

Appendix

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Appendix to: Doran CM, Byrnes JM, Cobiac LJ, et al. Estimated impacts of alternative Australian alcohol taxation structures on consumption, public health and government revenues. *Med J Aust* 2013; 199: 619-622. doi: 10.5694/mja13.10605.

Appendix 1

Economic modelling

The current taxation of alcoholic beverages in Australia is complex and inefficient. The only consistency is that all alcohol products are subject to a goods and service tax of 10%. Certain beverages (wine, fortified wine and cider) are subject to an ad valorem tax while other (beer, spirits and RTDs) are taxed volumetrically. The current value-based tax paid imposed on wine based products is referred to as a wine equalisation tax (WET). For wholesale sales, WET is paid on the selling price (excluding WET and goods and services tax) of the wine at the last wholesale sale. For untaxed wine sold by retail, WET is charged on a notional wholesale selling price. The rate of the WET is 29% of the wholesale value.

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Table 1 provides an overview of variations in excise rates according to beverage type. All beer benefits from a tax-free threshold on 1.15% of alcohol content. Low alcohol beer sold on-site (i.e. draught beer from a keg) is taxed at a rate 20% lower than beer sold off-site (i.e. beer packaged in cans or bottles). Mid-strength beer sold on-site is taxed at a rate 54% lower than beer sold off-site. Full strength alcohol beer sold on-site is taxed at a rate 70% lower than high strength beer sold off-site. Spirits and alcopops (or RTDs) are taxed at the highest rate (\$69.16 per litre of pure alcohol) and brandy is taxed at a slightly lower rate than spirits (\$64.57). RTDs are taxed at the same rate as spirits following the Government's equalisation of excise on all spirits based products in April 2008.

Table 1: Taxation and Duty Levies on Alcoholic Drinks 2010

| Beverage type | \$\$ per litre pure alcohol |
|---|--------------------------------|
| Low-alcohol beer (<3% ABV) and packaged in an individual container not exceeding 48 litres | \$35.03 |
| Low-alcohol beer (<3% ABV) and packaged in an individual container exceeding 48 litres | \$6.99 |
| Mid-strength beer (3.01-3.5% ABV) and packaged in an individual container not exceeding 48 litres | \$40.82 |
| Mid-strength beer (3.01-3.5% ABV) and packaged in an individual container exceeding 48 litres | \$21.96 |
| Full-strength beer (>3.5% ABV) and packaged in an individual container not exceeding 48 litres | \$40.82 |
| Full-strength beer (>3.5% ABV) and packaged in an individual container exceeding 48 litres | \$28.74 |
| Other excisable beverages not exceeding 10% by volume of alcohol | \$69.16 |
| Wine Equalisation Tax | 29% |
| Other excisable beverages exceeding 10% by volume of alcohol | \$69.16 |
| Goods and Services Tax | 10% |

In this analysis, base case taxation revenue for beer excise and wine equalisation tax were sourced from the Federal budget while excise spirits were calculated from Euromonitor International. Euromonitor International is a research organisation which uses a comprehensive and standardised methodology to collect detailed, extensive data on a wide range of topics including alcohol sales. Information on volume, value and price of all alcoholic beverages were sourced from Euromonitor International. These data for beer are reported for low-strength (including mid-strength and low-strength) and full strength (heavy) beer. Both onsite (i.e. licensed premises) and offsite (i.e. bottle shops) sales are reported. For the purpose of these analyses, onsite beer is assumed to be 100% keg/draught beer.

Although the Australian Bureau of Statistics publishes apparent consumption data for beer, wine, spirits and RTDs,² Euromonitor provides a more detailed breakdown of beverage consumption. Further, due to different data sources and methods, there are discrepancies in estimates of alcohol consumption. Euromonitor International data aligns closely to published beer consumption figures. However, there are some differences between the two because of the home-produced volume included in the ABS figures and fluctuating inventory levels. The ABS figures are compiled from the data provided by manufacturers and import/export data. The volumes of spirits and RTDs/high-strength premixes are reported in litres of alcohol by

the ABS. In contrast, Euromonitor International is reporting in terms of litres of as-marketed product. This difference makes any direct comparison near impossible. In wine there is little overall difference between the reported apparent consumption figures and existing Euromonitor International data.

To explore variations in consumption patterns as a consequence of varying beverage prices, published estimates of price elasticity were obtained for beer, wine, alcopops and spirits for on-site and off-site consumption and for two separate drinker risk profiles (moderate and hazardous).³ The percentage change in onsite and offsite consumption is calculated for both moderate drinkers and for heavy and hazardous drinkers using the relevant price elasticities for each class of drinker. Price elasticity estimates are provided in Table 2.

