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The Impact of the Minimum Legal Drinking Age on Alcohol Related Chronic Disease Mortality

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Abstract

Background—The minimum legal drinking age (MLDA) of 21 has been associated with a number of benefits compared to lower MLDAs, including long-term effects, such as reduced risk for alcoholism in adulthood. However, no studies have examined whether MLDA during young adulthood is associated with mortality later in life. We examined whether individuals exposed to permissive MLDA (< 21) had higher risk of death from alcohol-related chronic disease compared to those exposed to the 21 MLDA. Because prior work suggests that MLDA affects college students differently, we also conducted conditional analyses based on ever having attended college.

Methods—Data from the 1990 through 2010 U.S. Multiple Cause of Death files were combined with data on the living population and analyzed. We included individuals who turned 18 during the years 1967 to 1990, the period during which MLDA varied across states. We examined records on death from several alcohol related chronic diseases, employing a quasi-experimental approach to control for unobserved state characteristics and stable time trends.

Results—Individuals who reported any college attendance did not exhibit significant associations between MLDA and mortality for the causes of death we examined. However, permissive MLDA for those who never attended college was associated with 6% higher odds for death from alcoholic liver disease, 8% higher odds for other liver disease, and 7% higher odds for lip/oral/pharynx cancers (*OR* = 1.06, 95% CI [1.02, 1.10]; *OR* = 1.08, 95% CI [1.02, 1.14]; *OR* = 1.07, 95% CI [1.03, 1.12], respectively).

Conclusions—The 21 MLDA likely protects against risk of death from alcohol-related chronic disease across the lifespan, at least for those who did not attend college. This is consistent with other work that shows that the long-term association between MLDA and alcohol-related outcomes is specific to those who did not attend college.

Keywords

MLDA; Alcoholic liver disease; cancer; mortality; college drinking

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Introduction

The minimum legal drinking age (MLDA) in the United States determines the age at which individuals may legally purchase and possess alcohol in public. This policy varied by state after prohibition and many states reduced their drinking ages to as low as 18 during the Vietnam War era (Wagenaar and Toomey, 2002). Following passage of the National Minimum Drinking Age Act of 1984 (23 USC §158) all states adopted an MLDA of 21, a process that was complete by 1988 (with the exception of the state of Louisiana, which maintained a lower *de facto* purchase age until 1995; Ponicki, 2004; Scribner and Cohen, 2001).

Many studies have shown that higher MLDAs are linked to lower rates of motor-vehicle fatalities, alcohol consumption, and numerous other adverse alcohol-related outcomes among teenagers and adults under the age of 21 (see DeJong and Blanchette, 2014 and Wagenaar and Toomey, 2002 for reviews of this extensive literature). A handful of studies have also shown that the effects of the MLDA may persist into the early 20s; that is, those who were restricted from drinking at ages 18-20 were less likely to report heavy drinking episodes after age 21 (Cook and Moore, 2001; Moore and Cook, 1995; O'Malley and Wagenaar, 1991). Insofar as MLDA is a proxy for the age at which one has ready access to alcohol, this suggests that changes in access during late adolescence could have a persistent impact on one's drinking patterns.

The idea that the MLDA has persistent effects is intriguing because of neurobiological evidence that adolescence is a critical period during which vulnerability to substance use disorders are at their highest (Chambers et al., 2003). Motivated by this theory, recent work from our group has suggested that higher MLDAs do appear to have long-term benefit. Birth cohorts who were legally restricted from drinking prior to age 21 are less likely to drop out of high school, have alcohol use disorder (AUD), and engage in binge drinking behaviors in later adulthood (Norberg et al., 2009; Plunk et al., 2015, 2013). A protective cohort effect of higher MLDA on risk of death by suicide and homicide among adult women has also been suggested (Grucza et al., 2012). Others have shown that higher drinking ages appear to confer protection against fatal traffic accidents over the long term among men (Kaestner and Yarnoff, 2011). These studies of the long-term consequences of the MLDA have built upon a large body of literature showing that early drinking is an important marker, though not necessarily a contributing cause, of adult alcohol use disorders (DeWit et al., 2000; Grant and Dawson, 1997; Grucza et al., 2008; McGue et al., 2001; Prescott and Kendler, 1999; Ystrom et al., 2014). However, results of studies of the MLDA, which functions as a natural experiment of legally restricting access to alcohol during late adolescence, suggest that reductions in early drinking lead to decreased risk for alcohol problems well into mid-adulthood.

