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ORIGINAL ARTICLE

Per-capita Alcohol Consumption and All-cause Male Mortality in Australia, 1911–2006

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Abstract — **Aims**: Given the variety of relationships found between alcohol consumption and health using individual data (both negative and positive), the likely impact of changes in population-level alcohol consumption on health at the population level is not clear. This paper uses historical data from 1911 to 2006 to assess the relationship between changes in per-capita alcohol consumption on total male mortality in Australia. **Methods**: A longitudinal aggregate study using Australian per-capita alcohol consumption and mortality data from 1911 to 2006. Analysis is undertaken using autoregressive integrated moving average time-series methods. **Results**: Per-capita pure alcohol consumption has a significant association with male all-cause mortality, with an increase (decrease) of 1 l per-capita per year associated with a 1.5% increase (decrease) in male mortality (controlling for female mortality and smoking rates). The association between per-capita consumption and mortality was significant for all age groups, with a particularly strong effect among 15–29 year olds. **Conclusion**: These results place Australia in the group of countries for which a positive association between per-capita alcohol consumption and total mortality can be demonstrated. Thus, despite the beneficial effects of alcohol consumption on health found in many studies, increases in consumption at the population level in Australia are associated with declines in population health. Thus, per-capita alcohol consumption in Australia is a significant contributor to rates of male mortality, particularly among young adults, suggesting an interaction between per-capita consumption and risky episodic drinking. The policies aiming to reduce population-level alcohol consumption and episodic risky drinking have the potential to substantially improve population-health outcomes in Australia, particularly among young men.

INTRODUCTION

Alcohol is ranked in the top four risk factors in the global burden of disease (Rehm *et al*., 2006), and is estimated to be responsible for 3.3% of the total burden of disease in Australia (Begg *et al*., 2007). In spite of these considerable harms, the relationship between population-level alcohol consumption and health is a contentious one (Britton *et al*., 2003). Individual-level studies generally find a J-shaped relationship between levels of alcohol consumption and overall mortality (Holman *et al*., 1996; Klatsky and Udaltsova, 2007), with moderate drinkers living longer than abstainers and heavy drinkers. The lower mortality rates of moderate drinkers are largely driven by the reductions in heart-disease associated with moderate drinking (Ronksley *et al*., 2011), although the magnitude of these effects remain somewhat contested (Fillmore *et al*., 2006). The negative health effects of drinking, on the other hand, are spread across a range of acute and chronic conditions (Rehm *et al*., 2003). Given these varying effects of alcohol consumption on health outcomes, it is not immediately clear whether reductions in the amount of alcohol consumed at a population level would result in net benefits to health. Indeed, some studies from the UK have suggested that current levels of alcohol consumption are health protective, and that reductions may have net health costs (Britton *et al*., 2003), while others suggest 'optimal' levels of per-capita consumption are substantially lower (Nichols *et al*., 2012). As many of the alcohol policy levers available to government are aimed at the general population (e.g. taxation, advertising and availability restrictions), it is crucial for policy-makers to understand how changes in aggregate per-capita alcohol consumption relate to health outcomes. Furthermore, it is important to determine how different sub-sections of the population are affected by changes in aggregate consumption levels, to assess the likely impacts of changes in policy. Thus, this paper uses aggregate historical data to determine how excess mortality in Australia is associated with per-capita alcohol consumption and examines the differential impacts across age groups.

The current study is tackling a question that cannot be addressed using individual-level data—how do changes in the level of drinking in a population affect health outcomes for that population? This is basically a question that can only be assessed via aggregate studies—at the level of populations not individuals (Norström and Skog, 2001). Many researchers have raised concerns about the use of these kind of ecological designs in epidemiology (e.g. Robinson, 1950; Morgenstern, 2008). However, the approach taken in this study does not attempt to make cross-level inferences (i.e. to use ecological data to illuminate individual behaviour). Instead, the study draws population-level conclusions from population-level data and is thus less prone to issues relating to ecological fallacies.

