



Published in final edited form as:

Suicide Life Threat Behav. 2009 August ; 39(4): 452–459. doi:10.1521/suli.2009.39.4.452.

PER CAPITA ALCOHOL CONSUMPTION AND SUICIDE RATES IN THE US 1950-2002

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Abstract

The aim of this paper was to estimate how suicide rates in the United States are affected by changes in per capita consumption during the post-war period. The analysis included Annual suicide rates and per capita alcohol consumption data (total and beverage specific) for the period 1950-2002. Gender- and age-specific models were estimated using the Box-Jenkins technique for time series analysis. No significant estimate was found for males. For females the total alcohol estimate (0.059) was significant at the 10% level whereas the spirits estimate was significant with an effect of 0.152. The results imply that a change in US per capita consumption would result in a change in female suicide rates, whereas the male rates not would be affected.

Introduction

A large proportion of suicides in the United States are related to alcohol use. For example, a study by the Centers for Disease Control and Prevention (CDC) estimated that the number of alcohol-attributable deaths among the 30,622 suicides that occurred in 2001 amounted to 6,995 (23%). Further, a meta-analysis of individual level studies by Smith et al. (1999) estimated the mean proportion of Blood Alcohol Concentration (BAC) positive suicide victims to be around 29%, i.e., somewhat lower than the average of 37% that was estimated in a review of studies of acute alcohol use and suicidal behaviour by Cherpitel, Borges and Wilcox (2004).

These findings are also echoed at the aggregate level. Thus, Cases and Harford (1998), on the basis of time-series data for the period 1934 to 1987, found a statistically significant relationship between per capita alcohol consumption and total as well as gender specific suicide rates. The present paper can be seen as a continuation and refinement of the study by Cases and Harford, in that I test whether their results can be replicated using data for the post-war period (1950 to 2002), during which we witnessed less dramatic shifts in per capita consumption and suicide rates. In addition, I use more specific indicators of consumption (besides total consumption, measures of beverage-specific alcohol consumption are included) as well as of suicide mortality rates broken down by sex and different age-groups.

It is generally assumed that the alcohol-suicide relationship can be explained by two underlying causal mechanisms. The first mechanism, acute intoxication, is assumed to increase an individual's suicide risk by fostering psychological distress, impulsive behaviour, reducing self-control and triggering suicidal tendencies caused by other factors. The second mechanism concerns the more long-term effects of chronic abuse on suicidogenic factors such as mental problems and social disintegration (Skog, 1991). It should be noted that these two mechanisms may be placed within the conceptual framework of proximal- (acute effects of alcohol) and distal- (alcohol dependence) risk factors for suicidal behaviour, developed by Hufford (2001). Because changes in per capita consumption tend to reflect changes in both the frequency of intoxication occasions and the prevalence of heavy drinking in a population, the mechanisms

of acute intoxication and chronic abuse can be expected to operate on the aggregate level as well, and we should expect a positive relationship between changes in per capita alcohol consumption and suicide rates (Skog, 1985; 1991).

Most of the population level research on alcohol and suicide has been conducted on western European time-series data for the post-war period (e.g., Norström, 1995; Norström & Rossow, 1999; Mäkelä, 1996; Ramstedt, 2001; for a review, see Norström & Ramstedt, 2005). Taken together, these studies suggest a European north-south gradient in the alcohol effect on suicide mortality (Norström & Ramstedt, 2005), i.e. changes in population drinking tend to have a stronger effect on suicide mortality in northern Europe than in central and southern Europe (Norström, 1995; Norström & Rossow, 1999; Ramstedt, 2001). This finding is assumed to indicate the importance of drinking patterns, i.e. that the intoxication-oriented drinking patterns of northern Europe are more harmful also in social terms than are the more everyday, integrated drinking patterns of central and southern Europe. Some exceptions to this pattern should be noted, for example those found in the UK and Ireland, two countries with rather intoxication-oriented drinking patterns for which no significant population level association has been found (Ramstedt, 2001). Turning to studies outside Europe, Ramstedt (2005) reported a significant relationship between alcohol and suicide for Canada for the post-war period, as did Mann et al. (2006) for Ontario. The study by Cases and Harford (1998), using US data for the period 1934 to 1987, estimated that a 1-litre increase in per capita consumption was associated with an increase in male suicide rates of around 3% (after controlling for unemployment). Further, studies that have estimated beverage-specific effects on suicide have mostly found spirits to be the beverage most closely related to suicide (Norström & Rossow, 1999; Razvodovsky, 2001; Gruenewald et al., 1995).

