Estimating the Mortality Cost of Drinking: Evidence from the U.S. Minimum Legal Drinking Age

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Abstract

The Minimum Legal Drinking Age (MLDA) of 21 in the United States has been the subject of renewed debate, with organizations like the Amethyst Initiative calling for reconsideration of current policy [1]. This paper employs a regression discontinuity design to estimate the causal effects of the MLDA on both alcohol consumption and mortality outcomes. Using data from the National Health Interview Survey and Vital Statistics mortality records, we exploit the sharp change in legal drinking status at age 21 to identify these effects. Our analysis reveals that legal access to alcohol at age 21 increases drinking probability by approximately 9 percentage points and mortality by about 9.5 deaths per 100,000 person-years. The mortality increase is concentrated in external causes, particularly motor vehicle accidents and suicides. Through an instrumental variables approach using the age-21 threshold as an instrument for drinking behavior, we estimate that drinking increases mortality by over 100 deaths per 100,000 drinkers. Our identification strategy satisfies key validity tests, including balance in predetermined characteristics and a strong first stage. These findings suggest that the current MLDA policy prevents approximately 10 deaths per 100,000 young adults annually, providing evidence of substantial public health benefits.

1 Introduction

Understanding the causal impact of alcohol access on mortality is crucial for deciding public health policy. The Minimum Legal Drinking Age (MLDA) of 21 in the United States affects millions of young adults and remains one of the most prominent examples of age-based alcohol prohibition. Recent challenges to this policy, promoted by the Amethyst Initiative's call for reconsideration signed by over 100 college presidents, have reignited debates about whether the current drinking age effectively reduces harm or simply drives drinking underground [1]. This paper addresses two fundamental questions: How much does the MLDA reduce alcohol consumption among young adults? And more importantly, how many lives does this policy save?

These questions matter because alcohol-related deaths represent a leading cause of preventable mortality among young adults. Motor vehicle accidents, the primary cause of death in this age group, show strong *associations* with alcohol consumption, with 67% of nighttime fatal accidents involving alcohol [1].. If the MLDA effectively reduces drinking, it could prevent numerous premature deaths. However, establishing causality requires addressing the challenge that individuals who choose to drink may differ systematically from non-drinkers in ways that affect mortality risk.

We employ a regression discontinuity (RD) design that exploits the sharp change in legal drinking status at age 21. This approach compares individuals just under 21, who face legal restrictions, with those just over 21, who can drink legally. Our analysis involves several methodological choices to ensure robust results. We experiment with different bin widths for data aggregation, test various polynomial specifications (linear, quadratic, and cubic), and select appropriate age ranges and visualization parameters to best capture the discontinuity. We also construct balance tables to verify that baseline characteristics show no discontinuities at the threshold. One of the key assumptions is that individuals cannot manipulate their age,

making the assignment to treatment (legal drinking) as good as random near the threshold. We use data from the 2002 National Health Interview Survey (NHIS) to measure alcohol consumption and Vital Statistics mortality records from 1997-2003 to track deaths by cause.

Our analysis proceeds in two stages. First, we estimate the reduced-form effects of turning 21 on both drinking behavior and mortality. We find that legal access to alcohol increases the probability of drinking by 9.1 percentage points and raises all-cause mortality by 9.5 deaths per 100,000 person-years, with the increase concentrated in motor vehicle accidents and suicides. Second, we implement an instrumental variables (IV) strategy using the age-21 threshold as an instrument for alcohol consumption to estimate the mortality cost per drinker.

The IV analysis reveals that drinking increases mortality by approximately 105 deaths per 100,000 drinkers-a substantial effect suggesting each thousand young adult drinkers experience roughly one additional death per year. This estimate relies on the validity of the exclusion restriction (that turning 21 affects mortality only through drinking) and the relevance of the instrument (a strong first stage). We provide evidence supporting both assumptions through balance tests and statistical analysis. The results suggest that the MLDA generates substantial public health benefits, preventing approximately 10 deaths per 100,000 young adults annually. While these are local effects for people near age 21, they still offer compelling evidence that age-based drinking restrictions save lives.

2 Data

This analysis utilizes two primary datasets to examine the effects of the Minimum Legal Drinking Age (MLDA) on alcohol consumption and mortality outcomes. Both datasets provide nationally representative information that enables high quality causal inference through a regression discontinuity design.

2.1 National Health Interview Survey (NHIS)

The alcohol consumption data come from the 2002 National Health Interview Survey, an annual cross-sectional household survey conducted by the National Center for Health Statistics. The NHIS employs a complex, multistage probability design to select a representative sample of the civilian, non-institutionalized U.S. population [3]. Trained interviewers conduct in-person interviews, collecting detailed information on health behaviors, including alcohol consumption.