This kind of analysis has been undertaken in a range of other settings, with findings varying substantially. For example, in the European Comparative Alcohol Study (ECAS), Norström analysed data from 14 European countries finding positive relationships between per-capita consumption and all-cause mortality in eleven cases (Norström, 2001). Of note was the range of effect sizes, which were broadly aligned with the variation in drinking cultures. Alcohol's impact on mortality was almost three times as high in countries where drinking more often involved intoxication (e.g. Finland, Sweden and Norway) than in less intoxication-oriented drinking cultures (e.g. France, Spain, etc). Other studies on the basis of European data have pooled country data using cross-sectional time-series methods and produced similar results: overall detrimental

effects of alcohol consumption on mortality (Her and Rehm, 1998), with effect-sizes varying on the basis of the drinking pattern (Gmel *et al*., 2001). Studies using data from the former Soviet countries have found that alcohol has a substantial impact on all-cause mortality (Norström, 2006; Razvodovsky, 2008). Outside of Europe, Norström found a small but significant effect of per-capita alcohol consumption on Canadian mortality (Norström, 2004), while controlling for per-capita tobacco consumption.

It is not clear what association (if any) per-capita consumption in Australia will have with all-cause mortality. Per-capita consumption has historically been markedly higher in Australia than in Northern European countries such as Norway and Sweden, and is generally more oriented towards intoxication than Southern European countries such as Spain and Italy. In the ECAS study, the countries with the most similar-drinking cultures to Australia (the UK and Ireland) were two of the cases where per-capita alcohol consumption was not found to be associated with all-cause mortality, raising the possibility that per-capita consumption may not be related to mortality in Australia.

Given the relevance of this question for policy, the need for an Australian-specific study is clear. However, there has been little work in this area. The majority of Australian studies of alcohol and mortality are on the basis of individual-level data (Cullen *et al*., 1993) or are focussed on estimating the level of alcohol-attributable mortality (English *et al*., 1995) rather than examining how changes in aggregate alcohol consumption relate to mortality rates. There are a few exceptions. For example, in a study of liver cirrhosis mortality and alcohol consumption in four countries including Australia, Kerr *et al*. (2000) found a significant relationship between spirits consumption and cirrhosis mortality. A more recent study by Ramstedt (2011) demonstrated that per-capita consumption in Australia between 1950 and 2003 had a significant positive relationship with the homicide rate. However, as these studies both examine specific mortality outcomes they do not provide a means of addressing the overall health impacts of population-level alcohol consumption.

Thus, the current study on all-cause male mortality fills an important and policy-relevant gap in the literature.

METHOD

The study used time-series analysis to examine the relationship between per-capita alcohol consumption and all-cause male mortality for the period 1911–2006.

Data sources and preparation

A proxy for per-capita alcohol consumption was constructed using data on the sale of alcohol sourced from the Australian Bureau of Statistics. From 1960–1961 onwards, these data came from a recent synthesis of historical data (Australian Bureau of Statistics, 2011), while data from earlier years were extracted manually from the relevant yearbooks (Commonwealth Bureau of Census and Statistics, 1940) and converted from gallons or proof gallons to litres of pure alcohol. This was then converted to litres of pure alcohol per resident aged 15 and older, with population data provided by the Australian Institute of Health and Welfare (AIHW) (AIHW, 2008). The data were derived by the Australian Bureau of Statistics on the basis of alcohol sales. Beer and spirits producers have always paid an excise based on the amount of pure alcohol in their products, so relatively robust historical data are available on the amount of alcohol sold via these beverages. Estimates for wine are based on the volume of wine sold, and rely on historical estimates of the alcohol content of wine. For this reason, wine estimates are less robust before 1950, although it is worth noting that wine made-up ~15% of alcohol sold. These data relate to the amount of alcohol sold in a given year, and are thus only estimates of consumption, as they do not account for cellaring, spillage or other issues which mean alcohol sold in a given year is not consumed in that year (Robinson *et al*., 2012).

