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Alcohol and homicide in the United States – is the link dependent on wetness?

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Abstract

Introduction—Several aggregate-level studies have suggested that the relationship between alcohol and homicide is stronger in countries with an intoxication-oriented drinking pattern than in countries where drinking is more tempered. The present paper extends this research tradition by analysing the alcohol-homicide link in various regions in the U.S.

Design and Methods—I used annual time-series data for the U.S. states covering the period 1950-2002. Alcohol sales figures were used as proxy for alcohol consumption. Mortality data were used as indicators of homicide. The states were sorted into 3 groups labelled Dry, Moderate and Wet, where the last group has the highest prevalence of hazardous drinking according to survey data. Group-specific data were analysed using (i) ARIMA modelling and (ii) fixed-effects modelling. All modelling was based on differenced data, thus eliminating time trends and interstate correlations, both of which may bias estimates.

Results—The ARIMA estimates displayed a statistically significant gradient in alcohol effects; the effect was strongest in Wet, and weakest and insignificant in Dry states. The fixed-effects estimates showed a corresponding pattern, although the gradient was less steep and insignificant. The gradient was also weakened if the effects were expressed in absolute rather than relative terms. The spatial pattern revealed no ecological correlation between alcohol and homicide.

Conclusions—Results provided mixed support for the hypothesis that the relationship between alcohol and homicide is stronger in wet than in dry states in the U.S. Future research should probe more specific indicators of homicide as well as alcohol consumption.

Keywords

alcohol; violence; homicide; drinking patterns; United States

The present paper looks at the longitudinal association between population drinking and fatal violence, i.e., homicide, across three regions in the United States (U.S.). The paper is structured as follows. First, I outline a possible mechanism underlying the relation between alcohol and violence that, together with certain premises, comprises the theoretical basis for expecting a stronger association between alcohol consumption and homicide in intoxication-oriented drinking cultures. After a brief review of previous research on the alcohol-homicide link, the data and methods are presented. Next follow the results deriving from two different techniques for analysing aggregate time-series data. The paper concludes with a brief discussion of the findings, their limitations and implications for future research.

Conflicts of interest: none

Alcohol and Violence: Underlying Mechanisms

Several kinds of evidence suggest an association between alcohol consumption and various forms of violence, including homicide (for reviews, see Boles and Miotto [1] and Room and Rossow [2]). From the perspective of the group dynamics of drinking, one plausible mechanism underlying this relation is intoxication. If we imagine a specific drinking occasion, there are three elements that seem crucial to understanding the process of how such an occasion may escalate into violence: First, drinking is typically a social behaviour where ego's drinking affects the drinking of alter [3]. This means that if ego has been drinking to intoxication on this specific occasion, it is likely that his or her drinking companions have done so, too. Second, according to the influential theory outlined by Steele and Josephs [4], the state of intoxication tends to make individuals more short-sighted, rendering future costs less salient, while making situational cues more salient and important for people to act upon. Third, there is an elevated risk that the situational cues present in this wet drinking occasion will contain some kind of provocation from some of ego's drinking companions, simply because drunk people are more likely to engage in behaviours that are hostile and offensive [5–7].

The causal mechanism outlined above is consistent with the fact that a large proportion of the perpetrators of fatal violence [8] as well as the victims [8–10] are assessed as having been intoxicated prior to the event. Further, the scanty data that do exist [11,12] suggest that a fairly common scenario is one in which the perpetrator and the victim are acquaintances who have been drinking together prior to the fatal event.

Population Drinking and Violence: Cultural Contingencies

Because an increase in per capita alcohol consumption is associated with an increase in the frequency of drinking occasions and thereby also in the number of intoxication events, other things being equal, we should expect, according to the mechanism outlined above, a relation between alcohol and violence at the aggregate level as well. This has also been borne out in a large number of studies (for reviews, see Norström and Ramstedt [13] and Room and Rossow [2]). Studies based on U.S. aggregate data that document an association between alcohol and specifically homicide include Landberg and Norström [14], Parker [15], Parker and Cartmill [16] and Parker and Rebhun [17]. Time-series studies of other North American countries include Rossow [18], who reported a significant association for Canada during the post-war period.

