Cancers in Australia in 2010 attributable to the consumption of alcohol

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Both the International Agency for Research on Cancer (IARC) and the World Cancer Research Fund (WCRF) have concluded that there is sufficient and convincing evidence that alcoholic drinks cause cancers of the oral cavity, pharynx, larynx, colorectum and female breast.1-4 IARC also concluded there was sufficient evidence that alcohol causes liver cancer and oesophageal squamous cell carcinoma (SCC);1 while the WCRF concluded that alcohol causes oesophageal cancers (type not specified) and probably increases the risk of cancer of the liver, with cirrhosis being an essential precursor.2 The evidence relating alcohol to pancreatic cancer is less clear. High alcohol intake (more than about three drinks or 30 g ethanol per day) may be associated with a small increase in risk of pancreatic cancer,3,4 however, IARC noted that residual confounding by smoking could not be excluded.1 The conclusions of these two agencies are summarised in Table 1.

Abstract

Objective: To estimate the proportion and numbers of cancers occurring in Australia in 2010 that are attributable to alcohol consumption.

Methods: We estimated the population attributable fraction (PAF) of cancers causally associated with alcohol consumption using standard formulae incorporating prevalence of alcohol consumption and relative risks associated with consumption and cancer. We also estimated the proportion change in cancer incidence (potential impact fraction [PIF]) that might have occurred under the hypothetical scenario that an intervention reduced alcohol consumption, so that no-one drank >2 drinks/day.

Results: An estimated 3,208 cancers (2.8% of all cancers) occurring in Australian adults in 2010 could be attributed to alcohol consumption. The greatest numbers were for cancers of the colon (868) and female breast cancer (830). The highest PAFs were for squamous cell carcinomas of the oral cavity/pharynx (31%) and oesophagus (25%). The incidence of alcohol-associated cancer types could have been reduced by 1,442 cases (4.3%) – from 33,537 to 32,083 – if no Australian adult consumed >2 drinks/day.

Conclusions: More than 3,000 cancers were attributable to alcohol consumption and thus were potentially preventable.

Implications: Strategies that limit alcohol consumption to guideline levels could prevent a large number of cancers in Australian adults.

Key words: population attributable fraction, cancer, risk factor, alcohol, potential impact fraction

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Earlier studies have estimated the proportion of cancers in Australia attributable to alcohol consumption. Some are now out of date; others have used approaches that do not permit comparison with recent international efforts. We sought to estimate the proportion and numbers of cancers occurring in Australia in 2010 attributable to alcohol consumption. Specifically, we estimated the population attributable fraction for cancers of the oral cavity, pharynx, oesophagus (SCC), colon, rectum, liver, larynx and breast associated with various levels of alcohol exposure, with the reference category defined as zero alcohol intake.

**Methods**

**Relative risk estimates**

Relative risks for colorectal cancer were taken from the WCRF Continuous Update for Colorectal Cancer. The increased risks for colon and rectal cancer were modelled separately, as the risk associated with alcohol consumption is higher for rectal cancer than colon cancer. We used relative risks for breast cancer and liver cancer published in the WCRF/AICR report on Food, Nutrition, Physical Activity and the Prevention of Cancer. Hence, for cancers of the oral cavity, pharynx, larynx and oesophagus, we used relative risk estimates from recent meta-analyses (oral cavity, pharynx and larynx) and pooled analyses (oesophageal SCC) (Table 2). We did not use the WCRF relative risks for these cancers as the summary results were reported in terms of ‘alcoholic drinks/day’ rather than ‘grams of ethanol/day’. The relative risks for ‘drinks/day’ are imprecise as the size of each drink is unknown. In addition, the WCRF/AICR did not conduct separate analyses for oesophageal squamous cell carcinoma and adenocarcinoma. Where relative risks were reported as dose-response for 10 g/day or presented across categories, we assumed a log-linear relationship to convert the relative risk to an increase in risk per gram per day using the following formula:

\[ \text{Increase in risk per gram per day (Rg)} = \ln(RR_{10g}) / 10 \]

We performed sensitivity analyses using the same relative risks as used by the UK PAF Project. Thus, for most cancers we used relative risks published in an earlier meta-analysis. For breast cancer, relative risks were sourced from a meta-analysis of 53 studies by the Collaborative Group on Hormonal Factors in Breast Cancer, while relative risks for colorectal cancer were sourced from four meta-analyses (including WCRF).  