Our analysis sample includes 18,824 individuals aged 19 to 23 who provided complete information on drinking behavior. The key outcome variable is `drinks_alcohol`, a binary indicator equal to 1 if the respondent consumed at least 12 alcoholic drinks in the past year, and 0 otherwise.

The running variable is age in days relative to the 21st birthday (`days_21`), allowing precise measurement of distance from the MLDA threshold. Age in years is constructed as:

$$\texttt{age} = 21 + \frac{\texttt{days\_21}}{365}$$

Key demographic covariates include:

- `hs_diploma`: High school completion indicator
- `hispanic`, `white`, `black`: Race/ethnicity indicators
- `uninsured`: Lacks health insurance
- `employed`: Currently employed
- `married`: Marital status
- `working_lw`: Working a low-wage job
- `going_school`: Currently enrolled in school

- `male`: Gender indicator

2.2 Vital Statistics Mortality Data

Mortality outcomes are measured using the National Vital Statistics System mortality files from 1997–2003, which represent a complete census of all deaths in the United States. Death certificates are filed by funeral directors, attending physicians, medical examiners, and coroners, then compiled by state registrars and transmitted to the National Center for Health Statistics [4]. This administrative data provides highly reliable mortality counts with detailed cause-of-death coding.

We aggregate deaths by month of age for individuals aged 19 to 23, creating mortality rates per 100,000 person-years using Census population estimates. Deaths are classified according to the International Classification of Diseases, 10th Revision (ICD-10) into the following categories:

- `all`: All-cause mortality
- `internal`: Deaths from disease/natural causes
- `alcohol`: Alcohol-related deaths
- `suicide`: Suicide deaths
- `mva`: Motor vehicle accident deaths
- `drugs`: Drug-related deaths
- `external_other`: Other external causes

2.3 Data Quality and Limitations

Both datasets are quite strong for causal inference. The NHIS provides individual-level data with precise age measurement, while Vital Statistics offers complete coverage of mortality outcomes. However, self-reported alcohol consumption in surveys typically underestimates true consumption by 40–60% due to social desirability bias and recall errors[6, 2]. This underreporting may be particularly stronger for individuals under 21, for whom drinking is illegal. Additionally, alcohol-related deaths were likely undercounted during this period, as studies have found that alcohol involvement is often not recorded on death certificates even when present [5].

Despite these limitations, both datasets provide sufficient detail and quality for implementing the regression discontinuity design. The sharp age cutoff at 21 is measured precisely, and the large sample sizes give us enough precision detect policy-relevant effects.

3 Methods

Our strategy for this analysis uses a regression discontinuity (RD) design to estimate the causal effects of the Minimum Legal Drinking Age on alcohol consumption and mortality. The RD design exploits the sharp discontinuity in legal drinking status that occurs at age 21. Since individuals cannot manipulate their age, assignment to treatment (legal drinking) is effectively random near the threshold age.

3.1 Regression Discontinuity Implementation

The key to our RD design is selecting appropriate parameters and ranges for data visualization and analysis. Through systematic testing, we made several choices to optimize the tradeoff between bias and noise and clearly identify the discontinuity.

For data aggregation, we tested bin widths of 4, 20, 30, and 40 days. The 4-day bins

produced excessive noise that obscured underlying patterns, while 40-day bins oversimplified the data, potentially obscuring the true discontinuity. The 30-day bins provided optimal balance, showing the discontinuity clearly while maintaining sufficient data points for reliable estimation. Similarly, for the age range, we examined windows from the narrow (20.96-21.04 years, containing only one data point) to the complete broad age range available (17-27 years, making it hard to see local effects). The 19-23 year range was optimal, providing adequate observations on both sides of the threshold while maintaining focus on the local treatment effect.

For visualizing alcohol consumption rates, we also tested multiple Y-axis ranges to ensure accurate representation. The full [0-1] range compressed the data excessively. While the tight [0.45-0.75] range was a good option, it has the risk of slightly exaggerating the discontinuity. We selected the medium range [0.4-0.8] as it provides appropriate context while clearly displaying the size of the effect.

3.2 Regression Specification

Our regression discontinuity analysis involves two key equations. The **first stage** equation estimates the effect of turning 21 on alcohol consumption:

First Stage (FS):

$$D_i = \phi_0 + \phi_1 \cdot \mathbf{1}[Age_i \geq 21] + f(Age_i - 21) + \nu_i \tag{1}$$

where D_i is an indicator for whether individual i drinks alcohol, $\mathbf{1}[Age_i \geq 21]$ is an indicator for being 21 or older, and $f(Age_i - 21)$ is a flexible polynomial function of age centered at 21 that is allowed to differ on either side of the threshold.