However, in view of the great variation in drinking patterns across drinking cultures, it seems plausible that a given – say, 1-litre – increase in per capita consumption would yield a larger number of intoxication events in countries that have an explosive drinking pattern than in countries where drinking is more tempered. The corollary of this – that the magnitude of the relationship between population drinking and violence would vary across different drinking cultures - has also been borne out by the two comparative studies that explicitly address this issue. Thus, Lenke [19] found a stronger link between population drinking and violence in Sweden than in France. Similarly, Rossow [20], who performed time-series analyses on data for 14 European countries during the post-war period, found a gradient in the alcohol effect on homicide: It was strongest in northern Europe (with its intoxication-oriented drinking pattern), weakest in southern Europe (where the drinking pattern is more moderate), with mid-Europe falling in between. However, only one study has looked at the possible variation in the alcohol-homicide link within a country, that is, Rossow [18], who tested the hypothesis that the alcohol effect would be stronger in the Canadian provinces that have a more intoxication-oriented drinking pattern (as indicated by survey data). However, Rossow [18] points out that the differences among the provinces

with respect to drinking patterns are not very consistent, which may explain why the findings provided mixed support for the hypothesis. Another obstacle to region-specific analyses is that the populations of most countries are too small to obtain reasonably stable rates for geographical sub-units. However, the population of the U.S. should be of sufficient size to permit disaggregations of data into geographical clusters that are theoretically meaningful for analysing the contingency of the relationship between alcohol consumption and homicide.

This Study

The present study uses panel data for 48 U.S. states spanning the period 1950-2002 to test the hypothesis that the relationship between alcohol consumption and homicide is stronger in wet than in dry states. In this context, 'wet' refers to a hazardous drinking pattern, implying that the concept differs from how it is applied to western European countries. In the latter context, a wet drinking culture refers to countries with a high consumption level, but a low prevalence of hazardous drinking, with the Mediterranean countries as typical examples [21]. The methodological approach of the present study entails longitudinal analyses of time-series data, although the spatial pattern of the relationship between alcohol consumption and homicide also will be presented as a supplementary descriptive analysis.

Data

Mortality data were used as indicators of the homicide rate. Age-standardized rates for the total population 15-64 years specific to 48 U.S. states were constructed on the basis of sex-specific data in 5-year groups (source: Vital Statistics of the United States (1950-1967 [22,23]) and National Center for Health Statistics Compressed Mortality File (1968-2002 [24-26])). Alcohol sales figures were used as a proxy for alcohol consumption (expressed as litres of 100% alcohol per year and inhabitant 15 years and older). The data were obtained as industry statistics for the years 1950 to 1969 and from the Alcohol Epidemiologic Data System for the years 1970 to 2002 (for a detailed description, see Kerr et al. [27]). Two multi-state groups were created owing to the considerable cross-border trading of alcohol between New Hampshire and Massachusetts, and between the District of Columbia and Maryland and Virginia. Generally, the data spanned over the period 1950-2002, although there were missing data for alcohol consumption or homicide in the 1950s and early 1960s for Alaska, Hawaii, Mississippi and Oklahoma, making the series shorter for those states.

To test the hypothesis that the alcohol effect is contingent upon drinking patterns, the states were sorted into three regions labelled Wet, Moderate and Dry. This classification, which was made in a specific study by Kerr [28], was based on qualitative inspection of several factors, including geographic proximity and the following three measures: (i) the prevalence of heavy episodic drinking, as indicated by the percentage who had consumed 5 drinks or more in a day at least once during the past month; (ii) the prevalence of abstention; and (iii) sales of alcohol per capita in 2005. The grouping was primarily based on the 5+ measure, which (like the abstention measure) was based on survey data pertaining to 2005-2006; see Kerr [28] for a more detailed description. Dry states are thus characterized by a relatively high proportion of abstainers, lower per capita consumption and relatively low prevalence of heavy episodic drinking, while the reverse is true for states classified as wet. It should be noted that these definitions of wetness differ from those used in western Europe, where a wet drinking culture implies a high level of consumption, but where drinking is integrated into everyday life and is less likely to lead to negative outcomes. In contrast, a dry drinking culture in the European context is characterized by low level of consumption, but a high prevalence of binge drinking and ensuing problems.