<u>Table 2</u>: Own price and cross price elasticities of alcohol beverages

| | Beer | | Wine | | Spirits | | Alcopops | |
|------------------|----------|---------|----------|---------|----------|---------|----------|---------|
| Moderate | Off-Site | On-Site | Off-Site | On-Site | Off-Site | On-Site | Off-Site | On-Site |
| Beer offsite | -0.418 | 0.03 | 0.011 | 0.002 | 0.006 | 0.008 | 0.003 | 0.006 |
| Beer onsite | 0.013 | -0.369 | 0.011 | 0.002 | 0.005 | 0.015 | 0.003 | 0.008 |
| Wine offsite | 0.011 | 0.024 | -0.435 | 0.001 | 0.004 | 0.007 | 0.002 | 0.004 |
| Wine onsite | 0.002 | 0.025 | 0.005 | -0.261 | 0.004 | 0.008 | 0.001 | 0.005 |
| Spirits offsite | 0.012 | 0.029 | 0.011 | 0.001 | -0.517 | 0.006 | 0.002 | 0.004 |
| Spirits onsite | 0.002 | -0.009 | 0.008 | 0.004 | 0 | -0.98 | 0.001 | 0.01 |
| Alcopops offsite | 0.018 | 0.019 | 0.004 | 0.001 | 0.002 | 0 | -0.321 | 0.004 |
| Alcopops onsite | 0.003 | 0.012 | -0.001 | 0.006 | 0.002 | 0.011 | 0.001 | -0.321 |
| | Beer | | Wine | | Spirits | | Alcopops | |
| Hazardous | Off-Site | On-Site | Off-Site | On-Site | Off-Site | On-Site | Off-Site | On-Site |
| Beer offsite | -0.573 | 0.038 | 0.046 | 0.004 | 0.009 | 0.008 | 0.001 | 0.006 |
| Beer onsite | 0.038 | -0.582 | 0.056 | 0 | 0.011 | 0.008 | 0.002 | 0.005 |
| Wine offsite | 0.033 | 0.06 | -0.585 | 0.002 | 0.01 | 0.007 | 0.003 | 0.004 |
| Wine onsite | 0.008 | -0.004 | 0.006 | -0.395 | 0.009 | -0.008 | -0.001 | 0.006 |
| Spirits offsite | 0.014 | 0.041 | 0.023 | 0.003 | -0.635 | -0.001 | 0 | 0.001 |
| Spirits onsite | 0.023 | 0.002 | 0.013 | 0.012 | -0.003 | -1.993 | -0.001 | -0.01 |
| Alcopops offsite | -0.009 | 0.008 | 0.021 | 0 | 0.002 | 0.011 | -0.399 | 0 |
| Alcopops onsite | 0.006 | 0.017 | 0.007 | 0.008 | 0 | 0.017 | 0.001 | -0.399 |

Australian estimates of onsite and offsite alcohol sales by beverage class were provided by Euromonitor International. Onsite and offsite alcohol sales for each beverage class were then estimated for moderate and hazardous drinkers by applying the proportion of average alcohol consumption by hazardous drinkers. The proportion of alcohol consumption by hazardous drinkers for Australia was estimated using the equation below

$$h = q_H \times p_H / [(q_H \times p_H) + (q_M \times p_M)]$$

Where h is the proportion of average alcohol consumption by hazardous drinkers, q_H is the average quantity of alcohol consumption by hazardous drinkers, p_H is the prevalence of hazardous drinkers, q_M is the mean quantity of alcohol consumption by moderate drinkers and p_M is the prevalence of moderate drinkers. Average quantity of alcohol and prevalence of drinkers were estimated from the National Health Survey.⁴

Although the current taxation regime categorises beer into three categories of low-, mid- and heavy-strength, due to data constraints, our modelling is based on heavy- and low strength beer (i.e. combined low- and mid-strength) with the excise and subsequent onsite/offsite discounts based on the weighted averages (in volume) of mid- and low-strength beers.

Epidemiological modelling

The methodology used to model the taxation scenarios is based on the framework developed for the Alcohol Education Rehabilitation Foundation funded project entitled "ACE-Alcohol" (Assessing the Cost-Effectiveness of interventions to reduce the burden of harm from alcohol misuse). ACE-Alcohol was part of a larger priority-setting exercise funded by the National Health Medical Research Council (NHMRC) entitled "ACE-Prevention" (Assessing the Cost-Effectiveness of interventions to reduce the burden of harm from non-communicable diseases).