By focusing on the US post-war period, I can place the US experience of alcohol and suicide in an international perspective, in addition to checking whether the previously reported findings of Cases and Harford (1998) can be replicated. In one international comparative study of drinking patterns (Rehm et al., 2004), the US scored 2 on a 1 to 4 scale (where 1 designates the least hazardous patterns and 4 the most hazardous patterns). Because this is the same score Canada obtained, we should expect an alcohol effect for the US that is on a par with that of Canada. Further, because males generally show higher consumption and more often drink in binges than females do, the alcohol effect is expected to be stronger for men (although previous findings are mixed in this respect).

Data & Method

Age-standardised suicide mortality rates (number of suicides per 100 000 population 15 years and above) were constructed on the basis of mortality data (5-years groups) for men and women. The data were collected from Vital Statistics of the United States: 1950 to 1967 and National Center for Health Statistics Compressed Mortality File: 1968 to 2002. These sources report deaths according to underlying cause, rather than using multiple cause coding, so that each death is attributed to only one cause. The suicide mortality data corresponded to the detailed list of ICD codes: E950-E959 in ICD-7, ICD -8 and ICD-9 and X60-X84, Y87.0 in ICD-10.

Annual figures on alcohol consumption (expressed as litres of 100% alcohol per year and inhabitant 15 years and older) were based on beer, wine, and spirits sales figures from the Alcohol Epidemiologic Data System for the years 1970 to 2002 (Nephew, 2004) and US beer, wine and spirits industry statistics for the years 1950 to 1969. The percentage alcohol by volume for each beverage type and year was estimated using data from a variety of industry and government alcohol monopoly sources (see Kerr et al. 2006). These estimates utilized data on the %ABV (mean alcohol content by volume) of specific brands and their market share in the

US to estimate the mean %ABV of beer, wine and spirits sub-types such as light beer, or whiskey for each year. The market shares of these sub-types were then used to estimate the mean %ABV for each beverage in each year. These mean %ABV were then applied to alcohol sales figures to calculate yearly estimates of per capita consumption of beer, wine, spirits and total alcohol consumption.

The data were analysed using the technique for time-series analysis developed by Box and Jenkins (1976), often referred to as ARIMA modelling. The presence of strong time trends (see Figures 1) makes a filtering necessary to achieve the stationarity¹ required for the ARIMA-modelling. A simple differencing was sufficient to remove non-stationary trends; that is, rather than using raw series the yearly changes were analysed. This procedure has the advantage of reducing the risk of obtaining a spurious relationship, because an omitted variable is more likely to be correlated with alcohol consumption due to common trends than due to synchronization of the yearly changes (Skog, 1988). Further, the technique allows the noise term (which consists of measurement errors and casual factors not included in the model) to have a temporal structure that is modelled and estimated in terms of autoregressive or moving average parameters. Dummy variables were used to control for shifts between ICD versions: 1958, 1968, 1979 and 1999 (coded as zero before the change and one after the change), but were only kept in the models if they were significant. Previous research has suggested that divorce (Rossow, 1993) and unemployment (Gruenewald et al., 1995) may be related to suicide. Annual divorce rates (source: US Census Bureau) and unemployment (measured as the percentage of the labour force who are seeking work, source: US Bureau of Labor Statistics) were thus included as control variables in all models, but were only kept if they were significant.