The U.S. grouping scheme was validated by inspection of the between-states correlations in trends of beverage-specific alcohol sales [28]. As an additional indication of the validity of the scheme, it proved to be feasible when applied in an analysis of state panel data of the relationship between per capita alcohol consumption and ischemic heart disease mortality [29]. On the basis of the 5+ measure alone, I constructed an alternative grouping scheme which proved to differ quite little from Kerr's [28].

Statistical Analysis

The data were analysed by applying two different methodological techniques. The rationale for this is that triangulating findings from different methods should reduce the risk of obtaining method-bound results. The first was a time-series analysis of the region-specific alcohol and homicide indicators, which were obtained by computing unweighted averages of the state-specific series (SPSS 13.0 was used for this analysis). The second method was fixed effects modelling of the state-specific series, pooled into the three regions (STATA 11 was used for this analysis). A brief description of the two methods follows.

The time-series analysis was performed by applying the technique developed by Box and Jenkins [30], often referred to as ARIMA modelling (autoregressive integrated moving average). As Figure 1 shows, the time-series data are strongly trending. This requires some form of filtering (i.e., de-trending) to achieve the stationarity required for ARIMA modelling. In this case, a simple differencing was sufficient to remove non-stationary trends; that is, rather than using raw series, the yearly changes were analysed. Differencing greatly reduces the risk of obtaining spurious correlations, because an omitted variable is more likely to be correlated with the explanatory variable as a result of common trends than as a result of synchronisation in the yearly changes. Further, the noise (error) term, which includes explanatory variables not considered in the model, is allowed to have a temporal structure that is modelled and estimated in terms of autoregressive or moving average parameters. The model residuals should not differ from white noise; this is tested using the Box-Ljung Q statistics [31].

The second technique involves analyses of pooled cross-sectional time-series data. When such data are used for estimating the relationship between two variables, there are two obvious sources of bias that may distort the outcome. One is the possible presence of unobserved state differences that are linked to the dependent as well as the independent variables. The other threat to the validity of the results is the possibility that the X and Y variables have converging (or diverging) time trends that do not reflect a causal relationship, but rather the impact of other factors. In an assessment of alternative modelling techniques, Podesta [32] found that the use of differenced data to eliminate long-term trends was the most efficient device for avoiding spurious relationships due to trends. I thus chose to analyse the differenced data because the differencing not only eliminates the trends, but also means that only the intrastate co-variation over time is explored (fixed effects models), thus eliminating the first-mentioned source of bias as well. Further, the more conservative panel-corrected standard errors were used [33]. Finally, the models included panel-specific estimation of residual autocorrelation. In sum, the strongest possible model restrictions were imposed.

It is feasible to assume a multiplicative process in which the strength of the effect of alcohol consumption on homicide is conditioned by other causal factors not included in the model. Hence semi-log models were estimated, that is, with logged output series. This is also in accordance with most previous studies [14,18,20], thus facilitating comparisons. For the ARIMA analyses, the model has the following form:

 $\nabla \ln H_t = \beta^* \nabla A_t + \nabla N_t$

where *H* denotes the homicide rate, *A* is per capita alcohol consumption, and *N* is the noise term. The operator ∇ signifies that the data are differenced. The parameter to be estimated is β . The percentage increase in the homicide rate that is expected to follow from a 1-litre increase in per capita alcohol consumption is obtained by computing: $(\exp(\beta) - 1)*100$.

Results

There is an appreciable variation in homicide rates across states (Table 1); thus there is a 10fold difference between the highest rate (Mississippi = 19.3) and the lowest (North Dakota = 1.8). However, the regional pattern shows no match between homicide rates, on the one hand, and degree of wetness (Table 2) and per capita consumption (Figure 2), on the other. (The scatterplots between per capita consumption and homicide indicate zero-correlations also within the three regions of wetness; data not shown.)

