with the federal directive is also plausibly nonrandom. Second, there is not universal consensus that the national movement toward an MLDA of 21 did, in fact, significantly reduce alcohol consumption among the targeted groups. 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. And a Wagenaar and Toomey (2000) review of all published drinking age studies between 1960 and 1999 noted 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 (Wagenaar and Toomey 2002).[10] Finally, the literature that does find significant effects of drinking age on consumption produces a wide range of effect sizes. Cook and Moore (2001) find that a binding MLDA reduces drinking participation by 9 percent and binge drinking by about 17 percent. Dee (1999) finds smaller effects on heavy episodic

[9] In the case of the MLDA, this was done through the 1984 Minimum Legal Drinking Age Act. This stands in marked contrast to policies such as direct increases in state alcohol taxes and other forms of availability regulation, which are often instituted explicitly as a response to public health concerns.

[10] Wagenaar and Toomey's (2002) review included a comprehensive search of four databases, including: the National Institute on Alcohol Abuse and Alcoholism's alcohol problems science database (1960-1999), MEDLINE (1966-1999), Current Contents (1994-1999), and Social Science Abstracts (1983-1999). Their analysis yielded 132 drinking age studies.

drinking of about 8 percent among high school seniors, while Carpenter et al. (2007) find drinking age effects of about 4 percent. Thus, it is fair to say that the questions of whether and how much the drinking age affects alcohol consumption are not settled.

Other research has examined the impact of changes in the MLDA policies on mortality. Studies that use these age-based policies generally employ state/year panels of age-specific mortality outcomes that are plausibly linked to alcohol use to estimate reduced-form models that control for state and year fixed effects, other state demographics, and the MLDA. The literature on MLDA and age-specific traffic fatalities is far too large to review here, but the consensus is that age-based restrictions reduce motor vehicle fatalities. Wagenaar and Toomey's review, for example, noted that 58 percent of the 79 higher-quality studies found a significant inverse relationship between the legal drinking age and traffic accidents.

In contrast to the drinking age literature on highway fatalities, there is much less research on – and as a result less agreement about – the effects of the drinking age on other types of mortality.[11] With respect to suicides, for example, existing drinking age research reveals conflicting evidence, with some studies finding a strong relationship (e.g., Birckmayer and Hemenway 1999; Carpenter 2004) while other research with a similar design fails to find effects (Hingson et al. 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, Jones et al. 1992; Joksch and Jones 1993; and Howland et al. 1998).

This brief review highlights several key gaps in the literature on drinking ages, alcohol consumption, and mortality. First, although there is some credible evidence that the drinking age affects alcohol consumption, a clear consensus has not been reached. And second, the reviewed studies find only moderate consensus that the drinking age affects traffic accidents and no consensus that it affects nontraffic mortality. Given these challenges, it is perhaps not surprising that no previous drinking age study has provided a unified assessment of the implied magnitude of the effect of alcohol consumption on

[11] Wagenaar and Toomey's (2002) review, for example, identified only six drinking age studies that examined mortality outcomes other than traffic fatalities, each focusing on only one or two specific causes of death.

mortality.[12] We fill these gaps by: 1) using a design that removes bias from unobserved policy preferences; 2) considering a much more comprehensive range of alcohol consumption and mortality outcomes than previous research; and 3) directly providing an instrumental variables estimate of the effect of alcohol consumption on mortality among young adults.

2. 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) over the period 1997-2005.[13] 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.[14] For both parts of the analysis we focus on young adults age 19-22, inclusive.

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.[15] 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. Two screening questions were asked of all respondents. First: "In any one year, have you had at least 12 drinks of any type of alcoholic beverage?" And second: "In your entire

[12] One relevant study worth noting that does provide estimates of the elasticity of total mortality to per capita alcohol consumption is by Cook, Ostermann, and Sloan (2005). They use a 30-year state panel of total mortality and control directly for per capita ethanol consumption, producing an elasticity of .23. They also use an index of state alcohol taxes to estimate the reduced-form association with both total mortality and per capita ethanol consumption, producing an implied IV estimate of .33. They do not separately consider young adults.

[13] Our choice of data sources for evaluating the effects of age-based alcohol restrictions on consumption was driven by our need for large samples and the exact age in days of youths in the age range 19-22. The latter requirement eliminated nearly all standard sources of information on alcohol consumption, such as the Behavioral Risk Factor Surveillance System (BRFSS). The former requirement eliminated one-time surveys such as the National Longitudinal Alcohol Epidemiologic Survey (NLAES) and the National Epidemiologic Survey on Alcohol Related Conditions (NESARC).

[14] We also use population estimates from the census to convert mortality counts into rates.

[15] More detailed questions about alcohol consumption were asked as supplements in a handful of years. However, the sample sizes from these years are too small for meaningful analysis in the RD context.

life, have you had at least 12 drinks of any type of alcoholic beverage?" Individuals who had at least 12 drinks were then asked about the frequency and intensity of their alcohol consumption over the past year. We consider six 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.[16] We also examine three measures of drinking frequency and intensity: the percentage of days on which an individual drinks, the percentage of days on which an individual engages in "heavy" drinking, and the average number of drinks a person has on the days on which they drink.[17]

There are two potentially serious problems with estimating the change in alcohol consumption behavior that occurs at age 21 using the NHIS. The first is that the questions on alcohol consumption typically refer to the prior 12 months.[18] This feature implies that in the first months after turning 21 the reference period is largely composed of the person's 20th 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, 75 percent of the respondents answered using a reference period of either the past week or the past month.[19] A careful examination of Figures 1 and 2 reveals that the reference period creates a significant measurement problem only for

[16] A common variable in alcohol research is "binge" or "heavy episodic" drinking, which measures the consumption of 5 or more drinks *at one sitting*. The NHIS question asks respondents: "In the past year, on how many days did you have 5 or more drinks of any alcoholic beverage?" It is possible, though unlikely, that individuals could have consumed their 5 drinks evenly spaced across the day. For this reason we refer to this behavior as heavy drinking rather than binge drinking.

[17] 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.

[18] The actual NHIS drinking frequency and intensity questions are as follows. Respondents who have consumed at least 12 drinks in their lifetime (based on the screening questions described above) are 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. Drinkers were then asked: "In the past year, on those days you drank alcoholic beverages, on the average, how many drinks did you have?" Finally, individuals are asked: "In the past year, on how many days did you have 5 or more drinks of any alcoholic beverage?"

[19] For the question "In the past year, on how many days did you have 5 or more drinks of any alcoholic beverage?" 37 percent of respondents used a reference period of one week or one month rather than a year.

people just over 21. For all the measures of drinking intensity we examine, people interviewed in the first month after they turned 21 reported rates very similar to people interviewed just before they turned 21 and substantially lower than other 21-year-olds. The figures also reveal that people interviewed in the second month after turning 21 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.

The other potentially serious problem is the possibility that alcohol consumption 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. 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 will 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 21st 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. The pattern we actually observe is 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 over the

period 1997-2004.[20] 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.[21] 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.[22] To account for a pronounced 21st-birthday mortality effect (see the day-specific death rates in Appendix A), for all outcomes we report estimates from regression models that include dummy variables for the 21st birthday and the day after.[23] In nearly all cases the inclusion of these birthday dummies does not change the main results.

3. Methods

In this section we begin by describing how we estimate the reduced-form impact of the MLDA laws on alcohol consumption and mortality. We then describe how the reduced-form estimates can be combined to obtain the implied instrumental variables estimate of the impact of alcohol consumption on mortality.

3.1 Reduced Form

[20] The 2005 mortality data are not yet available. However, estimates using data from 1990-1996 are very similar to estimates using the 1997-2004 data (see Appendix I), suggesting that including the 2005 data – so as to match the data available from the NHIS exactly – would not significantly affect our results.

[21] 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. That is, 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 (Escobedo and Ortiz 2002, and others).

[22] Throughout, we use the International Classification of Diseases, 9th and 10th revisions. A full list of the cause-of-death codes is provided in Appendix E. 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.

[23] 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 we drop deaths occurring on the 21st birthday and the day immediately after the 21st birthday from the analysis.

Consider a simple reduced-form model:

$$(1) \qquad y_i = X_i \beta^y + g^y(a) + D_a \pi^y + v^y_i,.$$

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

$$(2) \qquad y = E[y_i] = k + g^y(a) + D_a \pi^y ,$$

where k is a constant. We will estimate the model above by modeling $g^y(a)$ as a low-order polynomial.[24]

In addition to estimating the discontinuity parametrically as described above, we also estimate it nonparametrically using local linear regression as detailed in Hahn, Todd, and van der Klaauw (2001). To estimate the discontinuity nonparametrically we implement a local linear regression procedure using the bandwidth selection procedure suggested in Fan and 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.

3.2 Implied Instrumental Variables Estimate

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. In particular, a structural model is needed to estimate how much mortality rates will increase

[24] We assess the fit of the polynomial both graphically and by comparing a series of nested models.

when alcohol consumption increases. Suppose that an increase in alcohol consumption is hypothesized to increase the probability that a person will die. Then an appropriate causal model is

$$(3) \qquad y_i = X_i\alpha + f(a) + C_i\delta + u_i ,$$

where y_i is the mortality probability of individual i, C_i is a measure of alcohol consumption by individual i, f(a) is the structural age profile, and u_i is an idiosyncratic error. Suppose in addition that alcohol consumption is determined by a simple (first-stage) model of the form

$$(4) \qquad C_i = X_i \beta^C + g^C(a) + D_a \pi^C + v^C_i ,$$

where β^C is a vector of coefficients, $g^C(a)$ is the age profile of alcohol consumption, and π^C represents the discrete increase in alcohol consumption that results from the increase in alcohol availability at age 21. Equations (3) and (4) imply a reduced-form model of the form of equation (1) in which the reduced-form effect of reaching age 21 on outcome y is $\pi^y = \pi^C \times \delta$. The causal effect of alcohol consumption on mortality, δ, can be estimated by forming the ratio of the estimated discontinuity in mortality at age 21 to the estimated discontinuity in alcohol consumption.

4. 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.

4.1 Alcohol Consumption

In this subsection we document how the frequency and intensity of alcohol consumption change when people turn 21.[25] In Figure 1 we present the age profiles for

[25] 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,

the proportion of respondents who reported consuming 12 or more alcoholic drinks in any one year, those who reported consuming 12 or more drinks over 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. Over these proportions we have superimposed the fitted lines from regressions on the underlying micro data.[26] 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 that there was not much increase in drinking at age 21. The figure also shows that people interviewed in the first month after their 21st birthday reported drinking behavior very similar to that reported by 20-year-olds. As noted above, this is probably due to the retrospective nature of the question. For this reason, in all the empirical specifications for the alcohol consumption variables we include a dummy variable for people interviewed in the 30 days after they turned 21.[27]

In the first panel of Table 1 we present the regression estimates of the increase in the proportion of people who reported having had 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.[28] In the second column of the panel we present the same regression using the NHIS sample weights to obtain the predicted jump at age 21 for the entire US population.[29] 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 21st birthday or the day immediately

---

these samples cannot be used to identify the effects of an age-21 drinking age. Moreover, one of the few studies to explicitly make use of young adults over age 21 to evaluate the effects of the drinking age on alcohol consumption found little evidence that drinking age laws significantly affect youth drinking (Kaestner 2000).

[26] The regression is a quadratic polynomial in age fully interacted with a dummy for over 21. 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.

[27] 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.

[28] Since the age variable in the regression is the number of days until (or since) the person's 21st 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.

[29] The means in all the figures derived from the NHIS are unweighted. Adjusting for sample weights increases the variance of the estimate and visibly increases the variation visible in the figure.

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.[30] 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.[31] 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.[32]

Figure 1 also plots the age profile of the proportion of people who reported having 12 or more drinks in one year. The regression line superimposed on the dark circles shows that there is a substantial jump in the proportion of people who reported having 12 or more drinks in one year. We present the corresponding regression results in the second panel of Table 1. The first specification confirms that the increase we saw in Figure 1 is about 8 percentage points and is statistically significant. The inclusion of weights in the second specification reduces the point estimate slightly, but the estimate remains statistically significant. The inclusion of covariates in the third specification has very little effect on the point estimate, again suggesting that these variables evolve

[30] Below, we directly show that these covariates are smoothly distributed across the age-21 discontinuity.

[31] The cubic polynomial is likely much more sensitive to the fact that (as observed in Figure 1) people interviewed just after their 21st birthday reported levels of alcohol consumption very similar to those reported by 20-year-olds.

[32] Note that lifetime abstainers are included in our analyses of all alcohol consumption outcomes; our only sample restriction is to drop those who refused to answer the alcohol screener questions. This is appropriate because we are interested in the effects of age-based restrictions on population outcomes (alcohol consumption and mortality).

smoothly through the discontinuity. The fourth specification reveals that the discontinuity estimate is somewhat sensitive to the inclusion of a cubic term in the polynomial. As with the lifetime drinking outcome, this is likely due to the retrospective nature of the question. Similarly, the local linear regression also returns a somewhat smaller estimate of the age 21 discontinuity than the preferred specification in column 3.

