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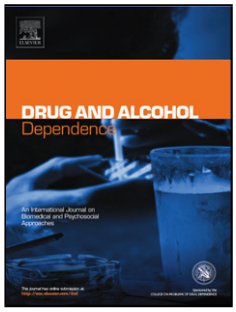

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Child injury deaths linked with adult alcohol consumption: A time series analysis*

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Highlights

- Novel study analysed 100 years of external child mortality and alcohol consumption.
- A 10% decrease in drinking was associated with a 3.3% reduction in child deaths.
- Compulsory seatbelts and random breath testing laws were effective.
- A decreased minimum drinking age increased some types of external child mortality.
- Alcohol control policies led to small but significant reductions in child deaths.

Abstract

Objective: This paper aims to quantify the population-level associations between child injury deaths and adult (aged 15+ years) per capita alcohol consumption (PCC) and between child injury deaths and the impact of major alcohol and safety policy changes in Australia.

Methods: All child deaths due to external causes during 1910-2013, and child deaths due specifically to road crashes, assaults, suicide and other external causes, were obtained from the Australian Institute of Health and Welfare. Child (0-14 year) mortality rates were analysed in relation to PCC using an Autoregressive Integrated Moving Average model.

Results: A positive association between PCC and overall child external mortality was identified. The estimated coefficient was 0.326 (p=.002), indicating that a 10% decrease in PCC was associated with a 3.3% reduction in child mortality. A positive association was identified for road traffic and other child mortality, but not assault injuries. The introduction of compulsory seatbelt legislation in combination with random breath testing was associated with a reduction in overall and road traffic child mortality. Decreasing the legal drinking age was associated with an increase in the rate of other external-cause child mortality.

Conclusion: Reducing PCC in Australia is likely to result in a small but significant reduction in the mortality rate of children aged 0-14 years.

Keywords: Per Capita Alcohol Consumption; Child Injury Mortality; Harm to Others

1. Introduction

Severe harms to children, including homicide (Cussen and Bryant, 2015), child abuse and neglect (Chaffin et al., 1996; Forrester and Harwin, 2008; Laslett et al., 2013; Laslett et al., 2012b), and unintentional injuries (Raitasalo and Holmila, 2017) have been associated with parents' and other adults' drinking in individual-level studies. In eight countries, episodic drinking of adult respondents (six drinks or more on one occasion in the last month or more frequently) was associated with a range of harms to children (Laslett et al., 2017). However, associations between population-level adult alcohol consumption (PCC), population-wide alcohol-related policy interventions, and harms to children have not been scrutinized; we address this research gap.

Although previous analyses have shown significant associations between PCC and adult external-cause injuries and deaths (Jiang et al., 2015; Norström, 2011; Ramstedt, 2011), no such analysis has been done among children aged 0-14. We hypothesise that population-level child external-cause injuries, such as traffic injuries, accidental falls, drownings, poisonings, and homicides, will demonstrate similar kinds of associations over time with PCC as have been measured in the adult population. Time series studies to date have unrealised potential to analyse the relationship between aggregate alcohol consumption, alcohol interventions, and harms to children from injuries associated with alcohol consumption by parents and other adults in Australian society.

1.1 Alcohol and child injuries

Injuries are among the leading causes of child and youth deaths worldwide (Patton et al., 2009; Peden, 2008). Children as passengers, pedestrians, and bicyclists are heavily involved in alcohol-related traffic crashes (Margolis et al., 2000; Quinlan et al., 2014). Child

injuries, including accidental falls, drownings, burns, and poisonings may result when parents' supervision is compromised, their attention and priorities are diverted (Schnitzer et al., 2015), and parents' or others' usual controls are disarmed (Famularo et al., 1992; Fillmore, 2012) in heavy drinking or other drinking situations. Alcohol is held responsible for a substantial proportion of child injuries using the aetiological fraction methodology. In New Zealand, 16-25% of assaults, 20% of road traffic injuries, 24% of fire injuries, and 6-19% of other unintentional injuries to children were attributed to alcohol (Connor et al., 2013). What is less well understood is how adult drinking levels and policy changes affect child deaths over time.

