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Alcohol policies and alcohol-attributable cancer mortality in U.S. States

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A B S T R A C T

Background: Although more restrictive alcohol control policies (e.g., higher alcohol taxes) are related to lower levels of alcohol consumption, little is known about the relationship between alcohol policies and rates of alcohol-attributable cancer.
Methods: State alcohol policy restrictiveness, as measured by a validated policy scale, were related to state rates of six alcohol attributable cancers in the U.S. from 2006 to 2010 in a lagged, cross-sectional linear regression that controlled for a variety of state-level factors. Cancer mortality rates were from the Center for Disease Control and Prevention's Alcohol-Related Disease Impact application, which uses population-attributable fraction methodology to calculate mortality from cancers of the esophagus, larynx, liver, oropharynx, prostate (male only) and breast (female only).
Results: More restrictive state alcohol policies were associated with lower cancer mortality rates for the six cancer types overall (beta [β] −0.33; 95% confidence interval [CI] −0.59, −0.07), and among men (β −0.45; 95% CI -0.81, −0.10) and women (β −0.21; 95% CI -0.40, −0.02). A 10% increase in the restrictiveness of alcohol policies (based on the mean APS among states) was associated with an 8.5% decrease in rates of combined alcohol-attributable cancers. In all analyses stratified by cancer subtype and sex, the associations were in the hypothesized direction (i.e., more restrictive state policy environments were associated with lower rates of alcohol-attributable cancers), with the exception of laryngeal cancer among women.
Conclusion: Strengthening alcohol policies is a promising prevention strategy for alcohol-related cancer.

1. Background

In North America, alcohol is a leading behavior-related risk factor for mortality [1,2] and cancer is a leading cause of death [3–6]. Although alcohol is a recognized human carcinogen, little is known about direct population-level relationships between alcohol consumption and the risk of alcohol-related cancers. Since alcohol control policies (e.g., alcohol taxes) can affect alcohol consumption, it is plausible that more restrictive policies may reduce alcohol-related cancer. However, the relationship between alcohol policies and mortality and incidence of alcohol-related cancers is unknown. Therefore, more restrictive alcohol policies constitute an overlooked but potentially impactful cancer prevention strategy.

Alcohol has emerged as a leading cancer risk factor, internationally and in the U.S [7,8]. Evidence for the relationship between alcohol and cancer comes from chemical toxicology studies, animal studies, and human epidemiological studies [9–20]. Alcohol is considered a Group I carcinogen (highest level) by the International Agency for Research on Cancer (IARC) of the World Health Organization (WHO) [21]. Based on meta-analyses of individual-level cohort studies, human cancers that are considered causally related to alcohol by IARC include cancers of the oral cavity, pharynx (throat), larynx (voice box), esophagus (squamous type only), liver, colon and rectum, and female breast. Other cancers that are associated with alcohol consumption and that are likely related to alcohol include cancers of the pancreas, prostate and stomach. In the U.S., alcohol consumption resulted in an estimated 18,200 to 21,300 cancer deaths annually, or 3.2%–3.7% of all U.S. cancer deaths [22].

While improving the diagnosis and treatment of cancer is essential, there is a compelling case for a public health approach to cancer prevention. For example, it is well-recognized that smoking is a behavior-related cancer risk factor [3,23], and that tobacco control policies may lower cancer mortality [24,25]. Furthermore, evidence among high-income nations finds that changes in alcohol consumption are related to corresponding changes in alcohol-related cancer mortality at the population level [16,22,26–28], and that reductions in alcohol consumption among individuals reduces their subsequent risk of alcohol-related aerodigestive cancers [29–32]. Multiple international cancer care and public health organizations recommend effective population-based policy strategies to reduce cancer risk [7,33–38].

In the U.S. most alcohol policies are promulgated at the state level [39], and across the 50 U S. states there is considerable policy heterogeneity [40]. This creates an opportunity to assess the relationship between state alcohol policies and cancer. Our research team developed

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and tested a series of 'Alcohol Policy Scales' to operationalize state alcohol policy environments. We found that more comprehensive policy scales had better goodness-of-fit to predict alcohol outcomes compared with models evaluating singular policies or simpler measures based on multiple policies [40,41]. More restrictive alcohol policies were associated with lower odds of binge drinking among adults, drinking and binge drinking among youth, drinking and driving, and greater likelihood of remission from alcohol dependence [42–44]. We also found that more restrictive policies were associated with reduced odds of alcohol-related crash fatalities among adults and youth, were protective of alcohol-involved homicides and suicides, and associated with lower rates of alcoholic cirrhosis mortality [45–50].