If ready access to alcohol at a younger age increases risk for alcohol use disorder and heavy drinking over the long term, then risk for alcohol-related chronic disease mortality should also be affected (Plunk et al., 2014a). In this work we undertake the first examination of the possible effects of the MLDA—and, by extension, changes in access to alcohol during a key

developmental period—on alcohol-related chronic disease mortality. This is significant for several reasons. First, there are relatively few studies of the long-term consequences of permissive (i.e., < 21) MLDA (DeJong and Blanchette, 2014). More specifically, if we find that the MLDA is associated with chronic disease mortality, it would show that MLDA policies protect not only adolescents and young adults from the harmful effects of alcohol, but that the influence of MLDA on health may last throughout the lifespan. Finally, this work is timely as debate about the minimum drinking age continues both in the U.S. and in other countries, where some argue that lower drinking ages will lead to safer drinking among youth and young adults (e.g., Christiansen, 2010; Cohan, 2014; Metherell, 2009; Paglia, 2014; Tracy, 2014; Young, 2012).

Our objective was to examine whether the ability to legally purchase alcohol prior to age 21 is related to increased chronic disease mortality later in life. We propose that permissive MLDA, a proxy for ready access to alcohol during the ages of 18-20, influences heavy drinking outcomes across the lifespan, which in turn influence mortality risk from chronic disease. Since this hypothesized association is indirect we expect to see significant findings only for diseases for which risk is substantially attributable to heavy alcohol use. We focused on mortality from the following chronic diseases that are in large part (> ~20%) attributable to alcohol: liver cirrhosis and other liver disease; cancers of the lip, oral cavity and pharynx; laryngeal cancer; and esophageal cancer (Boffetta et al., 2006; Haas et al., 2012; World Health Organization, 2014).

In addition to the proportion of mortality attributable to alcohol, other factors are likely to influence the magnitude of any potential association between MLDA and risk of death that we will be able to observe. First, MLDA exposure is determined by a person's year of birth and state of residence between the ages of 18-21, but it is not possible to directly observe this from mortality data. Instead, we must rely on subject's state of residence at time of observation as a proxy (Krauss et al., 2015; Norberg et al., 2009; Plunk et al., 2015, 2013). To determine if this is a reasonable way to estimate policy exposure we conduct a separate set of analyses on “likely non-movers.” This subset of the study sample resided in their birth state at time of observation and are much less likely to have ever migrated between states. Second, some studies have suggested that college campuses may be insulated from the effects of drinking age policy; that is, that the mix of legal and non-legal drinkers on school campuses provides ready access to alcohol, making youth-access policies like the MLDA less effective (Grucza et al., 2009; Johnston et al., 2015; Plunk et al., 2015; Wagenaar and Toomey, 2002). To ensure that we are capturing this potential campus insulation effect, we examine whether the potential impact of MLDA on chronic disease mortality is stronger among those who did not attend college. Thus, in the current study we use nationally representative mortality data to test three hypotheses: (1) MLDA is associated with increased risk for liver disease and alcohol-related cancers; (2) magnitudes of association are similar among likely non-movers, for whom we are less likely to misestimate MLDA policy exposure; and (3) the associations between MLDA and alcohol-related chronic disease mortality are stronger among individuals who never attended college.

Methods

Dependent variables and data sources

Our dependent variables were death due to: alcoholic liver disease; liver disease not specified as alcohol-related; cancers of the lip, oral cavity, and pharynx; esophageal cancer; and laryngeal cancer. Data on individual deaths in the U.S. were obtained from the Multiple Cause-of-Death files for 1990-2010, collected by the National Center for Health Statistics. These data are based on all death certificates filed in the U.S. by each state and the District of Columbia (Miniño et al., 2011). Files containing individual-level data through 2004 were obtained through the National Bureau of Economic Research (http://www.nber.org/data/multicause.html). For years beyond 2005, state-level geographic identifiers are not included in public use files, so customized files including geographic data were obtained by request through the National Association for Public Health Statistics and Information Systems. From the complete set of death records, we selected individuals who died from one of the causes of interest based on ICD-9 codes for years 1990-1998 and ICD-10 codes for years 1999-2010. Codes for each outcome are listed in Table 1. Each analysis included records from individuals for whom the cause of interest was listed as a contributing cause of death, alongside records from the living population, described below.