The time trends (Figure 2) in per capita consumption and homicide are more suggestive of a relationship between the two, and more so when we move from Dry (Panel A) to Wet (Panel C). However suggestive, the graphical data have little evidence value, and we proceed to the model estimations, starting with the outcome of the ARIMA analyses (Table 3). These results confirm the visual impression of Figure 2. All three estimates of the relationship between alcohol consumption and homicide are positive, but of varying strengths and statistical significance. In Dry the relationship is weak and insignificant, while it is fairly strong and highly significant in Wet. The middle region falls in between with respect to strength of the relationship and significance (borderline). No noise parameters were required in any of the estimated models, and the tests for residual correlation were satisfactory. The F-test indicates that the estimates are statistically different from each other. The outcome of the fixed effects modelling (Table 4) goes in the same direction as that of the ARIMA analyses, although the estimated relationships are generally weaker, and not statistically different from each other, according to the F-test. The estimates pooled across the three groups of states are strongly significant, and indicate that a 1-litre increase in consumption is associated with an increase in homicides of between 6% (fixed effects estimate) and 10% (ARIMA estimate).

The analyses reported in Tables 3-4 were replicated using the alternative grouping scheme that was based on the 5+ drinks measure alone. It appeared that this scheme did not yield larger region-specific differences in estimated alcohol effects than did the original grouping scheme.

Discussion

Previous comparative research on the association between alcohol consumption and homicide has revealed marked country differences in the magnitude of that association. The present study extends the comparative research tradition by exploring whether the association at issue is contingent on regional differences in drinking patterns *within* a country. This was accomplished by estimating the alcohol effect on homicide for three groups of U.S. states labelled as Dry, Moderate and Wet, where the last is assumed to represent the most hazardous drinking pattern (recall that this wet/dry classification differs from the one usually applied to western European countries). The findings from the groupspecific ARIMA analyses revealed a statistically significant gradient in the estimated alcohol effects in the expected direction; that is, the effect was stronger in Wet than in Dry. A corresponding — albeit not statistically significant — gradient was suggested by the fixed

effects models. It is further noted that the outcome of the longitudinal analyses was not echoed by the spatial pattern; there was thus no tendency for states with high per capita consumption to also have a high homicide rate. One plausible explanation for this is that any positive ecological association between homicide and alcohol is concealed by the latter factor's correlation with other determinants of homicide. This suggests that the choice of fixed effects models, i.e., discarding the spatial co-variation, seems appropriate.

The finding of a significant over-all (pooled) effect of alcohol on homicide accords with previous studies based on U.S. aggregate data [15-17]. More detailed comparisons with other findings are facilitated if we first average the estimates from the ARIMA and fixed effects modelling. These averages — 3% per litre for Dry, 6% for Moderate, and 14% for Wet— are fairly close to what Rossow [20] found for southern, mid- and northern Europe (6%, 9% and 13% per litre). The estimates reported above are also within the range of estimates reported in previous studies focusing on North America, including the estimate that Landberg and Norström [14] obtained for the United States as a whole (10%), and that Rossow [18] obtained for Canada as a whole (6%), as well as for the province of Ontario (10%).

Study limitations

Before concluding, some of the study's limitations should be pointed out. First, sales data are imperfect measures of actual alcohol intake due to, e.g., unrecorded consumption and crossborder trading. This may imply that the effect of alcohol consumption has been underestimated, as the presence of measurement errors in the independent variable yields a downward bias in estimates of the variable's effect on an outcome [34]. Second, the grouping according to drinking patterns is mainly based on survey data, with all its wellknown limitations. Further, these data refer to the end of the study period. Although drinking patterns may be assumed to be a fairly stable trait of a drinking culture, and the current categorization accords fairly well with those pertaining to earlier parts of the study period [35,36], it seems likely that some changes have occurred during this long period. These imperfections in the categorization scheme probably reduce the likelihood that we can demonstrate that the association between alcohol and homicide is contingent on drinking patterns. Third, with regard to the differences in estimated alcohol effects, one should be aware of the substantial differences in homicide rates between the three groups of states, the rate being 2.2 times higher in Dry than in Wet. This implies that if we express the change in the homicide rate per litre alcohol in absolute rather than relative terms, the gradient in the alcohol effect is greatly reduced. If we look at the ARIMA estimates (Table 2), there is a 5fold difference between Wet and Dry in the relative effects; this is lowered to a 2-fold difference when expressed in absolute effects. The less steep (and statistically insignificant) gradient in the fixed effects estimates (Table 3) disappears when effects are expressed in absolute terms.