**Exposure prevalence estimates**

The latent period between consumption of alcohol and onset of cancer is not known. We assumed a latent period of 10 years, and so used prevalence data from the 2001 National Health Survey Australia, Confidentialised Unit Record File (CURF) and cancer incidence data from 2010 to give a nominal latent period of about 10 years. To account for population ageing with time since exposure and the latent period, we used prevalence data for the age category that was 10 years younger than the corresponding cancer incidence age category. For example, cancer incidence in the 35–44 years age group in 2010 was attributed to alcohol consumption in the 25–34 years age group in 2001.

Participants in the 2001 National Health Survey aged 18 years and over, were asked how long ago they last had an alcoholic drink. Those who reported having an alcoholic drink within the previous week were asked what days in that week they had consumed alcohol (excluding the day of interview). For each of the last three days on which they drank, they were asked the types, number and size of drinks they had consumed and the brand name, where possible. They were further asked whether...

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**Table 1: Summary of Evidence (IARC and WCRF) – alcohol and the risk of cancer.**

| Cancer Site (ICD-10 Codes) | Level of Evidence – IARC | Level of Evidence – WCRF |
|----------------------------|--------------------------|--------------------------|
| Oral Cavity (C01-C06)      | Sufficient               | Convincing               |
| Pharynx (C09-C14)         | Sufficient               | Convincing               |
| Oesophagus (C15)          | Sufficient (squamous cell carcinoma only) | Convincing               |
| Colorectum (C18-C20)      | Sufficient               | Convincing (men) Probable (women) |
| Liver (C22)               | Sufficient               | Probable                 |
| Pancreatic (C25)          | Limited                  | Limited-suggestive (heavier drinking – more than about 3 drinks/day) |
| Larynx (C32)              | Sufficient               | Convincing               |
| Breast (C50)              | Sufficient               | Convincing               |

**Table 2: Summary of relative risks for the association between alcohol and site-specific cancers.**

| Cancer (ICD-10 code) | Source of Relative Risk | RR (95% CI) | Risk per g/day* |
|----------------------|-------------------------|-------------|-----------------|
| Oral cavity (C01-C06) and pharynx (C09-C14) | Meta-analysis of 45 case-control and 2 cohort studies (17,085 cases). A separate inference from cohort studies was not possible due to the small number of cases. Only 5 studies did not adjust for smoking. | 0 g/day Ref | 1.021 per g/day |
|                      |                         | 10 g/day | 1.29 (1.25-1.32) |
|                      |                         | 25 g/day | 1.85 (1.74-1.96) |
|                      |                         | 50 g/day | 2.34 (2.89-3.64) |
|                      |                         | 75 g/day | 5.42 (4.58-6.40) |
|                      |                         | 100 g/day | 8.61 (6.91-10.73) |
|                      |                         | 125 g/day | 13.02 (9.87-17.18) |
| Oesophagus ( SCC (C15)) | Pooled analysis of nine population-based case-control studies and two cohort studies (BEACON Consortium). 1,106 cases of ESCC and 9,253 controls, | 0 g/day Ref | 1.022 per g/day |
|                      |                         | >0-<7 g/day | 0.8 (0.56-1.14) |
|                      |                         | 7-<14 g/day | 1.23 (0.55-2.75) |
|                      |                         | 14-<42 g/day | 1.0 (1.10-5.96) |
|                      |                         | 42-<70 g/day | 2.0 (2.32-8.96) |
|                      |                         | ≥70 g/day | 7.17 (2.98-17.25) |
|                      |                         | ≥75 g/day | 9.62 (4.26-21.71) |
| Colon (C18, C19)     | Meta-analysis of 12 studies (10 cohort, 1 case-control). Moderate heterogeneity explained largely by region of study. | 0.91 (1.04-1.13) | 1.080 per g/day |
| Rectal (C20)         | Meta-analysis of 11 studies (10 cohort, 1 nested case-control). Low heterogeneity. | 1.09 (1.07-1.12) | 1.009 per g/day |
| Liver (C22)          | Meta-analysis of six cohort studies. No heterogeneity. | 1.10 (1.02-1.17) | 1.009 per g/day |
| Larynx (C32)         | Meta-analysis of 38 case-control and 2 cohort studies. | 0 g/day Ref | 1.013 per g/day |
|                      |                         | 12.5 g/day | 1.20 (1.15-1.25) |
|                      |                         | 25 g/day | 1.45 (1.33-1.57) |
|                      |                         | 37.5 g/day | 1.72 (1.52-1.90) |
|                      |                         | 50 g/day | 2.04 (1.76-2.36) |
|                      |                         | 100 g/day | 3.77 (2.93-4.86) |
| Breast (C50)          | Meta-analysis of nine cohort studies. High heterogeneity, partly explained by differential adjustment for age and reproductive history. | 1.10 (1.06-1.14) | 1.009 per g/day |

