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The legacy of minimum legal drinking age law changes: Long-term effects on suicide and homicide deaths among women

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Abstract

Background—Prior to the establishment of the uniform drinking age of 21 in the United States, many states permitted legal purchase of alcohol at younger ages. Lower drinking ages were associated with several adverse outcomes, including elevated rates of suicide and homicide among youth. The objective of this study is to examine whether individuals who were legally permitted to drink prior to age 21 remained at elevated risk in adulthood.

Methods—Analysis of data from the U.S. Multiple Cause of Death files, 1990–2004, combined with data on the living population from the U.S. Census and American Community Survey. The assembled data contained records on over 200,000 suicides and 130,000 homicides for individuals born between 1949 and 1972, the years during which the drinking age was in flux. Logistic regression models were used to evaluate whether adults who were legally permitted to drink prior to age 21 were at elevated risk for death by these causes. A quasi-experimental analytical approach was employed which incorporated state and birth year fixed effects to account for unobserved covariates associated with policy exposure.

Results—In the population as a whole, we found no association between minimum drinking age and homicide or suicide. However, significant policy-by-sex interactions were observed for both outcomes, such that women exposed to permissive drinking age laws were at higher risk for both suicide (OR=1.12; 95% CI 1.05, 1.18, p=0.0003) and homicide (OR=1.15; 95% CI 1.04, 1.25; p=0.0028). Effect sizes were stronger for the portion of the cohort born after 1960, whereas no significant effects were observed for women born prior to 1960.

Conclusions—Lower drinking ages may result in persistent elevated risk for suicide and homicide among women born after 1960. The national drinking age of 21 may be preventing about 600 suicides and 600 homicides annually.

Keywords

suicide; homicide; policy; women; epidemiology

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Introduction

Suicide and homicide are among the five leading causes of death in early to middle adulthood, together accounting for nearly 11% of years of potential life lost prior to age 65 (Centers for Disease Control and Prevention - National Center for Injury Prevention and Control, 1999–2007). In the U.S., deaths by both causes are associated with alcohol use and problem drinking. Specifically, rates of suicide and homicide are associated with per capita alcohol consumption (Cerda et al., 2010; Landsberg et al., 2008; Lester, 1995). In toxicology studies, alcohol is present in one-third to one-half of suicide and homicide victims (Darke, 2010; Karch et al., 2009; Kuhns et al., 2011). Psychological autopsy studies have reported that as many as 56% of people who committed suicide had alcohol use disorders (Conwell et al., 1996; Foster et al., 1997; Kung et al., 2003). Therefore, policies that reduce alcohol consumption may also reduce the likelihood of death by suicide or homicide.

Minimum legal drinking age (MLDA) policies are among the most widely studied alcohol control policies in the world, and most studies have found that higher MLDAs result in lower rates of alcohol consumption, later initiation of drinking, reduced frequency of heavy drinking, and reductions in other alcohol-related problems (Carpenter and Dobkin, 2009; Carpenter et al., 2007; Cook and Tauchen, 1984; Fell et al., 2008; Wagenaar and Toomey, 2002). Contemporaneous effects of MLDA on suicide and homicide among adolescents and young adults have been demonstrated, with lower drinking age predicting higher death rates by both causes (Birckmayer and Hemenway, 1999; Jones et al., 1992).

In addition to their clear relevance to public health, MLDA policies are attractive to study because changes in MLDA policies occurred in different states at different time periods, enabling quasi-experimental comparisons of state-by-state differences in MLDA. There have been two waves of change in the MLDA policy environment. The first occurred in the early 1970s, when drinking age laws were being liberalized in conjunction with broader social change. After several years of relative stability, a second wave of change occurred throughout the 1980s, when drinking ages were raised as a public health measure (Toomey et al, 1996).

While there are many studies examining contemporaneous or short-term effects of MLDA on drinking behaviors, the study of persistent or long-term effects of MLDA has only been undertaken recently. Our group has shown that adults who were legally permitted to drink prior to age 21 exhibited 1.3-fold higher odds for a past-year alcohol use disorder, suggesting that individuals exposed to an MLDA of under 21 may be at elevated risk for alcohol-related problems in later adulthood (Norberg et al., 2009).