Conclusions

In conclusion, the present findings revealed a statistically significant relationship between changes in per capita alcohol consumption and homicide during the postwar period in the United States. The findings further suggest that the strength of this relationship is contingent upon drinking patterns, such that it is stronger in wet states characterized by a higher prevalence of hazardous drinking. However, the latter conclusion should be regarded with great caution, as the pattern of estimated alcohol effects across groups of states with different degrees of wetness is sensitive to modelling technique and model specification.

In future research, it may be worthwhile to include more specific measures of the output, as well as the input, variables. It is thus well documented that homicide trends vary

substantially between population groups defined by age, gender and ethnicity. Similarly, previous research [16] suggests the existence of beverage-specific effects in the present context.

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References

- Boles SM, Miotto K. Substance abuse and violence. A review of the literature. Aggress Violent Beh. 2003; 8:155–74.
- 2. Room R, Rossow I. The share of violence attributable to drinking. J Subst Use. 2001; 6:218–28.
- 3. Skog OJ. The collectivity of drinking cultures: A theory of the distribution of alcohol consumption. Brit J Addict. 1985; 80:83–99. [PubMed: 3856453]
- Steele CM, Josephs RA. Alcohol myopia: Its prized and dangerous effects. Am Psychol. 1990; 45:921–33. [PubMed: 2221564]
- 5. Bushman BJ, Cooper HM. Effects of alcohol on human aggression: an integrative research review. Psychol Bull. 1990; 107:341–54. [PubMed: 2140902]
- Taylor SP, Chermack ST. Alcohol, drugs and human physical aggression. J Stud Alcohol. 1993; 11:78–88.
- Exum ML. Alcohol and aggression: an integration of findings from experimental studies. J Crim Justice. 2006; 34:131–45.
- Darke S. The toxicology of homicide offenders and victims: A review. Drug Alcohol Rev. 2010; 29:202–215. [PubMed: 20447230]
- Kuhns JB, Wilson DB, Clodfelter TA, Maguire ER, Ainsworth SA. A meta-analysis of alcohol toxicology study findings among homicide victims. Addiction. 2010 no. 10.1111/j. 1360-0443.2010.03153.x
- Smith G, Branings KC, Miller T. Fatal non-traffic injuries involving alcohol: A meta-analysis. Ann Emer Med. 1999; 33:699–702.
- Lindqvist P. Homicides committed by abusers of alcohol and illicit drugs. Br J Addiction. 1991; 86:321–26.
- Rying, M. Licentiat thesis. Department of Criminology, Stockholm University; 2000. Dödligt våld i Sverige 1990-1998. En deskriptiv studie (Fatal violence in Sweden. A descriptive study, in Swedish).
- 13. Norström T, Ramstedt M. Mortality and population drinking: a review of the literature. Drug Alcohol Rev. 2005; 24:537–47. [PubMed: 16361210]
- 14. Landberg, J.; Norström, T. Alcohol and homicide in Russia and the USA–a comparative analysis. Paper presented at the 35th Annual Alcohol Epidemiology Symposium of the Kettil Bruun Society for Social and Epidemiological Research on Alcohol; Copenhagen. 1-5 June, 2009;
- 15. Parker RN. Bringing "booze" back in: the relationship between alcohol and homicide. J Res Crime & Del. 1995; 32:3–38.
- Parker NA, Cartmill RS. Alcohol and homicide in the United States 1934-1995–or one reason why U.S. rates of violence may be going down. J Crim Law Crim. 1998; 88(4):1369–98.
- Parker, NA.; Rebhun, MA. Alcohol and Homicide: a deadly combination of two American traditions. Albany: State University of New York Press; 1995.
- Rossow I. Alcohol consumption and homicides in Canada, 1950-1999. Contemp Drug Probl. 2004; 31:541–59.
- Lenke, L. Alcohol and criminal violence: time series analysis in a comparative perspective. Stockholm: Almqvist and Wiksell International; 1989.