The third outcome in Figure 1 – represented by the crosses – shows the fraction of the sample reporting that they consumed 5 or more drinks on a single day at least once in the previous year (i.e., some "heavy" drinking). This variable exhibits a sharp increase at age 21 of about 7 percentage points. The corresponding regression estimates in the first column of the last panel of Table 1 confirm that the increase we observe in the figure is statistically significant. Even after we allow for the inclusion of covariates, the age-21 discontinuity estimate suggests a large increase (approximately16 percent relative to people just under 21) in the proportion of people who reported ever having engaged in heavy drinking.[33] Allowing for a cubic term in age in column 4 reduces the discontinuity estimate substantially, though again this is probably because this more flexible specification is more sensitive to the retrospective nature of the question.

In Figure 2 we present the age profiles of various measures of drinking intensity. In this figure the hollow squares denote the proportion of days on which a person drinks any alcohol. The figure reveals a discrete jump of about 2 percentage points in the proportion of days drinking. In the first column of the first panel of Table 2 we present the corresponding regression, which confirms that the increase we observe in the figure is about 2.5 percentage points. This is substantial given that the average 20-year-old reported drinking on 8.5 percent of days.[34] The inclusion of weights in the second specification has a substantial impact on the point estimate, though it remains significant at the 10 percent level. In the third specification we observe that the inclusion of birthday dummies and covariates does not further change the estimates (still indicating a 21 percent increase relative to people just under 21), while the fourth column reveals that the addition of a cubic term reduces the estimate of the jump at age 21 substantially. Again,

[33] This increase – though it is clearly visible in the figure – is only significant at the 10 percent level.

[34] That there is a discrete jump may be surprising given the long reference window (the past year). However, as described above, respondents were able to choose their reference period, and 75 percent of respondents reported about their behavior in the past week or month.

however, Figure 2 shows that this is likely due to the low level of reported drinking among people interviewed just after their 21st birthday – and importantly, the model with the cubic in age is not supported by the data. Finally, column 5 shows that, unsurprisingly, the local linear regression estimate is also smaller than the estimates from our parametric models.

Figure 2 also shows evidence of an increase in the proportion of days of heavy drinking. Though the corresponding regressions presented in the second panel of Table 2 reveal that the 22 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 age 21, these results suggest that the amount of heavy drinking increases in response to the easier alcohol availability that results from turning 21.

Having documented sharp increases at age 21 in a variety of alcohol consumption measures, we now turn to examining the number of drinks consumed on the days the individual drank. This outcome is also illustrated in Figure 2 (with units indicated on the right axis). This figure – and the associated regression estimates in Table 2 – does not provide evidence of a discrete break in the average number of drinks that people consume after they turn 21. All of the discontinuity estimates for the "drinks on days drinking" variable are 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 is statistically significant. 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 have also created the histogram of the number of average drinks per day for 20-year-olds and 21-year-olds in the NHIS sample. Figure 3 shows 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 in the first two panels of Table 2. 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.

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 educational attainment all evolve smoothly through the age 21 threshold.[35] We find no evidence of a discrete change at age 21 in any of these characteristics. While this is not surprising for the demographic characteristics, the null findings for employment and health insurance status in particular are comforting.[36] That these observable characteristics are 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.

4.2 Mortality

In this section we evaluate whether the increase in alcohol consumption documented above results in an increase in mortality rates. In Figure 4 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.[37] 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 external injuries. Figure 4 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 almost entirely to deaths due to external causes.

[35] We present the corresponding age profiles in Appendix D.
[36] For example, it might be plausible to find a discontinuity in health insurance since in some states people age out of Medicaid when they turn 21.
[37] The ICD-9 and ICD-10 that compose the external cause group are listed in Appendix E. The internal death category includes all deaths not coded as due to external causes.

In Table 4 we present the regression estimates corresponding to Figure 4. The dependent variable in the regression is the log of the counts of deaths occurring X days before or after the individual's 21st birthday, and we estimate the model over the 1,465 days between ages 19 and 22, inclusive.[38] 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.[39] In the second column we add a dummy for the 21st birthday and a dummy for the day immediately after.[40] In the third specification of each panel we add a cubic term to the polynomial. In the fourth column we present the estimate using a local linear regression with a rule-of-thumb bandwidth for each side of the age-21 cutoff (Fan and Gijbels 1996).[41] 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 4: there is a statistically significant 9 percent increase in overall mortality when people turn 21 that is almost entirely due to a 10 percent increase in deaths due to external causes.

Figure 5 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 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. In Tables 5 and 6 we present the corresponding regression results. These estimates confirm what we saw in Figure 5: deaths with an explicit mention of alcohol increase by over 30 percent at age 21, homicides exhibit essentially no change, and the suicide rate increases by over 15 percent. In Table 6 we document that deaths due to motor vehicle accidents increase by about 14 percent at age 21.[42] Estimates of

[38] 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.

[39] The figures in Appendix F suggest this is a sufficiently flexible model to fit the age profile of the death rate.

[40] As seen in Appendix A, there is a significant 21st-birthday effect.

[41] In Appendix G we present the sensitivity of the 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 4.

[42] 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

external deaths attributable to drugs are not precisely estimated, while deaths due to "other external causes" (i.e., all deaths due to external causes not included elsewhere in Tables 5 or 6) show evidence of a discrete increase of about 10 percent, an estimate that nears statistical significance in some of the models.[43] This result for "other external deaths," which includes deaths due to fire, falling, and drowning, provides some evidence that the strong correlation between alcohol consumption and these causes of deaths is causal.

In Table 7 we present the estimates of how the increase in total mortality varies by gender, race, and education.[44] The regressions stratified by gender reveal that men experience a 10 percent increase in mortality and account for the majority of the entire increase in mortality. The regressions stratified by race reveal that the increase in deaths is largely driven by the 14 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 16 percent increase in deaths among people attending college. 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.

5. 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

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.

[43] 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 Tables 5 and 6 we have added .5 to the dependent variable before taking the log. Appendix H shows that for these two outcomes the results are robust to two alternative approaches: adding 1 (instead of .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).

[44] For compactness we have included only the results from our preferred specification. 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 across subgroups in alcohol consumption.

21 reported drinking on 21 percent more days than individuals just under 21; those just over age 21 reported a similar increase in heavy drinking. 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 experiment with heavy drinking immediately after they turn 21. If the increase in mortality were caused by experimentation with heavy drinking, it should rise discretely at 21 and then return rapidly to the pre-21 level; Figures 4 and 5 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.[45] 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 .41 for individuals who change their drinking behavior because of the law. This elasticity suggests that a substantial proportion of deaths among 21-year-olds is directly due to alcohol consumption. It also suggests that the return to reducing underage drinking is potentially quite high.

An important question to consider when interpreting these results is: how persistent are the effects of easing access to alcohol at age 21? It is possible that the

[45] One potential 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.

MLDA laws simply shift the timing of deaths instead of reducing the number of deaths.[46] While our research design does not let us answer this question directly, we can provide some suggestive visual evidence on the degree to which the mortality effects persist after age 21. Specifically, in Figure 6 we generate a counterfactual age profile for people over 21 based on a quadratic polynomial fitted to the profile for people under 21. Despite the obvious limitations of this approach, it does suggest how alcohol consumption and mortality might evolve if the restrictions on alcohol were not lifted at age 21. The figure suggests that lowering the cost of drinking leads to an increase in alcohol consumption that persists for at least two years. The increase in deaths due to external causes also appears persistent.[47] That the increase in both drinking and mortality is persistent suggests that the MLDA laws are reducing lifetime drinking rather than just changing the age at which people begin to drink. This also suggests that a higher minimum drinking age actually reduces mortality, rather than just changing the age at which people die.

Finally, our finding that alcohol consumption increases mortality among young adults 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.

[46] For suggestive evidence in favor of the "shifting" hypothesis, see Dee and Evans (2001) and Males (1986). Other research does not find strong evidence of shifting (see, for example, Saffer and Grossman 1987 and Cook and Tauchen 1984).

[47] The projected mortality rate due to internal causes is very similar to the actual age profile, which suggests that creating a counterfactual age profile of mortality in this fashion is not unreasonable.

References

Baughman, Reagan, Stacy Dickert-Conlin, Michael Conlin, and John Pepper (2001). "Slippery When Wet: The Effects of Local Alcohol Access Laws on Highway Safety," *Journal of Health Economics*, 20(6): 1089-1096.

Birckmayer, Johanna, and David Hemenway (1999). "Minimum Age Drinking Laws and Youth Suicide 1970-1990," *American Journal of Public Health*, 89(9): 1365-1368.

Bonnie, R., and M. O'Connell, eds. (2004). *Reducing Underage Drinking: A Collective Responsibility*. Washington, DC: The National Academies Press.

Carpenter, Christopher (2004). "Heavy Alcohol Use and Youth Suicide: Evidence from Tougher Drunk Driving Laws," *Journal of Policy Analysis and Management*, 23(4): 831-842.

Carpenter, Christopher, Deborah D. Kloska, Patrick O'Malley, and Lloyd Johnston (2007). "Alcohol Control Policies and Youth Alcohol Consumption: Evidence from 28 Years of Monitoring the Future," *Berkeley Electronic Press Journals in Economic Analysis and Policy*, 7(1 Topics): Article 25.

Cook, Philip, and Michael Moore (2001). "Environment and Persistence in Youthful Drinking Patterns," in *Risky Behavior Among Youths: An Economic Analysis*, J. Gruber, ed. Chicago: University of Chicago Press.

Cook, Philip, and George Tauchen (1984). "The Effect of Minimum Drinking Age Legislation on Youthful Auto Fatalities," *The Journal of Legal Studies*, 13(1): 169-190.

----- (1982). "The Effect of Liquor Taxes on Heavy Drinking," *Bell Journal of Economics*, 13(2): 379-90

Cook, Philip, Jan Ostermann, and Frank Sloan (2005). "Are Alcohol Taxes Good for Us? Short and Long-Term Effects on Mortality Rates," NBER Working Paper #11138. Cambridge, MA: National Bureau of Economic Research.

Dee, Thomas (1999). "State Alcohol Policies, Teen Drinking and Traffic Fatalities," *Journal of Public Economics*, 72: 289-315.

Dee, Thomas, and William Evans (2001). "Behavioral Policies and Teen Traffic Safety," *American Economic Review*, 91(2): 91-96.

Escobedo, Luis, and Melchor Ortiz (2002). "The Relationship Between Liquor Outlet Density and Injury and Violence in New Mexico," *Accident Analysis and Prevention*, 34: 689-694.

Fan, J., and I. Gijbels (1996). *Local Polynomial Modeling and Its Applications*. London: Chapman and Hall.

Grossman, M., F. Chaloupka, H. Saffer, and A. Laixuthai (1994). "Effects of Alcohol Price Policy on Youth: A Summary of Economic Research," *Journal of Research on Adolescence*, 4: 347-364.

Hahn, Jinyong, Petra Todd, and Wilbert van der Klaauw (2001). "Identification and Estimation of Treatment Effects with a Regression-Discontinuity Design," *Econometrica*, 69(1): 201-209.

Hingson, R., D. Merrigan, and T. Heeren (1985). "Effects of Massachusetts Raising Its Legal Drinking Age from 18 to 20 on Deaths from Teenage Homicide, Suicide, and Nontraffic accidents," *Pediatric Clinics of North America*, 32: 221-232.

Howland, J., J. Birckmayer, D. Hemenway, and J. Cote (1998). "Did Changes in Minimum Age Drinking Laws Affect Adolescent Drowning (1970-1990)?" *Injury Prevention*, 4: 288-291.

Johnston, L. D., P. M. O'Malley, J. G. Bachman, and J. E. Schulenberg, (2005). *Monitoring the Future National Survey Results on Drug Use, 1975-2004. Volume I: Secondary School Students* (NIH Publication No. 05-5727). Bethesda, MD: National Institute on Drug Abuse.

Jones, N., C. Pieper, and L. Robertson (1992). "Effect of Legal Drinking Age on Fatal Injuries of Adolescents and Young Adults," *American Journal of Public Health*, 82: 112-115.

Joksch, Hans, and Ralph Jones (1993). "Changes in the Drinking Age and Crime," *Journal of Criminal Justice*, 21: 209-221.

Kaestner, Robert (2000). "A Note on the Effect of Minimum Drinking Age Laws on Youth Alcohol Consumption," *Contemporary Economic Policy*, 18(3): 315-325.

Males, Mike (1986). "The Minimum Purchase Age for Alcohol and Young-Driver Fatal Crashes: A Long-Term View," *The Journal of Legal Studies*, 15(1): 181-211.

Saffer, Henry, and Michael Grossman (1987). "Beer Taxes, the Legal Drinking Age, and Youth Motor Vehicle Fatalities," *The Journal of Legal Studies*, 16(2): 351-374.