The purpose of this study is to take advantage of the “natural experiments” afforded by changes in MLDA to assess long-term effects of state MLDA policies in effect during adolescence/early adulthood on later suicide and homicide rates. The quasi-experimental approach allows for inferences about the role of the policy environment, independent of genetic and other individual-level characteristics, providing that MLDA law can be considered to be randomly assigned at the individual level. Our focus is on the 1990–2004 period. During this time, MLDA changes had been completed, and any effect of having been legally permitted to drink prior to age 21 can only be attributed to earlier exposure to these policies.

Materials and Methods

Subjects

Data on suicide and homicide deaths were obtained from the Multiple-Cause of Death files for 1990–2004, collected by the National Center for Health Statistics. Files containing individual-level data were obtained through the National Bureau of Economic Research (http://www.nber.org/data/multicause.html). Data on deaths between 1990 and 2004 were compiled; 1990 was chosen as the start date because it was a census year, thus enabling a precise estimate of the population at-risk for the beginning of the observation period. The year 2004 was the last year for which publicly accessible, geographically identified mortality data was available.

In order to estimate risk ratios, it was necessary to merge death record data with corresponding records on the living population. Population data were culled from 1% samples of individual-level data from the 1990 U.S. Census, the 2000 U.S. Census, and the 2004 American Community Survey (ACS). The ACS is an ongoing population survey conducted by the U.S. Census Bureau, phased in as a replacement to the long form of the U.S. census. The 2004 sample contained data on 353,220 individuals in our targeted age-range, or 0.44% of the U.S. population. All population data were obtained from the Integrated Public Use Microdata Series maintained by the Minnesota Population Center (Ruggles et al., 2010).

Our analytical approach relies on differences in policy within birth cohorts, and requires that there are changes in policy over time. Therefore, we limited analyses to the population who approached age of majority during the period in which minimum legal drinking ages were in flux. This population consists of individuals who turned 18 during the years 1967 to 1990, corresponding to birth years 1949 to 1972. We also limited our analyses to records for individuals who could be classified as non-Hispanic White, non-Hispanic Black, or Hispanic of any known or unknown origin. We limited analyses to these large race/ethnicity categories to maximize comparability across death certificate codings from different jurisdictions and different eras. Inclusion of an “other” category would have resulted in a group that was heterogeneous with respect to race and, more problematically, would have changed significantly over time, both as a result of coding changes in the vital statistics system and demographic changes in the United States. We included only records for individuals born in the 50 U.S. states and the District of Columbia in order to facilitate estimation of MLDA exposure. Likewise, we tabulated only deaths occurring in those areas.

We used two approaches for estimating the “control” population, that is, the living population, and those who died from causes other than suicide and homicide. For our core analyses, we used the 1990 U.S. Census 1% micro-extract as the control population. Although this approach includes decedents from suicide and homicide as part of the control group, this represents only 0.25% of the population, so the error introduced by this approximation is negligible. The advantage of this approach is that the census extract is a precise 1% sample of the population born between 1949 and 1972 and alive at the beginning of our 1990–2004 observation window. The disadvantage of using single-year population data is that secular trends with respect to year-of-death (period effects) cannot be modeled because the controls yield no information about the population in the years beyond 1990. Hence, for regression analyses that included period as a covariate, we used an alternative approach to estimating the control population. In this second approach, we used estimates of the population for every year between 1990 and 2004 to produce a “time-averaged” population sample. The sample was assembled by combining the 1990 and 2000 U.S. census samples with the 2004 American Community Survey sample. Linear interpolation was used

to estimate population data for intercensal years. Further details are provided in the Supplemental Material.

Outcomes, Covariates, and Population Estimates

From the complete set of death records occurring between 1990 and 2004, we selected records for which either suicide or homicide was listed as the underlying cause, or among the contributing causes of death using ICD-9 and ICD-10 codes (World Health Organization, 1980; World Health Organization, 1992). For 1999–2004 records, ICD-10 codes for suicide include X60-X84 and Y87, corresponding to intentional self-harm and sequelae of intentional self-harm; ICD-10 codes for homicide included X85-Y09 and Y89 (assault and sequelae of assault). For 1990–1998, suicide records corresponded to ICD-9 codes of E950–E959, and homicide codes included E960–E969. ICD-9 to ICD-10 comparability ratios for both of these outcomes are very close to 1.0 (Anderson et al., 2001).