The **reduced form** equation estimates the direct effect of turning 21 on mortality:

Reduced Form (RF):

$$Y_a = \rho_0 + \rho_1 \cdot \mathbf{1}[Age_a \geq 21] + g(Age_a - 21) + \omega_a \tag{2}$$

where Y_a represents the mortality rate for age cell a, and $g(Age_a - 21)$ is similarly a flexible polynomial function of centered age.

In our implementation, we use quadratic polynomials for both $f(\cdot)$ and $g(\cdot)$, allowing for different slopes on either side of the age-21 threshold through interaction terms.

We estimate the effect of the MLDA using polynomial regression with age centered at 21. Define $AgeC_i$ as the centered age variable. Our main specification is:

$$Y_i = \beta_0 + \beta_1 Over21_i + \beta_2 AgeC_i + \beta_3 AgeC_i \times Over21_i + \beta_4 AgeC_i^2 + \beta_5 AgeC_i^2 \times Over21_i + \varepsilon_i \tag{3}$$

where Y_i represents either alcohol consumption or mortality outcomes, $Over21_i$ is an indicator equal to 1 for individuals aged 21 or older, and the interaction terms allow for different polynomial trends on either side of the threshold. The coefficient β_1 identifies the discontinuous jump in outcomes at age 21.

We tested linear, quadratic, and cubic polynomial specifications. While all three yielded similar point estimates for the discontinuity (8.7% for linear, 9.1% for quadratic, 8.1% for cubic), the quadratic specification was selected based on better fit to the data and lower standard errors compared to the cubic model. All specifications yielded identical R-squared values (0.025), suggesting the additional complexity of higher-order polynomials did not improve model fit.

3.3 Balance Tests and Validity

The validity of our RD design requires that predetermined characteristics are continuous and evolve smoothly through the age-21 threshold. We test this assumption by estimating equation (1) with demographic characteristics as dependent variables:

$$X_i = \alpha_0 + \alpha_1 Over21_i + \alpha_2 AgeC_i + \alpha_3 AgeC_i \times Over21_i + \alpha_4 AgeC_i^2 + \alpha_5 AgeC_i^2 \times Over21_i + u_i \tag{4}$$

where X_i includes gender, race/ethnicity indicators, education, employment status, marital status, and insurance coverage. The coefficient α_1 tests for discontinuous changes in characteristics that should not be affected by the MLDA.

3.4 Instrumental Variables Strategy

To estimate the effect of drinking on mortality, we implement a two-sample instrumental variables approach. This addresses endogeneity concerns arising from unobserved factors that may affect both drinking behavior and mortality risk. Our IV strategy uses the age-21 threshold as an instrument for alcohol consumption.

The first stage measures the effect of turning 21 on drinking probability:

$$Drinks_i = \phi_0 + \phi_1 Over21_i + \phi_2 AgeC_i + \phi_3 AgeC_i \times Over21_i + \phi_4 AgeC_i^2 + \phi_5 AgeC_i^2 \times Over21_i + \nu_i \tag{5}$$

The reduced form measures the effect of turning 21 on mortality:

$$Mortality_a = \rho_0 + \rho_1 Over21_a + \rho_2 AgeC_a + \rho_3 AgeC_a \times Over21_a + \rho_4 AgeC_a^2 + \rho_5 AgeC_a^2 \times Over21_a + \omega_a \tag{6}$$

where subscript a denotes age cells. The IV estimate of the effect of drinking on mortality is:

$$\hat{\beta}_{IV} = \frac{\hat{\rho}_1}{\hat{\phi}_1} \tag{7}$$

with standard errors calculated using the delta method:

$$SE(\hat{\beta}_{IV}) = \sqrt{\frac{[SE(\hat{\rho}_1)]^2}{\hat{\phi}_1^2} + \frac{\hat{\rho}_1^2 \times [SE(\hat{\phi}_1)]^2}{\hat{\phi}_1^4}} \tag{8}$$

This IV approach requires three assumptions: (1) Relevance - turning 21 significantly affects drinking behavior ($\phi_1 \neq 0$); (2) Exclusion restriction - turning 21 affects mortality only through its effect on drinking; and (3) Monotonicity - no individual reduces drinking upon turning 21. We assess these assumptions through statistical tests and examination of mortality patterns across different causes of death.

All regressions are estimated over the 48-month window from ages 19 to 23, use robust standard errors, and apply population weights for mortality regressions to account for group size variation.

4 Results

4.1 First Stage: Effect of MLDA on Alcohol Consumption

We begin by examining how the minimum legal drinking age affects alcohol consumption. Figure 1 presents the age profile of alcohol consumption from ages 19 to 23, with 30-day bins

and fitted quadratic regression lines on either side of the age-21 threshold.