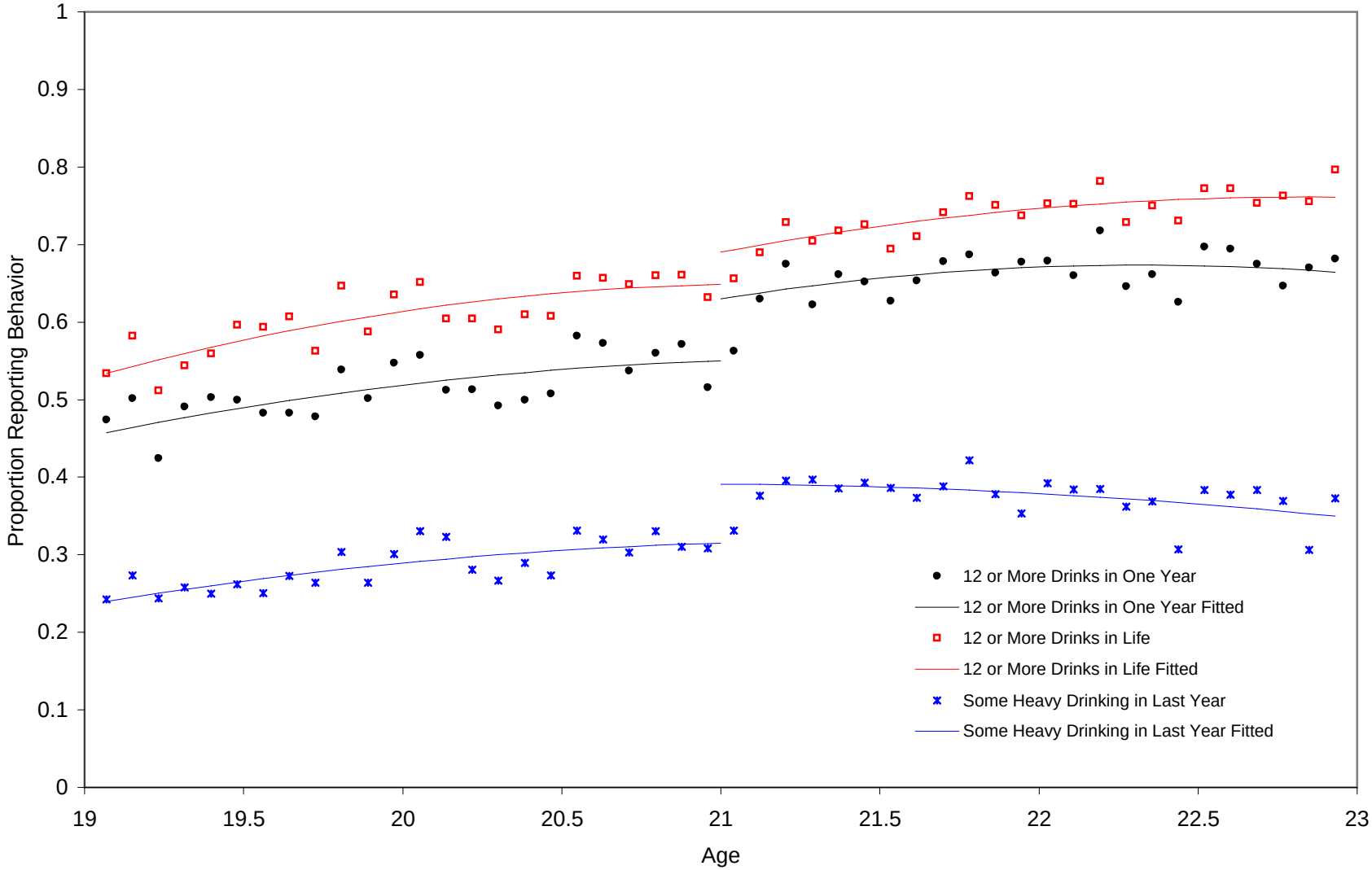
Thistlewaite, D., and D. Campbell (1960). "Regression-Discontinuity Analysis: An Alternative to the Ex Post Facto Experiment," *Journal of Educational Psychology*, 51: 309-317.

Wagenaar, Alexander, and Traci Toomey (2002). "Effects of Minimum Drinking Age Laws: Review and Analyses of the Literature from 1960 to 2000," *Journal of Studies on Alcohol*, Supplement No. 14: 206-225.

Young, Douglas, and Agnieska Bielinska-Kwapisz (2002). "Alcohol Taxes and Beverage Prices," *National Tax Journal*, 55: 57-73.

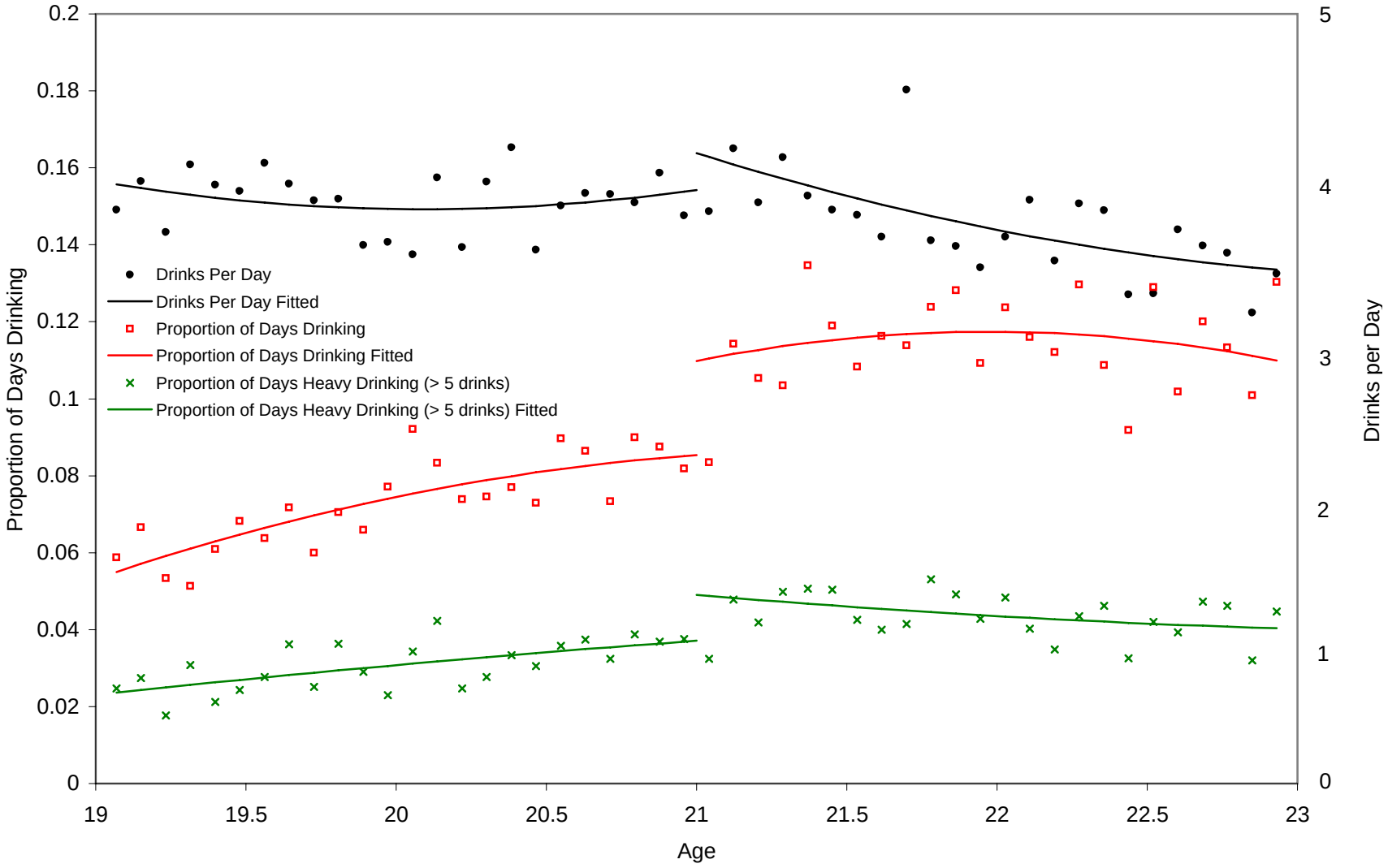
Zhu, L., D. Gorman, and S. Horel (2004). "Alcohol Outlet Density and Violence: A Geospatial Analysis," *Alcohol & Alcoholism*, 39(4): 369-37.

Figure 1: Age Profile of Drinking Participation



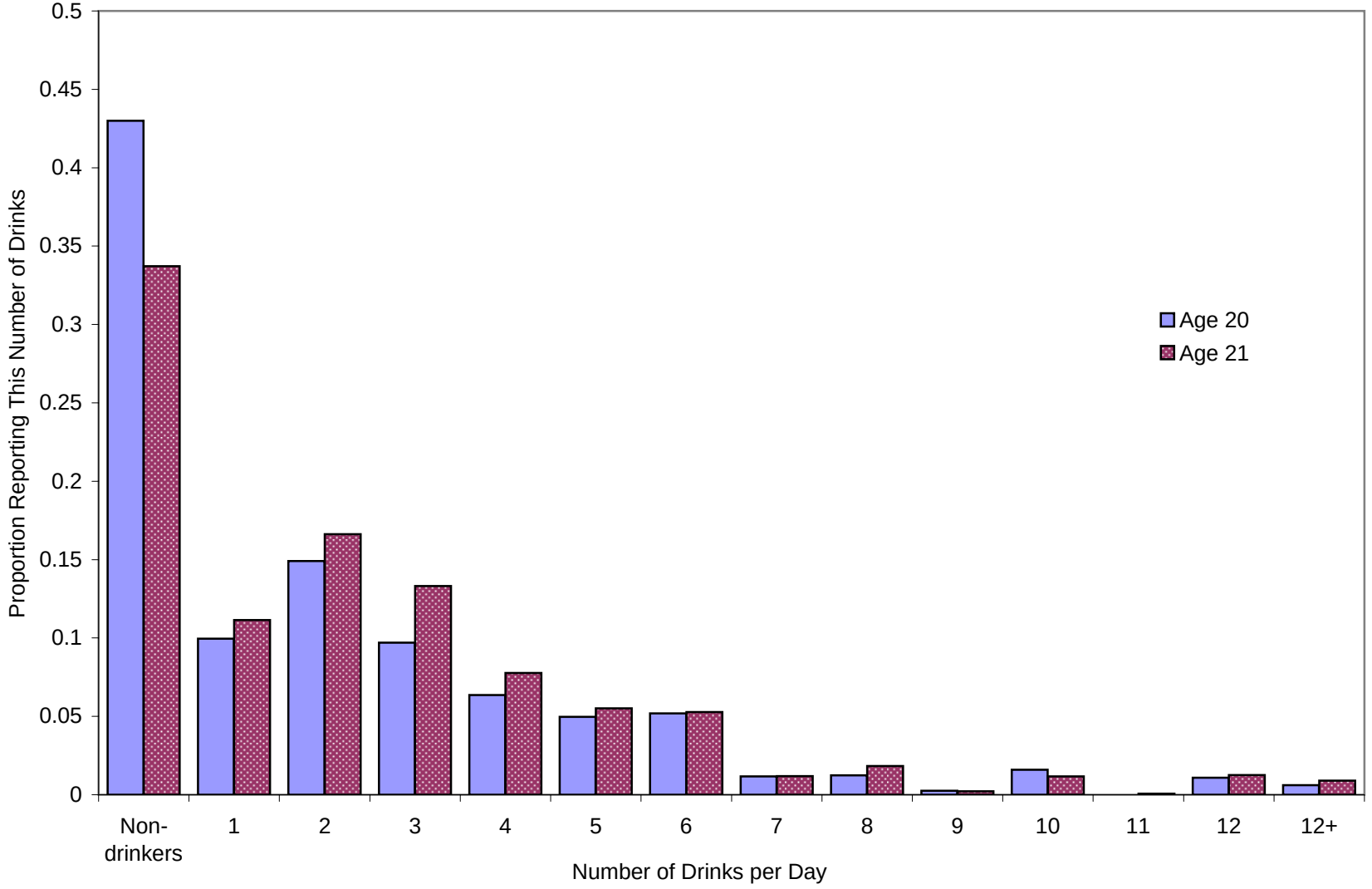
Notes: NHIS Sample Adult 1997-2005. Cells are the proportion of people in a 30-day block that report the behavior. The regression line is a second-order polynomial fitted on unweighted individual observations on either side of the age 21 cutoff.