Covariates included variables that were available in both the mortality and population data sources: year of birth, state of birth, whether the subject resided in their birth-state at time of death or census, year of death or year of census, sex, educational attainment, and race/ethnicity. Educational attainment was collapsed into a binary variable indicating more than 12 years of education (equivalent to a high school degree) or 12 or fewer years of education. Race/ethnicity was categorized as non-Hispanic White, non-Hispanic Black, and Hispanic categories. A dichotomous variable to indicate whether an individual resided in their birth state at time of death or census was created by comparing state of birth and state of residence variables. This variable was used as a measure of the likelihood that a person resided in their birth state when they were between 18- and 21-years-old. Those who resided in their state of birth as adults were assumed to be more likely to also have dwelled in their home state during early adulthood, although this does not preclude the possibility that such individuals left their state temporarily. We refer to these subjects as “likely non-movers”, and some analyses were limited to this subset of the data in order to test the robustness of our results to approximations described below.

Estimation of MLDA Exposure

We considered those who were permitted to drink legally prior to age 21 as having been “exposed” to permissive MLDA. Those who were not permitted to drink legally until age 21 were considered “unexposed”. To precisely assess exposure, we would need to know where individuals resided between the ages of 18 and 21. However, death and census records contain only state of birth and state or residence at time of observation. Therefore, we used state of birth as a proxy for state of residence during the ages of interest. Several tests were conducted to examine the validity of the proxy approach and the robustness of results to alternative approaches. First, we used state-of-residence at time of death or census as the proxy, rather than state-of-birth. Second, we conducted analyses that were limited to “likely non-movers” (individuals who resided in their state of birth at the time of their death or census). Finally, to quantitate the degree of error introduced into the exposure variable by the proxy approach, we culled 18–20 year old respondents from the 1980 and 1990 censuses to examine the percentage of those who lived in their birth states, and the percentages of those whose exposure would be correctly assigned under the assumption that they lived in their birth states, regardless of whether they had moved.

Precise dates of birth were not available from public-use death records, so date-of-birth was estimated as the mid-point in the reported year of birth. MLDA exposure was then estimated from state and year of birth. Month and year of changes in MLDA laws were coded based on data from published research articles and from data provided by the Statewide Availability Data System, a data source for alcohol policy and other alcohol-related

epidemiological information (DuMouchel et al., 1987; O'Malley and Wagenaar, 1991; Ponicki, 2004; Wagenaar, 1981–82).

Graphical Analysis of Age-adjusted Mortality Risk

In order to present a visual representation of the data, we prepared plots of the relative risks associated with MLDA in each of the 39 states that enacted MLDA changes that affected the birth cohorts analyzed here. These calculations, which were done only for the graphical analysis, were conducted separately for men and women. Specifically, we computed relative risks for suicide and homicide by state and sex. This was done using logistic regression to estimate the association between MLDA and each cause of death with year-of-birth, coded as a continuous variable and included as the only covariate. This covariate was included to account for the slightly lower risk for suicide and slightly higher risk for homicide associated with more recent birth years. The age-adjusted, sex-specific relative risk ratios were then plotted for each state.

Analytical Design and Statistical Methods

The analytical approach exploits changes in policy within states over time, and differences in policy between states at any given time, to estimate the policy effect independently of state and time confounders. (Theory behind this approach is provided in the Supplemental Material). The general model is outlined in Equation 1, below. It expresses the outcome variable, Y, as a function of (left-to-right): a vector of birth-year fixed effects (λ), a vector of state fixed effects (α), the policy effect (β) where policy ($MLDAX_{st}$) is a function of state and birth year, a vector of all other individual-level covariates such as demographic variables (X) and an error or disturbance term (ε).

$$\text{logit } Y_{ist} = \lambda_t + \alpha_s + \beta MLDAX_{st} + \eta X_{ist} + \varepsilon_{igt} \quad \text{[Equation 1]}$$

In this case, Y represents probability of death; $MLDAX_{st}$ is a binary indicator coded as one for subjects who were legally permitted to drink prior to age 21, and zero for those who were not. MLDAX is determined by state (s) and birth year (t), with β being the parameter of interest; namely, the log of the odds ratio for the association between exposure to MLDA<21 and death by suicide or homicide. For some analyses, we also included interactions between the state indicators (α) and birth-year coded as a continuous variable to account for unobserved state-level variables that may change over time.