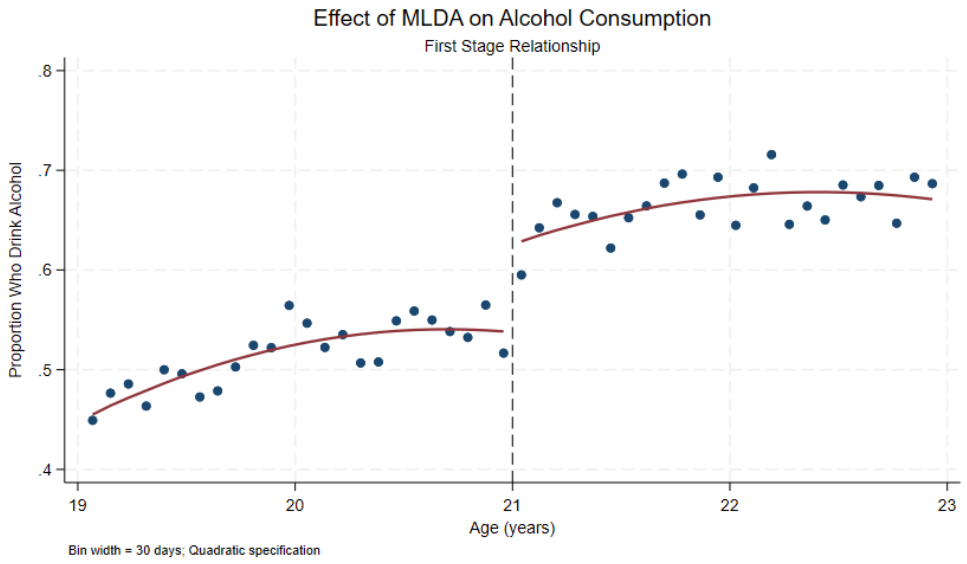

Figure 1: First Stage Relationship: Effect of MLDA on Alcohol Consumption

The figure reveals a clear discontinuous jump in the probability of drinking at age 21. The proportion of individuals reporting alcohol consumption increases sharply from approximately 54% just before age 21 to over 63% immediately after, representing a visually striking discontinuity.

Table 1: Effect of MLDA on Alcohol Consumption with Birthday Controls

VARIABLES	(1) Linear	(2) Linear w/ Birthday	(3) Quadratic	(4) Quadratic w/ Birthday	(5) Cubic	(6) Cubic w/ Birthday
Over 21	0.087***	0.087***	0.091***	0.090***	0.081***	0.079***
	(0.014)	(0.014)	(0.021)	(0.021)	(0.029)	(0.029)
Birthday		0.002		0.021		0.036
		(0.084)		(0.085)		(0.086)
Constant	0.559***	0.559***	0.536***	0.536***	0.532***	0.532***
	(0.010)	(0.010)	(0.015)	(0.015)	(0.021)	(0.021)
Observations	18,801	18,801	18,801	18,801	18,801	18,801
R-squared	0.025	0.025	0.025	0.025	0.025	0.025

Note: Dependent variable is "Drinks Alcohol" (0/1). Standard errors in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 1 presents regression estimates of this discontinuity under different polynomial specifications, both with and without controls for a birthday celebration effect (defined as the exact 21st birthday). Our preferred quadratic specification (Column 3) shows an increase of 9.1 percentage points in drinking probability at age 21 ($p < 0.01$) when not controlling for the birthday. When we include a dummy variable for the exact 21st birthday, the estimated MLDA discontinuity is 9.0 percentage points ($p < 0.01$). The coefficient for the birthday day itself (0.021) is small, positive, and not statistically significant.

This indicates that any temporary increase in drinking precisely on the 21st birthday is minimal and does not really affect the estimated impact of legally gaining access to alcohol. The stability of the "Over 21" coefficient across different polynomial models that include the birthday day control suggests that the observed discontinuity primarily shows a real change in drinking behavior due to the change in legal status, rather than being confounded by a celebration effect on the birthday itself. The MLDA estimates remain substantial and statistically significant across all models, ranging from an increase of 7.9 to 9.1 percentage points.

4.1.1 Measurement Error in Alcohol Consumption

An important consideration in interpreting our first-stage results is the presence of measurement error in self-reported alcohol consumption. The NHIS survey relies on respondents' self-reports, which are subject to several sources of measurement error:

Underreporting bias: Studies consistently find that survey respondents underreport alcohol consumption by 40-60% compared to alcohol sales data [6, 2]. This underreporting occurs due to social desirability bias, where respondents may feel uncomfortable admitting to behaviors that could be viewed negatively, and recall errors, where individuals genuinely forget or misremember their drinking patterns.