To model the living population, we combined data from the annual American Community Survey (ACS) for the years 2001-2010, obtained from the Integrated Public Use Microdata Series maintained by the Minnesota Population Center (Ruggles et al., 2010). Since the ACS was not administered annually prior to 2000 we used data from 1% samples of the 1990 and 2000 Census to estimate data for years 1991 through 1999 using a linear interpolation procedure fully described elsewhere (Grucza et al., 2015, 2009). Briefly, this was accomplished by creating a single record for each possible combination of covariate parameters in each Census data set (i.e., each combination of year, state, race/ethnicity, sex, age group and education) and assigning that record a weight corresponding to the population for that group. The weight for the corresponding record during non-observed years was estimated as: [(2000-year)*(1990 weight) + (year-1990)*(2000 weight)] / 10). We have shown elsewhere that this method is valid by comparing the results with U.S. Census Bureau estimates for intracensal years (Grucza et al., 2015). All observed and estimated living population data were combined and sample weights were divided by the number of data years. The living population data were then combined with the mortality data to create the final data set. Because our analytical approach relies on differences in policy exposure within birth cohorts, we limited analyses to those who turned 18 during the years 1967 to 1990 (birth years 1949 to 1972), the period in which the MLDA was in flux.

Independent variable: MLDA exposure—MLDA policy data were coded as described in our previous studies (e.g., Norberg et al., 2009; Plunk et al., 2015, 2013). We examined a period during which some states both increased and decreased their MLDAs, while others maintained a 21 MLDA throughout. Individuals who were legally permitted to purchase alcohol between the ages of 18-20 (i.e., those with “permissive MLDA exposure”) were contrasted with those who were restricted until age 21. Individuals who were exposed to an MLDA of 18, 19 or 20 were assigned a value of “1” and those who were unable to

purchase alcohol before the age of 21 were assigned a value of “0.” This means that individuals from the same state could have different MLDA exposure, depending on when they were age 18-20.

MLDA coding for each state was based on year of change (i.e., when a change occurred, MLDA was assigned based on what the MLDA was changing to at any time during that year). State of residence at the time of survey administration or death was also used as a proxy for state of residence at the age of potential exposure. Since mortality and census records do not contain residence history between the ages of 18 and 21, we used state of residence at time of observation as a proxy for state of residence during time of exposure. While this introduces error due to misclassification, the most likely effect is to reduce the estimated magnitude of any true association (i.e., bias toward the null hypothesis, or type II error). We have shown elsewhere that migration-induced error is unlikely to bias estimates toward type I error unless there is a strong correlation between disease status and change in policy exposure upon emigration (Grucza et al., 2012). Furthermore, the rate of misclassification is much lower than the rate of emigration since individuals may move to states with the same MLDA and thus migrate without changing their policy exposure. Elsewhere, using population migration data, we have estimated that misclassification to be approximately 11% (Krauss et al., 2015).

Covariates—Individual-level covariates extracted from mortality records included state of residence, year of birth, sex, age, race/ethnicity, educational attainment, and year of death. Race/ethnicity was coded as non-Hispanic White, non-Hispanic Black, Hispanic, and other. Because of the birth year inclusion criteria, age ranged from 28 to 61 years. Age categories corresponded to quartiles; ranges were 28-38, 39-44, 45-50, and 51-61 years old. Education was dichotomized such that those with no post-secondary education were coded as “no college” and those with one or more years of post-secondary education were classified as “any college.” We also included several time-varying state-level covariates to control for potential confounding due to factors that might have changed simultaneously with drinking age policies and might also influence alcohol-related mortality outcomes. These included a measure of citizen political ideology (Berry et al., 1998) state per-capita income, state annual unemployment rate, state beer excise tax at time of observation, state beer excise tax at time the respondent/decedent was age 21, an indicator for privatization of wine and spirits sales, percent of state population affiliated with Judeo-Christian religious denominations, and state annual unemployment rate. Per-capita income and unemployment measures were obtained from the University of Kentucky Center for Poverty Research.(University of Kentucky Center for Poverty Research, Gatton College of Business & Economics. University of Kentucky, Lexington, KY., n.d.). Data on beer taxes were obtained from the Statewide Availability Data System and from the Alcohol Policy Information System (National Institute on Alcohol Abuse and Alcoholism., n.d.; Ponicki, 2004). Wine and spirits sales privatization data were based on earlier studies examining these policy changes (Hahn et al., 2012; Wagenaar and Holder, 1995).

