Alcohol Consumption and Fatal Injuries in Australia Before and After Major Traffic Safety Initiatives: A Time Series Analysis

Article in Alcoholism Clinical and Experimental Research · January 2015
DOI: 10.1111/acer.12609

CITATION
1

READS
95

3 authors:

Heng Jiang
La Trobe University
30 PUBLICATIONS 56 CITATIONS
SEE PROFILE

Michael John Livingston
La Trobe University
117 PUBLICATIONS 1,652 CITATIONS
SEE PROFILE

Robin Room
La Trobe University
516 PUBLICATIONS 20,581 CITATIONS
SEE PROFILE

All content following this page was uploaded by Heng Jiang on 08 February 2015.
The user has requested enhancement of the downloaded file. All in-text references underlined in blue are added to the original document and are linked to publications on ResearchGate, letting you access and read them immediately.
Alcohol consumption and fatal injuries in Australia before and after major traffic safety initiatives: A time series analysis

Heng Jiang 1,2, * (Ph.D), Michael Livingston 1,3 (Ph.D) & Robin Room 1,4,5 (Ph.D)

1 Centre for Alcohol Policy Research, Turning Point, Melbourne, Victoria, Australia
2 Eastern Health Clinical School, Monash University, Australia
3 Drug Policy Modelling Program, National Drug and Alcohol Research Centre, The University of New South Wales, New South Wales, Australia
4 Melbourne School of Population and Global Health, University of Melbourne, Australia
5 Centre for Social Research on Alcohol and Drugs, Stockholm University, Sweden

Total page count: 17
Word count for abstract: 297
Word count for body text: 4490
References: 38
Figures: 4
Tables: 5

*Corresponding address: Heng Jiang,
Centre for Alcohol Policy Research, Turning Point
54 Gertrude Street,
Fitzroy, VIC, 3065
E: jasonj@turningpoint.org.au
P: 03 84138452
F: 03 94163420

Acknowledgements
The research was funded by a project grant from the Australian National Health and Medical Research Council (#566629). The Centre for Alcohol Policy is funded by the Foundation for Alcohol Research and Education (FARE), an independent, charitable organisation working to prevent the harmful use of alcohol in Australia (www.fare.org.au). ML is supported by a National Health and Medical Research Council Early Career Fellowship. RR’s position is supported by the Victoria Department of Health.
Abstract

Background: The associations between population level alcohol consumption and fatal injuries have been examined in a number of previous studies, but few have considered the external impacts of major policy interventions. This study aims to quantify the associations between per capita alcohol consumption and traffic and non-traffic injury mortality rates in Australia before and after major traffic safety initiatives (the introduction of Compulsory Seat Belt Legislation (CSBL) and Random Breath Testing (RBT) in 1970s).

Methods: Using data from 1924 to 2006, gender and age specific traffic and non-traffic mortality rates (15 years and above) were analysed in relation to per capita alcohol consumption using time series analysis. The external effects of policy interventions were measured by inserting a dummy variable in the time series models.

Results: Statistically significant associations between per capita alcohol consumption and both types of fatal injuries were found for both males and females. The results suggest that an increase in per capita alcohol consumption of 1 litre was accompanied by an increase in traffic mortality of 3.4 among males and 0.5 among females per 100 000 inhabitants and an increase in non-traffic mortality of 3.0 among males and 0.9 among females. The associations between alcohol consumption and fatal injury rates varied across age groups. The introduction of CSBL and RBT were associated with significant reductions in traffic crash mortality in Australia, particularly for males and young people.

Conclusions: The magnitude and distribution of the preventive effects from the reduction of population drinking on fatal injuries vary across different gender and age groups, with the strongest preventive impacts on fatal injuries among people age 15-29 years and 70 years and above. The mechanisms behind these effects are unclear from this study, but are likely to be due to the strong association between per-capita consumption and heavy drinking.

Key words: Alcohol consumption, traffic injury, non-traffic injury, time series analysis, intervention event
INTRODUCTION

It is well-recognized that alcohol intoxication contributes to a large proportion of traffic and non-traffic injuries in many countries (Skog, 2001a). Previous studies show that about 10% to 60% of all fatal traffic crashes appear to be alcohol-related and the countries with low alcohol consumption levels were found have low alcohol related traffic crash rates (Norström and Rossow, 2013). Furthermore, alcohol also plays a significant role in non-traffic injuries, such as accidental falls and drowning, injuries caused by fire, and other unintentional injuries (Rehm et al., 2010). In Australia, a recent study of alcohol’s contribution to the overall burden of disease estimated that alcohol contributed to more than 5,000 deaths and 55,000 hospitalisations, with more than 10% of this burden due to injuries (Gao et al., 2014).

There is significant evidence that the level of alcohol consumption in a given society is related to the rate of alcohol-related problems (Norström and Ramstedt, 2005). Studies examining whether unintentional injury mortality is related to population drinking levels are less common, but the existing evidence suggests a link, particularly in countries with intoxication-focussed drinking patterns. Two studies (Skog, 2001a; 2001b) on data from Europe demonstrated significant associations between per-capita consumption and unintentional injury mortality, with the effect sizes varying across countries. On the whole, changes in population drinking affected injury mortality more in Northern Europe than Southern Europe, but the effect on traffic injury mortality was higher in the Southern European countries. Other analyses have also shown a strong association between per-capita consumption and traffic injury mortality in North America, with significant positive effects also found for unintentional falls and “other fatal injuries” (Skog, 2003). However, to our knowledge, there have been no Australian studies in this area.