Mortality data were provided by the AIHW on the basis of historical death certificate data. As the outcome in question was all-cause mortality, there was no need to control for the impact of the variations in cause-of-death coding that have occurred over the study period. The death rates were age-standardized for men and women separately and expressed per 100,000 population, using indirect standardization to 2001 data from the Australian Census. (analyses were also run using crude death rates, with broadly similar results).

The use of time-series methods and differenced data reduce the need to control for potentially confounding variables in our analyses. Because we are modelling the annual changes in aggregate alcohol consumption and mortality rates, any omitted variable would need to be correlated with annual changes in both measures to bias our results (see Norström, 2001 for a full derivation). Thus we include only a limited number of control variables.

Firstly, analysis in this study uses male mortality as an outcome variable with female mortality incorporated as a control. Effectively, female mortality here serves as a proxy control variable for the range of unmeasured factors that would have impacted on overall mortality across the study period and may potentially be correlated with alcohol consumption (e.g. improvements in treatment, nutrition etc). This approach introduces a small downward bias on the effects of alcohol on mortality as it ignores the role of per-capita alcohol consumption in female mortality. Thus, our findings here represent a conservative estimate of the effect of per-capita consumption on male mortality (analyses presented here were re-run without the female mortality control, with findings as expected: larger effects for alcohol on mortality). This downward bias is the reason that we have not included models of the impact of per-capital alcohol consumption on excess female mortality. As changes in per-capita consumption will have a larger impact on men than women, a model that controlled for male consumption would likely estimate protective effects on women's mortality (relative to men's). It is worth noting that female mortality is not a perfect control, particularly for causes of death that are likely to be differentially affected by uncontrolled factors in this study (e.g. road deaths). However, it provides the most robust control available for the time period being analysed.

Age-specific analyses of male mortality were also undertaken for four-age groups: 15–29-year olds, 30–49-year olds, 50–69-year olds and those aged 70 or over. Descriptive analyses demonstrated a substantial outlier in the death rates for

all age groups aged 15–49 in 1919 and 1920, associated with the Spanish Flu pandemic. A dummy variable to control for this sharp spike in the death rate was included in all models. In addition, dummy variables for WW1 and WW2 (1914–1918 and 1939–1945) were included, as mortality data during these years excluded service personnel and are thus artificially low for young males (AIHW, 2006). Finally, to ensure that the effects of the depression, flu and world wars were not biasing the results, the models were re-estimated using only data from 1950 onwards. The results of these analyses followed the same pattern to those presented here, with smaller effect sizes and a non-significant association for 50–69 year olds.

Given the considerable impact of smoking on mortality and the likelihood that the use of tobacco and alcohol may co-vary, the per-capita consumption of tobacco (in kilograms) was used as a control variable in all analysis, with data sourced from the AIHW (AIHW, 2010). The per-capita tobacco measure was included using a distributed lag, although there is limited evidence for an appropriate approach to the lag structure here. Thus, the results for tobacco are unlikely to represent the actual impact of per-capita tobacco consumption on total mortality, but including it here will at least minimize the potential bias on the alcohol effects.

Mortality and tobacco data were provided on the basis of calendar year information, while alcohol consumption data (which is based on the tax collection) have been historically reported based on Australian financial years (July–June). To ensure that the data were as comparable as possible, the alcohol series was averaged across financial years to produce calendar year estimates. Thus, for example, alcohol data for 1990 were calculated as the average of the consumption estimates for the financial years 1989/1990 and 1990/1991. (We also analysed the data using the lagged financial year consumption data to predict mortality (e.g. data from 1989 to 1990 for 1990 mortality data). The models produced were strikingly similar to those presented here, with just a small reduction in effect sizes, suggesting that our findings are robust.). Descriptive data are presented in Figure 1.

Analysis

The analysis of time-series data using ordinary least squares (OLS) regression is likely to produce substantially biased results, as observations are not strictly independent (observations close in time are more like each other than those farther apart). As such, data in this study were analysed using auto-regressive integrated moving average (ARIMA) models as derived specifically by Box and Jenkins (Box and Jenkins, 1976) for the analysis of time-series data. This technique models the temporal structure of the error term, which is estimated in terms of auto-regressive and moving average components, ensuring that any correlations in the series over time do not bias the model coefficients.