The child injury death rate has been declining in many high-income countries around the world (National Public Health Partnership, 2004; Peden, 2008). Many regulations and laws support injury prevention interventions by, for instance, making seatbelts and child car restraints, childproof caps on medicines, and pool fencing mandatory (National Public Health Partnership, 2004; Peden, 2008). Alcohol-specific regulations and policies also seek to protect children (and adults). In Australia, the minimum legal drinking age (MLDA) is 18 years (Manton et al., 2014). However, children are still potentially influenced by others' drinking in public (e.g., in drunk driving crashes and assaults) and in their own homes.

1.2 Parental and other adults' drinking

Heavy drinking is widespread in Australian communities (AIHW, 2014) and amongst parents (Maloney et al., 2010). Substantial proportions of parents in other countries also drink; for example, Finland (Raitasalo, 2011), the United Kingdom (Manning et al., 2009) and the United States (Bijur et al., 1992). In Australia, 79% of drinkers living with children consume alcohol in their presence (FARE, 2013), 31% of respondents with children drink at short-term risky levels at least monthly (Maloney et al., 2010), and 17% of carers report

family members' heavy drinking has resulted in verbal abuse, exposure to domestic violence, poor supervision or physical injury to children (Laslett et al., 2012a).

Numerous plausible theories exist regarding why children are harmed when more alcohol is consumed by adults in proximity to children. The physical effects of intoxication include decreased reaction time, lack of coordination, adverse effects on cognition and memory, and eventually unconsciousness (Babor et al., 2010), none of which are compatible with supervision, the physical tasks involved in parenting, or being emotionally available. Alcohol is theoretically linked to child harms via disinhibition and expectancy theories wherein drinkers act out of character and do things they otherwise would not, or perceive that with alcohol comes some relaxation (within limits) of the social norms they would usually follow (Källmén and Gustafson, 1998). Alcoholic 'myopia' may also remove peripheral cues, as drinkers relax and focus on their own needs over those of others (Steele and Josephs, 1990).

In Australian child death reviews of children in contact with child protection systems, 19-50% of child deaths involved one or both carers having a history of alcohol abuse (NSW Child Death Review Team, 2003; Victorian Child Death Review Committee, 2011). Despite such knowledge, relatively few studies have focused on the role of alcohol in child mortality. In the United States, between 1982 and 2003, 29% of the 35,282 children killed in motor-vehicle crashes were in crashes that involved a drinking driver (Williams, 2006). In Norway, significant association between parental drinking to the point of intoxication and adolescent suicide was identified (Rossow and Moan, 2012).

1.3 Population drinking and mortality

To date, time series studies have consistently linked PCC with adult road crash, suicide, non-traffic unintentional injury, interpersonal violence, homicide, all injury and all-cause mortality (Arranz and Gil, 2009; Caces and Harford, 1998; Carpenter and Dobkin,

2007; Hingson and Howland, 1993; Jayasekara et al., 2013; Kerr et al., 2011; Koski et al., 2007; Landen et al., 1997; Leon et al., 1997; Lester, 1995; Mann et al., 2006; Norström, 2011; Norström and Ramstedt, 2005; Norton and Morgan, 1989; Pearn et al., 1989; Ramstedt, 2001; Ramstedt, 2004; Skog, 2003; Skog, 2001a,b). Despite these results, with one exception, the relationship between measures of alcohol availability and child deaths has not been analysed (Sen, 2006). Sen (2006) found a relationship in the U.S. between higher beer taxes and lower rates of child homicide and a direct relationship between alcohol retail outlet density and child homicides.

If causal associations exist, then restrictive alcohol policies should be effective in reducing alcohol-related harms to children, including fatalities (e.g., road traffic interventions should also be effective in reducing harms to children). There is good evidence that changes in PCC lead to changes in rates of adult alcohol-related injury and mortality (Babor et al., 2010). There is a strong correlation between alcohol sales and liquor licence density with a range of social problems including homicide, assault and domestic violence (Livingston, 2010; Rossow, 2000; Rossow, 2001). Given that Sen's study suggests that taxation and liquor licence density policies (presumably through their action on reducing sales and then consumption) may be linked to reductions in child mortality (Sen, 2006), more time series studies of alcohol's harm to others, alcohol consumption, and alcohol policy interventions are needed to inform such arguments.