* assuming a log-linear relationship
| Data Source a: Cancers in Australia in 2010 and strength of each drink are unknown.2 In risks for ‘drinks/day’ are imprecise as the size rather than ‘grams of ethanol/day’. The relative risks were reported in terms of ‘alcoholic drinks/day’ rather than ‘grams of ethanol/day’. The relative risks for these cancers as the summary results were reported in terms of ‘alcoholic drinks/day’ rather than ‘grams of ethanol/day’. The relative risks for ‘drinks/day’ are imprecise as the size and strength of each drink are unknown. In addition, the WCRF/AICR did not conduct separate analyses for oesophageal squamous cell carcinoma and adenocarcinoma. Where relative risks were reported as dose-response for 10 g/day or presented across categories, we assumed a log-linear relationship to convert the relative risk to an increase in risk per gram per day using the following formula:

\[ \text{Increase in risk per gram per day (Rg)} = \ln(RR_{10g}) / 10 \]
their consumption in that week was more, about the same, or less than usual. Reported quantities of alcoholic drinks were converted to millilitres of alcohol and a variable ‘average daily intake over week’ was derived (average consumption over 3 days x number of days consumed alcohol). We converted millilitres (mL) of alcohol to grams (g) of alcohol using 1 mL = 0.789 g of alcohol. Consumption was grouped into 12 categories; the median intake for each category was then calculated and extracted along with the proportion of the population in each category by age and sex. The 2001 National Health Survey questions focused on alcohol consumption in the previous week, resulting in a high proportion of people classified as ‘non-drinkers’ (47% of men and 60% of women age 18 years and over). The 2007-08 National Health Survey asked similar questions about alcohol consumption, but further identified three sub-categories for ‘non-drinkers’ (viz. last consumed alcohol 1 week to <12 months ago; last consumed alcohol >12 months ago; never consumed alcohol). This breakdown revealed that 57% of non-drinkers had actually consumed alcohol between 1 week and <12 months previously. Thus, because of concerns that infrequent drinkers may have been misclassified as ‘non-drinkers’ in the 2001 National Health Survey, we conducted sensitivity analyses in which we redistributed 57% of reported non-drinkers in the 2001 National Health Survey equally across the four lowest drinking categories.

Statistical analysis

The population attributable fraction (PAF) was calculated for each cancer site (Table 1) by age and sex category using the formula:\[ \text{PAF} = \frac{\sum (p_x \times ERR_x)}{\sum p_x \times ERR_x} \]
where \( p_x \) is the proportion of the population and \( ERR_x \) is the excess relative risk (RR, -1) in consumption category \( x \) (where \( x = 1 \) to 12). The excess relative risk (ERR) for each \( x \) level of alcohol consumption (Table 2) was calculated as:
\[ \text{ERR}_x = \exp(R_x \times G_x) - 1 \]
where \( R_x \) is the increase in risk per gram of alcohol consumption (Table 2) and \( G_x \) is the overall population median of consumption (in grams per day) in category \( x \).