Parameter estimates and standard errors were calculated using the generalized estimating equation method incorporated into SAS (Version 9, SAS Institute, Cary, NC) using state as the clustering unit (Arellano, 1987; Liang and Zeger, 1986). This approach accounts for intra-correlation of observations within clusters (states) in estimating standard errors, which is the promary consideration in standard error estimation when applying this quasi-experimental approach (Angrist and Pischke, 2008; Bertrand et al., 2004).

Results

Descriptive Statistics for Suicide and Homicide

Demographic distributions of suicide and homicide cases for individuals born in the U.S. between 1949 and 1972, and who died in the U.S. between 1990 and 2004, are listed in Table 1, along with the demographic distribution of the living population, based on the time-averaged population for the 1990–2004, estimated as described in the *Methods*. Demographic patterns for both suicide and homicide in these specific birth cohorts are comparable to those for full-population data in the U.S. (e.g., Heron et al., 2009). For

example, demographic risk factors for suicide included male sex and non-Hispanic White race/ethnicity. Homicide risk was associated with male sex, and non-Hispanic Black or Hispanic race/ethnicity. There were very small differences in suicide risk by birth year, age, or period. Younger people are generally at higher risk for homicide, but homicide rates have come down over time, leading to pronounced reductions in the percentage of homicides occurring during the later periods of the era that is the focus of our analyses (Klaus and Rennison, 2002).

Graphical Analysis of Death Rates

Nearly one-half of the U.S. population (48.9%) born between 1949 and 1972 were legally permitted to drink prior to age 21. To graphically examine the relationship between MLDA and deaths by suicide or homicide, we computed birth-year adjusted death rates for each birth year cohort, and relative risk ratios describing the association between death rate and MLDA exposure. Ratios were calculated separately by sex. Results are plotted in Figure 1. Among women, relative risks for suicide associated with MLDA <21 were greater than one in 37 of 39 states (all states except Montana and Oklahoma), and relative risks for women were higher than relative risks for men in all 39 states in which MLDA changes occurred. For homicide, relative risks for women were again greater than one in all states but one (Delaware), and in most cases (28 out of 39), relative risks for women were greater than for men. This descriptive analysis leaves a number of potential confounders unaccounted for, but nonetheless, the results demonstrate a pattern in which the association between suicide risk or homicide risk and MLDA is consistently higher for women then for men.

Fixed Effects Regression Models

Results of logistic regression analyses predicting 1990–2004 suicide rates from MLDA exposure, adjusting for birth state and birth year fixed effects are shown in Table 2. There was no significant main effect for MLDA on suicide (OR=1.02, p=0.35). Little change in the point estimate was observed when adjusting for race or sex. However, inclusion of a sex-by-MLDA interaction suggested that women who were exposed to lower MLDA had a statistically significant 12% elevated risk for suicide during this period (OR=1.12, 95% CI: 1.05, 1.18, p=0.0003). The odds ratio associating more permissive MLDA and suicide risk for men was slightly below one (0.97, 95% CI: 0.95, 1.00, p=0.048), and nominally statistically significant. The interaction between MLDA and sex was highly significant (p=<0.0001). Limiting the analysis to likely non-movers, i.e., individuals who died in their state of birth, or who lived in their state of birth for the 1990 census, had essentially no effect on parameter estimates (See Supplemental Table 1). Likewise, using state-of-residence, rather than state-of-birth, to estimate MLDA exposure had little impact (See Supplemental Table 2). Inclusion of educational attainment or linear state-by-year interactions (i.e., state-specific secular trends) as additional covariates had essentially no effect on the parameter estimates (results not shown).