The ACE–Alcohol method and several applications are reported in detail elsewhere. ACE–Alcohol evaluated the cost-effectiveness of eight different interventions for reducing harm attributable to alcohol consumption from an Australian health sector perspective. Health outcomes were evaluated in DALYs, using a multi-state, multiple cohort life table approach to determine changes in incidence, prevalence and mortality of alcohol-related diseases and injuries due to each intervention. Diseases evaluated in the model include ischaemic heart disease, ischaemic stroke, hypertensive heart disease, inflammatory heart disease, pancreatitis, cirrhosis, alcohol dependence and gallbladder and bile duct disease, as well as cancer of the breast (in women), mouth and oropharynx, oesophagus, liver and larynx. Injuries include road traffic accidents, falls, fires, burns and scalds, drowning, machinery accidents, suffocation and foreign bodies, suicide and self-inflicted injuries, and homicide and violence.

Population and all-cause mortality are derived, by age and sex, from Australian Bureau of Statistics data.^{10,11} Incidence and case fatality are derived, by age and sex, from Australian Burden of Disease study data and trend analyses.^{12,13} Average daily consumption of alcohol and prevalence of abstinent, low, hazardous or harmful levels of drinking are derived, by age and sex, from National Health Survey 2008 data, with adjustment for per capita consumption based on national sales data.^{11,14,15} These data are provided in Tables 3 and 4.

<u>Table 3</u>: Levels of alcohol consumption based on the average number of standard drinks consumed per day (1 standard drink = 10 grams of alcohol)

| | Abstain | Low | Hazardous | Harmful |
|-------|-----------|-----------|-----------|---------|
| Men | 0.00-0.25 | 0.26-4.00 | 4.01-6.00 | 6.01+ |
| Women | 0.00-0.25 | 0.26-2.00 | 2.01-4.00 | 4.01+ |

<u>Table 4</u>: Mean daily alcohol consumption (in grams), of Australian men and women aged 18+ years, by level of alcohol consumption

| | Abstain | Low | Hazardous | Harmful |
|-------|---------|-----|-----------|---------|
| Men | 1.8 | 10 | 35 | 99 |
| Women | 1.7 | 9.2 | 27 | 65 |

The costs of treating each case of disease or injury are derived, by age and sex, from Disease Costs and Impacts Study data, adjusted to 2009 dollars using health system deflators. ¹⁶⁻¹⁷ Intervention costs are derived from the WHO costing database adjusted to 2009 dollars using the Consumer Price Index. ¹⁸⁻¹⁹ The analyses are carried out in Microsoft Excel using the addin tool @Risk for uncertainty analysis. ²⁰ Cost-effectiveness ratios are derived from cost and health outcomes measured over the life-time of the Australian population in the baseline year of 2009. Future costs and health outcomes are discounted at 3% per annum. In ACE—Alcohol, cost-effectiveness ratios were evaluated for each intervention in comparison with current practice, which is equivalent to a 'do nothing' scenario apart from the current level of intensity of random breath testing. Probabilities of cost-effectiveness were reported against a cost-effectiveness threshold of \$50,000 per DALY averted, a threshold used in previous Australian priority setting studies. ⁶

Effect of change in alcohol consumption on disease

Excess consumption of alcohol increases the risk of ischaemic stroke, hypertensive heart disease, inflammatory heart disease, pancreatitis and cirrhosis, as well as cancer of the breast (in women), mouth and oropharynx, oesophagus, liver and larynx, ²¹ and has a protective effect against gallbladder and bile duct disease. ²² Consumption of alcohol also protects against ischaemic heart disease, but only at low levels of consumption; higher levels of consumption increase the risk of ischaemic heart disease. ²³

For all heart disease, stroke, digestive diseases and cancers, the effect of changing alcohol consumption on disease incidence was modelled by a modified version of the potential impact fraction,²⁴ in which the intervention effect changes the relative risk of disease in the intervention population rather than the population prevalence of alcohol consumption (Equation 1). This modification allows the tax impacts to be modelled from their effect on alcohol consumption (i.e. in standard units, serves or grams per day), a continuous measure of effect, rather than from their effect on population prevalence across consumption categories.

$$PIF = \frac{\sum_{i=1}^{n} p_{i} RR_{i} - \sum_{i=1}^{n} p_{i} RR'_{i}}{\sum_{i=1}^{n} p_{i} RR_{i}}$$
(1)

where:

PIF is the potential impact fraction;

 p_i is the prevalence of alcohol consumption at exposure level i;

 RR_i is the relative risk of disease associated with alcohol consumption at exposure level i; and

 RR'_i is the relative risk of disease associated with alcohol consumption after an intervention is implemented in the population at exposure level i.