Differential underreporting by age: Individuals under 21 face legal penalties for drinking, creating stronger incentives to underreport compared to those over 21. This underreporting would lead us to **overestimate** the first-stage effect of the MLDA on drinking. If 20-year-olds underreport more than 21-year-olds, the observed jump at age 21 could partially reflect changes in reporting behavior rather than actual consumption changes.

Implications for IV estimates: Since our IV estimate equals the reduced form effect divided by the first stage effect ($\beta_{IV} = \rho_1/\phi_1$), an overestimated first stage (inflated ϕ_1) leads to **underestimated** IV effects. Our estimate of 104.9 deaths per 100,000 drinkers may therefore represent a lower bound on the true mortality cost of drinking.

Binary vs. intensive margin: Our drinking variable captures only the extensive margin (whether someone drinks at all) rather than the intensive margin (how much they drink). Heavy drinking likely carries higher mortality risks than moderate drinking, but our binary measure treats all drinkers equally. This limitation means our IV estimates represent an average effect across all drinking intensities.

Despite these measurement challenges, our RD design remains valid for estimating the policy-relevant effect of the MLDA. The discontinuity we observe combines both real behavioral changes and reporting changes, both of which disappear when the drinking age is

enforced. From a policy perspective, this combined effect is what matters for evaluating the MLDA's impact.

4.2 Balance Tests

A key assumption of the regression discontinuity design is that predetermined characteristics should evolve smoothly through the age-21 threshold. Table 2 presents balance tests for demographic characteristics.

Table 2: Balance Table for Demographics

	Demographic Characteristics					
VARIABLES	High School	Hispanic	White	Black	Uninsured	Employed
Over 21	0.033*	-0.004	0.014	-0.024	-0.001	0.028
	(0.017)	(0.019)	(0.022)	(0.016)	(0.020)	(0.021)
Constant	0.793***	0.242***	0.554***	0.160***	0.309***	0.618***
	(0.012)	(0.013)	(0.016)	(0.011)	(0.014)	(0.015)
Observations	18,824	18,824	18,824	18,824	18,824	18,824
R-squared	0.003	0.000	0.000	0.000	0.001	0.014

	Additional Characteristics			
VARIABLES	Married	Working (Low Wage)	In School	Male
Over 21	-0.025	0.031	0.001	0.024
	(0.015)	(0.021)	(0.017)	(0.022)
Constant	0.158***	0.618***	0.165***	0.412***
	(0.011)	(0.015)	(0.012)	(0.016)
Observations	18,824	18,824	18,824	18,824
R-squared	0.019	0.014	0.019	0.000

Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Across demographic variables including gender, race/ethnicity, education, employment, marital status, and insurance coverage, we find no statistically significant discontinuities at age 21, with only high school completion showing marginal significance at the 10% level. This supports our identifying assumption.

When conducting multiple hypothesis tests, as is the case here with 10 covariates, the

probability of observing at least one statistically significant result purely by chance increases. In our initial assessment of Table 2, the 'High School' completion variable showed a coefficient of 0.033 (standard error = 0.017), which is shown to be significant at the 10% level (indicated by * in the table).

To formally address the issue of multiple inferences, we apply a Bonferroni correction. To maintain a family-wise error rate (FWER) of 0.10 across the 10 tests, the adjusted significance threshold for each individual test becomes more stringent: $\alpha_{adj} = 0.10/10 = 0.01$. The p-value for the 'High School' covariate (≈ 0.052) is greater than this Bonferroni-corrected threshold of 0.01. Consequently, after adjusting for multiple inferences, none of the 10 covariates show a statistically significant discontinuity at the age-21 threshold for an FWER of 10%. This adjustment provides stronger support for the identifying assumption that individuals on either side of the MLDA threshold are similar in terms of these observed characteristics.

4.3 Reduced Form: Effect of MLDA on Mortality

Figure 2 displays the age profile of all-cause mortality from ages 19 to 23.

Death rates jump from approximately 93 per 100,000 just before age 21 to around 105 per 100,000 immediately after, an increase of about 10%.

To better understand which types of mortality are driving this increase, Figure 3 compares mortality patterns for alcohol-related versus non-alcohol-related causes of death.

The stark contrast between alcohol-related and non-alcohol-related mortality is evident. While alcohol-related deaths (including motor vehicle accidents, suicides, direct alcohol deaths, and other external causes) show a clear discontinuity at age 21, non-alcohol-related deaths (internal causes and drug-related deaths) remain smooth through the threshold.

Figure 4 provides a detailed view of the specific alcohol-related causes driving the mortality increase.