The role of alcohol in unintentional injury mortality varies in different gender and age groups, and by socioeconomic status, ethnicity and historical periods, in the last instance reflecting changes in such factors as the drinking culture, behavioural patterns and norms, and policies. Gender differences in the association between per capita alcohol consumption and fatal injuries have been found in some
previous studies, such as Yang et al. (1977); Brismar and Bergman (1998); Skog (2003) and Ramstedt (2008), with effects on male mortality much higher than on female mortality. In a study of 14 European countries, estimated effect sizes of consumption on injury mortality were 3-4 times as large for males than for females (Skog, 2001b). These gender differences may relate to differences in risk-taking by gender (Byrnes et al., 1999; Cherpitel, 1993), or to the likelihood that changes in per-capita consumption in Europe are largely driven by changes in male drinking; a summary of European surveys estimated male drinking accounts for between 65% and 80% of total consumption (Knibbe and Bloomfield, 2001). Similarly, previous studies have found varying effects across age groups, with changes in per-capita alcohol consumption affecting traffic deaths among younger people (17-34-year-olds) more than older people (>34 years) in the U.S. (Ramstedt, 2008), while the effects on total injury mortality were smallest in the middle age group (30-49 years) compared with the young age group (15-29 years) and old age group (50-69 years) in European countries (Skog, 2001b). The relationship between alcohol and different types of injuries at the individual level has been systematically analysed, with roughly equivalent dose-response relationships for both road traffic and other injuries (Taylor et al., 2010).

Random Breath Testing (RBT) is a widely used drink-driving countermeasure and is particularly common in Australia (Rowland et al., 2012), where the acceptable blood alcohol content (BAC) is 0.05% in all states. RBT was introduced in Victoria in 1976, and gradually implemented in other states in Australia. A number of studies have demonstrated that the introduction of RBT significantly reduced alcohol-related crashes and fatalities (Henstridge et al., 1997; Peek-Asa, 1999). Surprisingly, given the significant literature examining the links between per-capita consumption and injury mortality and the studies focussing on the impact of RBT, no studies have explored whether or not the introduction of RBT moderates the relationship between population consumption and traffic deaths. Besides the RBT program, the implementation of Compulsory Seat Belt Legislation (CSBL) in the early 1970s in Australia has generated substantial reductions in traffic crashes and was successful enough become a standard feature in road safety (Conybeare, 1980; Milne, 1985; Hall et al., 2010). While the relationships between alcohol consumption and traffic and non-traffic injuries have been
widely discussed, few studies have considered the external impacts of intervention events in the
strength of the association.

The main aims of this study are to quantify the associations between per capita alcohol consumption
and fatal traffic and non-traffic injury mortality rates in Australia, and to assess how the
implementation of major policy measures aimed at reducing harm (i.e. RBT and seatbelt laws)
modifies these associations. We used autoregressive moving average (ARIMA) time-series methods
to control for trends across the time period, allowing for a more robust estimation of the association
between changes in population alcohol consumption and changes in injury mortality. The study was
further split into two time periods to explore whether the association between alcohol consumption
and injury mortality varies between the period before RBT law was introduced and the period after,
controlling for the effects of CSBL. Additionally, the estimation results were presented in a
comparative framework with previous international studies, to assess whether the links between
alcohol consumption and injury fatalities in Australia differ from those found elsewhere. The findings
added to the international literature on the links between per-capita alcohol consumption and injury,
and provided the first evidence of how these links are affected by policies aimed at reducing risk
behaviours other than alcohol consumption. This provided useful new evidence as to the relative
importance of policies targeting alcohol consumption and policies targeting particular types of risk-
taking behaviour for reducing injury rates.

METHODS

Data

A proxy for per-capita alcohol consumption was constructed using data on the sale of alcohol sourced
1960 were sourced from historical publications (Australian Year Book, e.g. Commonwealth Bureau of
Census and Statistics, (1960)) and were converted from gallons or proof gallons to litres of pure
alcohol. Conversion factors (i.e. alcohol content per volume) for the pre-1960 data were derived for beer and wine based on a report that provided snapshots of total volume and pure alcohol volume once per decade (Australian Bureau of Statistics, 1994). For beer, average alcohol content did not vary much (between 4.6% and 4.9%), whereas wine content varied sharply (from as low as 10.1% to a peak of 17.1%) over the century due to the rise and decline of fortified wine as a popular beverage. Linear trends in alcohol content were assumed between the available data points. Spirits data were always reported in proof litres (or gallons), enabling straightforward conversions to pure alcohol. These data were 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) (Australian Institute of Health and Welfare, 2008).

Age- and gender-specific mortality data were provided by the AIHW. They provide historical data on motor vehicle traffic injury mortality from 1924, based on the appropriate International Classification of Diseases codes. Data on all external cause mortality, homicide and suicide were also provided, allowing us to calculate a proxy series of non-traffic injury mortality (all external cause – [traffic mortality + homicide + suicide]). This series contains other transport injury mortality not included in motor vehicle traffic injuries, unintentional falls, poisoning and “other non-traffic injury mortality”.

The AIHW have developed standardised historical mortality databases which track the relevant cause of death codes across the various classification schemes used in Australia. Historical population data were provided by the AIHW. The death rates were age-standardised for men and women separately and expressed per 100,000 population, using indirect standardisation to the 2001 population age structure.