Figure 2: Age Profile of Drinking Intensity



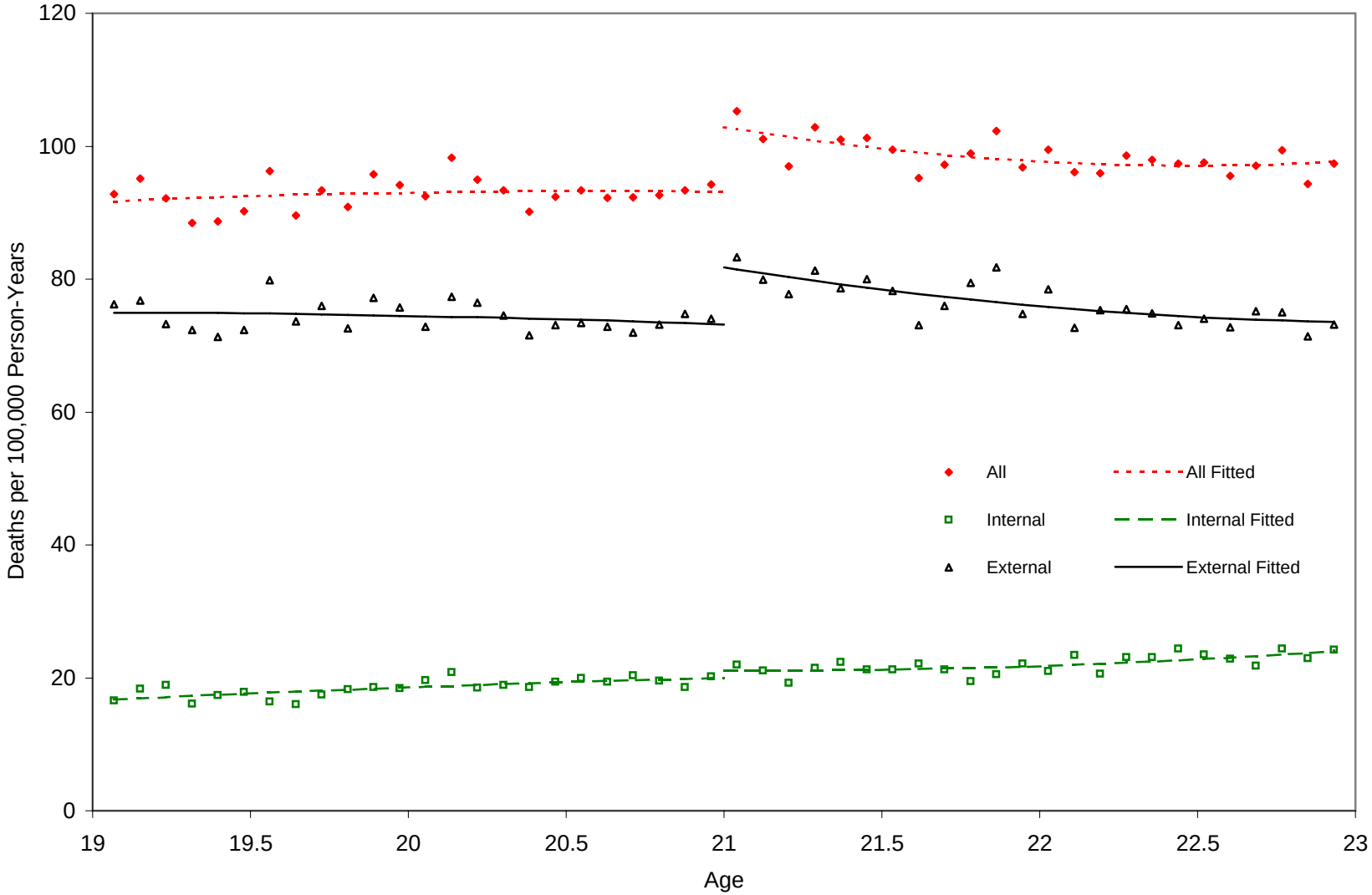
Notes: People can report their drinking for the last week, month, or year; 78% reported the number of days they drank in the last week or month rather than the last year. Average number of drinks per day is for people who reported some drinking.

Figure 3: Reported Average Number of Drinks per Day



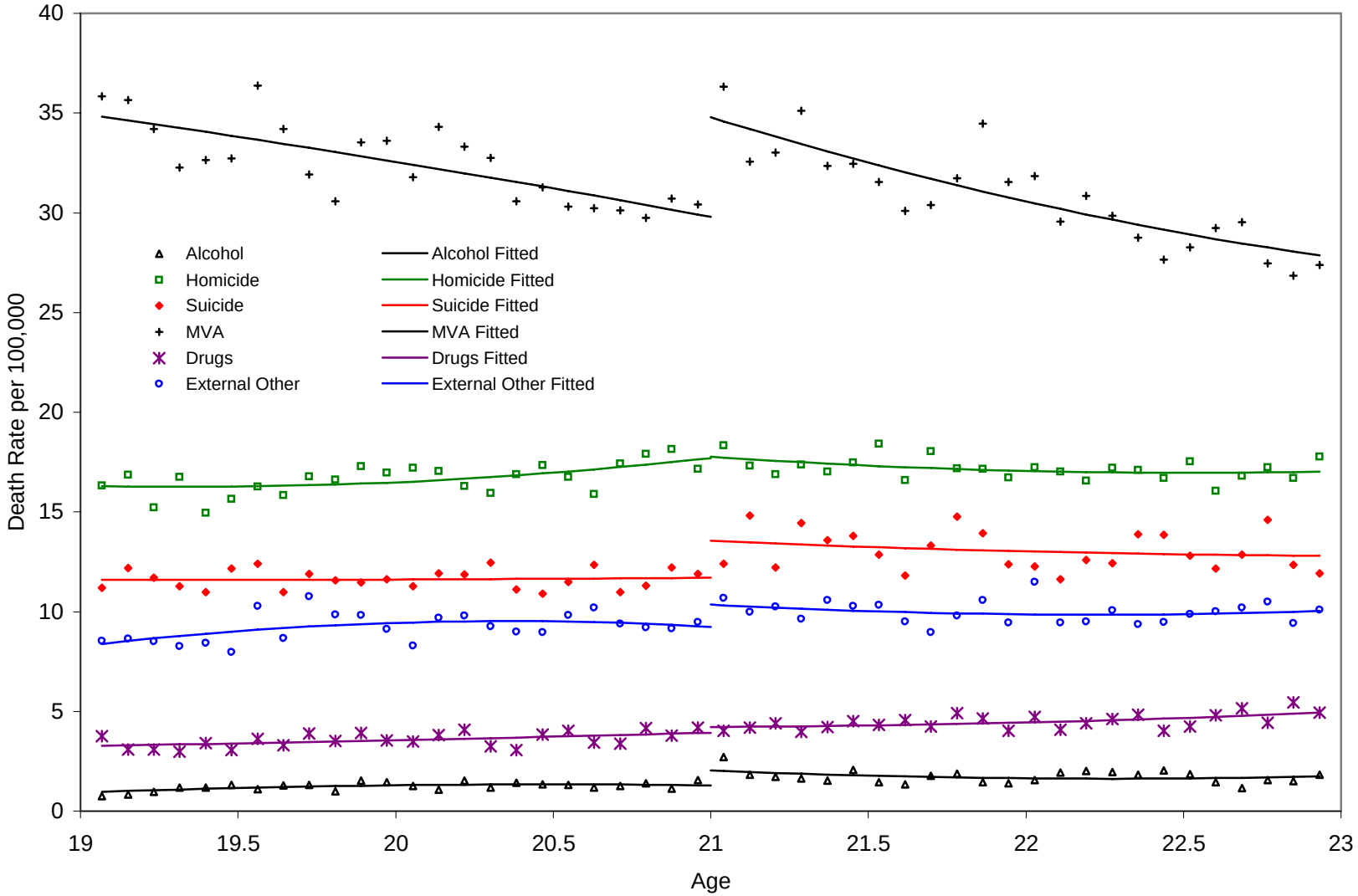
Notes: See notes from Figure 2. There are 3,950 twenty year olds in the sample and 4,206 twenty one year olds in the sample.

Figure 4: Age Profile for Death Rates



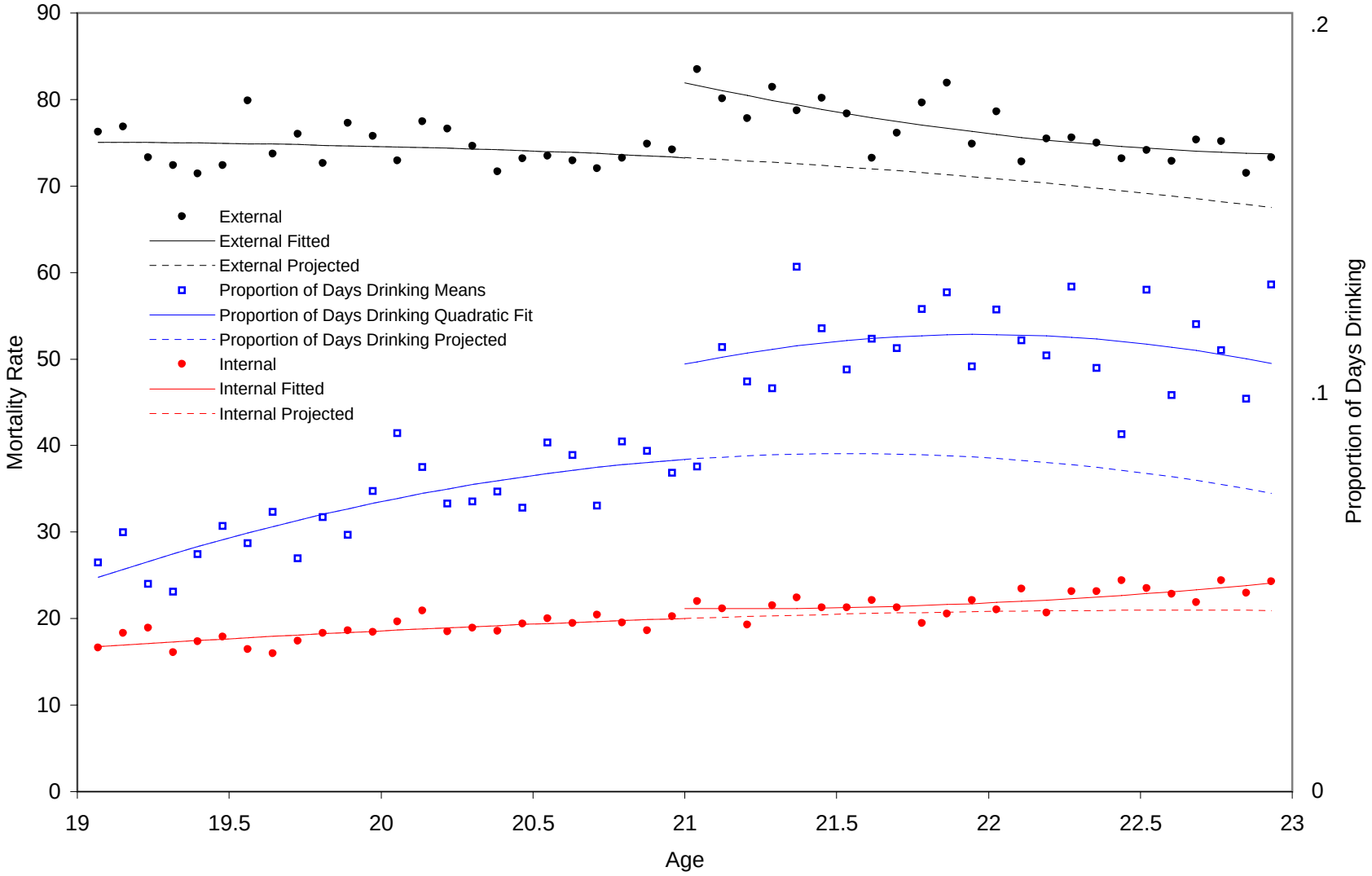
Notes: Deaths from the National Vital Statistics Records. Includes all deaths that occurred in the US between 1997-2003. The population denominators are derived from the census. See Appendix E for a list of causes of death.

Figure 5: Age Profiles for Death Rates by External Cause



Notes: See notes to Figure 4. The categories are mutually 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 E.

Figure 6: Projections of Alcohol Consumption



Notes: The dotted lines are the projection of the pre-21 age profile, which has been fitted with a quadratic polynomial.

Table 1: Alcohol Consumption - Measures of Participation

	12 or More Drinks in Lifetime					12 or More Drinks in One Year					Any Heavy Drinking in Last Year				
	Parametric Models				Local Linear	Parametric Models				Local Linear	Parametric Models				Local Linear
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
Over 21	0.0418	0.0316	0.0268	0.0198	0.0199	0.0796	0.0657	0.0611	0.0603	0.0461	0.0761	0.0527	0.0492	0.0262	0.0398
	[0.0242]	[0.0301]	[0.0292]	[0.0423]	[0.0179]	[0.0254]	[0.0313]	[0.0301]	[0.0438]	[0.0218]	[0.0248]	[0.0304]	[0.0291]	[0.0430]	[0.0201]
Age	0.0081	0.0293	0.0244	0.1235		0.0140	0.0273	0.0229	0.1198		0.0116	0.0688	0.0623	0.1403	
	[0.0385]	[0.0482]	[0.0471]	[0.1159]		[0.0396]	[0.0491]	[0.0477]	[0.1174]		[0.0361]	[0.0445]	[0.0431]	[0.1080]	
Age Sq	-0.0265	-0.0239	-0.0247	0.0980		-0.0176	-0.0173	-0.0167	0.1033		-0.0141	0.0098	0.0084	0.1048	
	[0.0190]	[0.0236]	[0.0231]	[0.1355]		[0.0194]	[0.0238]	[0.0232]	[0.1366]		[0.0175]	[0.0211]	[0.0205]	[0.1224]	
Age*Over21	0.0693	0.0446	0.0597	-0.0874		0.0521	0.0554	0.0675	-0.1058		-0.0139	-0.0683	-0.0544	-0.0853	
	[0.0544]	[0.0674]	[0.0655]	[0.1727]		[0.0571]	[0.0701]	[0.0678]	[0.1786]		[0.0554]	[0.0673]	[0.0646]	[0.1726]	
Age Sq*Over 21	0.0054	0.0096	0.0020	-0.0655		-0.0075	-0.0103	-0.0184	-0.0507		0.0041	-0.0127	-0.0181	-0.1686	
	[0.0259]	[0.0319]	[0.0311]	[0.1935]		[0.0272]	[0.0332]	[0.0321]	[0.2001]		[0.0261]	[0.0316]	[0.0303]	[0.1905]	
Month After 21	-0.0392	-0.0224	-0.0168	-0.0246		-0.0688	-0.0441	-0.0379	-0.0505		-0.0576	-0.0359	-0.0242	-0.0165	
	[0.0312]	[0.0378]	[0.0367]	[0.0419]		[0.0327]	[0.0394]	[0.0379]	[0.0432]		[0.0318]	[0.0384]	[0.0369]	[0.0424]	
21st Birthday			0.1290	0.1270				0.2186	0.2154				-0.3156	-0.3136	
			[0.1405]	[0.1406]				[0.1405]	[0.1406]				[0.1386]	[0.1387]	
21st Birthday + 1			-0.0470	-0.0487				-0.0437	-0.0466				0.1374	0.1393	
			[0.1492]	[0.1493]				[0.1592]	[0.1593]				[0.1671]	[0.1673]	
Age Cubic				-0.0582					-0.0677					-0.0148	
				[0.0624]					[0.0644]					[0.0607]	
Age^3*Over 21				0.0407					0.0398					0.0320	
				[0.0448]					[0.0451]					[0.0397]	
Constant	0.6483	0.6487	0.5503	0.5671		0.5503	0.5477	0.4470	0.4635		0.3150	0.3247	0.2389	0.2522	
	[0.0163]	[0.0204]	[0.0245]	[0.0303]		[0.0169]	[0.0211]	[0.0248]	[0.0308]		[0.0156]	[0.0199]	[0.0225]	[0.0287]	
Covariates	No	No	Yes	Yes	No	No	No	Yes	Yes	No	No	No	Yes	Yes	No
Weights	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes	No
Observations	16,107	16,107	16,107	16,107		16,107	16,107	16,107	16,107		16,107	16,107	16,107	16,107	
R-squared	0.02	0.03	0.1	0.1		0.02	0.03	0.11	0.11		0.01	0.01	0.1	0.1	
Prob > Chi-Squared			0.00	0.61				0.00	0.56				0.00	0.67	