Public health interventions, such as compulsory seatbelt legislation (CSBL) and child car restraints, have been effective in reducing the road death toll amongst adults (Crandall et al., 2001; Trinca and Dooley, 1975) and children (Elliott et al., 2006; Osberg and Di Scala, 1992). Alcohol-specific interventions such as the introduction of random breath testing (RBT) and increasing the minimum legal drinking age (MLDA) have also been shown to reduce rates of adult road traffic mortality (Erke et al., 2009; Jiang et al., 2014). However,

RBT is not only a traffic safety intervention but also an alcohol policy intervention. Although the RBT was designed to reduce drunk driving behaviour, it may also reduce adults' drinking as well. Furthermore, a reduction in the MLDA (from 21 to 18) is likely to not only increase PCC but decrease the age of drinking initiation among young adolescents. Indeed, evidence exists that an increase in young people's drinking and a decrease in the age of initiation will result in more violence towards children and adolescent traffic injuries (Hingson et al., 2001; Jones et al., 1992).

This paper aims specifically to examine the associations between population-level child external causes of mortality (including overall external causes of child mortality and specific types of external mortalities, such as traffic fatalities, assaults and "other" external mortalities) and PCC between 1910 and 2013 in Australia. It additionally aims to evaluate the effects of the implementation of alcohol policy and traffic safety interventions on child injury deaths.

2. Methods

2.1 Data

All child (0-14 year) deaths per annum due to external causes for the years 1910-2013, and, separately, sub-sets of child deaths due to road traffic crashes, assaults, suicide and other external causes of mortality (including accidental drowning, suffocation/strangulation/hanging, mechanical force, burn and scald, fall, poisoning and other causes) were obtained from the Australian Institute of Health and Welfare (AIHW) based on the appropriate International Classification of Diseases 10 codes (the standardised historical mortality databases were developed by AIHW, which track the relevant cause of death codes across the various classification schemes used in Australia). Historical data on land transport/traffic injury mortality was only available from 1924 to 2013. Data on all external cause, assault, traffic accident, and suicide mortality were provided, enabling calculation of a

proxy series for 'other' externally caused child mortality (i.e., equal to [all external cause – traffic injury – assault – suicide]). Mortality rates were age-standardized and expressed per 100,000 children (population aged 0-14 years), using indirect standardization to the 2001 population age structure. Initially the suicide sub-set of data was analysed separately, but it included around ten years with no deaths and numbers were too few to present reliable results.

A proxy for PCC was constructed using data on the sale of alcohol sourced from the Australian Bureau of Statistics (ABS, 2015). Early data before 1960 were sourced from historical publications (Australian Year Book, i.e., 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, wine and spirits based on a report that provided snapshots of total volume and pure alcohol volume once per decade (ABS, 1994).

2.2 ARIMA Model with intervention dummies

A two-stage analysis approach was undertaken in this study: first, a log-log autoregressive integrated moving average (ARIMA) model was employed to examine the relationship between changes in PCC and changes in child injury external mortality rates. Secondly, whether specific hypothesised alcohol policy (lowering the minimum drinking age) and traffic safety interventions (random breath testing and compulsory seatbelt legislation) could affect the changes in child mortality was analysed separately. Child injury deaths associated with adult drinking ought to occur soon after alcohol is consumed, so lagged effects were not considered. Adapting similar methods used in a previous study (Jiang et al., 2015), an ARIMA model applying the Box-Jenkins approach (Box and Jenkins, 1970) was

used to estimate the associations between PCC and children's external cause mortality. Further details regarding the methods are included in the Supplementary Information[1].

Because compulsory seatbelt legislation and random breath testing were both implemented in a similar timeframe in the 1970s, and both have been shown to have long-term and on-going preventive effects on traffic fatalities in Australia after the 1970s (Jiang et al., 2014; Trinca and Dooley, 1975), a joint-events dummy variable was constructed. This variable represents their combined impact on unintentional injuries and was constructed using the roll-out approach described by Jiang et al. (2015). Compulsory seatbelt legislation was first implemented in the Australian state of Victoria in 1970, and the joint-events dummy variable was coded as 0.25 (population weight of Victoria in Australia) in 1970 and 0 between 1924 and 1969. The joint-events dummy was recoded as 0.57 in 1971 and 1 in 1972, as the compulsory seatbelt legislation was implemented in New South Wales in 1971 and then fully implemented in all states in 1972. The random breath testing program was first introduced in Victoria in 1976, so the joint dummy variable was further coded as 1.25 in 1976. After that, the joint-events dummy was recoded, adding the population weight of each state when random breath testing was introduced in that state. The joint dummy was finally coded as 2 after 1987, as the two interventions were fully implemented in all states in Australia.