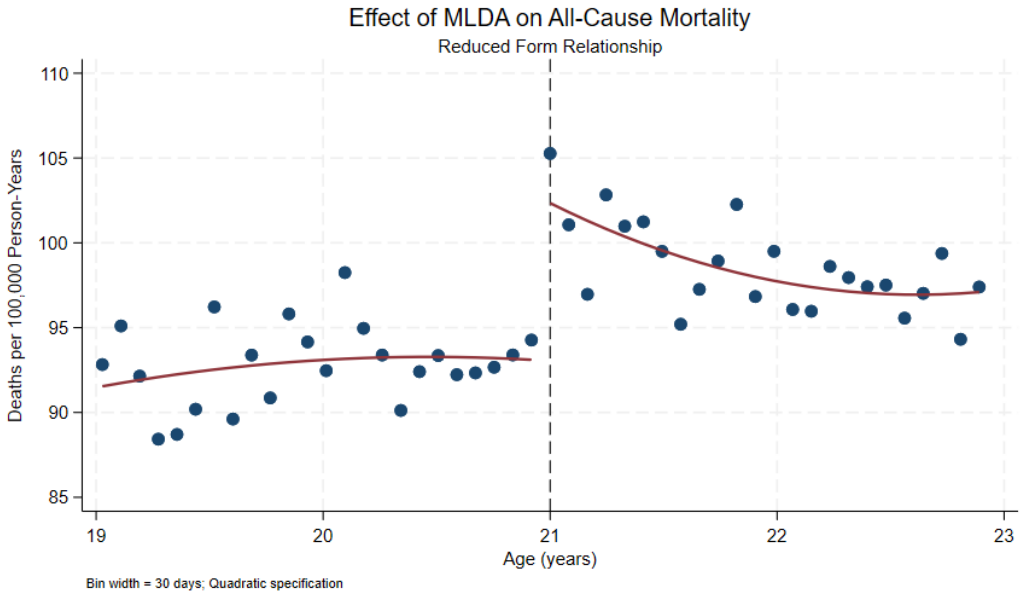

Figure 2: Effect of MLDA on All-Cause Mortality: Reduced Form Relationship

Figure 3: Mortality by Alcohol-Relatedness

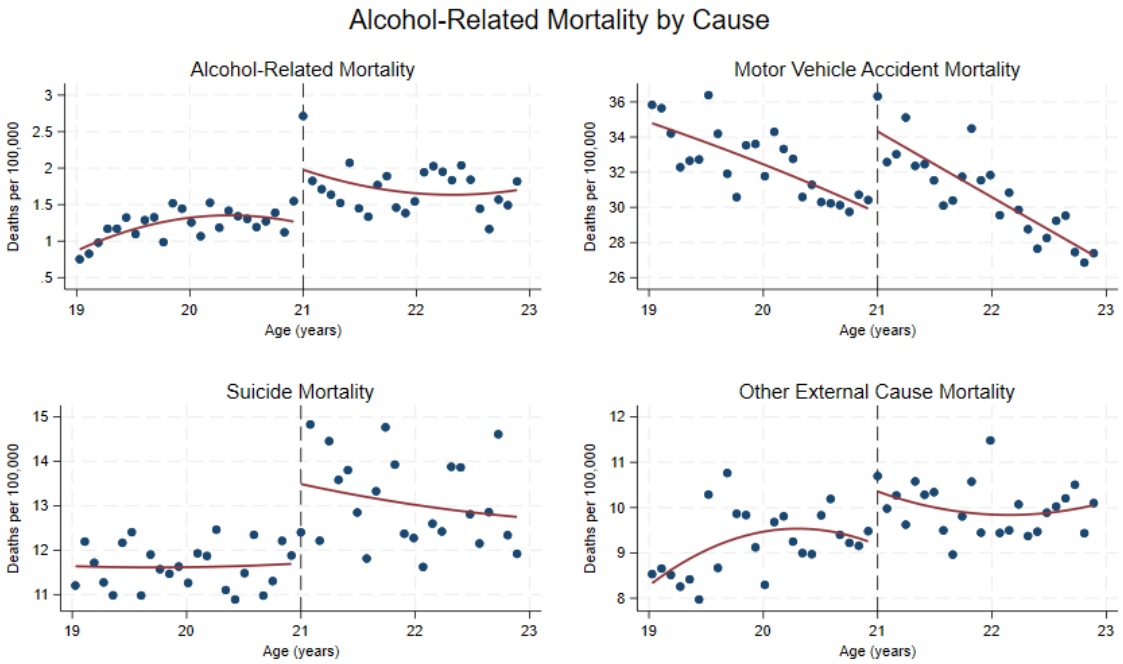

Figure 4: Alcohol-Related Mortality by Specific Cause

Motor vehicle accidents show the most dramatic increase at age 21, followed by substantial jumps in suicide rates and other external causes. Direct alcohol-related deaths also increase, though the baseline rates are lower. This pattern strongly suggests that access to alcohol at age 21 has immediate and severe consequences for young adult mortality.