ARIMA model with dummy variables

Fatal traffic and non-traffic injuries caused by intoxication ought to occur shortly after the alcohol has been consumed. Therefore, no lagged effects were considered in the current study (in contrast, studies
of liver disease mortality typically incorporate lag structures, e.g. (Jiang et al., 2013)). An autoregressive moving average model (ARIMA), using the Box-Jenkins (Box and Jenkins, 1970) approach, was employed to estimate the associations between alcohol consumption and traffic and non-traffic injury mortality in Australia. The prior condition for these analyses is that all the series be stationary, with time trends removed to avoid the risk of obtaining a spurious estimation (George, 1994). In most cases, a differencing of the time series is sufficient to eliminate non-stationarity (Norström, 2001). The Augmented Dickey-Fuller (ADF) unit root test, developed by Dickey and Fuller (1979), was employed to test for stationarity for the time series. An ARIMA model with dummy variables can be written as follows:

\[
\Delta Y_t = \alpha + \beta \Delta Y_{t-1} + \mu \Delta C_{i,t} + \sum_{j=1}^{n} \gamma_j D_{j,t} + \delta \Delta E_{t-1}
\]

where \( \Delta \) is the differencing operator, \( Y_t \) represents the dependent variable at time \( t \) (age-specific injury mortality in Australia), \( \alpha \) is a constant (which marks average annual changes due to other causes), \( \beta \) is the coefficient value of the AR(1) term (\( \Delta Y_{t-1} \)), \( \delta \) is the coefficient value of the MA(1) term (\( \Delta E_{t-1} \)), \( C_{i,t} \) are the control variables considered in the estimation, \( i \) is number of control variables, \( \mu \) is the coefficient values of the control variables, \( D_{j,t} \) is the one-off event dummy variable \( j \) at time \( t \), \( n \) is the number of dummy variables and \( \gamma_j \) is the estimates of the effect of the events or interventions. The dummy variable remains at value 1 for the duration of the presence of the event; otherwise, the dummy is value 0. A control variable based on annual numbers of registered vehicles (Australian Bureau of Statistics, 2012) was included in initial models to control for increases in car use over the study period, but there were no significant effects on traffic injury mortality rates, so it was excluded from the final models.

Initially, two separate intervention dummy variables were developed to represent the introduction of RBT and CSBL respectively. However, we found the estimates of two event dummy variables
interfered with each other in the model estimation: CSBL and RBT were both introduced in the 1970s, nearly in the same period, and both have long-term and on-going effects on traffic fatalities in Australia after the 1970s. In order to solve this problem and control for the effects of CSBL and RBT in the association estimation, a joint events dummy variable was constructed to represent the combined impacts of CSBL and RBT on unintentional injuries in Australia. In order to better control the joint effects of CSBL and RBT on injury fatalities, the joint CSBL and RBT dummy variable was constructed based on a roll-out approach, e.g. The CSBL was first implemented in Victoria state in Australian in 1970 and the joint events dummy variable is coded as 0.25 (population weight of Victoria in Australia) in 1970 and 0 between 1924 and 1969. The joint events dummy is recoded as 0.57 in 1971 and 1 in 1972 as the CSBL was implemented in New South Wales in 1971 and then fully implemented in all states in 1972. The RBT program was first introduced in Victoria State in 1976, so the joint dummy variable is further coded as 1.25 in 1976. After that, the joint events dummy is recoded, adding the population weight of each state when RBT was introduced in that state. The joint dummy is finally coded as 2 after 1987, as it was introduced in all states in Australia. The effects of World War 2 on injury mortality are also controlled by setting up a WW2 dummy variable, as mortality data during these years excluded service personnel and are thus artificially low for young males (as can be seen in the negative associations between WW2 and young male mortality presented in Figures 1-4). The development of event dummy variables for the proposed time series models is presented in Table 1. A further set of analyses were conducted attempting to further control for the effects of petrol rationing during and after the war period and the impacts of lowering minimum legal drinking age (MLDA) from 21 to 18 in four Australian states, but these effects did not significantly alter the final models and are not presented here. The model fit was evaluated with the aid of the Box-Ljung portmanteau test of the first 10 autocorrelations, $Q(10)$. The model structures used are reported below, alongside the output of the models.

<Insert Table 1 about here>
RESULTS

Alcohol consumption and fatal injuries in Australia

The trends in alcohol consumption and male and female fatal injuries in different age groups are presented in Figs 1-4. Both male and female traffic fatalities dramatically decreased during the Second World War, for the reasons discussed above, and there was an obvious climb after the war. Per capita alcohol consumption increased significantly between 1950 and 1970, as did traffic fatalities. Trends in fatal traffic and non-traffic injury mortalities and per capita alcohol consumption declined and levelled off in a similar way after 1974. However, there is less convergence between traffic fatalities and alcohol consumption between 1996 and 2006, as male and female traffic fatalities decreased slightly but alcohol consumption stayed at the same level.

The associations between alcohol consumption and fatal injuries

The results of the time series analyses between per capita alcohol consumption and age-specific male and female traffic injury mortality rates are presented in Table 2. The findings suggest that there were positive associations between alcohol consumption and total traffic injury mortality among both men and women 15 years and older. These results suggest that an increase in population drinking would lead to a greater effect on traffic injury mortality for male (3.4) than for female (0.5). The age-specific effects are positive but statistical significance is only found in the younger age groups. The estimates indicate that an increase of 1 litre in per capita alcohol consumption was associated with an increase
in male traffic injury mortality by about 7.3 deaths per 100,000 population among 15-29 year olds, 2.4
among those 30-49 years and 2.2 among those 50-69 years, but with no significant effects on 70 years
and older group. In contrast, the effects for women were only significant in two younger age groups
(15-29 and 30-49 years) with 0.5 and 0.3 per 100,000 population.