The effects of World War II (WWII) on injury mortality were also controlled by setting up a WWII dummy variable. A further set of analyses was conducted in an attempt to control for the effects of petrol rationing during and after the war period and the impacts of lowering minimum legal drinking age from 21 to 18 years in four Australian states. Protective effects were assumed in the period of a higher minimum legal drinking age, while lower minimum legal drinking ages after 1967 were expected to increase alcohol-related

[1] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi…

traffic fatalities (Smith, 1986). Therefore, the minimum legal drinking age dummy variable started at 1 between 1924 and 1967 (the prevention event on) and then changed from 1 to 0.6 when the minimum legal drinking age reduced (the prevention event off) in the four Australian states between 1967 and 1974. The minimum legal drinking age in the other four states has been 18 years of age since the 1900s. The dummy variables developed for these major events and alcohol policy changes that were applied in the time series models are described in Table 1.

Furthermore, the introduction of the Swimming Pools Fencing Act in 1992 (Government of New South Wales, 1992) and Mandatory Bicycle Helmet laws in 1990 (Centers for Disease Control Prevention, 1993) in Australia were also converted to intervention dummies in our models, but we found no significant impact on overall, traffic and other external child injury mortalities. Thus, the impacts of these two policy interventions were not considered in the final estimation.

The model fit for each time series analysis was evaluated using the Box-Ljung portmanteau test of the first 10 autocorrelations, $Q(10)$.

3. Results

Trends for PCC and children's overall and specific types of external causes of mortality are presented in Figure 1. PCC fluctuated between 1910 and 2013; consumption bottomed during the Second World War (1939-1945), peaked in the 1970s, declined in the 1980s, and has been relatively steady in the past 20 years. Children's all-external-causes-of-mortality rates have declined steadily in the last 100 years from 54 to 4 per 100,000 children in Australia. In contrast, children's traffic mortality rates increased between 1924 and 1970 and decreased gradually after the 1970s.

Figure 2 shows the scatter of changes (both variables first differenced) in PCC and overall child mortality during 1910 and 2013. A positive correlation is suggested between

changes in PCC and changes in overall child mortality. Using first differenced data, long-term trends in alcohol consumption and child external cause mortality rates were removed to achieve stationarity.

Estimated associations between PCC and overall and specific external causes of mortality without controlling for any potential confounders are summarized in Table 2. The estimated coefficient was 0.326 (p=.0002) in the overall external cause mortality model, suggesting that if PCC is decreased by 10%, the child mortality rate will be reduced by 3.3%. Specific associations between PCC and traffic crashes (0.686, p<.01) and other external mortality models (0.199, p<0.1) indicated that for a 10% decline in PCC, mortality rates of children's traffic crashes and other external causes would be reduced by almost 7% and 2%, respectively.

Table 3 shows the effects of key alcohol policy and traffic safety interventions on overall and specific external causes of mortality. There was a significant negative impact associated with WWII on children's traffic and other external cause mortality. The introduction of compulsory seatbelt legislation and random breath testing in the 1970s was associated with significant reductions in both children's overall and traffic crash mortality. Lowering of the minimum legal drinking age led to a significant increase in the child mortality rate from other external causes.

Given that societal conditions over a long period of time can change, we conducted a sensitivity analysis (see Supplementary Information, Tables A1-A4[2]) to test the robustness of our findings and applied ARIMA to a shorter and more recent period from 1960 onwards. Findings for the full sample and shorter sample time series models were similar including, critically, PCC and overall child external cause mortality and the effects of key alcohol policy and traffic safety interventions.

[2] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi…

The models presented here have not controlled for a range of factors (e.g., macroeconomic conditions, unemployment and motor vehicle ownership rates), nor have they included and studied simultaneously the effects of key alcohol policy or traffic safety regulation changes over and above the effects of PCC. Tables A3 and A4 in the Supplementary Information[3] present combined models.