In line with previous studies (Norström, 1995; Ramstedt, 2001), semi-logarithmic models were used. The model can be written as follows:

$$\nabla \ln S_t = a + b \cdot \nabla A_t + c \cdot \nabla D_{it} + \ln N_t$$

Where ∇ refers to the differencing operator, S is suicide, a is a constant (which marks average annual changes in S due to other causes), A is alcohol consumption, b is the effect parameter to be estimated. The percentage effect of a 1-litre change in consumption is obtained by applying the formula: $(\exp(b)-1) \cdot 100$. The parameter c is the potential effect on S due to changes in ICD versions, modelled by a set of dummy variables (D). Finally, N refers to the noise term in which other casual factors are included. The Box-Ljung statistic was used to test for residual autocorrelation.

Gender-specific models were estimated for both total alcohol consumption and beverage specific indicators. In addition age-specific models were estimated for men and women in the following age groups 15-34, 35-54, 55-74 and 74+ (using the total alcohol indicator only).

Results

Trends and average levels in per capita alcohol consumption and suicide rates

Trends in per capita alcohol consumption and male and female suicide rates are presented in Figure 1. The per capita alcohol consumption increased up till the early 1980s, peaked in 1981 and then decreased and leveled off for the remainder of the period. The male suicide rates have been rather stable over the period (except a decline during the 1990's). Female suicide rates, on the other hand, increased up to the mid-1970s, after which they decreased and levelled off

¹Stationarity is a statistical term which refers to “a condition of mean, variance and covariance equilibrium” in a time-series (Yaffe & McGee, 2000)

from the mid-1990s onwards, and thus appear to have developed more similarly to the trend in alcohol consumption.

Estimation of time series models

Results of the gender-specific time-series models are presented in Table 1. As can be seen, neither total alcohol consumption nor any of the beverage specific indicators had any statistically significant effect on male suicide rates. Unemployment proved to be significant (with the expected positive effect) in the total alcohol model for males. For females the estimated effect of total alcohol consumption was significant at the 10%-level only. The estimate of 0.059 implies that a 1-litre² increase in overall per capita consumption is associated with an increase in female suicide rates of around 6%. Of the beverage specific indicators, only spirits had a statistically significant effect on female suicide rates, with an estimate of 0.15, this means that a 1-litre increase in the per capita spirits consumption on average was followed by an increase of female suicide mortality rates of 16%. None of the control variables showed significant estimates for females.

The estimates of the age specific models for men and women are reproduced in Table 2. No significant estimates were obtained for any of the male age-group. For females the estimates were generally positive (except for the 74+ age group) and ranging from 0.062 to 0.086, thus implying an increase in female suicide rates in the different age-groups of between 6.5% and 9% following a 1-litre increase in per capita consumption, however the only significant estimate was found in the 55-74 age-group.

Discussion

This study has addressed the population level association between alcohol and suicide in the US. By means of time-series analyses it has been estimated to what extent suicide rates are affected by changes in the per capita consumption. The analyses extended beyond previous research by utilizing data for the post-war period and using more detailed indicators of alcohol consumption as well as suicide rates.

The results did not support the hypothesis of an aggregate alcohol - suicide relationship among US males. In fact, the estimates for males were non significant for all beverages and in all age-groups. On the other hand, the findings did suggest a positive relationship between changes in per capita consumption (in particular spirits consumption) and female suicide mortality rates. According to the estimations, female suicide rates increase by approximately 6% if overall per capita consumption increases by 1 litre; the corresponding figure for a 1-litre change in the per capita spirits consumption was 16%. Further, the age-specific analyses suggested that the alcohol effect is fairly evenly distributed over the different age groups for females (except the oldest).

Given that, in the US, females drink less and are less prone to binge drinking than men are (Substance Abuse and Mental Health Services Administration, 2003), the finding that changes in per capita consumption only affect female suicide rates in the US is rather unexpected. The observed gender difference in the alcohol effect on suicide accords, however, with results of Ramstedt's (2005) and Mann et al.'s (2006) studies of Canadian data. They, too, found stronger alcohol effects on female suicide rates whereas the effects among males tended to be weaker and/or non significant. Further, a recent meta-analysis by Wilcox et al. (2004) showed that the Standardized Mortality Ratio for suicide among subjects with alcohol dependence syndrome was higher for women (1690) than for men (438), i.e. approximately 17 and 4.4 times higher,