The aim of this study was to assess the relationship between the restrictiveness of the alcohol policy "environment" (i.e., that comprised of the implementation and relative efficacy of multiple existing policies) and rates of alcohol-attributable cancer mortality overall, and by cancer mortality type in the 50 states and Washington, DC. We hypothesized that states with more restrictive alcohol-policy environments would have lower alcohol-attributable cancer mortality rates.

2. Methods

2.1. Study setting and time period

This study assessed the association between alcohol policies from 2005 to 2009 and alcohol-attributed cancer mortality rates in 50 US states and Washington DC from 2006 to 2010. Policy scores and alcohol cancer mortality rates were averaged during this time period, and the association was estimated using a cross-sectional study design with a one-year lag between policies and cancer outcomes. Effects for all persons, males, and females were estimated separately.

2.2. Exposure variable: state-level alcohol policy environments

The Alcohol Policy Scale (APS) was used to characterize the alcohol policy environment in U.S. states. APS scores by state-year are derived by summing any of 29 present alcohol control policies after weighting each policy by its relative efficacy and completeness of its implementation using ratings developed in conjunction with 10 alcohol policy experts [40]. The APS score was scaled to range from 0 to 100, with higher scores indicating a more restrictive policy environment [40].

2.3. Outcome variable: age-adjusted alcohol attributable cancer death rates

The Center for Disease Control's Alcohol-Related Disease Impact Application (ARDI) data was used to calculate the age-adjusted death rates (ADR) per 100,000 population for alcohol related cancers for the 50 states and Washington, DC [1]. ARDI is an online application that provides national and state estimates of alcohol related health impacts by sex and age groups. The most recent update of ARDI included pooled mortality data from 2006 to 2010, and was used for this study.

Population-attributable fraction methodology, which utilizes relative risk estimates from published meta-analyses combined with prevalence estimates of alcohol consumption at various thresholds among the U.S. adults, were used to calculate alcohol-attributable fractions for the six cancers. These included breast cancer (female only); esophageal cancer; laryngeal cancer; liver cancer; oropharyngeal cancer; and prostate cancer (male only) [1]. Because the ARDI application reports deaths by state from 2006 to 2010, we divided the total by five to determine the average annual rate of cancer deaths, in total and by subtype, in each state. We then calculated the age-adjusted cancer mortality rate for each state and cancer type using the average of the intercensal estimate of the resident population across the five-year period 2006–2010 by age group for all persons and by sex [1]. The average annual state cancer mortality rate for each cancer from ARDI are shown in Table 1.

Table 1
Age-adjusted death rates[a] of alcohol-attributable cancer, overall and by 6 cancer subtypes, average Alcohol Policy Scale scores,[b] and distribution of state-level covariates used in adjusted models.

Cancer Mortality Outcomes[a]	Mean ± Standard Deviation
All Six Cancer Types Combined	4.39 ± 0.94
Women	2.31 ± 0.68
Men	6.52 ± 1.29
Breast Cancer	0.56 ± 0.17
Women	1.12 ± 0.32
Men	0.00 ± 0.00
Esophageal Cancer	1.45 ± 0.36
Women	0.38 ± 0.18
Men	2.53 ± 0.60
Laryngeal Cancer	0.40 ± 0.13
Women	0.09 ± 0.05
Men	0.71 ± 0.22
Liver Cancer	1.23 ± 0.33
Women	0.56 ± 0.18
Men	1.91 ± 0.53
Oropharyngeal Cancer	0.45 ± 0.10
Women	0.16 ± 0.08
Men	0.75 ± 0.19
Prostate Cancer	0.30 ± 0.06
Women	0.00 ± 0.00
Men	0.61 ± 0.12
Alcohol Policy Score[b]	42.88 ± 8.56
Covariates	
Age (median)	37.15 ± 2.23
Black (%)	11.94 ± 11.93
Hispanic (%)	9.85 ± 9.77
White (%)	79.54 ± 14.13
Income (median USD)	55,967.71 ± 8,232.65
Employed (%)	60.45 ± 3.78
Overweight (%)	36.40 ± 1.11
Smoking Tobacco (%)	19.15 ± 3.18
Protestant Religion (%)	52.37 ± 16.92
Roman Catholic Religion (%)	21.59 ± 10.16
Other religion (%)	26.04 ± 12.39
Health Insurance coverage[c] (%)	85.91 ± 3.90
Non-alcohol related cancer mortality rate[d]	156.16 ± 24.63

[a] Death rates per 100,000 population. Age-adjusted deaths were based on CDC Alcohol Related Disease Impact (ARDI) application; rates were then calculated for each state and cancer type using the average of the intercensal estimate of the resident population across the 5 years period 2006–2010 by age group for all persons and by sex.