The estimates of event dummies indicate that both World War 2 and the joint events of CSBL and
RBT had significant negative effects on both male and female traffic injury mortality in Australia.
The estimates of the joint events dummy variable suggest that the introduction of CSBL and RBT in
the 1970s in Australia has led to a reduction in total male and female traffic injury mortality of about
0.8 and 0.3 per 100,000 among the population aged 15 years and older. It had greater prevention
effects on both male (-1.5) and female (-0.5) fatal traffic injuries in the youngest age group than for
middle and older age groups (30 year olds and above).

The estimated effects of alcohol consumption on age- and gender-specific fatal non-traffic injury
mortality during 1924-2006 are presented in Table 3. A stronger association was found between
population drinking and non-traffic injury mortality for older people than for younger people both for
males and females. The estimates indicate that an increase of 1 litre in per capita alcohol consumption
was associated with an increase in male and female non-traffic injury mortality of about 9.1 and 7.3
per 100,000 for males and females aged 70 and over, which is significantly higher than the effects for
younger age groups. The coefficient values of the joint dummy of CSBL and RBT indicate that, as
expected, the introduction of CSBL and RBT in 1970s had no significant effects on non-traffic injury.
However, the World War 2 years had significantly lower total injury mortality, for the reasons
discussed earlier.

The estimated effects of alcohol consumption on age- and gender-specific fatal non-traffic injury
mortality during 1924-2006 are presented in Table 3. A stronger association was found between
population drinking and non-traffic injury mortality for older people than for younger people both for
males and females. The estimates indicate that an increase of 1 litre in per capita alcohol consumption
was associated with an increase in male and female non-traffic injury mortality of about 9.1 and 7.3
per 100,000 for males and females aged 70 and over, which is significantly higher than the effects for
younger age groups. The coefficient values of the joint dummy of CSBL and RBT indicate that, as
expected, the introduction of CSBL and RBT in 1970s had no significant effects on non-traffic injury.
However, the World War 2 years had significantly lower total injury mortality, for the reasons
discussed earlier.
Changes in the effects of alcohol consumption on fatal injuries

It can be observed that per capita alcohol consumption in Australia increased steadily from about 6 litres per capita to 13 litres between 1931 and 1975. After the RBT program was first introduced in Victoria in 1976, per capita alcohol consumption decreased slightly from 13 to 12.5 litres per capita by 1982. After 1982, as the RBT program moved to being fully implemented in all states and territories, the per capita alcohol consumption decreased steadily from 12.5 to 10 litres by 1991. After that, alcohol consumption has been reasonably steady (about 10 litres per capita) up to 2006. In a previous study exploring the association between per-capita consumption and liver disease mortality, we found a substantially different relationship between 1976 and 2006 than before 1976, with alcohol consumption seemingly having less impact on mortality at the population level post-1975 (Jiang et al., 2013). We conduct similar analyses here to explore whether the links between per-capita consumption and injury mortality differ by time period. This has added value in this case as the latter period incorporates the majority of the road traffic policy interventions of interest, which may have mediated the links between population drinking and traffic injury deaths. Thus, we analyse the data for two periods: 1924-1975 (the period before the introduction of RBT) and 1976-2006 (the period following the introduction of RBT).

The two ARIMA models for these periods are presented in Table 4. The results show that weaker associations were found between per capita alcohol consumption and both traffic and non-traffic injury mortality for both genders in Model 2 compared with Model 1. Particularly, the estimated effects of per capita alcohol consumption on male traffic injuries dropped from 4.3 in the earlier period to 3.2 in the later period. Furthermore, the effects of alcohol consumption on female traffic injury mortality also became weaker in the later period compared with the effects in the earlier period (2.1 vs 3.3). These changes in the link between per-capita consumption and fatal injury mortality suggest that alongside the reduction of traffic fatalities due to the introduction of compulsory seat belt laws and the reduction in driving after drinking following the roll-out of RBT programs, RBT may also have more generally affected alcohol consumption in Australia, further helping to prevent
alcohol-related traffic injuries. However the changes in non-traffic injury mortality may imply that broader shifts in how alcohol is consumed in Australia, as well perhaps as in emergency responses to injuries, are responsible for the varying relationships over time.

<Insert Table 4 about here>

Comparison of Australian findings and previous international studies

The results of comparison between Australian findings and previous international studies are presented in Table 5, which shows the effect of alcohol consumption on male traffic injury mortality in Australia (3.4) is higher than for Northern Europe (0.1) (i.e. Finland, Sweden and Norway), Central Europe (2.1) (i.e. Austria, Belgium, Denmark, Ireland, Netherlands, Germany and the U.K), and Southern Europe (0.8) (i.e. France, Italy, Portugal and Spain), but roughly the same as the effects in the U.S. (3.2) and Canada (3.6) (Skog, 2001a; 2003; Ramstedt, 2008). The estimated effect of alcohol on female traffic injury mortality in Australia is smaller than for Canada, Northern Europe and the U.S, and higher than for Central Europe and Southern Europe, although there is not much variation across these studies. In contrast, the estimated effect of alcohol on female non-traffic injury mortality in Australia is the strongest among all selected countries or regions, while the effect on male non-traffic injury mortality is only weaker than Northern Europe and stronger than all other selected countries or regions. These comparisons in general place Australia at the higher end of the spectrum internationally, suggesting that changes in population consumption are likely to have large impacts on injury mortality.