To obtain the number of cancers attributable to alcohol consumption, we summed the age- and sex-specific incidence rates between 2006 and 2008 to the 2010 Australian estimated resident population (by age and sex) to estimate the number of incident oesophageal SCCs in 2010. The total number of cancers attributable to alcohol consumption was also expressed as a percentage of the total number of all incident cancers (excluding basal cell and squamous cell carcinoma of the skin) recorded in Australian adults aged over 25 years in 2010. In our primary analysis, we assumed risk increased with any amount of alcohol consumption (greater than 0 g/day). In our sensitivity analyses, we also modelled the possibility that low levels of drinking (<2 g/day and <5 g/day) confer no increased risks of cancer.

Potential impact of reducing alcohol consumption

Complete elimination of alcohol consumption is unlikely, so we modelled the potential impact on cancer incidence assuming intake had not exceeded the levels recommended by The Australian Guidelines to Reduce Health Risks from Drinking Alcohol, as:
1. No Australian adults drinking >4 standard drinks/day (40 g alcohol)
2. No Australian adults drinking >2 standard drinks/day (20 g alcohol).

To model each scenario, we assumed the level of alcohol consumption in each category above the respective threshold had been equal to the recommended maximum (i.e. 40 g or 20 g) and estimated the new relative risks for these categories compared to never drinkers. We then calculated the potential impact fraction (PIF) using the formula of Barendregt and Veerman:
\[ \text{PIF} = \frac{\sum p_x \times R_x - \sum p_x \times RR_x}{\sum p_x \times RR_x} \]
where \( p_x \) is the proportion of the population in each age and sex category and alcohol consumption category \( x \), \( RR_x \) is the relative risk for that category compared to never drinkers at the observed level of alcohol consumption and \( RR_x \) is the new relative risk assuming a maximum intake of 20 g (or 40 g) per day.

For each cancer site, we calculated the number of cases that would have occurred in Australia in 2010, assuming that the alternative scenario of alcohol consumption had prevailed. The PIF is then the proportional difference between the numbers of cancers observed and the numbers expected under the alternative exposure scenarios.

Results

The 2001 National Health Survey reported that 53% of men and 40% of women regularly consumed alcohol, with median daily intake varying markedly by age group and sex. More women than men were non-drinkers across all age groups. The highest prevalence of heavy drinking (>4 standard drinks/day) was seen among males in the 25-64 age years categories (14-15%), see supplementary file: Table 51, available with the online version of this paper.

The estimated numbers and proportions of cancers attributed to alcohol consumption are presented in Table 3. In 2010, there were 33,527 diagnoses of cancers of the oral cavity, larynx, pharynx, oesophagus (SCC), colon, rectum and liver in Australians aged >25 years, of which we estimated 3,208 (10%) were attributable to alcohol consumption (1,976 in men and 1,232 in women). This corresponds to 2.8% of all cancer cases (excluding basal cell carcinoma and squamous cell carcinoma of the skin) in Australian adults >25 years (3.0% in men and 2.5% in women). Cancer sites with the highest proportion of cases attributable to alcohol were oral cavity and pharynx (31%), oesophageal squamous cell carcinoma (25%) and larynx (20%). Cancers with the greatest number of cases attributable to alcohol were colon (868) and breast (830). Across all cancer sites, there were marked sex differences in the PAF. On average, more than 80% of alcohol-attributable cancers at each site occurred in men (ranging from 80% of colorectal cancers to 94% of laryngeal cancers). In women, 67% of all cancers attributed to alcohol were cancers of the breast (830 female breast cancers out of 1,232 excess female cancers attributed to alcohol) (Table 3).

Sensitivity analyses

The UK PAF project used relative risks sourced from earlier reports; these were systematically lower than the relative risks used for our primary analyses. Reanalysing the Australian data using the same relative risks as the UK PAF project reduced the fraction of overall cancers attributable to alcohol from 2.8% to 2.4% (i.e. 463 fewer cancers overall were attributable to alcohol using the earlier summary risk estimates). The sites for which the PAFs differed most between primary vs. sensitivity analyses were cancers of the oesophagus (SCC) (25% vs. 14%) and liver (13% vs. 8%).