Stratification Variables—As previously discussed, one limitation of our analytical approach is that we do not know the state of residence of each individual during late

adolescence, and so we approximate MLDA exposure using state of residence at observation (i.e., at time of survey or census for the living population and time of death for decedents). One way to examine whether this approximation induces bias into our estimates is to conduct additional analyses limited to "likely non-movers" (i.e., those who resided in their birth state at the time of observation). These individuals are less geographically mobile, and therefore more likely to have lived in their current state during the period in which they would have been subject to MLDA policies (Plunk et al., 2014b). All analyses conducted on the full data set were subsequently conducted on this subset. We also examined whether the relationship between MLDA and mortality status differed by educational attainment. As discussed earlier, MLDA may have less influence on college campuses, and therefore, MLDA-mortality associations may be stronger among those who did not attend college. Lower educational attainment is also associated with lower cross-state mobility, so migration-related error is also likely lower among this group (Kaestner and Yarnoff, 2011).

Educational attainment was dichotomized as having any education beyond a high-school diploma vs. having no post-secondary education. Information on education was missing for 7.1% of decedents. Therefore, education was multiply imputed from other demographic variables for those observations. Five imputations were carried out. These data were used only for analyses stratified by education, and reported standard errors reflect the additional variance introduced by the imputation process.

Empirical Strategy

Our objective was to examine whether permissive MLDA exposure (<21) during adolescence is associated with increased likelihood of death from the selected alcohol-related chronic diseases in adulthood. Our quasi-experimental study design is based on the difference in differences approach, which models exposure to a policy change by comparing pre- and post-policy implementation differences in an outcome for exposed groups to those for unexposed comparison groups (Wooldridge, 2010). In effect, we use changes in MLDA to reflect within-state variation in alcohol availability over time for individuals under the drinking age. This approximates an experimental design if there are no unobserved confounders related to both policy exposure and the outcome in question. The plausibility of this "exogeneity assumption" rests on whether or not the policy change came about independently of other factors that also influence the outcome; for example, MLDA has been used as an exogenous variable representing a change in availability by many researchers since MLDA changes were driven by national trends (Dee and Evans, 2003).

Statistical analysis

We used logistic regression to model death due to the chronic diseases listed in Table 1. Fixed-effects regression models were used to control for the impact of unobserved, time-invariant state factors and national secular trends. This is accomplished by including dummy variables for state and birth year in regression models (Allison, 2009; Wooldridge, 2010). Final models included the primary independent variable (MLDA exposure), as well as state and birth-year fixed effects, participant demographics (sex, race, age category, year of observation, and education), and state-level variables selected as described previously (unemployment rate, per capita income, political ideology, beer excise taxes, wine and

spirits privatization, and religious affiliation). Parameter estimates and clustered standard errors were calculated using the SAS (Version 9.2, SAS Institute, Cary, NC) procedure "surveylogistic" employing state as the clustering unit (Angrist and Pischke, 2008; Arellano, 1987; Bertrand et al., 2004).

Results

Table 2 describes the demographic characteristics of the sample broken down by mortality status for each of the causes of death analyzed. The number of decedents ranged from 8,397 for laryngeal cancer to 115,841 for alcoholic liver disease. Men were substantially over-represented among decedents for each outcome, comprising between 70.1 and 80.5% of decedents. Blacks were over-represented among deaths from lip/oral/pharynx cancer, laryngeal cancer and liver disease not specified as alcoholic, while Whites were over-represented among esophageal cancer decedents. Hispanics were under-represented among all types of cancers, but over-represented among liver disease decedents. The proportion of death from each cause was also higher for older individuals and those who resided in their state of birth at time of observation.

Results of logistic regression analyses modeling risk for death from each cause as a function of permissive MLDA exposure are displayed in Table 3 (full models with estimates for all covariates are described in Tables S1-S5). Mortality risk was positive and statistically significant for alcoholic liver disease and for other liver disease (i.e., not specified as alcoholic); permissive MLDA exposure was associated with 5% and 6% greater odds of death from those two causes, respectively (*OR* = 1.05, 95% CI [1.01, 1.10]; *OR* = 1.06, 95% CI [1.01, 1.12]). A significant association between higher risk of death and permissive MLDA was also observed for lip/oral/pharynx cancers (*OR* = 1.05, 95% CI [1.01, 1.10]). The association between MLDA and esophageal and laryngeal cancers did not approach statistical significance. Analyses conditioned on sex did not suggest any meaningful between-groups differences. These analyses were then repeated for likely non-movers, for whom permissive MLDA was significantly associated with 6% greater odds of mortality from alcoholic liver disease (*OR* = 1.06, 95% CI [1.01, 1.11]), but not with other outcomes. However, point estimates were largely similar to those observed in the full sample. As with the full-sample analyses, there were no notable between-groups differences when likely non-mover men and women were analyzed separately.