<Inset Table 5 about here>

DISCUSSION
This paper analyses to what extent changes in per capita alcohol consumption influence mortality rates due to fatal traffic and non-traffic injuries in Australia between 1924 and 2006. The results show significant positive associations between per capita alcohol consumption and traffic and non-traffic injury mortality among most gender- and age-specific groups in Australia when controlling for the impacts of World War 2 and introduction of Compulsory Seat Belt Legislation and Random Breath Testing, with the exception of females in the 50-69 and 70 years and over groups. The parameter estimates are approximately seven times larger for males than for females for traffic injury mortality, and about four times as large for non-traffic injury mortality. This suggests more drink-driving as well as more drinking by males than by females in Australia.

This study revealed similarities with findings in relationship between aggregate alcohol consumption level and traffic injuries in some other countries (e.g. Canada and the U.S.), but stronger than in European countries. One possible reason is the greater amount of driving that takes place in Australia as well as in U.S. and Canada compared to European countries. For example, in 2010, the number of motor vehicles per 1000 people was 698, 797 and 607 in Australia, the U.S. and Canada respectively, compared with 547 in European countries (The World Bank, 2013). Furthermore, the associations between aggregate alcohol consumption level and fatal non-traffic injuries were stronger than in most of the selected countries. This suggests that the fatal unintentional falls, poisonings, fires, drownings and other types of non-traffic fatalities in Australia are more strongly related to alcohol consumption than in the U.S and Europe. In spite of these variations, the overall impact of alcohol on injury deaths in Australia was broadly comparable to the U.S. and much of Europe. Using methods applied in previous studies [e.g. Skog (2001b)], alcohol consumption (average 8 litres per capita during the study period) in Australia produced about 26 male and 5 female injury deaths per 100,000 inhabitants per year respectively, contributing to 41% of total male and 20% of total female injury deaths. These findings are similar to the attributable fractions for males estimated in Northern (45%), Central (35%) and Southern Europe (40%) and in the U.S. (39%) [Skog (2001b), Ramstedt (2008)].
In terms of the effects of alcohol consumption on age-specific injury mortality, the results reveal that an increase in alcohol consumption was associated with a higher increase in traffic injury mortality among 15-29-year olds than in the other three age groups (30-49, 50-69 and 70+ years). This finding is consistent with a similar study using data from the U.S. (Ramstedt 2008). However, the findings for age-specific non-traffic injury mortality suggest that the effects are larger in older age groups (70 years +) than in younger groups (15-29, 30-49, 50-69 years). These results make intuitive sense given the prevalence of traffic injury mortality in the younger age groups and of fall-related mortality in the older groups. Thus, the findings suggest that reducing per capita alcohol consumption may reduce fatal traffic injuries among young Australians and fatal non-traffic injuries among older Australians, although this assumes that the relationship between per-capita consumption and heavy drinking in these groups remains unchanged. The non-traffic findings presented here are different from previous studies in Europe and the U.S., which both suggested that middle age groups (35-54 years) showed greater effects than younger (15-34 years) and older age groups (55-74 years) (Skog, 2001b; Lunetta et al., 2001; Ramstedt, 2008).

The findings of this study also emphasise the significant impacts of CSBL and RBT on traffic injury mortality. However, even following the introduction of RBT, population alcohol consumption was still significantly associated with fatal traffic injury mortality, albeit with weaker effects during the RBT period.

The study has a number of important limitations that should be kept in mind when interpreting the findings. In the first place, while the use of an overall per-capita measure of alcohol consumption has important implications for policy, it hides substantial variation in drinking patterns across population sub-groups and over time. The key mechanism linking per-capita consumption to injuries is intoxication, and changes in drinking patterns alter the strength of this link. Thus, the differential effect sizes reported for different age/sex sub-groups and in the two different time periods may be driven partly by changes in the distribution of drinking, whereby different proportions of per-capita consumption are associated with intoxication. This is particularly relevant given some recent findings.
of polarised drinking or divergence between rates of harm and consumption (Livingston et al., 2010; Hallgren et al., 2012), although these have been contested (Rossow et al., 2014; Norström and Svensson, 2014). Further, while the use of differenced data somewhat reduces the likelihood of confounding (Norström and Svensson, 2014), this is an aggregate time-series study and the associations identified may be influenced by unmeasured factors associated with both per-capita consumption and injury mortality.

It is also worth noting that the effect sizes for the joint intervention of CSBL and RBT produced here may be over-estimated, as the impacts of other traffic injury control policies were not included in the estimation (e.g. changes in blood alcohol content levels, probationary licensing for young drivers, promoting safety features on motor vehicles and enforcing speed limits and etc.). On the other hand, the indirect impact of RBT on traffic mortality via RBT’s impact on consumption behaviour is controlled out, which may mean the effects are under-estimated.