In further sensitivity analyses, we assumed that low levels of drinking (<2 g/day and <5 g/day) conferred no risks of cancer development, but
Table 3: Population attributable fraction (PAF) and estimated number of cancers diagnosed in Australia in 2010 attributable to alcohol consumption.

| Age at outcomea | Oral cavity (C01-C06) and Pharynx (C09-C14)b | Oesophagus (SCC) (C15)b,c | Colon (C18, C19)b | Rectum (C20)b | Liver (C22)b | Larynx (C32)b | Breast (C50)b | All cancerd | PAF Obs. Exc. | PAF Obs. Exc. | PAF Obs. Exc. | PAF Obs. Exc. | PAF Obs. Exc. | PAF Obs. Exc. |
|----------------|---------------------------------------------|---------------------------|-------------------|----------------|------------|-------------|-------------|-------------|--------------|--------------|--------------|--------------|--------------|--------------|
| Males          |                                             |                           |                   |                |            |             |             |             |              |              |              |              |              |              |
| 25–34 yrs      | 31.3 17                                  | 4 12.2                     | 31.7 6           | 1 38.5        | 2 0          | 1 22.4      | 2 8.7       | 1 28.6      | 1 2.9        | 0 0          | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 35–44 yrs      | 41.4 86                                  | 5 6.5                      | 41.4 8            | 19 37.2       | 4 26.6       | 1 17.9      | 2 8.6       | 1 37.9      | 1 12.7       | 1 22.4      | 2 8.7        | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 45–54 yrs      | 41.1 124                                 | 12 10.1                    | 41.1 10           | 19 37.2       | 2 8.6       | 1 17.9      | 2 8.6       | 1 37.9      | 1 12.7       | 1 22.4      | 2 8.7        | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 55–64 yrs      | 31.5 429                                 | 12 9.6                     | 31.5 12           | 19 37.2       | 2 8.6       | 1 17.9      | 2 8.6       | 1 37.9      | 1 12.7       | 1 22.4      | 2 8.7        | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 65–74 yrs      | 43.8 336                                 | 10 8.0                     | 43.8 13           | 19 37.2       | 12 10.1     | 2 8.6       | 1 17.9      | 2 8.6       | 1 37.9      | 1 12.7       | 1 22.4      | 2 8.7        | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 75–84 yrs      | 31.9 146                                 | 70 61.0                    | 31.9 31           | 19 37.2       | 4 26.6       | 1 17.9      | 2 8.6       | 1 37.9      | 1 12.7       | 1 22.4      | 2 8.7        | 1 25.6       | 1 12.7       | 1 24.4       |              |
| 85+ yrs        | 1.1 48                                   | 1 0.2                      | 1.1 1             | 0.3 0.3       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| Total          | 30.0 1,040                               | 317 21.7                   | 30.0 15           | 10.0 5.8      | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| Females        |                                             |                           |                   |                |            |             |             |             |              |              |              |              |              |              |              |
| 25–34 yrs      | 11.0 15                                  | 2 1.3                      | 11.0 11           | 17 5.3        | 2 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 35–44 yrs      | 13.0 44                                  | 6 1.1                      | 13.0 10           | 17 5.3        | 2 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 45–54 yrs      | 16.9 100                                 | 17 5.7                     | 16.9 14           | 23 5.6        | 2 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 55–64 yrs      | 16.2 145                                 | 24 6.8                     | 16.2 24           | 6 5.4         | 6 0.8       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 65–74 yrs      | 14.0 128                                 | 18 7.3                     | 14.0 16           | 7 5.5         | 2 1.6       | 0 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 75–84 yrs      | 10.9 106                                 | 12 11.4                    | 10.9 14           | 7 5.5         | 2 1.6       | 0 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2       | 1 0.2        | 1 0.2        | 1 0.2        | 1 0.2        |              |
| 85+ yrs        | 0.2 54                                   | 0.3 0.0                    | 0.2 0             | 0.1 0.0       | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      |              |
| Total          | 8.9 529                                  | 2.5 0.5                     | 8.9 22            | 2.0 0.4       | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      |              |
| Persons        | 6.1 258                                  | 0.7 0.0                     | 6.1 12            | 0.3 0.0       | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0     | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      | 0.2 0.0      |              |