4. Discussion

In line with previous individual child protection cases and injury case series reports involving others' drinking and child injury (NSW Child Death Review Team, 2003; Victorian Child Death Review Committee, 2011), this study provides the first research evidence that population-level alcohol consumption is significantly associated with overall child injury mortality. This association is consistent with the literature that has examined the association between PCC and adult traffic crash fatalities (Jiang et al., 2015) and all-cause mortality among adults (Jayasekara et al., 2014; Roerecke and Rehm, 2013). The association between child assault deaths and PCC was not significant, although child assault deaths were infrequent. This is inconsistent with an association identified previously between PCC and adult homicide among Australian adults in Australia (Ramstedt, 2011) and event level information, where in 35% of non-fatal family violence incidents involving children, the victim reported that the perpetrator was under the influence of alcohol or drugs at the time (Durose et al., 2005). In our study, the mortality rate of children who died from "other external causes of injury" was associated with overall PCC (at the $p<0.10$, a marginal level, but one that has been used in other time series studies, e.g., Kerr et al., 2011; Norström, et al., 2001). The major causes of other external cause child mortality injuries in Australia are drownings, accidental suffocation/strangulation/hanging, animate or inanimate forces (e.g., hit by rocks, sports equipment, crushing injuries), burns, falls and poisonings, (ABS, 2006).

[3] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi…

The aetiology of these injuries involves the critically important role of supervision, which is affected by parental drinking and intoxication (Peden et al., 2016).

Turning to the major interventions tested in the model, the introduction of compulsory seatbelt legislation, in combination with random breath testing, was significantly associated with a decrease in child traffic fatalities as has been identified previously for adult road traffic deaths (Jiang et al., 2014; Jiang et al., 2015). A decrease in the minimum legal drinking age from 21 to 18 years in four of the eight Australian states and territories was not associated with overall increases in child mortality but was associated with an increase in child injury deaths due to other causes. This leads us to question how young people's alcohol consumption might affect other children (i.e., siblings) in the family and whether removal of restrictions on young parental drinking affects behaviour and supervision which in turn increase children's risk of unintentional injuries.

A decrease in the rate of child traffic fatalities was identified during World War II, potentially affected because fewer young men were present, and as both alcohol and petrol were rationed.

Figure 1 shows increasing alcohol consumption between 1930 and 1978 and decreasing external cause child mortality. At face value, this is inconsistent with the model we have included in Tables 2 and 3. However, differencing the series to achieve stationarity means that longer-term trends that may be related to a host of causes (e.g., antibiotics, improving medical care) are set aside, while relationships underpinned by changes that occur each year and from one year to the next allow a focus on the impact of alcohol consumption-related changes and child external cause deaths.

In the Supplementary Information (Table A3[4]) we present a more complex model that estimates the associations between adult alcohol consumption and child mortality, controlling for the effects of key alcohol policy and traffic safety interventions. The results demonstrate that there is negligible difference between controlled and uncontrolled models, supporting the consistency of our findings. However, the slight attenuation of the effect of PCC in the adjusted model provides some evidence that the mechanism via which the alcohol policy interventions work is by reducing PCC.

4.1 Limitations

A considerable strength of this study is that it applies time series data from an entire country, over a hundred-year period, enabling investigation of a range of policies. Nevertheless, even though it is unlikely that child injury deaths influence changes in PCC, the study design only allows a test of association and not causality. Due to a lack of information over the long time period of this study, we have not been able to include data on a range of potential factors. For instance, changes in gendered drinking patterns may have contributed to level of supervision; however, data did not differentiate men's and women's drinking patterns. The consumer price index of alcohol has varied over time, but data are only available from 1972. The proportion of the population that drinks riskily or heavily is also not availabl,e as surveys of drinking patterns did not commence in Australia until 1985 (Australian Institute of Health and Welfare, 2014). Per capita consumption has been shown to be related to heavy drinking levels within a country and was the best measure available to us (Livingston, 2015). A range of single parent pensions and family services introduced (and retracted) in Australia in the last 100 years may also have contributed to declining death rates among children. Finally, recent increases in injury surveillance and prevention and public awareness of child safety, e.g., pool fencing, and child resistant medicine caps, are

[4] Supplementary material can be found by accessing the online version of this paper at http://dx.doi.org and by entering doi…

recommended as effective interventions to address other specific child injuries (Peden, 2008), but the relationship between these environmental countermeasures and child death rates has been difficult to prove effective given that the time points for their introduction is uncertain (Thompson and Rivara, 1998). Nevertheless, use of differenced data in the ARIMA models reduces the risk of this type of confounding.