In spite of these limitations, this study has presented analyses of what the magnitude and distribution of the reduction in injury mortality might be per litre less of per-capita alcohol consumption, suggesting that reducing alcohol consumption across the population will lead to the greatest preventive impact on fatal traffic injuries among young people and fatal non-traffic injuries among older people. Given the likely mechanism of these associations is via the injured person’s own consumption, policies that reduce consumption, particularly to intoxication, within these particular age groups are likely to be particularly effective, although there are limited examples of such targeted policies in the empirical literature. The drinking of older populations in Australia is particularly invisible in policy and research (Lynskey et al., 2003), and these findings suggest policies aimed at reducing drinking in this group could prevent a significant amount of non-traffic injury mortality. The study also finds strong evidence that policies aimed at reducing risk without necessarily reducing drinking (in this case, RBT and seatbelt laws) have the potential to produce major health benefits. Further research into potentially effective harm reduction policies for non-traffic injuries is necessary to assess the benefits of alcohol harm reduction policies on injury rates. Similarly future research into
the relationship between population level consumption and alcohol-related harms should consider the
mediating effects of harm reduction policies where appropriate. More broadly, the ongoing shifts in
alcohol consumption patterns among young people in Australia (Livingston, 2014), highlights the
potential limitations of focussing solely on per-capita consumption. Future research should
incorporate specific proxy measures of drinking pattern (potentially over shorter time-series where
data are available) and attempt to assess the impact of demographic shifts in drinking (e.g. the rise of
women’s drinking across the 20th Century).
REFERENCES


Figure legends

Figure 1. Age specific male fatal traffic injury mortality per 100,000 inhabitants and per capita alcohol consumption per inhabitant aged 15 years and above

Figure 2. Age specific female fatal traffic injury mortality per 100,000 inhabitants and per capita alcohol consumption per inhabitant aged 15 years and above

Figure 3. Age specific male fatal non-traffic injury mortality per 100,000 inhabitants and per capita alcohol consumption per inhabitant aged 15 years and above

Figure 4. Age-specific female fatal non-traffic injury mortality per 100,000 inhabitants and per capita alcohol consumption per inhabitant aged 15 years and above
Table Legends

Table 1 The development of intervention dummy variables
CSBL: Compulsory Seat Belt Legislation; RBT: Random Breath Testing
Note: * the joint events dummy is recoded by adding the population weight of each state when CSBL and RBT were introduced in that state in Australia.

Table 2 Estimated effects of a 1-litre increase in per capita male alcohol consumption on age and gender specific fatal traffic injury mortality during 1924-2006 #
WW 2: World War 2; CSBL: Compulsory Seat Belt Legislation; RBT: Random Breath Testing
Note: * p<0.05, ** p<0.01, (*p) p<0.10; # Box-Ljung Q-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with p> 0.10.

Table 3 Estimated effects of a 1-litre increase in per capita alcohol consumption on age and gender specific fatal non-traffic injury mortality during 1924-2006 #
WW 2: World War 2; CSBL: Compulsory Seat Belt Legislation; RBT: Random Breath Testing
Note: * p<0.05, ** p<0.01, (*p) p<0.10; # Box-Ljung Q-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with p> 0.10.

Table 4 Estimated effect of a 1-litre increase in per capita alcohol consumption on fatal injury mortality in periods of pre and post RBT introduction #
ARIMA: Autoregressive moving average; RBT: Random Breath Testing; S.E.: Standard Error
Note:*p<0.05, **p<0.01, (*p) p<0.10. # Box-Ljung Q-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with p> 0.10.

Table 5 The estimated effects of a 1-litre increase in per capita alcohol consumption on fatal injury mortality in Australia and other countries (age 15 year and above)
Note:*p<0.05, **p<0.01, (*p) p<0.10.
**Table 1** The development of intervention dummy variables

<table>
<thead>
<tr>
<th>Year</th>
<th>Dummy of World War 2</th>
<th>Joint events dummy of CSBL and RBT</th>
<th>Intervention events</th>
</tr>
</thead>
<tbody>
<tr>
<td>1924</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1939</td>
<td>1</td>
<td>0</td>
<td>Start of World War 2</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1945</td>
<td>1</td>
<td>0</td>
<td>End of World War 2</td>
</tr>
<tr>
<td>1946</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1970</td>
<td>0</td>
<td>0.25</td>
<td>CSBL was first introduced in Victoria state in 1970</td>
</tr>
<tr>
<td>1971</td>
<td>0</td>
<td>0.57</td>
<td>CSBL was introduced in New South Wales state in 1971</td>
</tr>
<tr>
<td>1972</td>
<td>0</td>
<td>1</td>
<td>CSBL was fully implemented in all Australian States in 1972</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1975</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>1976</td>
<td>0</td>
<td>1.25</td>
<td>RBT was first introduced in Victoria state in 1976</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>1.25</td>
<td></td>
</tr>
<tr>
<td>1980</td>
<td>0</td>
<td>1.26</td>
<td>RBT was introduced in Northern Territory 1980</td>
</tr>
<tr>
<td>1981</td>
<td>0</td>
<td>1.33</td>
<td>RBT was introduced in South Australia state in 1981</td>
</tr>
<tr>
<td>1982</td>
<td>0</td>
<td>1.67</td>
<td>RBT was introduced in New South Wales and Australian Capital Territory in 1982</td>
</tr>
<tr>
<td>1983</td>
<td>0</td>
<td>1.69</td>
<td>RBT introduced in Tasmania state in 1983</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>1.69</td>
<td></td>
</tr>
<tr>
<td>1988</td>
<td>0</td>
<td>2</td>
<td>RBT was introduced in Western Australian and Queensland states, and fully implemented in all Australian states and territories in 1988</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>2006</td>
<td>0</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

Note: *a* the joint events dummy is recoded by adding the population weight of each state when CSBL and RBT were introduced in that state in Australia.
Table 2: Estimated effects of a 1-litre increase in per capita male alcohol consumption on age and gender specific fatal traffic injury mortality during 1924-2006#