Abbreviations: Obs. = observed cancers in 2010; exc. = excess cancers in 2010 attributable to alcohol; PAF = population attributable fraction (expressed as a percentage); PAFaw = age-weighted population attributable fraction (expressed as a percentage)

a: Prevalence data age groups are 10 years younger than cancer incidence age groups assuming a 10 year latent period between exposure and outcome (see text)
b: International Classification of Diseases Code (ICD-10)
c: Oesophageal cancer (squamous cell carcinoma) with histology codes 8050-8082
d: Excluding basal cell carcinoma and squamous cell carcinoma of the skin

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Cancers in Australia in 2010

Attributable to consumption of alcohol
these changes had minimal impact, reducing the PAF estimates by less than 2% and 4% for <2 g/day and <5 g/day, respectively, across all cancers except for breast cancer, where the PAF was reduced by 3% for the <2 g/day scenario and 9% for the <5 g/day scenario. Overall, assuming zero-risk of cancer for consumption levels below 2 g/day and 5 g/day, the total estimated number of cancers attributed to alcohol was reduced by 31 and 136 cases, respectively.

Finally, we assessed the impact of redistributing a proportion of ‘non-drinkers’ across the four lowest drinking categories. This increased the fraction of total cancers attributable to alcohol from 2.8% to 3.0% (i.e. 300 more cancers overall were attributable to alcohol). This redistribution had the greatest impact on women, with 253 more cancers overall attributable to alcohol, compared to only 47 in men.

**Potential impact of reducing alcohol consumption**

We assessed the potential impact on cancer incidence of reducing alcohol consumption in Australian adults (see online supplementary file: Table S2). If no Australian adult had consumed more than four standard drinks per day in 2001, we estimate 745 fewer cancers would have occurred in 2010 (PIF 2.2%), see online supplementary file: Table S2. If alcohol consumption had been even lower, such that no Australian adult had consumed more than two standard drinks per day in 2001, we estimate 1,442 fewer cancers would have occurred in 2010 (PIF 4.3%; 45% of all cancers attributable to alcohol). The proportional reductions were greatest for squamous cell cancers of the upper aerodigestive tract, while the greatest absolute reduction was for colon cancer.

**Discussion**

We estimated that more than 3,000 cases of cancer that occurred in Australian adults in 2010 were attributable to alcohol consumption. The PAF was highest for cancers of the oral cavity and pharynx (31%) and oesophageal squamous cell carcinoma (25%). In absolute terms, the greatest numbers of the oral cavity and pharynx (31%) and aerodigestive tract, while the greatest absolute reduction was for colon cancer.

using a burden of disease approach, estimated that alcohol accounted for 3.1% of the 'total health loss' due to cancers in Australia, similar to our overall estimate of 2.8%. In its 2011 position statement, Cancer Council Australia suggested that up to 5% of cancers in Australia may be attributable to alcohol, although this was estimated by extrapolating PAF estimates from international studies to the Australian population and was not intended to be definitive. In 2014, VicHealth and Turning Point published a report entitled *Alcohol's burden of disease in Australia*. It did not report an overall PAF for cancer, but it did report the proportion of site-specific cancer deaths attributable to alcohol for Australian men as: colon 5.7%; larynx 28.8%; liver 14.9%; oesophagus 28.1%; oral cavity and pharynx 45.8%; and rectum 8.7%. Those PAF estimates differ somewhat from those in our report. In part, the differences are explained by the use of older risk estimates that pre-date the effect estimates we used, and the fact that cancer mortality rather than incidence was used as the outcome measure. However, the most important methodological difference was in measuring exposure; the 2014 report used alcohol sales and consumption data rather than self-reported survey data to estimate alcohol exposure prevalence in the Australian population. We used estimates of alcohol consumption for the Australian population by age and sex obtained from the Australian National Health Survey. That survey recruited a nationally representative sample and asked respondents to self-report their alcohol intake during one week in 2001. Those estimates of alcohol consumption were not validated independently and so some degree of misclassification of alcohol intake (under-reporting) is possible. Indeed, national sales data indicate that alcohol consumption is likely to be considerably higher than suggested by self-reported surveys. However, the cancer risk estimates we used to calculate the population attributable fraction were also generated from self-reported consumption data, which are likely to overestimate the risk related to actual (as opposed to self-reported) alcohol consumption. We therefore considered it most appropriate to use prevalence data that most closely matched the exposure data used to generate the cancer risk estimates (i.e. self-reported data).