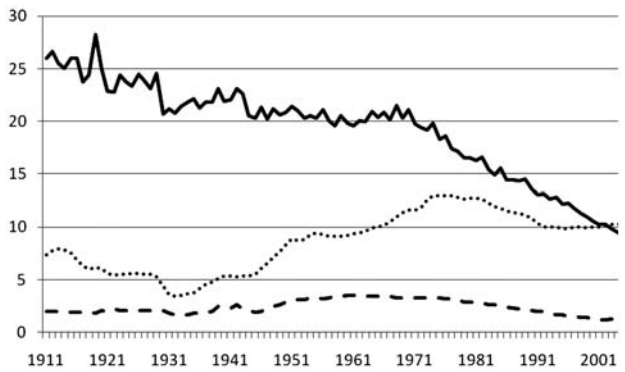

Fig. 1. Age-standardized male mortality rate, per 1000 populations (solid line), alcohol consumption, litres pure alcohol per capita 15 and older (dotted line), tobacco consumption, kilograms per capita (dashed line), 1911–2006.

ARIMA models require stationary series, and strong trends were present in the main series of interest (see Fig. 1). These trends were removed by differencing the series, resulting in significant Dickey–Fuller tests for the stationarity of the data. In other words, the data analysed in this study are the annual changes in each of the series rather than the raw data. In addition to validating a key requirement of ARIMA modelling, this differencing also reduces the risk of spurious effects due to unobserved factors. An omitted variable would need to be correlated with the annual differences in both alcohol consumption and mortality for it to introduce bias to the results, whereas a variable that was correlated with trends in both could potentially bias the results of un-differenced models (see Norström and Skog, 2001 for the mathematical derivation of this statement).

One of the key issues raised in previous work is whether the effect of alcohol consumption on all-cause mortality is distributed over time. Some studies have found that the effects of per-capita alcohol consumption in preceding years (as well as the current year) impact on current mortality rates (Norström, 2001), while others studies have found that models without lagged effects fit their data better, even for chronic conditions like cirrhosis (Norström, 1988; Gruenewald and Ponicki, 1995). For this study, the analysis was conducted both ways (with and without lagged effects). The lagged approach, following Norström's work (Norström, 2001) used a geometric lag scheme with a lag parameter (λ) of 0.7. This implies that one-third of the effect of alcohol consumption on all-cause mortality is due to consumption in the current year, a quarter because of consumption in the year before, one-sixth from consumption in the year before that and so on (up to a maximum of five years). This contrasts with the non-lagged approach which implies that the effects of changes in per-capita consumption on mortality all occur in the current year. The non-lagged approach provided the best fit to the data in this study (based on the Akaike Information Criteria measure of goodness of fit), and thus the results reported here are based on this specification.

Due to the large proportion of alcohol-related causes of death that have exponential risk functions, previous aggregate studies in this field have used a semi-logarithmic model specification (Norström and Ramstedt, 2005). This approach was also used here, with the natural log of the mortality data used as the dependent variable and the independent variables unlogged. Using this specification, the interpretation of the alcohol parameter (β) is relatively straightforward: the percentage increase in mortality that is expected to follow from a 1-litre increase in per capita alcohol consumption is obtained by computing: $(\exp(\beta)-1)*100$.

The specific structures of the ARIMA models (in terms of auto-regressive and moving average parameters) were derived from exploratory descriptive analyses and tested to ensure that no temporal structure remained in the residuals.

The residuals of all models produced non-significant Portmanteau Q statistics, indicating they were indistinguishable from white noise. The model structures used are reported below, alongside the final model output.

RESULTS

The general trends in the three series of interest are displayed in Figure 1. Male mortality rates have declined steadily across the study period, with a particularly steep fall from the 1970s onwards. Per-capita alcohol consumption declined between 1911 and the early 1930s, before increasing steadily to a peak in the late 1970s, gradually declining to the early 1990s and then stabilizing. Tobacco consumption increased steadily to a peak in the late 1970s and then declined over subsequent decades.