²1 litre (metric measure) equals approximately 0.26 US gallons.

respectively, than expected in a standard population. This suggests that the association between alcoholism and suicide may be more pronounced among women than among men. One possible explanation for this, which also has been brought up by Ramstedt (2005), is gender differences in stigmatization of alcohol abuse, i.e., that stereotypical masculine behaviour such as e.g. alcohol abuse and aggression are more socially disapproved of in women and that females engaging in high levels of such behaviour may be more stigmatized and thus of higher suicide risk compared to men that engage in the same kind of behaviour. This idea finds support in a study by Conner et al. (2001) which found that violent behaviour was a significant predictor of suicide and more so for women than for men. It does also seem reasonable to assume, however, that some of the alcohol effect on female suicide rates is due to the social and psychological harm brought on by the heavy drinking of male partners.

If we consider the magnitude of the alcohol effect on US female suicide rates, the estimate (around 6%) was stronger than the effects previously estimated for southern and central Europe and somewhat weaker than the effects for Canada and Northern Europe (Ramstedt, 2001, 2005). The insignificant alcohol effect on male suicides echoes the results reported by Ramstedt (2001) for southern and mid-Europe, and by Gmel et al. (1998) for Switzerland. However, it is at variance with the results of Cases and Harford (1998) mentioned above, that is, the significant estimate based on US data for an earlier period (1934 -1987). This suggests that something took place during the present observation period (1950-2002) that affected the aggregate alcohol-suicide link for males, causing it to either disappear or, more likely, to become too weak to emerge in these kinds of aggregate analyses. It seems doubtful that the individual level relationship would have disappeared; recent individual level studies do suggest that excessive alcohol use is related with increased suicide risk among US men (see e.g., Kung et al., 2003). Moreover, a recent study by Ramstedt (2008), utilizing the same technique and time-period as the present study, found a significant association between per capita consumption and US male accident mortality rates, which indicates that the prevalence of intoxication occasions and heavy drinking still can be expected to reflect changes in the total consumption. One possible explanation could be that the present observation period has seen the emergence of some confounding factor (or several factors) which tends to mask the link between alcohol and suicide. One such factor could be increased/improved alcoholism treatment, which has been analysed and discussed by Holder and Parker (1992) in relation to changes in per capita consumption and liver cirrhosis mortality rates and by Mann et al. (2006) in relation to per capita consumption and suicide rates in Ontario. Another factor which is often brought up in discussions on recent declines in suicide rates, is the growing use of antidepressants (see e.g. McKeown et al, 2006 and Grunebaum et al, 2004). It is difficult, however, to explain how any of these factors only would have affected the aggregate alcohol-suicide association among men and not among women.

Moreover, it is possible, that in the US, the cross-sectional differences between states regarding drinking patterns and drinking levels as well as suicide mortality rates confound the national level association for males and that a significant aggregate alcohol-suicide link would emerge if the states were analysed separately (or in groups). Therefore, a recommendation for further research would be to analyse the association at the state-level.

Acknowledgments

This research was supported by research grant R01 AA014362 from the National Institute on Alcohol Abuse and Alcoholism.