Notes: The first column of each block contains the regression from the corresponding figure. Standard errors in brackets. Birthday is a dummy for 21st birthday and Birthday + 1 is a dummy for the day after the 21st birthday. Covariates include dummies for Census Region, Race, Gender, Health Insurance, Employment Status 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 (not necessarily in one sitting) are coded as heavy drinkers. Bandwidths from the left and the right for each outcome are: 12 or more One Year (0.81,0.75), 12 or More in Lifetime (1.28, 0.88) and proportion reporting Heavy Drinking (1.06, 0.72).

Table 2: Alcohol Consumption - Measures of Intensity

	Proportion of Days Drinking					Proportion of Days Heavy Drinking					Drinks per Day on Days Drinking				
	Parametric Models				Local Linear	Parametric Models				Local Linear	Parametric Models				Local Linear
	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)	(1)	(2)	(3)	(4)	(5)
Over 21	0.0245	0.0180	0.0182	0.0119	0.0107	0.0120	0.0075	0.0075	0.0021	0.0026	0.2387	0.2068	0.2465	0.2806	0.1886
	[0.0086]	[0.0097]	[0.0095]	[0.0135]	[0.0072]	[0.0061]	[0.0063]	[0.0062]	[0.0091]	[0.0048]	[0.2810]	[0.3403]	[0.3291]	[0.4782]	[0.2024]
Age	0.0056	0.0085	0.0068	-0.0071		0.0056	0.0057	0.0045	0.0134		0.2774	0.6407	0.4749	0.7006	
	[0.0116]	[0.0138]	[0.0137]	[0.0338]		[0.0081]	[0.0088]	[0.0088]	[0.0216]		[0.3538]	[0.4608]	[0.4358]	[1.1732]	
Age Sq	-0.0052	-0.0042	-0.0045	-0.0217		-0.0007	-0.0007	-0.0013	0.0097		0.1531	0.3581	0.2595	0.5438	
	[0.0055]	[0.0066]	[0.0065]	[0.0388]		[0.0039]	[0.0041]	[0.0041]	[0.0249]		[0.1730]	[0.2144]	[0.2035]	[1.2958]	
Age*Over21	0.0101	0.0140	0.0168	0.0724		-0.0123	-0.0034	-0.0014	0.0087		-0.8909	-1.4498	-1.1870	-1.7552	
	[0.0192]	[0.0220]	[0.0216]	[0.0558]		[0.0133]	[0.0144]	[0.0142]	[0.0370]		[0.5763]	[0.6656]	[0.6372]	[1.7127]	
Age Sq*Over 21	-0.0029	-0.0055	-0.0065	-0.0371		0.0019	-0.0012	-0.0012	-0.0340		-0.0387	-0.1217	-0.0498	0.0571	
	[0.0090]	[0.0104]	[0.0101]	[0.0625]		[0.0062]	[0.0067]	[0.0066]	[0.0408]		[0.2608]	[0.2937]	[0.2836]	[1.8047]	
Month After 21	-0.0245	-0.0220	-0.0147	-0.0078		-0.0163	-0.0118	-0.0070	-0.0039		-0.3742	-0.5580	-0.3151	-0.3719	
	[0.0111]	[0.0120]	[0.0122]	[0.0138]		[0.0066]	[0.0067]	[0.0067]	[0.0080]		[0.3226]	[0.3317]	[0.3222]	[0.3904]	
21st Birthday			-0.0758	-0.0740				-0.0463	-0.0455				-2.3836	-2.3989	
			[0.0200]	[0.0201]				[0.0132]	[0.0133]				[0.6654]	[0.6681]	
21st Birthday + 1			-0.0497	-0.0481				-0.0239	-0.0231				-1.7425	-1.7555	
			[0.0287]	[0.0288]				[0.0074]	[0.0075]				[0.3517]	[0.3547]	
Age Cubic				0.0209					0.0033					-0.2196	
				[0.0201]					[0.0131]					[0.5550]	
Age^3*Over 21				-0.0057					0.0036					0.0956	
				[0.0127]					[0.0081]					[0.4105]	
Constant	0.0853	0.0836	0.0602	0.0579		0.0371	0.0340	0.0188	0.0203		3.8570	3.8943	2.9378	2.9747	
	[0.0051]	[0.0059]	[0.0072]	[0.0090]		[0.0036]	[0.0039]	[0.0048]	[0.0058]		[0.1550]	[0.2174]	[0.2161]	[0.2919]	
Covariates	No	No	Yes	Yes	No	No	No	Yes	Yes	No	No	No	Yes	Yes	No
Weights	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes	No	No	Yes	Yes	Yes	No
Observations	16,107	16,107	16,107	16,107		15,825	15,825	15,825	15,825		9,906	9,906	9,906	9,906	
R-squared	0.02	0.02	0.07	0.07		0	0.01	0.05	0.05		0.00	0.00	0.07	0.07	
Prob > Chi-Squared			0.00	0.56				0.00	0.72				0.00	0.92	

Notes: See notes to Table 1. People can report their drinking for the last week, month or year. For people who reported any drinking 78% of people just over 21 reported about their drinking in the last week or month rather than the last year. The probability of reporting drinking over the last week or last month goes up at age 21. For the dependent variable "Drinks per Day" only people who reported drinking are included in this analysis, so there is a composition change in the sample at age 21 due to the increase in the number of people who reported that they drink. The dependant variable is the number of drinks the respondent reported drinking on average on the days that they drank. Bandwidths from the left and the right for each outcome are: percent days drinking (0.87, 0.75), drinks per day on days drinking (0.64,1.42), and days heavy drinking (1.02, 0.73).

Table 3: Age Profile of Demographic Characteristics from the NHIS

	Male	White	Black	Hispanic	No HS Diploma	HS Diploma	Employed	Looking for Work	No Health Insurance
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(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]
Age	0.0025	0.0152	-0.0139	0.0068	0.0435	0.0074	0.0033	0.0299	-0.0151
	[0.0395]	[0.0451]	[0.0321]	[0.0296]	[0.0373]	[0.0457]	[0.0475]	[0.0263]	[0.0440]
Age Sq	0.0092	0.0185	-0.0120	-0.0025	0.0403	0.0189	-0.0238	0.0189	-0.0201
	[0.0194]	[0.0217]	[0.0156]	[0.0143]	[0.0186]	[0.0224]	[0.0232]	[0.0130]	[0.0211]
Age*Over21	-0.0167	-0.0333	0.0571	-0.0406	-0.0934	0.0095	0.0430	-0.0596	0.0289
	[0.0585]	[0.0657]	[0.0474]	[0.0450]	[0.0519]	[0.0660]	[0.0676]	[0.0382]	[0.0656]
Age Sq*Over 21	-0.0056	-0.0076	-0.0083	0.0144	-0.0225	-0.0365	0.0197	-0.0045	0.0152
	[0.0278]	[0.0311]	[0.0224]	[0.0212]	[0.0248]	[0.0314]	[0.0320]	[0.0183]	[0.0309]
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	16107	16107	16107	16107	16107	16107	16107	16107	16107
R-squared	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 brackets. The regressions do not include covariates. Since Age = persons age - 21 the constant is the predicted value for people about to turn 21.

Table 4: Discontinuity in Log Deaths at Age 21

	Deaths Due to All Causes				Deaths Due to External Causes				Deaths Due to Internal Causes			
	Parametric Models			Local Linear	Parametric Models			Local Linear	Parametric Models			Local Linear
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
Over 21	0.0960	0.0870	0.0910	0.0740	0.1100	0.1000	0.0960	0.0824	0.0630	0.0540	0.0940	0.0656
	[0.018]**	[0.017]**	[0.023]**	[0.0161]	[0.022]**	[0.021]**	[0.028]**	[0.0207]	[0.040]	[0.040]	[0.053]	[0.0309]
Age	-0.0270	-0.0270	-0.0740		-0.0480	-0.0480	-0.0730		0.0220	0.0220	-0.1280	
	[0.028]	[0.028]	[0.069]		[0.034]	[0.034]	[0.085]		[0.066]	[0.066]	[0.174]	
Age Sq	-0.0110	-0.0110	-0.0710		-0.0110	-0.0110	-0.0410		-0.0280	-0.0280	-0.2160	
	[0.014]	[0.014]	[0.083]		[0.016]	[0.016]	[0.098]		[0.032]	[0.032]	[0.201]	
Age*Over21	-0.0660	-0.0470	0.0210		-0.0590	-0.0400	0.0320		-0.0560	-0.0390	0.0240	
	[0.043]	[0.041]	[0.101]		[0.050]	[0.048]	[0.119]		[0.090]	[0.090]	[0.232]	
Age Sq*Over 21	0.0340	0.0260	0.0600		0.0270	0.0190	-0.0100		0.0700	0.0620	0.3590	
	[0.020]	[0.020]	[0.118]		[0.024]	[0.023]	[0.137]		[0.044]	[0.044]	[0.269]	
21st Birthday		0.4400	0.4430			0.4850	0.4930			0.2720	0.2570	
		[0.013]**	[0.018]**			[0.015]**	[0.020]**			[0.027]**	[0.036]**	
21st Birthday + 1		0.3390	0.3420			0.3210	0.3290			0.4320	0.4180	
		[0.013]**	[0.018]**			[0.015]**	[0.019]**			[0.027]**	[0.035]**	
Age Cubic			-0.0200				-0.0100				-0.0630	
			[0.027]				[0.032]				[0.066]	
Age^3*Over 21			0.0280				0.0300				0.0270	
			[0.039]				[0.045]				[0.088]	
Constant	4.3790	4.3790	4.3710		4.1340	4.1340	4.1290		2.8090	2.8090	2.7840	
	[0.011]**	[0.011]**	[0.015]**		[0.015]**	[0.015]**	[0.020]**		[0.029]**	[0.029]**	[0.039]**	
Obs	1460	1460	1460		1460	1460	1460		1460	1460	1460	
R-squared	0.04	0.05	0.05		0.06	0.08	0.08		0.10	0.10	0.10	
Prob > Chi-Squared		0.000	0.735			0.000	0.788			0.000	0.525	

Notes: Robust standard errors in brackets. The dependent variable is the log of the number of deaths that occurred x days from the persons 21st birthday. External deaths include all deaths with mention of an injury, alcohol use, or drug use. The Internal Death category includes all deaths not coded as external. The first three columns give the estimates from polynomial 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 fourth column we present the results of a local linear regression procedure with a rule-of-thumb bandwidth for each side of age 21. For this procedure, we follow Fan and Gijbels (1996) and fit a 4th order polynomial 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 ($\sigma 2$). 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 polynomial 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 2 fewer observations because the 21st birthday and the day after the 21st birthday have been dropped. The bottom row gives the results of a Wald test of the hypothesis that the coefficients on the variables added to the model in that column are all equal to 0. The running variable, age -21, is measured in years so it has a span of 2 on either side of the discontinuity. For the local linear regression the bandwidth (h) from the left and from the right for each variable are as follows All Cause (0.55, 0.96); External Causes (0.53, 0.84); Internal Causes (1.07, 1.17). Death Rate 20 is the death rate at age 20 and 11 months.