Table 4 describes results from analyses conditioned on educational attainment. Individuals who reported any college attendance did not exhibit significant associations between permissive MLDA and any of the causes of death we examined (although deaths from laryngeal cancers approached nominal significance criteria for likely non-movers who had gone to college). However, individuals who reported not attending college exhibited significant associations between permissive MLDA and several causes of death in the full sample: 6% higher odds for death from alcoholic liver disease, 8% higher odds for other liver disease, and 7% higher odds for lip/oral/pharynx cancers (*OR* = 1.06, 95% CI [1.02, 1.10]; *OR* = 1.08, 95% CI [1.03, 1.13]; *OR* = 1.07, 95% CI [1.03, 1.12]). Estimates based on the likely non-mover subsample did not differ significantly from those derived from the full sample.

Discussion

Our findings suggest that individuals who were legally permitted to purchase alcohol prior to age 21 had modest, but statistically significant, increased risk of death from alcoholic liver disease, other liver disease, and lip/oral/pharyngeal cancers relative to those who were not permitted to purchase alcohol until age 21. These apparent policy effects were driven by individuals without any college education—those who reported having attended college did not exhibit increased mortality risk related to permissive MLDA exposure.

These findings are consistent with earlier studies from our group suggesting that permissive MLDA exposure during adolescence and young adulthood is related to increased risk of alcohol misuse later in life (Norberg et al., 2009; Plunk et al., 2013). The finding that MLDA only seems to have impacted alcohol-related mortality for non-college educated individuals is also consistent with our past work. It may be that the 21 drinking age confers protection against heavy drinking among non-college educated individuals, but not among those who attended college (Grucza et al., 2009). If life-long drinking habits are formed during these years, MLDA-related differences may extend into adulthood (Plunk et al., 2013).

The idea that college attendance could be associated with decreases in the effectiveness of the MLDA is also consistent with other research. For example, binge drinking has decreased in the general population, but is more common on college campuses, where the campus environment likely insulates against policies aimed at curbing underage drinking due to easy access to alcohol coupled with a culture that promotes drinking to excess (Grucza et al., 2009; Johnston et al., 2015). Other researchers have noted that underage college students report being able to obtain alcohol very easily and that legal-age drinkers are their primary source (Wagenaar et al., 1996; Wechsler et al., 2002). Further, most legal-aged college students also report frequently providing alcohol to underage peers (Brown et al., 2009). Our current findings are consistent with this proposed campus insulation effect: the MLDA seems to be less effective on college campuses, where underage drinkers have ready access to alcohol.

It is also possible that the positive impact of college education on health could be moderating the negative effects of permissive MLDA exposure on the outcomes we examined. However, regardless of the exact mechanism involved, our findings suggest that the long-term impact of lowering the drinking age as a response to risky college drinking would primarily be experienced by those without a college education.

Based on current death rates, and assuming that the effects documented above are generalizable to the current population, we estimate that the 21 drinking is preventing 900 deaths per year from alcoholic liver disease, 1,000 deaths per year from liver disease not specified as alcoholic, and 400 deaths annually from lip/oral/pharynx cancers. These effects were observed only among individuals who never attended college. Based on U.S. Census Bureau estimates, as of 2014 there were approximately 62 million adults age 25 and over in the U.S. without any form of post-secondary education (roughly 30% of the adult population). Further, while heavy drinking has declined across the population, there are still important between groups differences based on college attendance. According to Monitoring

the Future data, college student heavy drinking (5+ drinks in a row sometime in the prior two weeks) has declined nine percentage points (from 44% to 35%) from 1980 to 2014. Heavy drinking declined by 12 percentage points (from 41% to 29%) during the same period for non-college respondents (Johnston et al., 2015). These differences suggest that adolescents and young adults who do not attend college likely continue to benefit from the 21 MLDA.