Table 3 presents regression estimates of the discontinuous jump in mortality rates at age 21 using our preferred quadratic RD specification. Each column shows the result for a different cause of death. Due to the aggregated publicly available mortality data (by months of age), a direct "birthday" control similar to that used in the first-stage drinking regressions with individual-level survey data is not applied here. Thus, these reduced form estimates focus on the discontinuity at the 21-year threshold.

Table 3: Reduced Form Effects of MLDA on Mortality (Deaths per 100,000)

VARIABLES	(1) All	(2) Internal	(3) Alcohol	(4) Suicide	(5) MVA	(6) Drugs	(7) External Other
Over 21	9.548***	1.073	0.745***	1.814**	4.663***	0.306	1.178**
	(1.985)	(0.909)	(0.221)	(0.699)	(1.155)	(0.289)	(0.533)
Constant	93.073***	20.068***	1.255***	11.698***	29.809***	3.917***	9.215***
	(1.404)	(0.642)	(0.156)	(0.494)	(0.817)	(0.204)	(0.377)
Observations	48	48	48	48	48	48	48
R-squared	0.682	0.808	0.574	0.489	0.722	0.723	0.398

Note: Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.
Dependent variables are cause-specific mortality rates per 100,000 person-years for individuals aged 19-23.

The regression results in Table 3 confirm the visual patterns observed in the figures. Turning 21 is associated with a statistically significant increase of 9.548 deaths per 100,000 person-years in all-cause mortality ($p < 0.01$). This effect is driven primarily by significant increases in:

- Motor vehicle accidents: an increase of 4.663 deaths per 100,000 ($p < 0.01$)
- Suicides: an increase of 1.814 deaths per 100,000 ($p < 0.05$)
- Alcohol-related deaths: an increase of 0.745 deaths per 100,000 ($p < 0.01$)
- Other external causes: an increase of 1.178 deaths per 100,000 ($p < 0.05$)

Consistent with expectations, internal causes of death do not show a significant discontinuity, nor do drug-related deaths.

Non-alcohol-related causes show no significant effects:

- **Internal causes**: No significant change (coefficient = 1.1, $p > 0.10$). Deaths from disease and natural causes should not be directly affected by alcohol access, and indeed we find no discontinuity.

- **Drug-related deaths**: No significant change (coefficient = 0.3, $p > 0.10$). While there's a chance that substance use problems may be correlated, access to alcohol at 21 does not directly affect drug-related mortality.

4.4 Instrumental Variables Estimates

To estimate the mortality effect per drinker, we use the MLDA as an instrument for alcohol consumption in a two-sample IV framework. The first stage effect from our analysis of NHIS data (Table 1, Column 3) shows that turning 21 increases the probability of drinking by 9.1 percentage points. Table 4 presents the IV estimates of the effect of drinking on mortality, with standard errors calculated using the delta method.

Table 4: Instrumental Variables Estimates: Effect of Drinking on Mortality (Deaths per 100,000 Drinkers)

Cause of Death	(1) All	(2) Internal	(3) Alcohol	(4) Suicide	(5) MVA	(6) Drugs	(7) External Other
IV Estimate	104.9***	11.8	8.19***	19.9**	51.2***	3.36	12.9**
IV Std. Error	(32.6)	(10.4)	(3.08)	(8.95)	(17.3)	(3.27)	(6.58)
First Stage Effect (from Table 1)				0.091*** (0.021)			

Note: Standard errors for IV estimates calculated via the delta method in parentheses. ***p<0.01, ** p<0.05, * p<0.1.

The IV results in Table 4 suggest that drinking increases all-cause mortality by approximately 104.9 deaths per 100,000 drinkers ($p < 0.01$)]. This effect is primarily driven by statistically significant increases in mortality from:

- Motor vehicle accidents: 51.2 deaths per 100,000 drinkers ($p < 0.01$)
- Suicides: 19.9 deaths per 100,000 drinkers ($p < 0.05$)
- Alcohol-related deaths: 8.19 deaths per 100,000 drinkers ($p < 0.01$)
- Other external causes: 12.9 deaths per 100,000 drinkers ($p < 0.05$)

The IV estimates for internal causes and drug-related deaths remain statistically insignificant. Despite the larger standard errors in IV estimation, the key effects linking drinking to increased mortality risk are statistically significant and practically large.