<table>
<thead>
<tr>
<th></th>
<th>15-29 years</th>
<th></th>
<th>30-49 years</th>
<th></th>
<th>50-69 years</th>
<th></th>
<th>70 years and plus</th>
<th></th>
<th>Total (15 years and plus)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Male fatal traffic injuries</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>7.30**</td>
<td>2.03</td>
<td>2.40*</td>
<td>1.08</td>
<td>2.19*</td>
<td>1.33</td>
<td>2.64</td>
<td>2.28</td>
<td>3.37**</td>
<td>0.98</td>
</tr>
<tr>
<td>WW 2 (1939-45)</td>
<td>-6.28**</td>
<td>2.07</td>
<td>-2.69*</td>
<td>1.06</td>
<td>-3.59**</td>
<td>1.25</td>
<td>-4.61</td>
<td>2.08</td>
<td>-3.46**</td>
<td>1.00</td>
</tr>
<tr>
<td>CSBL and RBT in 1970s</td>
<td>-1.44*</td>
<td>0.71</td>
<td>-0.56(0)</td>
<td>0.36</td>
<td>-0.89*</td>
<td>0.43</td>
<td>-1.47</td>
<td>0.69</td>
<td>-0.80**</td>
<td>0.34</td>
</tr>
<tr>
<td>Constant</td>
<td>1.32</td>
<td>0.84</td>
<td>0.54</td>
<td>0.43</td>
<td>0.78</td>
<td>0.51</td>
<td>1.15</td>
<td>0.82</td>
<td>0.71</td>
<td>0.41</td>
</tr>
<tr>
<td>(Q) (lag 10)</td>
<td>5.42, (p=0.86)</td>
<td></td>
<td>4.10, (p=0.90)</td>
<td></td>
<td>2.24, (p=0.69)</td>
<td></td>
<td>14.08, (p=0.12)</td>
<td></td>
<td>7.47, (p=0.68)</td>
<td></td>
</tr>
<tr>
<td>Model specification</td>
<td>(0,1,0)</td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td>(0,1,0)</td>
<td></td>
</tr>
</tbody>
</table>

| Female fatal traffic injuries |             |            |             |            |             |            |                   |            |                          |            |
| Algebraic consumption | 0.51(0)     | 0.32       | 0.34(0)     | 0.19       | 0.38        | 0.51       | 1.28              | 0.99       | 0.46*                    | 0.24       |
| WW 2 (1939-45)   | -1.09**     | 0.29       | -0.58**     | 0.16       | -1.01*      | 0.41       | -3.01**           | 0.91       | -0.94**                  | 0.22       |
| CSBL and RBT in 1970s | -0.49 ** | 0.09       | -0.21**     | 0.05       | -0.44*      | 0.18       | -0.33*            | 0.30       | -0.34**                  | 0.07       |
| Constant         | 0.47*       | 0.11       | 0.18        | 0.06       | 0.35        | 0.23       | 0.64(0)          | 0.35       | 0.29*                    | 0.08       |
| \(Q\) (lag 10)  | 5.45, \(p=0.79\) |           | 3.03, \(p=0.93\) |           | 4.39, \(p=0.82\) |           | 5.59, \(p=0.78\) |           | 12.75, \(p=0.12\)     |           |
| Model specification | (0,1,1) |            | (2,1,1)     |            | (2,1,1)     |            | (0,1,1)         |            | (2,1,1)                  |            |

Note: * \(p<0.05\), ** \(p<0.01\), (0) \(p<0.10\); *Box-Ljung \(Q\)-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with \(p> 0.10\).
### Table 3 Estimated effects of a 1-litre increase in per capita alcohol consumption on age and gender specific fatal non-traffic injury mortality during 1924-2006