Regression results for homicide largely paralleled those for suicide: there was no significant main effect of MLDA (OR=1.04, p=0.35) but there was a significant interaction by sex with corresponding increased risk for death by homicide for women exposed to MLDA < 21 (OR=1.14, p=0.006 for women, interaction p=0.003), and no significant effect for men. Restricting the analysis to non-movers (Supplemental Table 1) resulted in an increased odds-ratio estimate for women (OR=1.20, p=0.002) and for the interaction term (p=0.0009). Replacing state-of-birth with state-of-residence as the predictor variable had little effect on the results (Supplemental Table 2). Likewise, these results were essentially unchanged after inclusion of education, or linear state-by-year interactions as covariates (results not shown).

Interactions of MLDA with Period and Birth Year

Table 3 describes analyses to determine whether the effects documented above differ by period (i.e., year of death) or birth year. For each of these analyses, the sample was split roughly into halves. The first analysis split death-events into those occurring between 1990 and 1996 and those occurring from 1997 to 2004. During the 1990–96 period, the study sample was between the ages of 18 and 47; during 1997 to 2004 the age range was 25 to 55 years old. For both suicide and homicide, the odds ratio associated with lower MLDA exposure was significant for women during both periods, although there was a slight trend toward lower odds ratios for women during the second period – this was more apparent for homicide than for suicide.

Splitting the sample by year of birth revealed several notable differences. For birth years 1949–1959, there were was no significant effect of MLDA exposure on suicide or homicide deaths for men or women. In contrast, for the later cohort, born between 1960 and 1972, effects for both outcomes were significant for women and larger than those estimated in the full cohort analyses. This younger birth cohort were affected by MLDA changes that came about in the late 1970s through late 1980s, when drinking age laws were raised both as state-initiated public health measures, and in response to the uniform legal drinking age act of 1984.

Accuracy of the State of Birth Approximation

Ideally, to assess exposure to permissive MLDA policy, we would like to know where an individual dwelled when they were between the ages of 18 and 20. Unfortunately, this information is not available in vital statistics records, and so we use state of birth as a proxy. To check the accuracy of this approximation, we examined U.S. Census data on individuals who were between the ages of 18 and 20 during 1970 and 1980, the two census years that correspond to the historical period in which we are interested. Observed MLDA exposure was assessed based on their year and quarter of birth, and the state in which they resided at the time of census. We then imputed exposure under the assumption that state of birth was equivalent to state of residence at the time of census, and compared the proxy-imputed exposure with the observed exposure. From the total sample of N=196,620 observations, 72.4% resided in their state of birth, leaving 27.6% susceptible to misclassification when using the state-of-birth proxy. However, imputed MLDA exposure matched observed MLDA exposure in 88.0% of subjects, indicating that 15.6% of the population had emigrated to states with similar MLDA policy as their birth state. Using the proxy approach to predict observed exposure, the positive predictive value was 88.4%, while the negative predictive value was 87.4%. (These values correspond to the percentage of correctly classified exposed and unexposed individuals, respectively). Hence, this approximation appears to exhibit little bias towards positive or negative misclassification, and exhibits good overall precision. These analyses are discussed in more detail in the Supplemental Material.

Discussion

Summary of Findings

We hypothesized that the elevated risks for suicide and homicide linked to MLDA exposure might persist into adulthood. In the full population, we do not find this to be the case. We found important differences, however, between men and women in the association between MLDA and risk of death from either cause. Specifically, we observed a significant MLDA x sex interaction in the prediction of both suicide and homicide that corresponded to a 12% elevation in suicide risk and a 15% elevation in homicide risk for women exposed to MLDA < 21.

When the sample was split (roughly in half) by period, the MLDA-associated risk for women was significant both in the 1990–1996 and 1997–2004 periods, though there was a very small tendency toward lower risk in the latter period, suggesting MLDA effects may wane with age. This trend of lower risk in the latter period was more pronounced in the case of homicide (Table 3). When splitting the sample by birth cohort, there was no significant MLDA-associated risk observed for women (or men) born between 1949 and 1959 -- the birth cohorts who came of age during a period of liberalized drinking age laws. In contrast, the odds ratios for the 1960–1972 birth cohort -- who came of age as drinking age laws were being raised in response to public health concerns -- tended to be larger than those observed in the full sample analyses, and significantly higher than those observed for the 1949–1959 birth cohort. Hence, there are two interactions that need to be discussed: the interaction between MLDA and sex, and for women, the interaction between MLDA and birth cohort (technically, a three-way MLDA x sex x birth cohort interaction). Moreover, there is a need to understand why these interactions were observed for both outcomes, even though suicide and homicide are fundamentally different causes of death with regard to other risk factors.