5 Conclusion

This paper investigates the causal impact of the U.S. Minimum Legal Drinking Age of 21 on both alcohol consumption and crucial mortality outcomes among young adults. Employing a regression discontinuity design that leverages the sharp change in legal drinking status at this age threshold, our findings provide clear answers to key public health questions.

Our analysis of NHIS data reveals that the MLDA significantly reduces alcohol consumption. Upon turning 21, young adults show a substantial and statistically significant increase in the probability of drinking by approximately 9.1 percentage points. This finding is robust across various polynomial specifications and remains stable even when accounting for potential birthday-specific celebration effects, suggesting a behavioral shift due to legal access rather than a temporary rise in consumption.

Furthermore, using Vital Statistics mortality data, we find evidence that the MLDA has a life-saving effect. Gaining legal access to alcohol at age 21 is associated with a statistically significant increase of approximately 9.5 deaths per 100,000 person-years in all-cause mortality. This increase is not uniform across causes of death; rather, it is concentrated in external causes linked to alcohol consumption. Specifically, we see significant increases in mortality from motor vehicle accidents (4.6 deaths per 100,000), suicides (1.8 deaths per 100,000), and directly classified alcohol-related deaths (0.7 deaths per 100,000). There are no significant discontinuities in internal causes of death or drug-related mortality, which lends further support to the causal relationship between alcohol access and these specific outcomes.

To show the exact mortality risk per drinker, we implemented a two-sample instrumental

variables strategy, instrumenting for alcohol consumption with the age-21 MLDA threshold. Our IV estimates suggest that drinking increases all-cause mortality by a substantial 104.9 deaths per 100,000 drinkers.

The validity of this IV approach is based on key assumptions. First, instrument relevance is strongly supported by our first-stage finding of a 9.1 percentage point increase in drinking at age 21. Second, the underlying RD assumption of continuity of potential outcomes is proven by our balance tests, which, after applying a Bonferroni correction for multiple comparisons, show no statistically significant discontinuities in a range of demographic characteristics at age 21. Third, the exclusion restriction—that turning 21 affects mortality only through alcohol consumption—is not perfectly satisfied in this context. Several factors may violate this assumption:

1. Celebration effects: The 21st birthday is a significant milestone often marked by celebration, which could independently increase risky behaviors and mortality risk. While we attempted to control for this by including a birthday month dummy in our first-stage analysis, the effect was small and statistically insignificant. However, birthday celebrations may extend beyond the exact birthday, creating a discontinuity in risk-taking that operates outside the alcohol channel.

2. Changes in social context: Legal drinking age affects where and how young adults socialize. Those over 21 can enter bars and clubs, exposing them to different environments that may carry risks (e.g., late-night driving, different peer groups) beyond the direct effect of alcohol consumption.

3. Psychological and behavioral changes: Turning 21 represents a transition to full legal adulthood in the United States, which may affect behavior through channels beyond alcohol access. Young adults may feel more mature and engage in other risky behaviors, or conversely, feel more responsible. These psychological changes could influence mortality risk independently.

4. Enforcement and visibility effects: When individuals can drink legally, their consumption may become more visible and occur in regulated establishments rather than hidden settings. This could have ambiguous effects on safety—bar service might reduce binge drinking compared to unsupervised settings, but it might also normalize heavy drinking.

5. Complementary behaviors: Legal alcohol access might affect consumption of other substances. While we found no effect on drug-related deaths, alcohol could influence other behaviors (e.g., smoking, risky sexual behavior) that affect mortality through different channels with varying time lags.

Given these potential violations, our IV estimate of 104.9 deaths per 100,000 drinkers should be interpreted cautiously. It likely represents an upper bound if celebration effects and other risky behaviors at 21 contribute to mortality independently. However, the fact that mortality increases are concentrated in alcohol-related causes (MVA, suicide, direct alcohol deaths) while non-alcohol-related causes show no effects suggests that alcohol consumption is indeed the primary, or at least the largest, channel. The policy-relevant **reduced form effects**—that the MLDA prevents approximately 9-10 deaths per 100,000 young adults—remain valid regardless of the exact channels involved.

In conclusion, our analysis demonstrates that the Minimum Legal Drinking Age of 21 effectively reduces alcohol consumption among young adults and, importantly, is associated with a significant reduction in mortality rates, preventing approximately 9-10 deaths per 100,000 young adults annually. The instrumental variable analysis further highlights the considerable mortality risks directly attributable to drinking behavior in this age group. While debates surrounding the optimal drinking age persist, often fueled by arguments about responsible drinking and civil liberties [1], our findings highlight the substantial public health benefits of the current MLDA policy in the United States. Any proposed policy changes aimed at lowering the drinking age must disprove the strong evidence presented here and in the original literature [1] that such a change would likely lead to significant increases in

preventable deaths among young adults.

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