Alcohol consumption was divided into four levels of alcohol use – abstinence, low, hazardous and harmful (Table 3) – based on number of standard drinks consumed per day. We used Australian National Health Survey data to determine prevalence across the four levels of consumption (Table 5) and average consumption within each category (Table 4).

We derived relative risks of disease from existing meta-analyses of data describing the relationship between alcohol consumption and the risk of alcohol related conditions (Table 6).

The change in relative risk of each disease was calculated from the change in alcohol consumption due to the tax changes, by assuming a linear increase (or decrease) in disease risk with increasing alcohol consumption between each of the four levels of alcohol consumption.

For alcohol dependence, which is wholly attributable to excess alcohol consumption, the intervention effect on disease incidence was modelled, by age and sex, as a reduction in the incidence of alcohol dependence that is proportional to the change in consumption at a harmful level of alcohol consumption (Equation 2). Incidence of alcohol dependence was assumed to be negligible below a harmful level of consumption.

$$i' = i \left(1 - \frac{\Delta c_{Harm}}{c_{Harm} - c_{Abs}} \right) (2)$$

where:

i' is the incidence of alcohol dependence after an intervention is implemented in the population;

i is the current incidence of alcohol dependence;

 Δc_{Harm} is the average change in alcohol consumption in the population due to an intervention, in g/day, among those currently drinking at a harmful level;

 $c_{\textit{Harm}}$ is the average alcohol consumption, in g/day, among those currently drinking at a harmful level; and

 c_{Abs} is the average alcohol consumption, in g/day, among those currently defined as abstinent.

Effect of change in alcohol consumption on injuries

Excess consumption of alcohol increases the risk of injury.²⁵ Injuries associated with at least 5% of death or disability due to alcohol consumption in Australia include road traffic accidents, falls, fires, burns and scalds, drowning, machinery accidents, suffocation and foreign bodies, suicide and self-inflicted injuries, and homicide and violence.

In contrast to modelled diseases, injuries are acute in nature. Thus, in the Australian Burden of Disease study, disability and mortality due to injury are derived from *incidence* of fatal and non-fatal injuries in the population, rather than from *prevalence* of injuries and their sequelae. For this reason, we modelled changes in injury outcomes due to the tax changes via direct

changes in injury-related mortality and disability. All mortality and disability rates in the current population were derived from the Australian Burden of Disease study. 25

<u>Table 5</u>: Prevalence of alcohol use in Australian men and women aged 18+ years

| Age group | | Men | | | | Women | | | | |
|-----------|---------|-----|-----------|---------|---------|-------|-----------|---------|--|--|
| (years) | Abstain | Low | Hazardous | Harmful | Abstain | Low | Hazardous | Harmful | | |
| 18-24 | 7% | 45% | 33% | 15% | 14% | 61% | 17% | 8% | | |
| 25-29 | 8% | 44% | 36% | 12% | 14% | 68% | 12% | 6% | | |
| 30-34 | 8% | 50% | 29% | 13% | 20% | 62% | 12% | 5% | | |
| 35-39 | 11% | 49% | 29% | 11% | 20% | 60% | 16% | 5% | | |
| 40-44 | 9% | 48% | 32% | 11% | 15% | 61% | 18% | 6% | | |
| 45-49 | 12% | 44% | 33% | 10% | 19% | 59% | 16% | 6% | | |
| 50-54 | 12% | 42% | 34% | 12% | 14% | 64% | 15% | 7% | | |
| 55-59 | 10% | 53% | 27% | 10% | 20% | 59% | 14% | 7% | | |
| 60-64 | 9% | 48% | 32% | 11% | 20% | 56% | 20% | 4% | | |
| 65-69 | 10% | 47% | 32% | 11% | 18% | 56% | 22% | 5% | | |
| 70-74 | 18% | 49% | 29% | 4% | 24% | 53% | 15% | 7% | | |
| 75-79 | 22% | 46% | 31% | 2% | 25% | 61% | 11% | 4% | | |
| 80-84 | 16% | 48% | 33% | 3% | 28% | 52% | 19% | 1% | | |
| 85+ | 11% | 63% | 24% | 2% | 43% | 47% | 9% | 2% | | |