Table 5: Discontinuity in Log Deaths by External Cause of Death (Alcohol, Homicide, and Suicide)

	Alcohol				Homicide				Suicide			
	Parametric Models			Local Linear	Parametric Models			Local Linear	Parametric Models			Local Linear
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
Over 21	0.336	0.297	0.440	0.427	0.009	0.002	-0.003	-0.014	0.160	0.154	0.135	0.105
	[0.115]	[0.113]	[0.148]	[0.109]	[0.045]	[0.045]	[0.061]	[0.041]	[0.059]	[0.059]	[0.086]	[0.045]
Age	-0.216	-0.216	-0.056		0.077	0.077	-0.046		-0.041	-0.041	-0.017	
	[0.187]	[0.187]	[0.455]		[0.072]	[0.072]	[0.182]		[0.103]	[0.103]	[0.279]	
Age Sq	-0.159	-0.159	0.040		0.026	0.026	-0.127		-0.015	-0.015	0.015	
	[0.090]	[0.090]	[0.528]		[0.034]	[0.034]	[0.206]		[0.048]	[0.048]	[0.309]	
Age*Over21	-0.040	0.039	-1.130		-0.180	-0.165	0.109		-0.024	-0.012	0.057	
	[0.266]	[0.263]	[0.652]		[0.102]	[0.102]	[0.258]		[0.133]	[0.134]	[0.354]	
Age Sq*Over 21	0.256	0.223	1.280		0.004	-0.003	-0.037		0.022	0.017	-0.127	
	[0.129]	[0.128]	[0.764]		[0.048]	[0.048]	[0.295]		[0.063]	[0.063]	[0.398]	
21st Birthday		1.738	1.568			0.607	0.633			0.166	0.182	
		[0.078]	[0.101]			[0.031]	[0.042]			[0.038]	[0.051]	
21st Birthday + 1		1.502	1.335			0.020	0.045			0.309	0.325	
		[0.078]	[0.100]			[0.031]	[0.042]			[0.037]	[0.051]	
Age Cubic			0.066				-0.051				0.010	
			[0.174]				[0.067]				[0.099]	
Age^3*Over 21			-0.484				0.113				0.028	
			[0.253]				[0.096]				[0.128]	
Constant	0.216	0.216	0.243		2.687	2.687	2.667		2.245	2.245	2.248	
	[0.082]	[0.082]	[0.108]		[0.032]	[0.032]	[0.044]		[0.046]	[0.046]	[0.069]	
Obs	1460	1460	1460		1460	1460	1460		1460	1460	1460	
R-squared	0.03	0.03	0.04		0.01	0.01	0.01		0.02	0.02	0.02	
Prob > Chi-Squared		0.000	0.070			0.000	0.495			0.000	0.8922	

Notes: See notes to Table 4. There are 188 observations where there are no deaths coded as due to alcohol; for this variable .5 was added to the dependent variable before taking the log. The running variable, age -21, is measured in years so it has a span of 2 on either side of the discontinuity. For the local linear regression the bandwidth (h) from the left and from the right for each variable are as follows: alcohol (0.94, 0.56); homicide (0.63, 0.99); suicide (1.68, 0.63).

Table 6: Discontinuity in Log Deaths by External Cause of Death (MVA, Drugs, External Causes Other)

	MVA				Drugs				External Causes Other			
	Parametric Models			Local Linear	Parametric Models			Local Linear	Parametric Models			Local Linear
	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)	(1)	(2)	(3)	(4)
Over 21	0.158	0.143	0.145	0.139	0.097	0.093	0.001	-0.023	0.087	0.098	0.098	0.074
	[0.033]	[0.032]	[0.044]	[0.032]	[0.082]	[0.083]	[0.107]	[0.081]	[0.060]	[0.059]	[0.075]	[0.043]
Age	-0.133	-0.133	-0.217		0.102	0.102	0.308		-0.075	-0.075	0.002	
	[0.052]	[0.052]	[0.136]		[0.148]	[0.148]	[0.357]		[0.092]	[0.092]	[0.218]	
Age Sq	-0.019	-0.019	-0.124		0.009	0.009	0.267		-0.059	-0.059	0.037	
	[0.025]	[0.025]	[0.152]		[0.074]	[0.074]	[0.431]		[0.045]	[0.045]	[0.262]	
Age*Over21	-0.026	0.004	0.155		-0.125	-0.118	0.015		-0.028	-0.051	-0.205	
	[0.078]	[0.075]	[0.187]		[0.196]	[0.198]	[0.481]		[0.139]	[0.138]	[0.322]	
Age Sq*Over 21	0.026	0.014	0.036		0.025	0.021	-0.658		0.098	0.107	0.107	
	[0.037]	[0.036]	[0.216]		[0.098]	[0.098]	[0.571]		[0.066]	[0.066]	[0.376]	
21st Birthday		0.589	0.600			0.386	0.443			0.075	0.062	
		[0.022]	[0.029]			[0.055]	[0.074]			[0.045]	[0.058]	
21st Birthday + 1		0.663	0.674			-0.066	-0.010			-1.024	-1.036	
		[0.022]	[0.028]			[0.055]	[0.073]			[0.045]	[0.057]	
Age Cubic			-0.035				0.086				0.032	
			[0.049]				[0.145]				[0.088]	
Age^3*Over 21			0.063				0.054				-0.064	
			[0.071]				[0.190]				[0.125]	
Constant	3.220	3.220	3.206		1.226	1.226	1.261		2.024	2.024	2.037	
	[0.023]	[0.023]	[0.033]		[0.062]	[0.062]	[0.077]		[0.038]	[0.038]	[0.048]	
Obs	1460	1460	1460		1460	1460	1460		1460	1460	1460	
R-squared	0.15	0.16	0.16		0.04	0.04	0.04		0.01	0.01	0.01	
Prob > Chi-Squared		0.000	0.666			0.000	0.440			0.000	0.877	

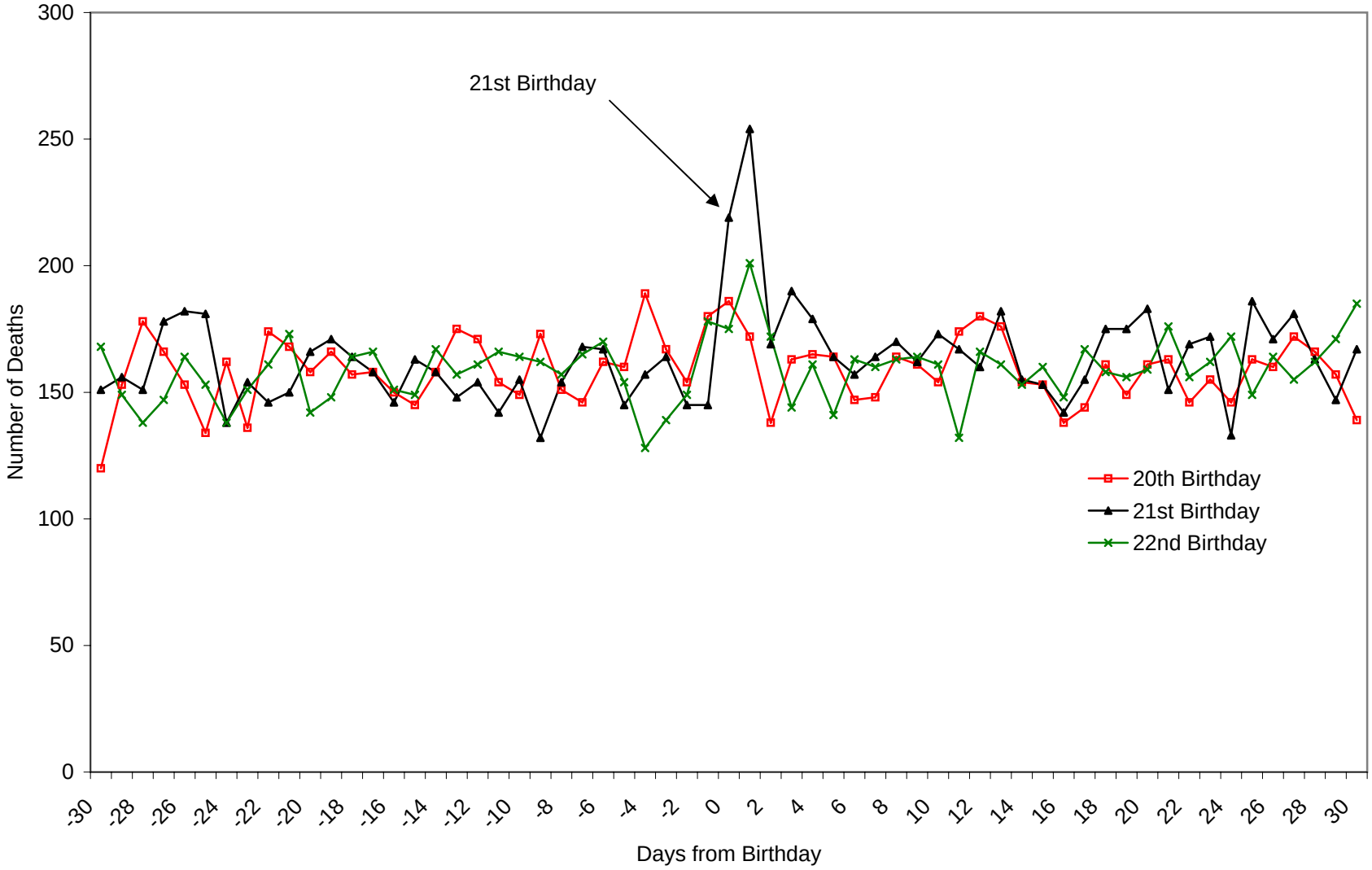
Notes: See notes to Table 4. There are 23 observations where there are 0 deaths coded as due to drug use ,for this variable .5 was added to the count before taking the log. The running variable, age -21, is measured in years so it has a span of 2 on either side of the discontinuity. For the local Linear Regression the bandwidth (h) from the left and from the right for each variable are as follows: MVA (0.66, 0.67); Drugs (0.59, 0.95); External Causes Other (0.98, 1.25).

Table 7: Discontinuity in Log Deaths by Gender, Race and Education

	Gender		Race			Education			
	Male	Female	White	Black	Hispanic	HS Drop Out	HS Completed	Some College	Education Missing
Over 21	0.097	0.057	0.144	0.019	0.006	0.043	0.088	0.155	0.083
	[0.021]	[0.035]	[0.022]	[0.039]	[0.044]	[0.039]	[0.027]	[0.039]	[0.050]
Age	-0.033	-0.005	-0.106	0.054	0.128	-0.009	-0.054	-0.152	0.068
	[0.033]	[0.063]	[0.036]	[0.061]	[0.074]	[0.056]	[0.042]	[0.070]	[0.081]
Age Sq	-0.016	0.004	-0.033	-0.001	0.040	-0.029	0.032	0.035	-0.284
	[0.016]	[0.031]	[0.017]	[0.030]	[0.036]	[0.088]	[0.061]	[0.092]	[0.117]
Age*Over21	-0.054	-0.041	0.012	-0.085	-0.231	0.043	0.009	-0.207	0.017
	[0.048]	[0.085]	[0.053]	[0.090]	[0.105]	[0.027]	[0.020]	[0.034]	[0.039]
Age Sq*Over 21	0.033	0.010	0.044	0.018	-0.008	-0.065	-0.021	0.258	0.080
	[0.023]	[0.042]	[0.025]	[0.044]	[0.052]	[0.042]	[0.029]	[0.046]	[0.057]
21st Birthday	0.564	-0.106	0.479	0.629	0.069	0.589	0.308	0.609	0.345
	[0.015]	[0.024]	[0.017]	[0.030]	[0.031]	[0.030]	[0.019]	[0.025]	[0.036]
21st Birthday + 1	0.346	0.336	0.479	-0.218	0.306	0.184	0.388	0.204	0.718
	[0.015]	[0.024]	[0.017]	[0.029]	[0.031]	[0.030]	[0.019]	[0.025]	[0.036]
Constant	4.100	2.939	3.772	2.908	2.633	2.951	3.475	2.819	2.312
	[0.014]	[0.026]	[0.015]	[0.025]	[0.032]	[0.025]	[0.018]	[0.030]	[0.035]
Obs	1460	1460	1460	1460	1460	1460	1460	1460	1460
R-squared	0.06	0.00	0.07	0.03	0.01	0.08	0.05	0.29	0.02
Rate per 100,000	138	46	80	154	102	NA	NA	NA	NA