The sex specificity of the MLDA-suicide and MLDA-homicide associations may relate to unique alcohol-related risks faced by women. One of the most thoroughly documented characteristics of suicide is that attempts are more common in women, whereas completions are more prevalent in men (Kessler et al., 1999; Krug et al., 2002; Moscicki, 2001; Weissman et al., 1999). Suicide attempts that occur under the influence of alcohol are more lethal than those not occurring during alcohol use (DeJong et al., 2010; Sher, 2006; Sher et al., 2009). If higher rates of alcohol problems associated with MLDA result in an overall increase in the lethality of suicide attempts, this effect might be more apparent in women than in men, for whom the lethality of suicide is relatively high. A possible sex-specific link between alcohol involvement and homicide is the differential rate of intimate partner violence (Tjaden et al., 2000). Approximately two-thirds of intimate partner violence homicides in the U.S. are perpetrated against women (Karch et al., 2010; Paulozzi et al., 2001) and one in three homicides involving female victims is committed by a current or former spouse or partner, compared to just one in twenty men (Federal Bureau of Investigation - U.S. Department of Justice, 2009). Excessive alcohol consumption by the victim, aggressor, or both is thought to exacerbate relationship stressors and increase the occurrence and severity of intimate partner violence (Foran and O'Leary, 2008; World Health Organization, 2005). Hence, increases in alcohol problems associated with lower MLDA may elevate rates of partner violence and thus influence rates of female homicide deaths.

Another key observation is that MLDA was not associated with suicide or homicide rates for women born between 1949 and 1959, but there was a substantial association between MLDA and both outcomes for women born between 1960 and 1972 (Table 3). Multiple authors have documented a "closing gender gap" in alcohol use disorders and related problems in recent decades (Greenfield et al., 2003; Holdcraft and Iacono, 2002; Keyes et al., 2008). This trend may have started with women born after World-War II, with increases in alcohol dependence particularly noticeable among women born in the 1960s and beyond (Grucza et al., 2008). It may be that excessive drinking was simply not part of the culture for women born in earlier years of the cohorts examined here, and that changing cultural norms may be a key contributor to MLDA-associated suicide and homicide risk observed for more recent birth years. In any event, the fact that MLDA effects were not the same for all birth years indicates that the lower MLDA alone does not result in elevated suicide and homicide risk, but that its effects may be dependent on other aspects of the cultural environment. Nonetheless, our data is consistent with the interpretation that the *de facto* national drinking age of 21 has resulted in fewer deaths by suicide and homicide among women.

Limitations and Conclusion

The incorporation of state and birth year fixed effects accounts for any unobserved state-specific covariates that do not change over time, and any birth-year-specific covariates that are constant across state. While this controls for many potential confounders, it falls short of a controlled-experiment, as does any observational study. There are additional limitations that relate to the accuracy of the effect size estimate, including the lack of exact birth date for decedents, and the lack of information on state of residence during the ages 18 to 21. As discussed at length the Supplemental Material, we expect this to lead to an underestimate of the effect size for the association between MLDA and death by suicide or homicide.