Limitations and conclusion

We make several assumptions which could bias our results if violated and thus represent potential limitations of our study. First, we assume that MLDA did not change because of unobserved confounding factors that varied by state, but rather came about due to national trends. This assumption is supported by past research suggesting that MLDA laws are suitable exogenous predictors of alcohol use (Dee and Evans, 2003). We also assume that any error introduced by retrospectively estimating policy exposure was essentially random. While we do introduce error by estimating exposure, bias toward false positive associations would require that other factors related to increased risk of death from the outcomes we examined were correlated with the with the decision to move to states with permissive MLDAs. Our conditional analyses for likely non-mover and individuals who did not attend college, two sub-groups less likely to migrate between states, suggest that this is not the case. To the degree that our assumptions are reasonable, our results represent the average effect of MLDA exposure in addition to these other unmeasured factors.

Even in light of these limitations, our findings suggest that the long-term effects of permissive MLDAs—which represent increased access to alcohol at an earlier age—extend to alcohol-related chronic disease. We also offer additional evidence that increases in the MLDA significantly protected individuals who did not attend college, which would need to be addressed by those who argue that lower drinking ages are justified because of the prevalence of heavy drinking on college campuses. These analyses have focused only on diseases with a high degree of alcohol-attributable mortality. The 21 drinking age likely protects against other chronic diseases as well, and thus represents a major protective factor against alcohol-related morbidity and mortality in the United States.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

ICD Codes used to extract mortality records

Outcome	ICD-10 Codes	ICD-9 Codes
Alcoholic Liver Disease	K70	571.0-571.3
Other Liver Disease	K73-K74	571.4-571.9
Lip/Oral/Pharynx Cancers	C00-C14	140-149
Esophageal Cancer	C15	150
Laryngeal Cancer	C32	161

Table 2

Demographic characteristics of the living population and decedents from alcohol-related chronic disease

	Living Population (Weighted)		Alcoholic Liver Disease		Other Liver Disease		Lip/Oral/Pharynx Cancers		Esophageal Cancers		Laryngeal Cancer	
	N	%	N	%	N	%	N	%	N	%	N	%
Sex												
Women	50,439,012	50.5	33,153	28.6	25,146	29.9	5,695	23.7	4,374	15.1	1,641	19.5
Men	49,500,311	49.5	82,688	71.4	58,967	70.1	18,301	76.3	24,616	84.9	6,756	80.5
Race												
White	70,243,997	70.3	79,612	68.7	56,780	67.5	16,256	67.7	22,269	76.8	5,607	66.8
Black	11,849,740	11.9	13,213	11.4	12,713	15.1	5,213	21.7	4,826	16.7	2,215	26.4
Hispanic	11,762,690	11.8	17,404	15.0	11,584	13.8	1,164	4.9	1,301	4.5	462	5.5
Other	6,082,895	6.1	5,612	4.8	3,036	3.6	1,363	5.7	591	2.0	113	1.3
Age												
21-29	14,561,338	14.6	982	0.9	1,006	1.2	288	1.2	98	0.3	18	0.2
30-39	35,573,013	35.6	19,762	17.1	15,311	18.2	2,343	9.8	1,721	5.9	365	4.3
40-49	35,237,270	35.3	57,274	49.4	39,707	47.2	9,723	40.5	10,886	37.6	3,201	38.1
50-59	14,053,099	14.1	36,787	31.8	27,091	32.2	11,165	46.5	15,441	53.3	4,581	54.6
60-61	514,603	0.5	1,036	0.9	998	1.2	477	2.0	853	2.9	232	2.8
Education												
No College	48,221,541	48.3	77,620	71.7	56,999	73.5	15,415	68.4	17,600	64.2	6,242	80.2
Any College	51,717,781	51.8	30,718	28.3	20,556	26.5	7,115	31.6	9,818	35.8	1,543	19.8
Moved from Birth State												
Yes	47,395,326	47.4	51,422	44.4	35,508	42.2	10,405	43.4	11,615	40.1	3,264	38.9
No	52,543,996	52.6	64,419	55.6	48,605	57.8	13,591	56.6	17,375	59.9	5,133	61.1
Total	99,939,322	100.0	115,841	100.0	84,113	100.0	23,996	100.0	28,990	100.0	8,397	100.0

Note: Individuals born in the U.S, 1949 through 1972; observations from 1990 through 2010.