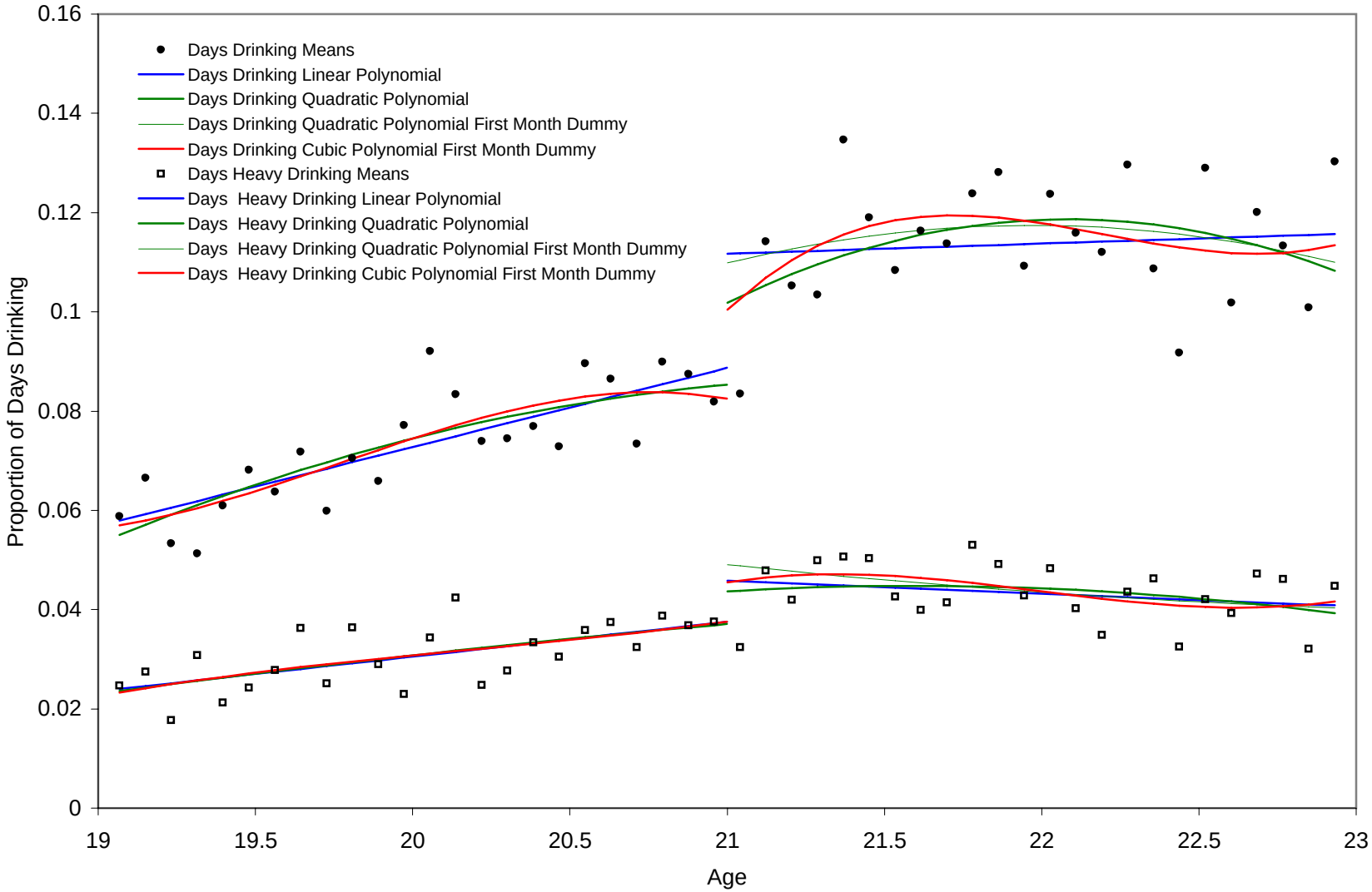
Notes: See notes to Table 4.

Appendix A: Deaths by Days to Birthday

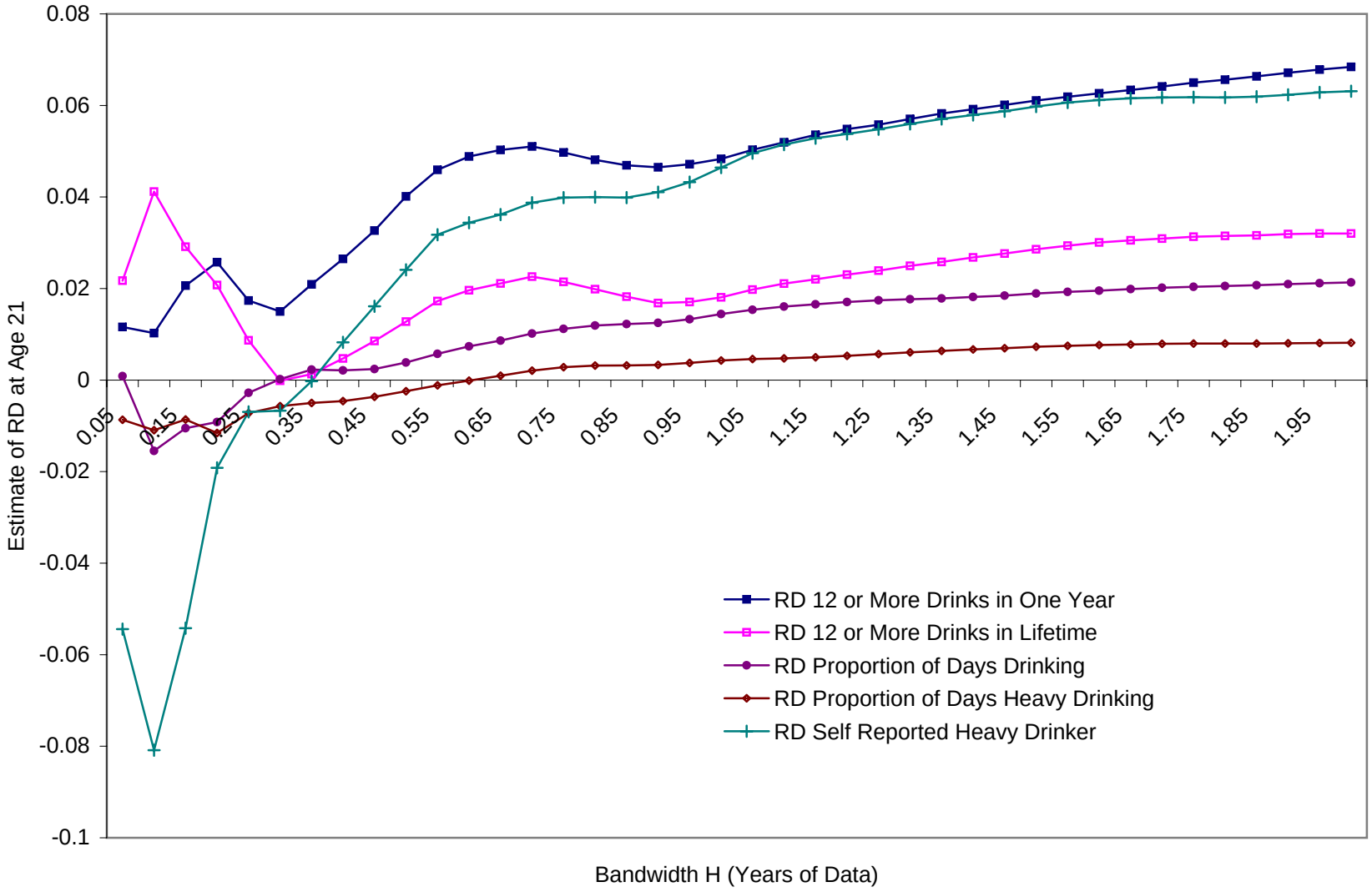


Notes: Number of deaths in the US between 1997 and 2003 occurring x days from the person's birthday.

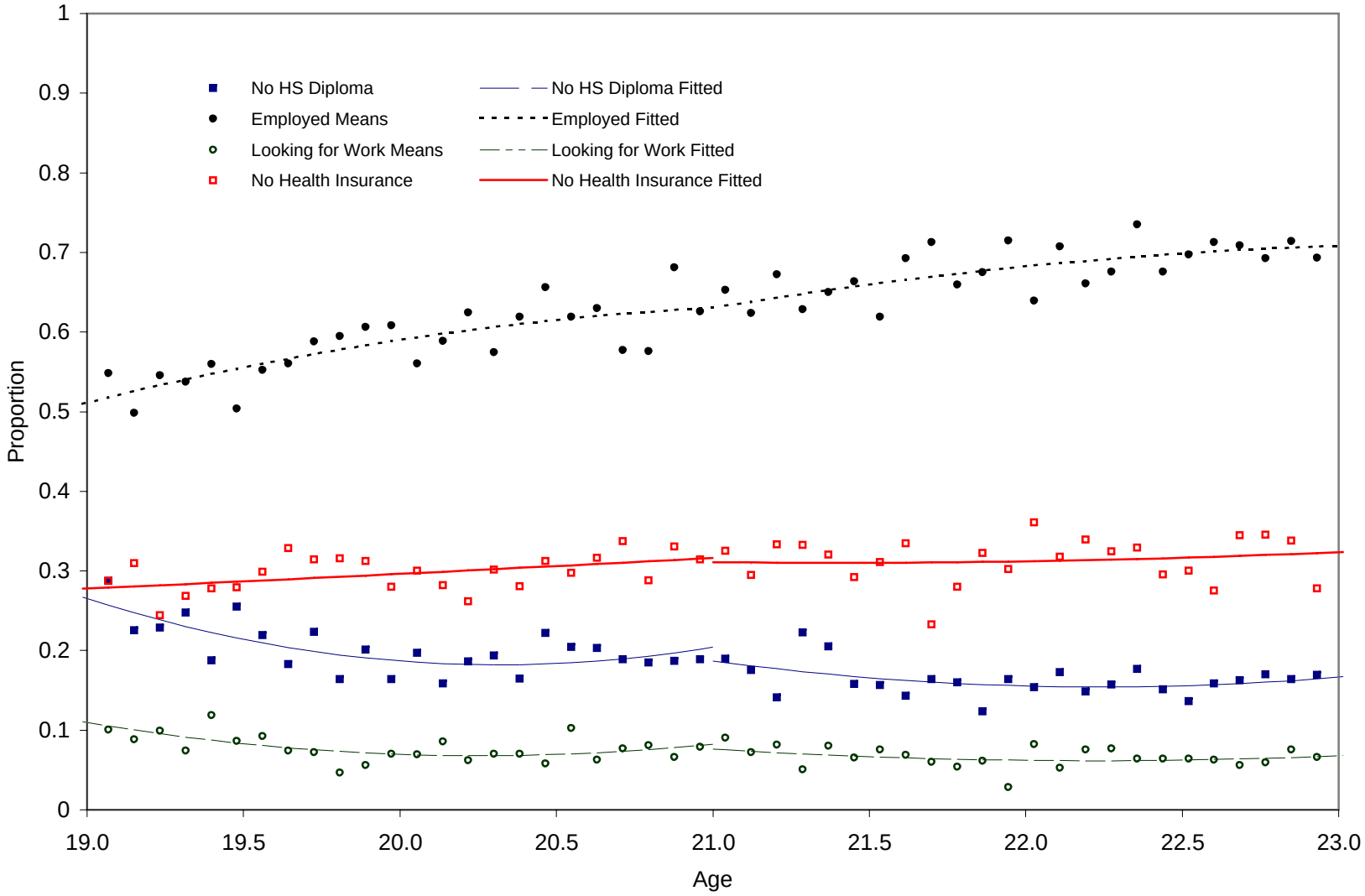
Appendix B: Assessing Fit of Different Polynomials on Age Profile of Drinking



Appendix C: Evaluating the Sensitivity of the Local Linear Regressions to the Choice of Bandwidth