Limitations notwithstanding, the findings from this quasi-experimental study provides stronger evidence of MLDA effects than analyses of single cross-sections would. Recent reports estimate the number of suicides and homicides for women born after 1960 in the United States to be about 3,600 and 2,700, respectively for 2007 (Crosby et al., 2011; Logan et al., 2011). Based on these numbers, and the effect size estimates in Table 3, we estimate that the 21 MLDA may be preventing more than 600 suicides and more than 600 homicides annually among individuals of all ages (i.e., those currently restricted from purchasing alcohol prior to age 21 as well as those who are now past the age of majority, but were restricted from purchase as minors). This interpretation must be tempered by considering the previously mentioned limitations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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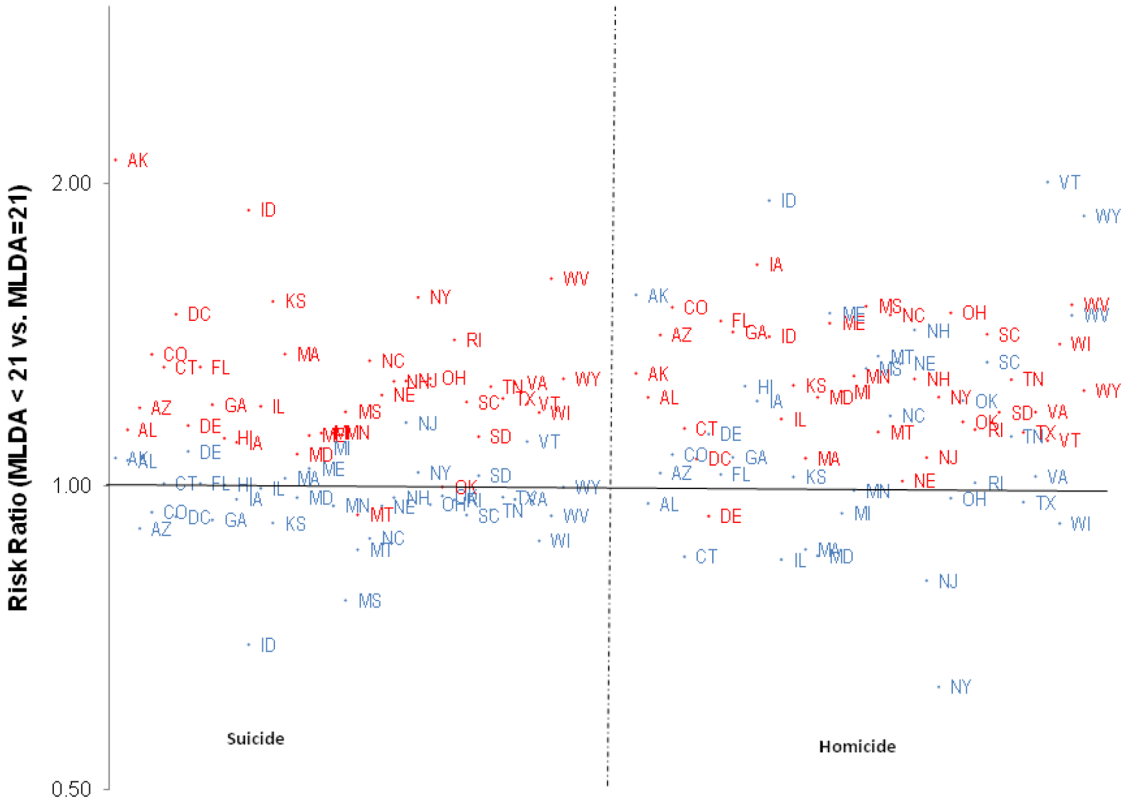

Figure 1.

Age-adjusted relative risk ratios for the association between MLDA (under 21 vs. 21) and suicide (left) or homicide (right), computed separately by sex (blue=men, red=women) for each of the 39 states that underwent one or more changes in MLDA between 1967 and 1989

Table 1

Demographic Descriptions of Suicide Cases, Homicide Cases, and the General Population Individuals born in the U.S. between 1949 and 1972, death records from 1990 to 2004.