<u>Table 6</u>: Relative risks of disease due to alcohol consumption

| | | Alcohol intake level | | | | | |
|-----------------------------------|--------|----------------------|------------------|------------------|-------------------|--|--|
| Disease | Sex | Abstinent | Low | Hazardous | Harmful | | |
| Ischaemic heart disease | Male | 1.00 | 0.85 (0.82–0.88) | 0.84 (0.80–0.87) | 1.00 (0.94–1.07) | | |
| | Female | 1.00 | 0.87 (0.84–0.90) | 0.92 (0.87–0.96) | 1.20 (1.06–1.35) | | |
| Ischaemic stroke* | Male | 1.00 | 1.02 (0.84–1.21) | 1.44 (1.15–1.79) | 1.84 (1.02–3.04) | | |
| | Female | 1.00 | 0.62 (0.50–0.77) | 0.77 (0.52–1.09) | 1.47 (0.41–3.77) | | |
| Breast cancer | Male | _ | - | - | - | | |
| | Female | 1.00 | 1.14 (1.09–1.20) | 1.41 (1.32–1.50) | 1.59 (1.43–1.78) | | |
| Mouth and oropharynx cancer | Male | 1.00 | 1.58 (1.35–1.87) | 2.95 (1.92–4.63) | 5.41 (1.78–16.53) | | |
| | Female | 1.00 | 1.32 (1.11–1.63) | 2.01 (1.44–2.85) | 3.89 (1.97–10.62) | | |
| Oesophagus cancer | Male | 1.00 | 1.32 (1.19–1.46) | 2.17 (1.71–2.75) | 4.42 (0.91–2.57) | | |
| | Female | 1.00 | 1.18 (1.11–1.26) | 1.56 (1.38–1.76) | 2.05 (1.65–2.57) | | |
| Liver cancer | Male | 1.00 | 1.13 (1.07–1.20) | 1.39 (1.21–1.60) | 1.79 (1.23–2.57) | | |
| | Female | 1.00 | 1.07 (1.04–1.11) | 1.22 (1.13–1.31) | 1.49 (1.29–1.75) | | |
| Larynx cancer | Male | 1.00 | 1.13 (1.07–1.20) | 1.49 (1.21–1.81) | 2.08 (1.40–3.08) | | |
| | Female | 1.00 | 1.07 (1.04–1.11) | 1.23 (1.14–1.32) | 1.63 (1.37–1.97) | | |
| Hypertensive heart disease | Male | 1.00 | 1.26 (1.20–1.32) | 1.97 (1.76–2.23) | 4.03 (2.93–5.53) | | |
| | Female | 1.00 | 1.14 (1.11–1.18) | 1.45 (1.35–1.55) | 2.45 (2.15–2.84) | | |
| Inflammatory heart disease** | Male | 1.00 | 1.00 (1.00–1.00) | 1.43 (1.33–1.54) | 2.24 (1.93–2.55) | | |
| | Female | 1.00 | 1.00 (1.00–1.00) | 1.87 (1.65–2.09) | 3.49 (2.86–4.11) | | |
| Gallbladder and bile duct disease | Male | 1.00 | 0.82 (0.76–0.90) | 0.68 (0.55–0.84) | 0.50 (0.33–0.75) | | |
| | Female | 1.00 | 0.82 (0.76–0.90) | 0.68 (0.55–0.84) | 0.50 (0.33–0.75) | | |
| Pancreatitis | Male | 1.00 | 1.19 (1.10–1.30) | 1.78 (1.35–2.40) | 3.15 (1.77–5.47) | | |
| | Female | 1.00 | 1.11 (1.06–1.16) | 1.34 (1.21–1.50) | 2.10 (1.61–2.79) | | |
| Cirrhosis | Male | 1.00 | 1.39 (1.17–1.67) | 2.36 (1.43–3.91) | 4.33 (1.32–13.61) | | |
| | Female | 1.00 | 1.36 (1.08–1.82) | 2.14 (1.39–3.45) | 5.21 (2.18–21.39) | | |

NB. Values are mean relative risk and 95% confidence interval at average alcohol consumption for intake category.

^{*} Weighted average of relative risks for ischaemic and haemorrhagic stroke (weighting based on incidence).

^{**} Due to an absence of relative risk studies for inflammatory heart disease, relative risks were derived from the proportion of inflammatory heart disease deaths with alcoholic cardiomyopathy as an underlying cause and prevalence of alcohol use, assuming linearly increasing risk with increasing consumption above low levels of alcohol use.

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