[b] Average alcohol policy scale scores among all states from 2005 to 2009 (i.e., based on 1-year average lag between policies and cancer outcomes).

[c] State average percent with health insurance during study period.

[d] Overall unadjusted cancer mortality rate per 100,000 population, after excluding six alcohol attributable cancers.

2.4. Covariates

Because ARDI reports the average deaths of alcohol attributable cancers over the five-year period (2006–2010), we averaged all covariate data across the same five-year period and used those values in our statistical model. We controlled for population median age, median household income, religion, race/ethnicity, percent of urbanization, and health insurance coverage for each model using data from the American Community Survey (ACS) [51]. Race/ethnicity was coded as population percentage of White, Black, Hispanic and other races. Religion data was based on data from the Religious Landscape Survey [52]. We sourced data on smoking prevalence, overweight and obesity prevalence from the Behavioral Risk Factor Surveillance System (BRFSS) [53]. The mean state values of these covariates is shown in Table 1.

To control for additional state level factors that could contribute to cancer mortality more generally, we used total cancer deaths data from

the CDC Wonder United States Cancer Statistics (USCS) to calculate the cancer mortality rate by state that excluded the six alcohol-attributable cancers mortality rates assessed in this study [54].

2.5. Statistical analysis

Using linear regression models, we related average APS score from 2005 to 2009 to the average alcohol-attributable cancer mortality rate from 2006 to 2010 (i.e., using a 1-year lag between policies and outcomes). The crude and adjusted association between a 10% point increase in the mean APS score and the mean alcohol-attributable cancer mortality rate was estimated for all alcohol-related cancers, by cancer subtype, and among men and women. After assessing crude associations, covariates were entered into the model individually to identify those which modified the association by 10% or more; those covariates that modified the association were kept in the final adjusted models. The first adjusted model (Model A) controlled for all covariates described above. Model B further controlled for state-level cancer mortality rates minus the mortality rates for the six cancers assessed in the study. This was done to account for unmeasured state-level factors that may have contributed to cancer mortality more generally (i.e., apart from alcohol consumption). Microsoft Excel 2013 and SAS 9.3 software were used to conduct analyses.

We evaluated the relative percent change in alcohol-related cancer mortality rates that corresponded to a 10% point change in APS by calculating the change in alcohol-related mortality rate associated with a 10% point increase in APS (from Model B) as a percent of the baseline mortality rate calculated holding the APS score and covariates at their mean values.

3. Results

3.1. Alcohol policy scores distribution

There was considerable variability in the restrictiveness of state policy environments during the study period, with state APS scores ranging from 26.5 to 66.1. The mean ± standard deviation APS score was 42.82 ± 8.56 out of a possible score of 100 (Table 1). Scores were normally distributed.

3.2. Age-adjusted alcohol attributable cancers mortality rates

The distribution of average annual alcohol-attributable cancer mortality rates based on the ARDI application overall and by cancer type, are presented in Table 1. Esophageal (1.45 per 100,000) and liver (1.23 per 100,000) cancers had the highest alcohol-attributable mortality rates. Esophageal cancer had the highest alcohol-attributable mortality rate (2.53 per 100,000) among men, and breast cancer had the highest among women (1.21 per 100,000). There were considerable differences in alcohol-attributable cancer rates across the states, ranging from 1.52 in Utah to 7.3 in the District of Columbia (Fig. 1). Regions of the United States showed variations in age-adjusted mortality rates, with higher rates in the Upper-Midwest and New England, and lower rates in the Southeast.

3.3. Associations between alcohol policies and alcohol-attributable cancer mortality

A correlation analysis showed an inverse relationship between APS score and alcohol-attributable cancer mortality rates (Pearson $r = -0.25$, $p = 0.08$, Fig. 2). States with low policy scores and high cancer mortality included Nevada, Wisconsin, and District of Columbia. States with high scores and low mortality included Utah, Tennessee, and Oklahoma.