The results of the time-series analysis of total mortality are presented in Table 1. The effect of per-capita alcohol consumption on male mortality is positive and significant, suggesting that each 1 l increase (decrease) in per-capita consumption of pure alcohol is associated with a 1.5% increase (decrease) in male mortality. The effect estimate of per-capita cigarette consumption was positive, but non-significant. Note that these effects are the net effect on male mortality, with female mortality controlled for, which may explain the lack of a significant relationship between cigarette consumption and mortality in the overall population. Furthermore, the lags between smoking and mortality are significantly longer than those modelled here. Building in bigger lags would have limited the data available for our analyses, and the five-year lags constructed at least control for concurrent changes in alcohol and tobacco consumption likely to impact on our estimates of alcohol's relationship with mortality.

The results of age-specific models are presented in Table 2. The age-specific models demonstrate that the effect of per-capita alcohol consumption on male mortality is strongest amongst the youngest age group (15–29 year olds), with a 1 l increase in consumption associated with a 12.5% increase in mortality. Alcohol effects for the other age groups were all positive and significant but substantially smaller (between 0.9 and 3.2% increases in mortality for a 1 l increase in consumption). The effect of cigarette consumption on mortality was positive and significant for the 50–69-year old age group, with each extra kilogram of tobacco consumed associated with a 6.8% increase in excess male mortality.

Table 1. Semi-logarithmic ARIMA model of the estimated effect of alcohol consumption on all-cause male mortality, controlling for female mortality and cigarette consumption, 1911–2006, Australia

	Effect (β)	Standard error
Alcohol consumption (l)	0.015***	0.004
Female mortality	0.920**	0.045
Cigarette consumption (kg)	0.031	0.021
War years	–0.004	0.008
1919 Flu	–0.002	0.037
Constant	0.0003	0.001
Portmanteau Q test for residual autocorrelation (5 lags)	$Q = 3.608$ ($P = 0.607$)	
Model specification	(0,1,1) $\lambda = 0.0$	

**$P < 0.01$, *$P < 0.05$ and (*)$P < 0.10$.

CONCLUSIONS

This study has established that the amount of alcohol consumed at the population level in Australia is significantly associated with total male mortality. In other words, despite the potential health benefits from moderate drinking found in many studies, increases in per-capita alcohol consumption in Australia have negative effects on population health. The findings for Australia overall, that each additional litre of pure alcohol consumed per-capita is associated with a 1.5% increase in total male mortality, are broadly in line with studies in other settings. The magnitude of the effect found here is higher than that found in Middle- and Southern European countries (1.1 and 1.0%, respectively), but lower than the effects found in Northern Europe and Canada (2.9 and 2.3%) (Norström, 2001, 2004).

The age-specific findings highlight striking differences in effect size of per-capita alcohol consumption on different sub-populations. The mortality of young men (15–29 year olds) is substantially affected by alcohol consumption, with a 1 l increase in per-capita consumption associated with a 12.5% increase in mortality in this age group. It is worth noting that this group consumes around one-third of the alcohol consumed in Australia (unpublished analyses of National Drug Strategy Household Survey, 2007), which is less than those aged 30–59, but that they are the group most likely to drink at levels that put them at risk of short-term harm (AIHW, 2009). These findings (along with the lack of

Table 2. Age specific semi-logarithmic ARIMA models of the estimated effect of alcohol consumption on all-cause male mortality, controlling for female mortality and cigarette consumption, 1911–2006, Australia