Importantly, while these data are specific to Australia, our investigation of the temporal relationship between adult consumption of alcohol and child injury deaths and the impact of the implementation of alcohol policies designed to reduce childhood injury mortality is relevant to other countries which report similar concerns about alcohol-related child injury deaths.

Conclusion

This study supports an emerging body of evidence of the impact of adult drinking on children, which suggests that policy interventions to reduce population drinking levels could lead to a significant reduction in child mortality.

Author Disclosures

Contributors

Laslett conceptualised and wrote the majority of the paper. Jiang undertook the time series analysis and described the results. Chikritzhs contributed to the writing of the introduction, results and discussion, and edited multiple drafts of the paper. All authors have approved the final article.

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Conflict of Interest

No conflict declared by any of the authors.

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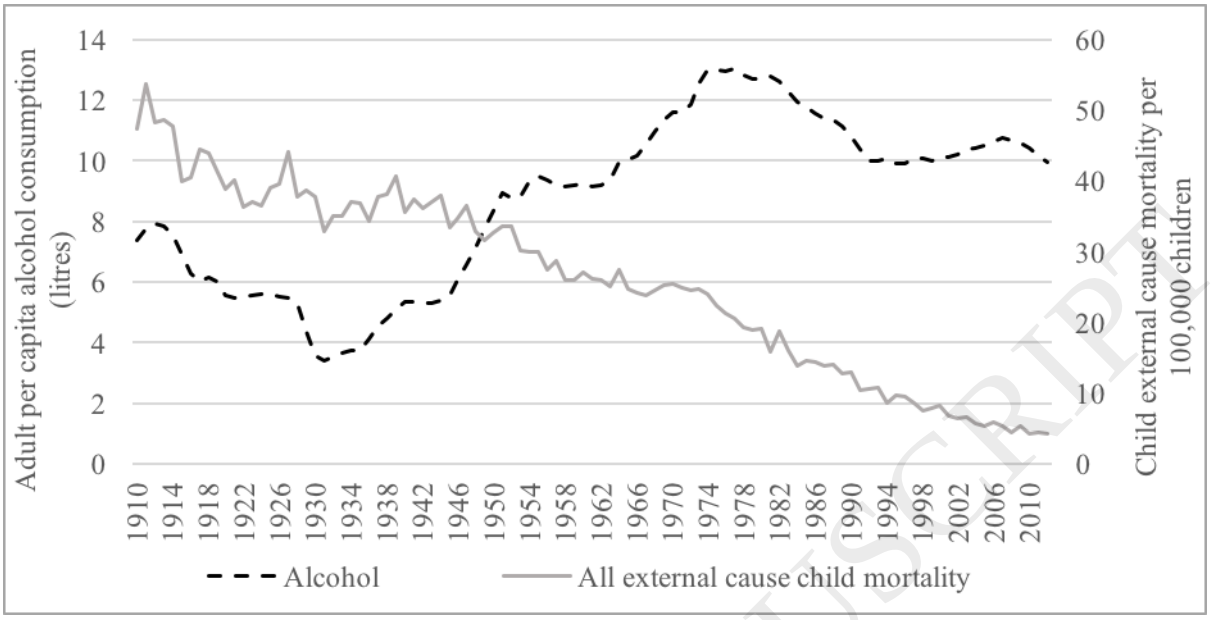
Figure Legends

Figure 1. Adult per capita alcohol consumption (15+) and overall and children's (0-14 yrs) specific types of child external cause mortality rates (per 100,000 children) in Australia, 1910-2013

Figure 2. Scatter chart of changes in (first differenced) adult per capita alcohol consumption (15+) and changes in overall child external cause mortality rate (0-14 years) per 100,000 children in Australia, 1910-2013

Figure 1.