	Suicide		Homicide		Living Population[1]	
	N	Col %	N	Col %	N	Col %
Sex						
Women	41,131	20.5%	32,154	23.4%	41,828,860	50.8%
Men	159,540	79.5%	104,927	76.6%	40,477,566	49.2%
Race						
White	177,311	88.4%	51,513	37.6%	67,259,800	81.7%
Black	16,034	8.0%	75,824	55.3%	10,705,125	13.0%
Hispanic	7,326	3.6%	9,744	7.1%	4,341,501	5.3%
Birth Year[2]						
1949–1960	105,781	52.7%	53,994	39.4%	41,737,328	50.7%
1961–1972	94,890	47.3%	83,087	60.6%	40,569,098	49.3%
Age at Death or Census[2]						
18–19	2,017	1.0%	3,046	2.2%	658,451	0.8%
20–29	40,429	20.2%	46,435	33.9%	1,6378,979	19.9%
30–39	83,004	41.4%	56,898	41.5%	34,486,392	41.9%
40–49	62,251	32.5%	27,844	20.3%	26,338,056	32.0%
50–55	9,970	5.0%	2,858	2.1%	4,444,547	5.4%
Moved from Birth State						
No	122,840	61.1%	96,804	70.6%	51,015,093	63.1%
Yes	77,831	38.9%	40,277	29.4%	30,390,443	36.9%
Period (Year of Death or Census)[2]						
1990–1996	93,886	46.8%	83,773	61.1%	43,657,687	53.0%
1997–2004	106,785	53.2%	53,308	38.9%	38,648,739	47.0%
Total	200,671	100.0%	137,081	100.0%	82,306,426	100.0%

[1] Based on an estimated time-averaged population for the years 1990–2004 generated as described in *Methods*.

[2] Birth year and period categories are used for stratification analyses, and comprise approximate sample splits. Age categories were chosen for descriptive purposes only

Table 2

Fixed Effects Logistic Regression – Suicide and Homicide Predicted from MLDA exposure. State and year of birth included in all models.

	Suicide		Homicide	
Additional Covariates:	**Odds Ratio (95% CI)**	**P**	**Odds Ratio (95% CI)**	**P**
None	1.02 (0.98, 1.06)	0.35	1.04 (0.96, 1.14)	0.35
Sex	1.00 (0.97, 1.02)	0.78	1.03 (0.95, 1.12)	0.50
Sex, Race/Ethnicity	1.00 (0.98, 1.02)	0.91	1.02 (0.95, 1.09)	0.58
Sex, Race/Ethnicity, Sex x MLDA Interaction:				
MLDA: Women	1.12 (1.05, 1.18)	0.00031	1.15 (1.04, 1.25)	0.0028
MLDA: Men	0.97 (0.95, 1.00)	0.048	0.96 (0.90, 1.03)	0.25
Interaction (Men vs. Women)	0.87 (0.82, 0.93)	<0.0001	0.83 (0.74, 0.94)	0.0023

Table 3

Fixed Effects Logistic Regression – Suicide and Homicide Predicted from MLDA Exposure, with Sex Interaction, Among Split-Cohorts.

	Suicide		Homicide	
	Odds Ratio (95% CI)	**P**	**Odds Ratio (95% CI)**	**P**
Death Years 1990–1996				
Women	1.13 (1.05, 1.21)	0.00083	1.18 (1.06, 1.31)	0.0022
Men	0.97 (0.94, 1.00)	0.032	0.96 (0.89, 1.04)	0.32
Interaction (Men vs. Women)	0.86 (0.80, 0.92)	<0.0001	0.82 (0.73, 0.92)	0.0011
Death Years 1997–2004				
Women	1.10 (1.04, 1.17)	0.00099	1.12 (1.03, 1.22)	0.0076
Men	0.98 (0.95, 1.01)	0.15	0.95 (0.89, 1.03)	0.20
Interaction (Men vs. Women)	0.89 (0.83, 0.95)	0.00052	0.85 (0.76, 0.96)	0.0070
Birth Years 1949–1959				
Women	0.97 (0.92, 1.02)	0.25	0.98 (0.89, 1.09)	0.75
Men	0.98 (0.95, 1.01)	0.15	0.96 (0.90, 1.03)	0.29
Interaction (Men vs. Women)	1.02 (0.96, 1.08)	0.54	0.98 (0.95, 1.01)	0.15
Birth Years 1960–1972				
Women	1.17 (1.10, 1.24)	<0.0001	1.24 (1.10, 1.40)	0.0004
Men	0.98 (0.94, 1.02)	0.23	1.00 (0.93, 1.09)	0.93
Interaction (Men vs. Women)	0.83 (0.78, 0.89)	<0.0001	0.81 (0.71, 0.93)	0.0037

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