Crude and adjusted associations between policy scores and cancer mortality are presented in Table 2. Although effect estimates for Model A and Model B were similar, we focused on interpreting the results from Model B since this model might control for unmeasured state-level factors that might promote or protect against cancer mortality. In the fully adjusted Model B, there was a negative association of the APS policy scores (higher scores indicating more restrictive policy environments) with the rate of all alcohol-attributable cancers combined (beta [β] −0.33; 95% confidence interval [CI] −0.59, −0.07). A 10% increase in the restrictiveness of alcohol policies (based on the mean APS among states) was associated with an 8.5% decrease in rates of alcohol-attributable cancers. Results for total alcohol-attributable cancer death rates were consistent, with negative associations found for men (β −0.45; 95% CI -0.81, −0.10) and women (β −0.21; 95% CI -0.40, −0.02).

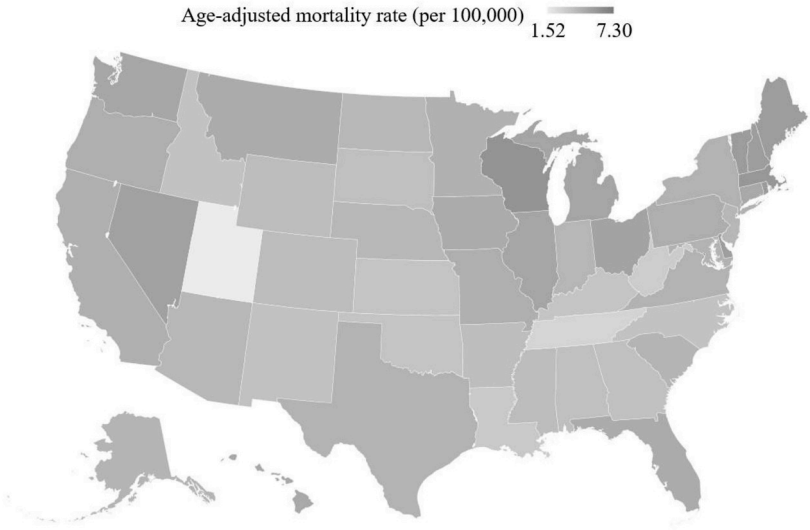
By cancer subtype, more restrictive alcohol policies were associated with reductions in liver cancer mortality for all persons (β −0.11; 95% CI -0.21, −0.01), women (β −0.06; 95% CI -0.12, −0.004) and men (β −0.17; 95% CI -0.32, −0.02) (Table 2). Reductions were also found in oropharyngeal cancer mortality rates for all persons (β −0.04; 95% CI -0.07, −0.01), women (β −0.03; 95% CI -0.05, −0.0004), and men (β −0.08; 95% CI -0.13, −0.03). Reductions were found in prostate cancer morality (β −0.05; 95% CI -0.08, −0.01). Our estimate of the association between alcohol policies and female breast cancer was also negative (β −0.08; 95% CI -0.17, 0.01). In all analyses stratified by cancer subtype and sex, the associations were in the hypothesized direction (i.e., more restrictive state policy environments were associated with lower rates of alcohol-attributable cancers), with the exception of laryngeal cancer among women.

4. Discussion

To our knowledge, this study is the first effort to examine the relationship between the restrictiveness of state alcohol polices and rates of alcohol-attributable cancer mortality in the U.S. Overall, states with more restrictive alcohol polices had lower rates of alcohol-attributable cancer mortality, including significant associations overall and for men and women for combined cancer mortality from all six cancers assessed. With the exception of laryngeal cancer among women, by individual cancer subtype all associations were in the hypothesized direction (i.e., more restrictive state policy environments were associated with lower rates of alcohol-attributable cancers), and were significant for liver and oropharyngeal cancers for both sexes combined, and for prostate cancer among men.

Because CDC estimates of alcohol-attributable cancer mortality were pooled across five years, this was a cross-sectional study. Therefore, observed associations should be considered hypothesis-generating and not of causal inference. Future studies with multiple years of state and policy data would allow more precise estimates that account for change over time in order to enable causal inference. Nonetheless, our findings are plausible given associations between alcohol policies and various measures of alcohol consumption, which may be related to population-level mortality rates for certain cancers. Furthermore, our findings were generally consistent across the various cancer subtypes, and were generally similar for men and women. Finally, the relative reduction in cancer mortality we observed based on a 10% point increase in the restrictiveness of state alcohol policies was similar in direction and magnitude to those we have observed in studies of rates and odds of binge drinking, alcoholic cirrhosis mortality, and the odds of alcohol involvement in motor vehicle crash fatalities and homicide victimization [42–50].