- 20. Rossow I. Alcohol and homicide: a cross-cultural comparison of the relationship in 14 European countries. Addiction. 2001; 96(Suppl.1):77–92.
- 21. Room R, Mäkelä K. Typologies of the Cultural Position of Drinking. J Stud Alcohol. 2000; 61:475–83. [PubMed: 10807222]
- National Office of Vital Statistics. Vital Statistics of the United States, 1950–1958. Washington, DC: U.S. Public Health Service; 1954–1960.
- 23. National Center for Health Statistics. Vital Statistics of the United States. 1961–1969.
- 24. National Center for Health Statistics. Compressed Mortality File 1968-1988. Hyattsville, MD: National Center for Health Statistics; 2000.
- 25. Compressed Mortality File 1989-1998. National Center for Health Statistics; Hyattsville, MD: National Center for Health Statistics; 2003.
- 26. Compressed Mortality File 1999-2002. National Center for Health Statistics; Hyattsville, MD: National Center for Health Statistics; 2004.
- Kerr WC, Greenfield TK, Tujague J. Estimates of the mean alcohol concentration of the spirits, wine and beer sold in the U.S. and per capita consumption: 1950 to 2002. Alcohol Clin Exp Res. 2006; 30:1583–91. [PubMed: 16930221]
- Kerr WC. Categorizing US state drinking practices and consumption trends. Int J Environ Res Public Health. 2010; 7:269–83. [PubMed: 20195444]
- 29. Kerr WC, Karriker-Jaffe K, Subbaraman M, Ye Y. Per capita alcohol consumption and ischemic heart disease mortality in a panel of US states from 1950 to 2002. Addiction. 2010 no. 10.1111/j. 1360-0443.2010.03195.x
- Box, GEP.; Jenkins, GM. Time-Series Analysis: forecasting and control. London: Holdens Day, Inc.; 1976.
- 31. Ljung GM, Box GEP. On a measure of lack of fit in time-series models. Biometrika. 1978; 65:297–303.
- 32. Podesta F. Comparing time series cross-section model specifications: the case of welfare state development. Qual Quant. 2006; 40:539–59.
- 33. Beck N, Katz JN. What to do (and not to do) with time-series cross-section data. Am Polit Sci Rev. 1995; 89:634–47.
- 34. Greene, WH. Econometric Analysis. 5th. New Jersey: Prentice Hall; 2002.
- 35. Room, R. Region and urbanization as factors in drinking practices and problems. In: Kissin, B.; Begleiter, H., editors. The Pathogenesis of Alcoholism: Psychosocial factors. Vol. 6. New York: Plenum Press; 1983. p. 555-604.
- Hilton ME. Regional diversity in United States drinking practices. Br J Addict. 1988; 83:519–32. [PubMed: 3382810]

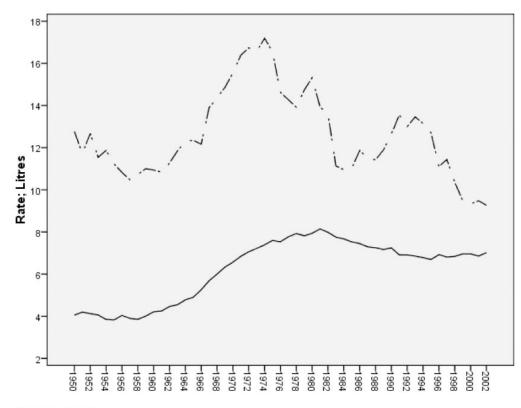


Figure 1, Panel A.