We used a similar methodology to the PAF project undertaken in the United Kingdom, except that the relative risks used in our analysis were obtained from more recent meta-analyses than in the UK study. Even with different risk estimates and different underlying distributions of alcohol consumption, the proportions of cancers attributable to alcohol were quite similar for Australia and the UK (online supplementary file: Table S3). Our PAF estimates for Australia differed from the WCRF/AICR preventability estimates for the US and UK populations, however, owing to quite different methodologies (online supplementary file: Table S3). The WCRF/AICR calculated PAFs using ‘high’, ‘medium’ and ‘low’ levels of alcohol consumption, with the definition of ‘high’ intake varying across cancer sites: oral cavity and pharynx (≥37 g/day); liver (≥30 g/day); colorectum (≥20 g/day); breast (≥15 g/day); and oesophagus (drinkers versus non-drinkers). The PAF estimates for the French population used statistics on the production, sales, imports and exports of alcohol to derive estimates of the prevalence of personal consumption. This methodology yields consistently higher estimates of intake than methods based on self-reported individual consumption, with consequent effects on the PAF estimates (online supplementary file: Table S3).

Our estimates of PAF assume that the effect of alcohol is independent of other causal factors. Thus, we used relative risk estimates that were adjusted for the potentially confounding effects of other exposures, although it is possible that some residual confounding by factors such as smoking, poor diet and physical inactivity remains. Similarly, we were unable to model possible interactions between smoking and alcohol that are likely to affect cancers of the aero-digestive tract, especially the oral cavity, pharynx and oesophagus. This is because reliable estimates of relative risk are not available for these interactions. As such, some of the effect reported here for alcohol is likely to be due to smoking. In addition, we had no data on the prevalence of binge drinking, nor secure measurement of its cancer-related risks.

Finally, we assumed log-linear associations between alcohol intake and cancer risk, which may not describe the true biological association for different sites. Indeed, there is a concern that possible adverse health effects associated with low levels of alcohol consumption may have been estimated poorly in epidemiologic studies. We do not have data to address this directly, but additional sensitivity analyses suggested that the cancer burden attributable to alcohol consumption
Cancers in Australia in 2010

Monitoring of trends in risk factor prevalence underscoring the importance of continued prevalence of all causal factors for that cancer, in cancer incidence at a particular site will while others less so. Thus, any future trends in cancers causally associated with alcohol-related cancers could be anticipated. In particular, rising alcohol consumption in young women could lead to increased numbers of cancers of the breast and other organs in the future. It is worth noting, however, that all of the cancers causally associated with alcohol are also caused by other factors, some of which are becoming more prevalent while others less so. Thus, any future trends in cancer incidence at a particular site will reflect the cumulative effects of changes in the prevalence of all causal factors for that cancer, underscoring the importance of continued monitoring of trends in risk factor prevalence and cancer incidence. While total abstinence from alcohol is an achievable target, these analyses suggest that reducing alcohol intake among heavy drinkers from current levels to those recommended by national guidelines could prevent almost 1,500 cancers each year in Australia.

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Supporting Information

Additional supporting information may be found in the online version of this article:

Supplementary Table 1: Proportion of adult Australians (age and sex) by average daily intake (grams) of alcohol over a week, 2001.

Supplementary Table 2: Impact of alternative alcohol consumption distributions: numbers of cancers and potential impact fractions (Pf).

Supplementary Table 3: Comparison of calculated PAFs for Australia with other international reports.