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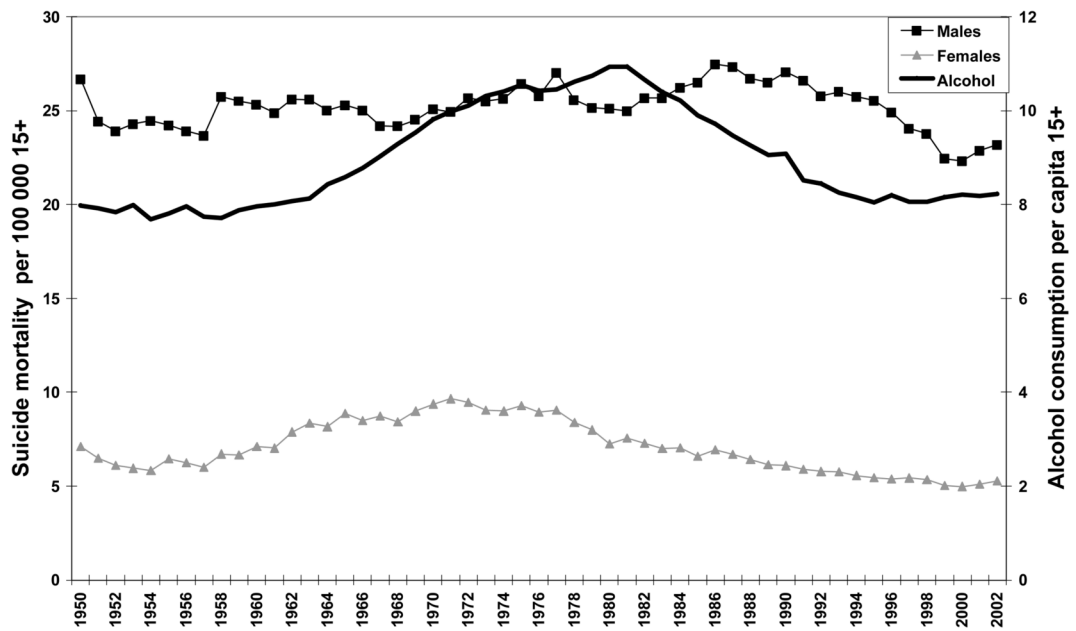


Figure 1. Trends in per capita alcohol consumption and gender-specific suicide mortality rates, 1950-2002.

Table 1

Total alcohol and beverage-specific ARIMA models (semi-logarithmic) for males and females.

	<i>Est</i>	<i>SE</i>	<i>Model</i> ¹	<i>Divorce</i>	<i>Unempl.</i>	<i>Q</i> ⁺	<i>p</i> [§]
Men							
Total alcohol	0.000	0.017	0.1.0; D7, D10	--	0.008*	1.076	0.956
Beer	-0.14	0.044	0.1.0; D7, D10	--	--	0.633	0.986
Spirits	-0.031	0.038	0.1.0; D7, D10				
Wine	0.133	0.095	0.1.0; D7, D10				
Women							
Total alcohol	0.059 ^(*)	0.034	0.1.0; D7	--	--	6.682	0.245
Beer	-0.034	0.084	0.1.0; D7	--	--	7.353	0.196
Spirits	0.152*	0.072	0.1.0; D7				
Wine	0.024	0.177	0.1.0; D7				

D7, D10 = dummies for ICD-changes.

^(*) $p < 0.10$

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

¹ ARIMA models are indicated by (in turns): order of autoregressive parameters (AR), order of differencing and order of moving-average parameters (MA)

⁺ Box-Ljung test for residual autocorrelation, lag 5

[§] p -values of the Box-Ljung tes.

Table 2

Age-specific ARIMA models (semi-logarithmic) of total alcohol consumption for males and females.

	<i>Est</i>	<i>SE</i>	<i>Model^l</i>	<i>Divorce</i>	<i>Unempl.</i>	<i>Q⁺</i>	<i>p[§]</i>
Men							
15-34	-0,021	0,033	0,1,1 D7	0,165*	--	1,665	0,893
35-54	-0,017	0,013	0,1,1 D7	--	0,003*	2,077	0,838
55-74	-0,022	0,028	0,1,0	--	0,005*	3,957	0,556
75+	-0,035	0,036	0,1,0	--	--	6,156	0,291
Women							
15-34	0,069	0,043	0,1,0 D7	--	--	5,349	0,375
35-54	0,062	0,041	0,1,0 D7	--	--	6,242	0,283
55-74	0,086*	0,040	0,1,0 D8	--	--	1,883	0,865
74+	-0,32	0,048	0,1,1	--	--	4,697	0,454

(*) $p < 0.10$

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

^l ARIMA models are indicated by (in turns): order of autoregressive parameters (AR), order of differencing and order of moving-average parameters (MA)

⁺ Box-Ljung test for residual autocorrelation, lag 5

[§] p -values of the Box-Ljung tes.