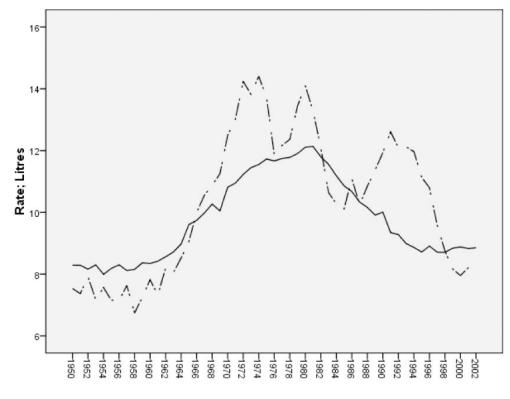


Figure 1, Panel B.

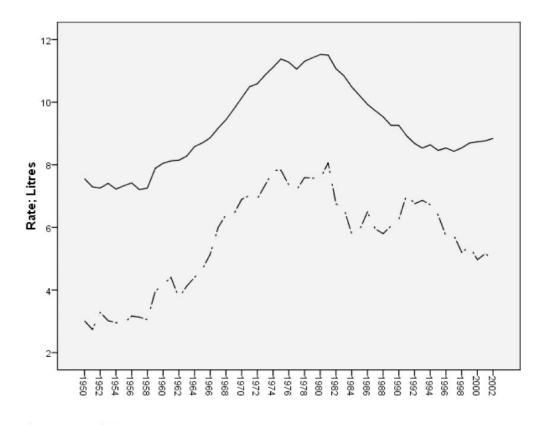


Figure 1, Panel C.

Figure 1.

Trends in per capita alcohol consumption (litres per capita 15+, solid line) and homicide rates (per 100 000 15-64 years of age, broken line) in three wetness regions. A=Dry, B=Moderate, C=Wet.

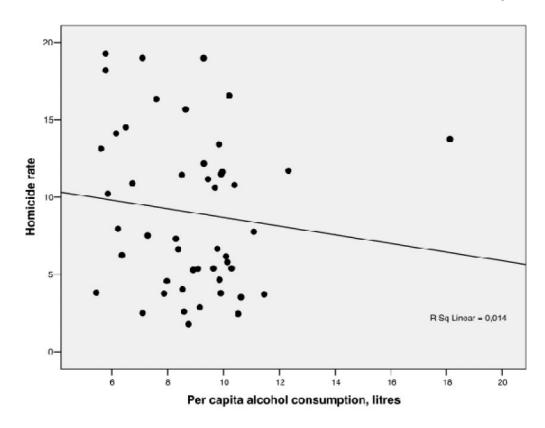
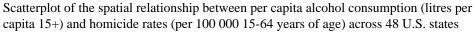


Figure 2.



alcohol per capita 15+). Percentage who at least once had consumed 5 drinks (based on survey data 2005-2006), abstention rate (%; based on survey data Period means (1950-2002) for homicide rate (number of homicides per 100 000 15-64 years of age) and per capita alcohol consumption (litres of 100% 2005-2006), and scores on Wetness (Dry=1, Moderate=2, Wet=3).

State	Homicide	Alcohol	5+	Abstention	Wetness
Alabama	18.20	5.78	18.74	57.55	1
Alaska	11.70	12.33	21.58	47.26	ю
Arizona	11.63	9.96	22.87	45.63	2
Arkansas	13.13	5.61	20.71	57.39	1
California	10.78	10.40	20.81	49.67	2
Colorado	6.66	9.78	24.68	41.41	ю
Connecticut	4.66	9.86	25.14	39.23	2
Delaware	7.77	11.09	21.29	48.49	7
DC/Maryland/Virginia	13.41	9.84	23.44	44.92	2
Florida	16.56	10.21	22.34	46.57	2
Georgia	18.99	7.10	19.65	55.15	1
Hawaii	5.40	10.29	21.41	53.30	2
Idaho	4.57	7.97	21.89	53.48	2
Illinois	11.49	9.92	25.46	47.53	ю
Indiana	7.52	7.29	21.10	50.60	1
Iowa	2.50	7.11	27.14	46.91	ю
Kansas	6.25	6.36	25.43	46.81	ю
Kentucky	10.89	6.74	21.79	57.53	1
Louisiana	18.98	9.29	23.53	50.93	5
Maine	2.60	8.59	22.43	45.24	ю
Mass/New Hampshire	3.54	10.62	24.42	40.68	3
Michigan	11.16	9.45	25.37	43.80	ю
Minnesota	2.88	9.15	27.86	38.26	ю
Mississippi	19.28	5.78	18.40	63.07	1
Missouri	11.43	8.51	23.86	50.09	ю
Montana	5.80	10.14	28.57	43.29	ю