<table>
<thead>
<tr>
<th>Gender</th>
<th>City</th>
<th>15-29 years</th>
<th>Coef.</th>
<th>S.E.</th>
<th>30-49 years</th>
<th>Coef.</th>
<th>S.E.</th>
<th>50-69 years</th>
<th>Coef.</th>
<th>S.E.</th>
<th>70 years and plus</th>
<th>Coef.</th>
<th>S.E.</th>
<th>Total (15 years and plus)</th>
<th>Coef.</th>
<th>S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male fatal non-traffic injuries</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td></td>
<td></td>
<td>3.52**</td>
<td>0.91</td>
<td></td>
<td>3.32**</td>
<td>1.00</td>
<td></td>
<td>3.58**</td>
<td>1.37</td>
<td></td>
<td>9.12*</td>
<td>3.63</td>
<td></td>
<td>2.97**</td>
<td>0.62</td>
</tr>
<tr>
<td>WW 2 (1939-45)</td>
<td></td>
<td></td>
<td>-2.18**</td>
<td>0.82</td>
<td></td>
<td>-1.82*</td>
<td>0.98</td>
<td></td>
<td>-0.35</td>
<td>1.33</td>
<td></td>
<td>3.39</td>
<td>3.35</td>
<td></td>
<td>-0.47</td>
<td>0.57</td>
</tr>
<tr>
<td>CSBL and RBT in 1970s</td>
<td></td>
<td></td>
<td>0.62</td>
<td>0.26</td>
<td></td>
<td>0.69</td>
<td>0.33</td>
<td></td>
<td>0.98</td>
<td>0.44</td>
<td></td>
<td>2.42</td>
<td>1.05</td>
<td></td>
<td>0.79</td>
<td>0.18</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td></td>
<td>-1.03**</td>
<td>0.31</td>
<td></td>
<td>-1.07</td>
<td>0.40</td>
<td></td>
<td>-1.88**</td>
<td>0.53</td>
<td></td>
<td>-3.81**</td>
<td>1.27</td>
<td></td>
<td>-1.27**</td>
<td>0.22</td>
</tr>
<tr>
<td>Q (lag 10)</td>
<td></td>
<td></td>
<td>12.04, p=0.21</td>
<td></td>
<td></td>
<td>8.04, p=0.53</td>
<td></td>
<td></td>
<td>12.70, p=0.18</td>
<td></td>
<td></td>
<td>11.71, p=0.19</td>
<td></td>
<td></td>
<td>11.68, p=0.17</td>
<td></td>
</tr>
<tr>
<td>Model specification</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td></td>
<td>(1,1,0)</td>
<td></td>
<td></td>
<td>(2,1,1)</td>
<td></td>
<td></td>
<td>(2,1,1)</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
</tr>
<tr>
<td>Female fatal non-traffic injuries</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td></td>
<td></td>
<td>0.38**</td>
<td>0.10</td>
<td></td>
<td>0.54*</td>
<td>0.24</td>
<td></td>
<td>0.79(*)</td>
<td>0.44</td>
<td></td>
<td>7.24(*)</td>
<td>4.64</td>
<td></td>
<td>0.86*</td>
<td>0.40</td>
</tr>
<tr>
<td>WW 2 (1939-45)</td>
<td></td>
<td></td>
<td>-0.27**</td>
<td>0.06</td>
<td></td>
<td>-0.22</td>
<td>0.21</td>
<td></td>
<td>-0.00</td>
<td>0.40</td>
<td></td>
<td>7.44</td>
<td>4.38</td>
<td></td>
<td>0.38</td>
<td>0.38</td>
</tr>
<tr>
<td>CSBL and RBT in 1970s</td>
<td></td>
<td></td>
<td>0.11</td>
<td>0.02</td>
<td></td>
<td>0.12</td>
<td>0.12</td>
<td></td>
<td>0.13</td>
<td>0.12</td>
<td></td>
<td>1.06</td>
<td>1.39</td>
<td></td>
<td>0.19</td>
<td>0.12</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td></td>
<td>-0.13**</td>
<td>0.02</td>
<td></td>
<td>-0.14</td>
<td>0.07</td>
<td></td>
<td>-0.30*</td>
<td>0.14</td>
<td></td>
<td>-2.20</td>
<td>1.68</td>
<td></td>
<td>-0.25</td>
<td>0.15</td>
</tr>
<tr>
<td>Q (lag 10)</td>
<td></td>
<td></td>
<td>7.39, p=0.60</td>
<td></td>
<td></td>
<td>12.09, p=0.21</td>
<td></td>
<td></td>
<td>8.55, p=0.48</td>
<td></td>
<td></td>
<td>8.89, p=0.18</td>
<td></td>
<td></td>
<td>7.52, p=0.58</td>
<td></td>
</tr>
<tr>
<td>Model specification</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
<td></td>
<td>(1,1,2)</td>
<td></td>
<td></td>
<td>(0,1,1)</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** *p<0.05, **p<0.01, (*)p<0.10. **Box-Ljung Q-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with p> 0.10.
Table 4 Estimated effect of a 1-litre increase in per capita alcohol consumption on fatal injury mortality in periods of pre and post RBT introduction

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effects</td>
<td>S.E.</td>
</tr>
<tr>
<td>Male</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Traffic injuries</td>
<td>4.32**</td>
<td>1.30</td>
</tr>
<tr>
<td>Non-traffic injuries</td>
<td>3.25**</td>
<td>0.67</td>
</tr>
<tr>
<td>Female</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Traffic injuries</td>
<td>0.79*</td>
<td>0.54</td>
</tr>
<tr>
<td>Non-traffic injuries</td>
<td>0.94*</td>
<td>0.46</td>
</tr>
</tbody>
</table>

Note: *p<0.05, **p<0.01, 'p<0.10. Box-Ljung Q-tests were satisfactory with regards to autocorrelation of residuals, and non-normal errors in the residuals with p> 0.10.
Table 5 The estimated effects of a 1-litre increase in per capita alcohol consumption on fatal injury mortality in Australia and other countries (age 15 year and above)

<table>
<thead>
<tr>
<th>Countries and authors</th>
<th>Period</th>
<th>Traffic Male</th>
<th>Traffic Female</th>
<th>Non-traffic Male</th>
<th>Non-traffic Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>1924-2006</td>
<td>3.37**</td>
<td>0.46*</td>
<td>2.97**</td>
<td>0.86*</td>
</tr>
<tr>
<td>Canada; Skog (2003)</td>
<td>1950-2002</td>
<td>3.60*</td>
<td>0.70*</td>
<td>0.30*</td>
<td>0.10</td>
</tr>
<tr>
<td>14 European Countries; Skog (2001a)</td>
<td>1950-1995</td>
<td>1.28**</td>
<td>0.38**</td>
<td>0.95**</td>
<td>0.23</td>
</tr>
<tr>
<td>Northern Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Southern Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The U.S; Ramstedt (2008)</td>
<td>1950-2002</td>
<td>3.18*</td>
<td>0.73*</td>
<td>1.10*</td>
<td>0.29</td>
</tr>
</tbody>
</table>

Note: *p<0.05, **p<0.01, [*] p<0.10.