	15–29 year olds		30–49 year olds		50–69 year olds		70+ year olds	
	Effect (β)	Standard error	Effect (β)	Standard error	Effect (β)	Standard error	Effect (β)	Standard error
Alcohol consumption (l)	0.125**	0.025	0.036**	0.011	0.021**	0.008	0.009*	0.005
Female mortality	0.255**	0.079	0.371**	0.081	0.724**	0.074	0.957**	0.047
Cigarette consumption (kg)	–0.126	0.088	–0.007	0.045	0.068**	0.026	0.025	0.022
War years	–0.099**	0.024	–0.045**	0.015	–0.021(*)	0.012	0.010	0.012
1919 Flu	0.471**	0.080	0.409**	0.052	–0.046	0.051	–0.023	0.056
Constant	–0.005	0.008	–0.011**	0.003	–0.003(*)	0.002	0.001	0.001
Q test for residual autocorrelation (5 lags)	$Q = 3.325$ ($P = 0.65$)		$Q = 2.336$ ($P = 0.801$)		$Q = 6.295$ ($P = 0.279$)		$Q = 4.055$ ($P = 0.542$)	
Model specification	(0,1,0) $\lambda = 0$		(0,1,1) $\lambda = 0$		(0,1,1) $\lambda = 0$		(0,1,1) $\lambda = 0.0$	

**$P < 0.01$, *$P < 0.05$ and (*)$P < 0.10$.

lagged effects) suggest that changes in per-capita alcohol consumption have substantial impacts on acute alcohol-related mortality (e.g. accidents, suicide and homicide) and less impact overall on mortality related to chronic disease. This implies that the association between mortality and per-capita consumption is significantly linked with episodic risky drinking. Thus, a theoretical change in per-capita consumption that did not affect rates of episodic risky drinking would most likely have a far smaller impact on mortality rates than suggested by these results. However, the link between per-capita consumption and rates of risky drinking is relatively strong, so it is likely that any marked change in per-capita consumption will see a broadly similar change in rates of episodic risky drinking and thus mortality.

The current study has a number of limitations. The most important one is the inability of sales data to provide per-capita consumption estimates for gender or age-based subgroups of the population. However, given that many population-health policies similarly cannot be specifically targeted at the population subgroups, this does not detract from the policy relevance of the approach taken, and the finding that changes in per-capita consumption particularly relate to mortality in young people has particular implications for policy. Further, data provided by governments on per-capita alcohol consumption have a range of limitations (see for example Chikritzhs *et al*., 2011), however this type of data is generally considered the most robust option available for estimating population-drinking levels (World Health Organization, 2000) and is the only available source of consumption data over long periods of time. Obviously the use of an aggregate measure of consumption also means that changes in *patterns* of drinking cannot be considered. Future analyses of shorter time-series may benefit from the combination of aggregate and survey-derived consumption data to build more robust understandings of how aggregate consumption and episodic drinking co-vary. Finally, given the limited number of control variables included, there is some risk that the effects found for alcohol consumption here are because of some other unmeasured factor. However, the modelling approach taken ensures that this is only plausible in the case where the unobserved factor is correlated with the annual changes in both per-capita alcohol consumption and mortality. The most obvious candidate for this is smoking, which has been controlled for, meaning that the findings should be considered reasonably robust.

The findings of this study have significant implications for alcohol policy. They suggest that alcohol policies that aim to reduce total population consumption have the potential to make significant positive impacts on the health of Australia, particularly where these changes also result in reductions in episodic risky drinking. There is reasonably robust evidence that population-based policy interventions such as taxation and availability restrictions impact on both per-capita consumption and heavy drinking (Babor *et al*., 2010). For example, two recent meta-analyses demonstrate that both heavy drinkers and young adults reduce their consumption in response to price increases (Gallet 2007; Wagenaar *et al*., 2009). Similarly, recent Australian studies have shown that the density of outlets and the trading hours of late-night drinking venues are directly linked with both acute and chronic alcohol-related harms (Livingston et al., 2008; Kypri, 2011; Livingston 2011). The findings of this study provide support to population-based policy interventions which reduce total consumption and particularly consumption by young adults. This policy focus is widely supported by the existing research evidence (Babor *et al*., 2010), but it is worth re-emphasizing given the ongoing discussion about alcohol's health benefits and the evidence that Australian per-capita alcohol consumption has recently begun to increase after a period of relative stability (Chikritzhs *et al*., 2011).

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