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State	Homicide	Alcohol	5+	Abstention	Wetness
Nebraska	4.04	8.53	26.69	45.97	3
Nevada	13.74	18.12	23.75	48.00	2
New Jersey	6.17	10.10	22.29	43.53	2
New Mexico	12.17	9.30	20.30	54.17	2
New York	10.62	9.70	23.47	45.25	2
North Carolina	14.51	6.50	19.46	56.42	1
North Dakota	1.79	8.75	30.32	41.76	3
Ohio	7.31	8.30	24.45	48.43	3
Oklahoma	10.22	5.86	21.14	58.15	1
Oregon	5.29	8.92	21.61	45.33	2
Pennsylvania	6.63	8.38	23.09	47.70	2
Rhode Island	3.79	9.91	27.12	38.77	3
South Carolina	16.34	7.60	20.88	54.74	2
South Dakota	3.77	7.87	28.14	41.30	ю
Tennessee	14.12	6.16	20.52	57.59	1
Texas	15.68	8.64	24.06	50.51	2
Utah	3.83	5.44	17.38	67.60	1
Vermont	2.46	10.53	25.99	39.58	3
Washington	5.37	9.09	22.80	45.55	2
West Virginia	7.95	6.22	18.90	63.60	1
Wisconsin	3.72	11.46	29.41	36.86	3
Wyoming	5.39	9.64	25.21	43.64	3

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Period means (1950–2002) for homicide rate (number of homicides per 100 000 15–64 years of age), and per capita alcohol consumption (litres of 100% alcohol per capita 15+). Percentage who had consumed 5 drinks or more in a day at least once during the past month (based on survey data 2005-2006), abstention rate (%; based on survey data 2005-2006) in three groups of U.S. states representing different wetness regions.

		H	Homicide	A	Alcohol		5+	Ab	Abstention
Wetness	Z	Mean	(range)	Mean	(range)	Mean	Wetness N Mean (range) Mean (range) Mean (range) Mean (range)	Mean	(range)
Dry	=	12.60	3.83-19.28	6.23	5.44-7.29	19.80	11 12.60 3.83–19.28 6.23 5.44–7.29 19.80 17.38-21.79 58.61 50.60-67.60	58.61	50.60-67.60
Moderate	18	10.32	4.57–18.98	9.93	7.60–18.12	22.50	7.60–18.12 22.50 20.30-25.14 48.17	48.17	39.23-54.74
Wet	19	5.70	19 5.70 1.79–11.70 9.31	9.31	6.36–12.33	26.01	6.36–12.33 26.01 21.58-30.32 43.56 36.86-50.09	43.56	36.86-50.09
Total	48	9.01	1.79-19.28	8.84	5.44-18.12	23.27	48 9.01 1.79–19.28 8.84 5.44–18.12 23.27 17.38-30.32 48.73 36.86-67.60	48.73	36.86-67.60

Estimated effects of per capita alcohol consumption on homicide rates. ARIMA-models (semi-log) estimated on U.S. regional data for the period 1950–2002.

Wetness	Est	SE	р	Q*
Dry	0.035	0.047	0.465	3.21; p > 0.61
Moderate	0.071	0.037	0.063	11.08; p > 0.05
Wet	0.174	0.045	< 0.001	7.58; p > 0.18
Pooled est.	0.093	0.025	< 0.001	
F-test for heterogeneity	2.056		0.02	

* Box-Ljung test for autocorrelated residuals

Estimated effects of per capita alcohol consumption on homicide rates. Fixed effect models (semi-log) estimated on U.S. state data for the period 1950–2002.

Wetness	Est	SE	р
Dry	0.029	0.024	0.210
Moderate	0.048	0.019	0.015
Wet	0.085	0.027	0.002
Pooled est.	0.054	0.014	< 0.001
F-test for heterogeneity	2.056		0.13