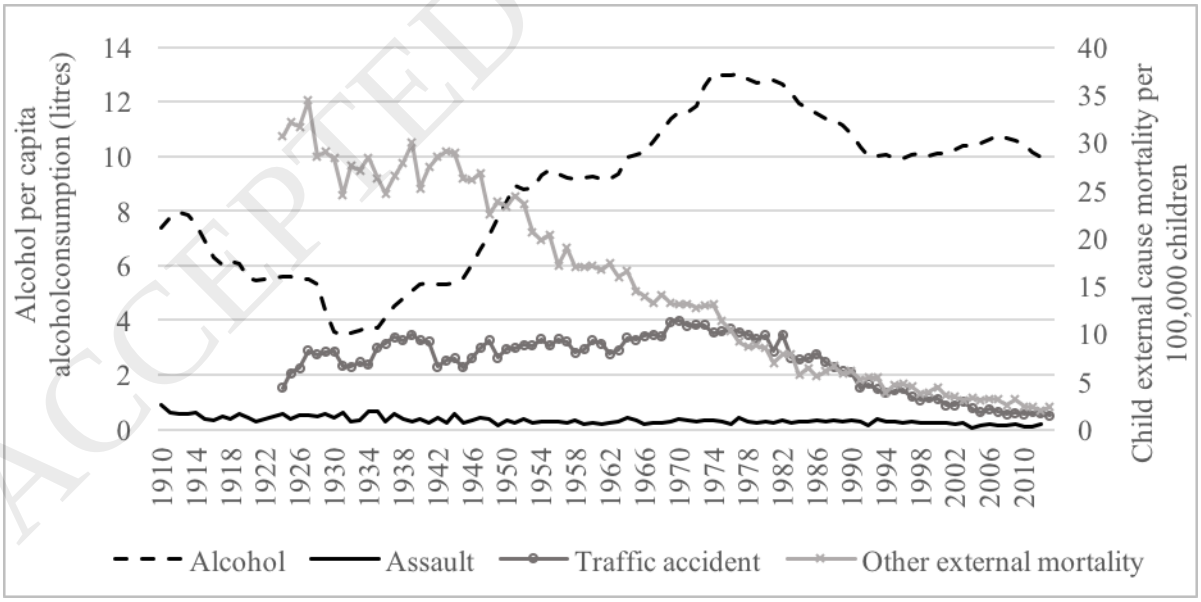


Figure 2.

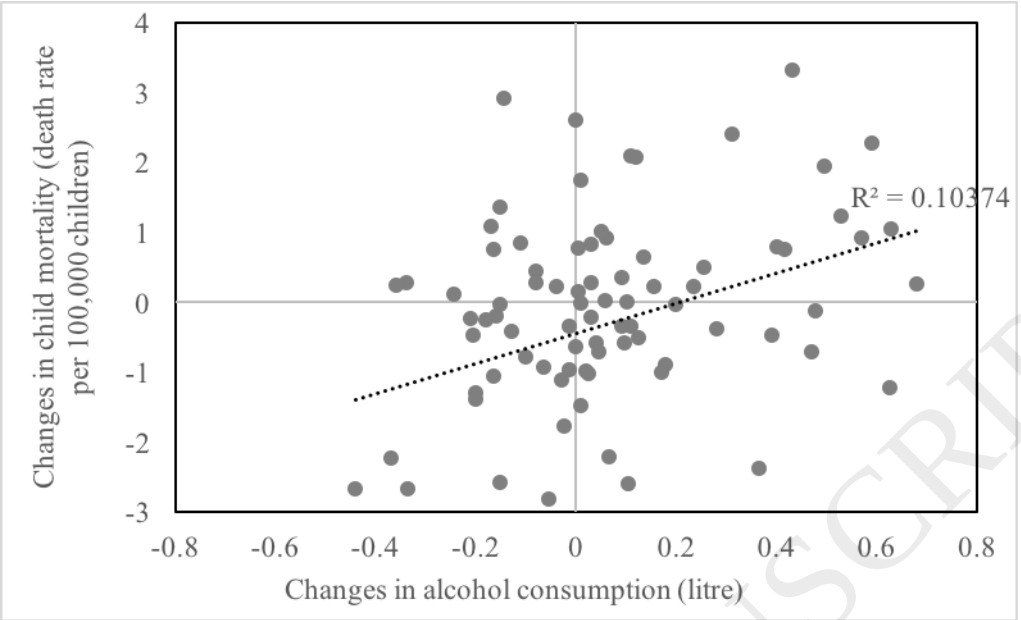


Table 1. Intervention dummy variables for major events and alcohol policy changes