Appendix D: Age Profile of Sample Surveyed in NHIS

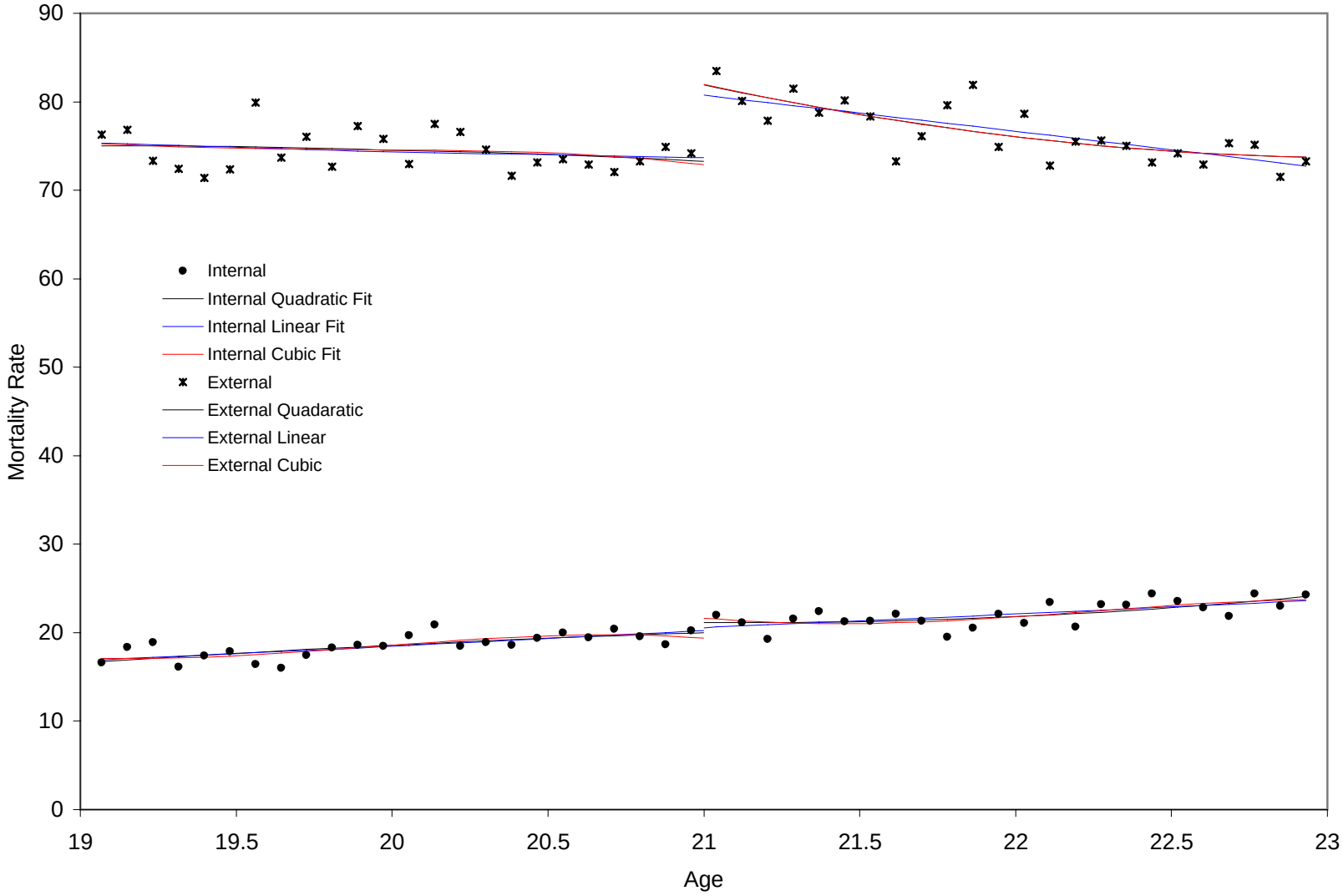


Appendix E: ICD-9 and ICD-10 Codes Used to Create Cause-of-Death Categories

	ICD-9	ICD-10
External Deaths	'8', '9', '292', '304', '305', '850', '851', '852', '853', '854', '855', '856', '857', '858', '3321', '3576', '291', '303', '305', '860', '3575', '4255', '5353', '5710', '5711', '5712', '5713', '7903'	'V', 'W', 'X', 'Y', 'Z', 'F10', 'K70', 'X45', 'X65', 'Y15', 'Y91', 'K70', 'T51', 'X46', 'X65', 'Y15', 'Y90', 'Y91', 'G312', 'G621', 'I426', 'K292', 'R780', 'E244', 'G721', 'K852', 'K860', 'Z502', 'Z714', 'Z721', 'K860', 'T518', 'T519', 'F11', 'F12', 'F13', 'F14', 'F15', 'F16', 'F17', 'F18', 'F19', 'F55', 'T40', 'T41', 'T43', 'F55', 'X40', 'X42' and not in 'F116', 'F126', 'F136', 'F146', 'F156', 'F166', 'F176', 'F171', 'F172', 'F186', 'F196'
Subcategories of External Deaths		
Mention of Alcohol	'291', '303', '3050', '860', '3575', '4255', '5353', '5710', '5711', '5712', '5713', '7903'	'F10', 'K70', 'X45', 'X65', 'Y15', 'Y91', 'K70', 'T51', 'X46', 'X65', 'Y15', 'Y90', 'Y91', 'G312', 'G621', 'I426', 'K292', 'R780', 'E244', 'G721', 'K852', 'K860', 'Z502', 'Z714', 'Z721', 'K860', 'T518', 'T519'
Homicide	'96'	'X85', 'X86', 'X87', 'X88', 'X89,"X9', 'Y0'
Suicide	'95'	'X6', 'X7', 'X80', 'X81', 'X82', 'X83', 'X84', 'X870'
MVA	'81', '820', '821', '822', '823', '824', '825'	'V0', 'V1', 'V2', 'V3', 'V4', 'V5', 'V6', 'V7', 'V8'
Deaths with a Mention of Drugs	'292', '304', '305', '850', '851', '852', '853', '854', '855', '856', '857', '858', '3321', '3576'	'F11', 'F12', 'F13', 'F14', 'F15', 'F16', 'F17', 'F18', 'F19', 'F55', 'T40', 'T41', 'T43', 'F55', 'X40', 'X42' and not in 'F116', 'F126', 'F136', 'F146', 'F156', 'F166', 'F176', 'F171', 'F172', 'F186', 'F196'
Other	External Deaths group - (Mention of alcohol, Homicide, Suicide, MVA, Deaths with a mention of drugs)	External Deaths group - (Mention of alcohol, Homicide, Suicide, MVA, Deaths with a mention of drugs)

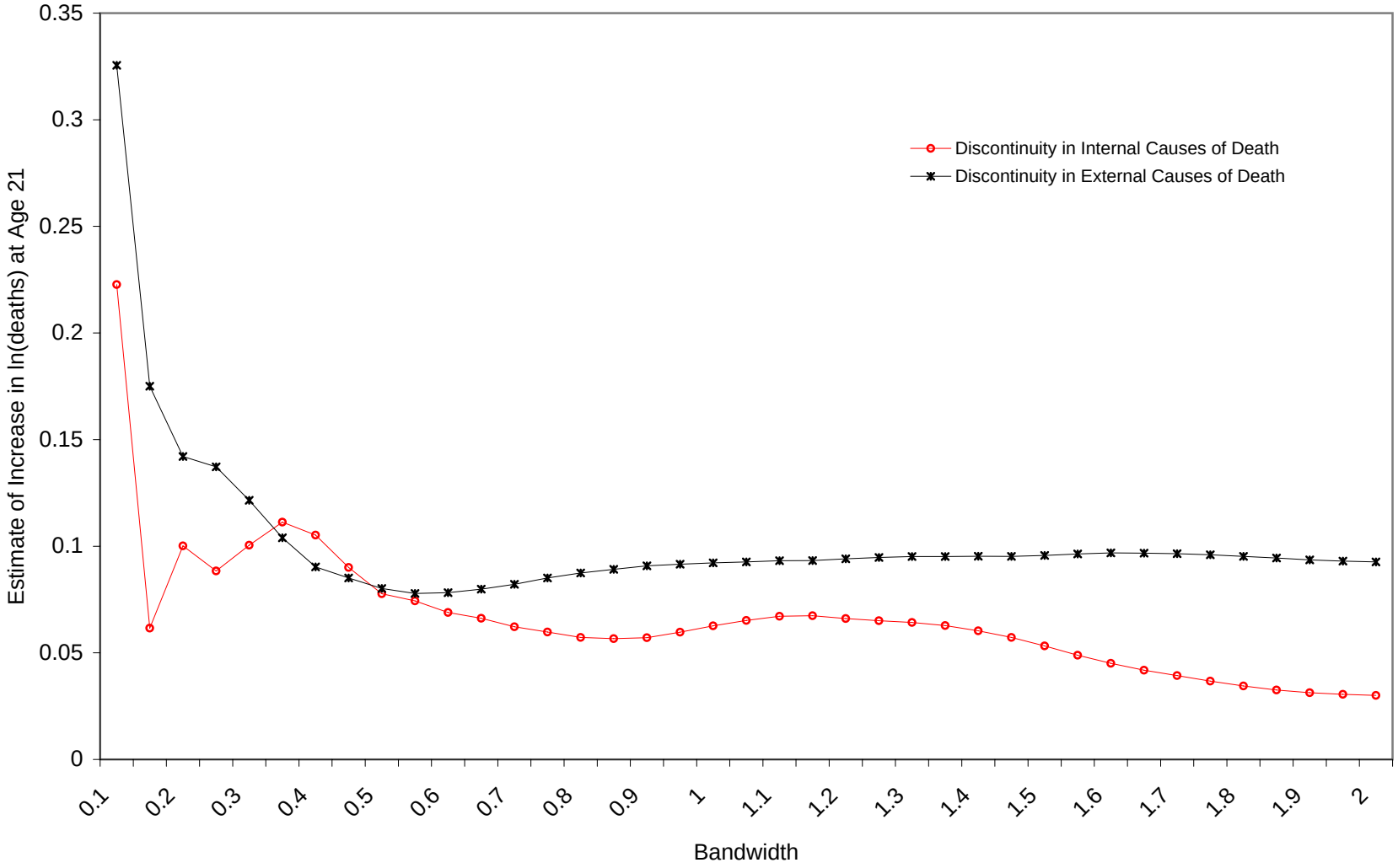
Notes: The order of precedence is homicide, suicide, MVA, deaths with a mention of alcohol, deaths with a mention of drugs. A death is coded as due to a particular ICD code if the code appears on any line of the death certificate.

Appendix F: Comparison of the Fit of Different Polynomials for Internal and External Causes of Death



Notes: See notes from Figure 4, All three polynomials are fully interacted with a dummy for over 21.

Apppendix G: Nonparametric Estimates of Discontinuity in Internal and External Deaths with Different Bandwidths



Notes: See notes to Table 4. The range of the running variable (age) is equal to 2. The parametric estimate for external deaths is 0.104 and for internal deaths 0.022

Appendix H: RDD Estimates for Outcomes With 0 Deaths in Some Periods

	Alcohol			Drugs		
	Log(x+.5)	Log(x+1)	Level	Log(x+.5)	Log(x+1)	Level
	(1)	(2)	(3)	(1)	(2)	(3)
Over 21	0.2970	0.2130	0.4990	0.0930	0.0740	0.2430
	[0.113]**	[0.081]**	[0.193]**	[0.083]	[0.068]	[0.279]
Age	-0.2160	-0.1400	-0.2240	0.1020	0.0840	0.3370
	[0.187]	[0.130]	[0.281]	[0.148]	[0.120]	[0.454]
Age Squared	-0.1590	-0.1050	-0.1850	0.0090	0.0080	0.0510
	[0.090]	[0.062]	[0.132]	[0.074]	[0.060]	[0.222]
Age*Over 21	0.0390	0.0170	-0.0640	-0.1180	-0.0950	-0.3870
	[0.263]	[0.187]	[0.436]	[0.198]	[0.162]	[0.652]
Age Sq*Ov 21	0.2230	0.1490	0.2820	0.0210	0.0180	0.0990
	[0.128]	[0.091]	[0.207]	[0.098]	[0.080]	[0.321]
Birthday	1.7380	1.4800	7.4000	0.3860	0.3340	1.3630
	[0.078]**	[0.057]**	[0.146]**	[0.055]**	[0.046]**	[0.198]**
Birthday + 1	1.5020	1.2570	5.4010	-0.0660	-0.0710	-0.6370
	[0.078]**	[0.057]**	[0.145]**	[0.055]	[0.046]	[0.197]**
Constant	0.2160	0.6100	1.1010	1.2260	1.3840	3.3950
	[0.082]**	[0.057]**	[0.125]**	[0.062]**	[0.050]**	[0.197]**
Observations	1460	1460	1460	1460	1460	1460
R-squared	0.03	0.04	0.07	0.04	0.04	0.04

Notes: See notes to Table 5. There are 188 observations with 0 deaths due to alcohol and 23 observations with no deaths due to drugs. Model (1) in each panel has the log of the number of deaths + .5 which is the same specification as model (2) in for the corresponding outcome in Table 6 or 7. Model (2) has the log of the number of deaths + 1 as a dependent variable. Model (3) in has the dependent variable in levels. The level regressions imply a similar sized change in percentage terms as the regressions in logs. For the alcohol case the levels model implies a 45.3 percent change, which is larger than the 34 percent (%change = exp(B)-1) change in the regression with Log(x-.5) as a dependent variable. In the Drugs regression the levels model implies a 9.7% change and in the "External Other Deaths" the level model implies a 7.1% change.

Appendix I: RDD Estimates of Mortality for Different Time Periods

	All Deaths			All External Deaths			All Internal Deaths		
Years	1990-2004	1990-1996	1997-2004	1990-2004	1990-1996	1997-2004	1990-2004	1990-1996	1997-2004
	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
Over 21	0.081	0.073	0.087	0.095	0.089	0.100	0.032	0.011	0.054
	[0.013]	[0.018]	[0.017]	[0.015]	[0.020]	[0.021]	[0.031]	[0.042]	[0.040]
Age	-0.030	-0.029	-0.027	-0.051	-0.052	-0.048	0.038	0.063	0.022
	[0.020]	[0.028]	[0.028]	[0.024]	[0.032]	[0.034]	[0.048]	[0.070]	[0.066]
Age Squared	-0.015	-0.017	-0.011	-0.015	-0.018	-0.011	-0.023	-0.017	-0.028
	[0.010]	[0.014]	[0.014]	[0.011]	[0.015]	[0.016]	[0.023]	[0.034]	[0.032]
Age*Over 21	-0.025	-0.007	-0.047	-0.023	-0.009	-0.040	-0.013	-0.012	-0.039
	[0.030]	[0.041]	[0.041]	[0.034]	[0.045]	[0.048]	[0.069]	[0.101]	[0.090]
Age Sq*Ov 21	0.026	0.024	0.026	0.021	0.022	0.019	0.047	0.039	0.062
	[0.014]	[0.020]	[0.020]	[0.016]	[0.022]	[0.023]	[0.033]	[0.049]	[0.044]
Birthday	0.274	0.061	0.440	0.291	0.039	0.485	0.220	0.181	0.272
	[0.010]	[0.013]	[0.013]	[0.010]	[0.014]	[0.015]	[0.022]	[0.031]	[0.027]
Birthday + 1	0.423	0.513	0.339	0.407	0.498	0.321	0.502	0.605	0.432
	[0.010]	[0.013]	[0.013]	[0.010]	[0.014]	[0.015]	[0.022]	[0.031]	[0.027]
Constant	5.034	4.297	4.379	4.802	4.076	4.134	3.436	2.642	2.809
	[0.009]	[0.012]	[0.011]	[0.011]	[0.014]	[0.015]	[0.021]	[0.029]	[0.029]
Observations	1460	1460	1460	1460	1460	1460	1460	1460	1460
R-squared	0.09	0.05	0.05	0.11	0.04	0.08	0.20	0.12	0.10

Notes: See notes to Table 4.