In additional to the cross sectional study design, this study is subject to additional caveats and limitations. While the alcohol policy scores we used to characterize state policy environments have been carefully developed, validated and used in multiple studies, it is difficult to operationalize the aggregate policy environment. To the extent that the scales are an imperfect representation of state alcohol policies, it is likely that this resulted in type II error (i.e., have obscured 'true'



Data Source: Centers for Disease Control and Prevention. Alcohol Related Disease Impact (ARDI) application. 2013. www.cdc.gov/ARDI.

Fig. 1. Distribution of Age-Adjusted Alcohol Attributable Cancer Mortality Rates by U.S States (All Persons)
Data Source: Centers for Disease Control and Prevention. Alcohol Related Disease Impact (ARDI) application. 2013. www.cdc.gov/ARDI.

associations between policies and cancer). Another limitation is that ARDI data uses six types of cancer, one of which is not considered definitely causally related to alcohol consumption by IARC (prostate cancer), and which omits another cancer type (colorectal cancer) that is considered conclusively causally related to alcohol consumption by IARC. In addition, rates of alcohol-attributable cancers in ARDI are lower than other estimates because consumption prevalence estimates from the BRFSS survey used to derive alcohol-attributable fractions are not adjusted for per capita consumption (i.e., consumption based on sales tax and shipment data), which is substantially higher than average consumption based on survey data, including BRFSS. However, this would not affect relative differences in cancer mortality rates between states, since the CDC methodology was applied uniformly across states. Despite controlling for a variety of state-level cancer risk factors including demographic characteristics, obesity and tobacco use, there may be other state-level confounding factors that could influence the rate of alcohol-related cancers for which we could not account. This was mitigated by controlling for mortality rates from all cancers other than those assessed in the study, which could control for unmeasured state-level factors that promote or protect against cancer. Finally, the use of a one-year lag between policy exposures and cancer outcomes does not preclude reverse causation as an explanation for our findings.

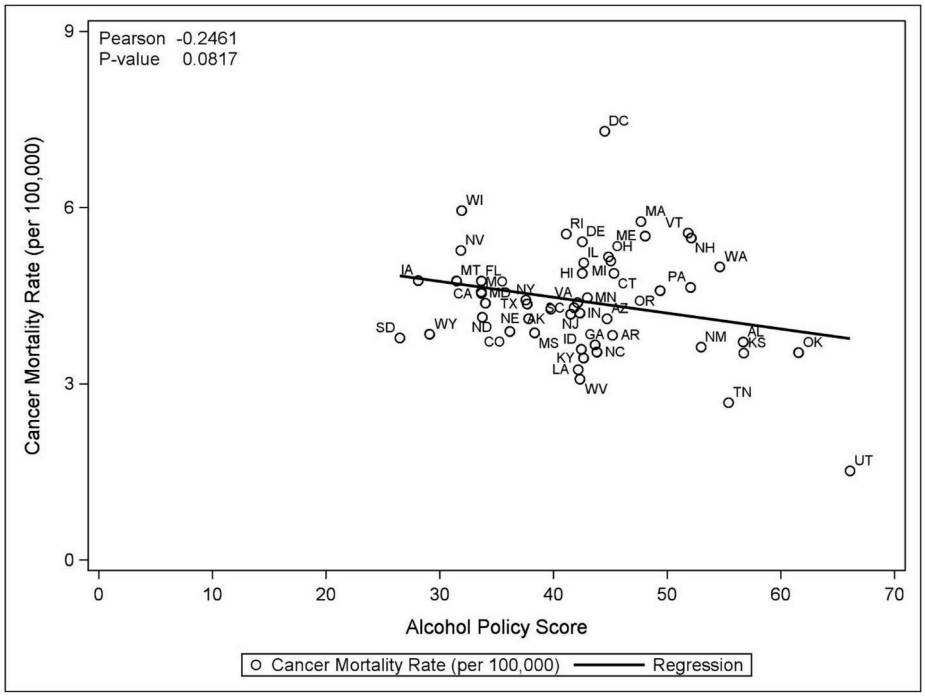


Fig. 2. Mean Alcohol Policy Scale score 2005–2009 and mean age-adjusted alcohol attributable cancer mortality rate 2006–2010, by U.S. State.