Table 3

Mortality from selected alcohol-related chronic disease as a function of under 21 MLDA exposure

Outcome	Full Sample			Men Only			Women Only		
	OR	95% CI	p	OR	95% CI	p	OR	95% CI	p
Full Sample									
Alcoholic Liver Disease	1.05	(1.01, 1.09)	0.021	1.05	(1.00, 1.10)	0.035	1.04	(0.99, 1.10)	0.132
Other Liver Disease	1.05	(1.01, 1.10)	0.034	1.05	(0.99, 1.11)	0.081	1.05	(1.00, 1.11)	0.046
Lip/Oral/Pharynx Cancer	1.05	(1.01, 1.10)	0.030	1.05	(1.00, 1.10)	0.071	1.06	(0.97, 1.15)	0.220
Esophageal Cancer	1.01	(0.96, 1.05)	0.805	1.00	(0.95, 1.05)	0.923	1.05	(0.94, 1.16)	0.408
Laryngeal Cancer	1.03	(0.95, 1.12)	0.433	1.02	(0.93, 1.12)	0.716	1.10	(0.96, 1.25)	0.160
Likely Non-Movers									
Alcoholic Liver Disease	1.06	(1.01, 1.11)	0.026	1.06	(1.00, 1.12)	0.052	1.05	(0.98, 1.13)	0.154
Other Liver Disease	1.06	(1.00, 1.12)	0.064	1.07	(0.99, 1.16)	0.084	1.02	(0.96, 1.07)	0.594
Lip/Oral/Pharynx Cancer	1.02	(0.97, 1.08)	0.436	1.02	(0.95, 1.08)	0.631	1.05	(0.93, 1.20)	0.431
Esophageal Cancer	1.02	(0.96, 1.08)	0.587	1.01	(0.93, 1.08)	0.892	1.08	(0.93, 1.25)	0.320
Laryngeal Cancer	1.10	(0.98, 1.23)	0.106	1.08	(0.95, 1.23)	0.265	1.19	(0.97, 1.48)	0.100

Note: All models include state and birth year indicators, age category, sex, race, education, year of observation, state unemployment rate, state per-capita income, citizen political ideology, state beer excise tax rate, state beer excise tax rate at the time the respondent was 21 years old, presence of state controlled liquor and wine sales at the time the respondent was 21 years old, and percent of state population affiliated with Judeo-Christian religious denominations. Likely non-movers are those who lived in the state in which they were born at time of observation.

Table 4

Mortality from selected alcohol-related chronic disease as a function of under 21 MLDA exposure, conditioned on college attendance

Outcome	No College			Any College		
	OR	95% CI	p	OR	95% CI	p
Full Sample						
Alcoholic Liver Disease	1.06	(1.02, 1.10)	0.001	1.02	(0.96, 1.07)	0.570
Other Liver Disease	1.08	(1.03, 1.13)	0.002	0.99	(0.94, 1.05)	0.822
Lip/Oral/Pharynx Cancers	1.07	(1.03, 1.12)	0.001	1.00	(0.91, 1.09)	0.946
Esophageal Cancer	0.99	(0.94, 1.04)	0.723	1.04	(0.98, 1.10)	0.239
Laryngeal Cancer	1.02	(0.94, 1.10)	0.707	1.13	(0.95, 1.34)	0.175
Likely Non-Movers						
Alcoholic Liver Disease	1.08	(1.03, 1.13)	0.003	1.01	(0.95, 1.08)	0.714
Other Liver Disease	1.08	(1.02, 1.15)	0.016	0.99	(0.92, 1.07)	0.885
Lip/Oral/Pharynx Cancers	1.04	(0.97, 1.10)	0.277	0.99	(0.88, 1.12)	0.914
Esophageal Cancer	1.03	(0.95, 1.11)	0.551	1.00	(0.93, 1.08)	0.970
Laryngeal Cancer	1.08	(0.97, 1.21)	0.171	1.22	(0.98, 1.51)	0.079

Note: All models include state and birth year indicators, age category, sex, race, year of observation, state unemployment rate, state per-capita income, citizen political ideology, state beer excise tax rate, state beer excise tax rate at the time the respondent was 21 years old, presence of state controlled liquor and wine sales at the time the respondent was 21 years old, and percent of state population affiliated with Judeo-Christian religious denominations. Likely non-movers are those who lived in the state in which they were born at time of observation.