Year	World War II	Joint events of CSBL and RBT [a]	MLDA [a]	Intervention events
1910	0	0	1	
⋮	0	0	1	
1939	1	0	1	Start of World War II
⋮	1	0	1	
1945	1	0	1	End of World War II
1946	0	0	1	
⋮	0	0	1	
1967	0	0	0.98	The MLDA was lowered from 21 to 20 in Tasmania in 1967.
1968	0	0	0.95	The MLDA was lowered from 21 to 20 in South Australia in 1968.
1969	0	0	0.95	
1970	0	0.25	0.85	CSBL was first introduced in Victoria state in 1970; The MLDA was lowered from 21 to 18 in Western Australia in 1968.
1971	0	0.57	0.80	CSBL was introduced in New South Wales state in 1971; The MLDA was lowered from 20 to 18 in South Australia in 1971.
1972	0	1	0.80	CSBL was fully implemented in all Australian States in 1972.
⋮	0	1	0.80	
1974	0	1	0.60	The MLDA in Tasmania was lowered from 20 to 18 in 1974 and in Queensland it was lowered from 21 to 18.
1975	0	1	0.60	
1976	0	1.25	0.60	RBT was first introduced in Victoria state in 1976
⋮	0	1.25	0.60	
1980	0	1.26	0.60	RBT was introduced in Northern Territory 1980
1981	0	1.33	0.60	RBT was introduced in South Australia state in 1981
1982	0	1.67	0.60	RBT was introduced in New South Wales and Australian Capital Territory in 1982
1983	0	1.69	0.60	RBT introduced in Tasmania state in 1983
⋮	0	1.69	0.60	
1988	0	2	0.60	RBT was introduced in Western Australian and Queensland states, and fully implemented in all Australian states and territories in 1988
⋮	0	2	0.60	
2013	0	2	0.60	

Note: CSBL: compulsory seatbelt legislation; RBT: random breath testing; MLDA: minimum legal drinking age. [a] the joint event and MLDA dummies is recoded by adding the population weight of each state when CSBL and RBT were introduced and the MLDA was changed in that state in Australia. The MLDA in the other four states has been 18 years since the 1900s.

Table 2. Adult per capita alcohol consumption (15 yrs+) and external cause child (0-14 yrs) mortality in Australia without considering the impacts of key events or policy interventions (log-log ARIMA model) between 1910 and 2013

	Overall external cause mortality		Traffic accident		Assault		Other external cause mortality	
	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.
Alcohol consumption	0.326**	0.103	0.686**	0.188	0.045	0.229	0.199$^{(*)}$	0.117
Constant	-0.026**	0.005	-0.027	0.008	-0.015*	0.006	-0.030**	0.004
Model specification	(3,1,1)		(3,1,1)		(0,1,1)		(0,1,1)	
Q (lag 10)	12.398, *p*=0.134		8.379, *P*=0.397		6.056, *p*=0.733		8.428, *p*=0.492	

Note: $^{(*)}p$ <0.10, *p <0.05, **p <0.01; Other external cause mortality (including accidental falls, drowning, poisoning and other external causes of mortalities); D indicates an intervention dummy variable.

Table 3. Effects of key alcohol policy and traffic safety interventions on external cause child (0-14 yrs) mortality in Australia between 1910 and 2013

	Overall external cause mortality		Traffic accident		Assault		Other external cause mortality	
	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.
D(World War II)	0.007	0.011	-0.036**	0.011	-0.043	0.034	0.030*	0.014
D(CSBL & RBT)	-0.019*	0.009	-0.052**	0.008	-	-	-	-
D(MLDA)	-0.006	0.047	-0.066	0.041	0.043	0.031	0.038**	0.014
Constant	-0.007	0.047	0.081*	0.040	-0.047	0.025	-0.062**	0.011
Model specification	(0,1,1)		(0,1,1)		(0,1,1)		(0,1,1)	
Q (lag 10)	9.001, *p*=0.437		12.213, *P*=0.142		5.143, *p*=0.822		4.776, *p*=0.853	

Note: $^{(*)}p$ <0.10, *p <0.05, **p <0.01; Other external cause mortality (including accidental falls, drowning, poisoning and other external causes of mortalities); D indicates an intervention dummy variable. CSBL: compulsory seatbelt legislation; RBT: random breath testing; MLDA: minimum legal drinking age.