Table 2
Associations between Alcohol Policy Scale scores and age-adjusted alcohol-attributable cancer mortality rates, and relative percent change in the rate of alcohol-attributable cancer deaths based on a 10% increase in policy restrictiveness,[a] U.S. states, 2006–2010.

	Crude Association[b] β (95% CI)	Model A[b] β (95%CI)	Model B[b] β (95%CI)	Relative change in rate (%)[b]
All Alcohol-Attributable Cancers				
All Persons	−0.27 (−0.58,0.04)	−0.31 (−0.58,-0.04)	−0.33 (−0.59,-0.07)	−8.5%
Women	−0.18 (−0.40,0.04)	−0.20 (−0.40,0.002)	−0.21 (−0.40,-0.02)	
Men	−0.37 (−0.79,0.04)	−0.42 (−0.80,-0.05)	−0.45 (−0.81,-0.10)	
Esophageal Cancer				
All Persons	−0.10 (−0.22,0.02)	−0.06 (−0.16,0.04)	−0.06 (−0.16,0.04)	−4.4%
Women	−0.02 (−0.08,0.04)	−0.02 (−0.08,0.04)	−0.02 (−0.08,0.04)	
Men	−0.16 (−0.36,0.04)	−0.08 (−0.24,0.09)	−0.09 (−0.25,0.08)	
Laryngeal Cancer				
All Persons	−0.02 (−0.06,0.02)	−0.03 (−0.07,0.01)	−0.03 (−0.07,0.01)	−9.2%
Women	0.003 (−0.02,0.02)	0.004 (−0.01,0.02)	0.004 (−0.01,0.02)	
Men	−0.03 (−0.10,0.04)	−0.04 (−0.11,0.02)	−0.05 (−0.11,0.02)	
Liver Cancer				
All Persons	−0.07 (−0.18,0.05)	−0.12 (−0.23,-0.02)	−0.11 (−0.21,-0.01)	−7.7%
Women	−0.05 (−0.11,0.01)	−0.07 (−0.13,-0.01)	−0.06 (−0.12,-0.004)	
Men	−0.08 (−0.26,0.09)	−0.18 (−0.34,-0.03)	−0.17 (−0.32,-0.02)	
Oropharyngeal Cancer				
All Persons	−0.03 (−0.06,0.01)	−0.04 (−0.07,-0.01)	−0.04 (−0.07,-0.01)	−8.3%
Women	−0.03 (−0.06,-0.01)	−0.03 (−0.05,-0.0004)	−0.03 (−0.05,0.0004)	
Men	−0.04 (−0.11,0.02)	−0.08 (−0.13,-0.02)	−0.08 (−0.13,-0.03)	
Breast Cancer				
Women	−0.08 (−0.19,0.03)	−0.08 (−0.18,0.01)	−0.08 (−0.17,0.01)	−7.3%
Prostate Cancer				
Men	−0.06 (−0.10,-0.02)	−0.05 (−0.08,-0.01)	−0.05 (−0.08,-0.01)	−8.5%

[a] Mortality rates are per 100,000 population. Crude rate refers to the unadjusted association between mean state Alcohol Policy Scale scores from 2005 to 2009 in relation to mean state-level cancer mortality rates from 2006 to 2010 (i.e., using a 1 year lag between policy exposure and cancer outcomes). Model A adjusted for the following state-level covariates: median age, median household income, religious composition, race/ethnicity, percent of population living in urbanized locations, percent of the population with health insurance, smoking prevalence and obesity prevalence. Model B further controlled for state-level cancer mortality rates minus the mortality rates for the six cancers assessed in the study.

[b] Percent change calculated based on 10% point increase from mean APS score mean for model B.

However, other studies of lag times for alcohol-cancer outcomes find similar results with varying lag times ranging from 0 to 10 years [22,26,55,56].

5. Conclusion

Despite reduction in age-adjusted cancer mortality, cancer is still a leading a cause of death in the U.S. Major cancer risk factors including obesity, smoking and alcohol are targeted through individual level interventions, but can also be targeted through population-level strategies. In this lagged cross-sectional study, we found that more restrictive state alcohol policies were associated with lower rates of alcohol-attributable cancers. This suggests that strengthening alcohol control policies may be a promising cancer prevention strategy.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cbi.2019.108885.

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