Cancers in Australia in 2010 attributable to the consumption of alcohol

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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

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;[1,5] however, IARC noted that residual confounding by smoking could not be excluded.[1] The conclusions of these two agencies are summarised in Table 1.

Alcohol is not directly mutagenic; however, there is evidence that reactive metabolites of alcohol, such as acetaldehyde, have carcinogenic properties.[2] It is speculated that alcohol may potentiate cancer development indirectly, e.g. by acting as a solvent for ingested carcinogens or through chemical processes such as prostaglandin production or generating free-radical oxygen species. It is commonly observed that people who consume large volumes of alcohol often smoke or have a diet lacking essential nutrients, placing them at increased risks of cancer.[2] Disentangling the independent effects of alcohol consumption from the carcinogenic actions of tobacco smoke and other factors is not straightforward, particularly for cancers of the upper airways, digestive tract and pancreas.[6,7]

The *Australian Guidelines to Reduce Health Risks from Drinking Alcohol*,[8] released by the National Health and Medical Research Council of Australia (NHMRC) in 2009, recommend that healthy men and women, aged 18 years and over, drink no more than two standard drinks on any day to reduce the lifetime risk of harm from alcohol-related disease or injury; and no more than four standard drinks on a single occasion to reduce the risk of alcohol-related injury arising from that occasion. One standard drink is defined as 10 g of alcohol (equivalent to 12.5 mL of pure alcohol). The guidelines emphasise that these recommendations do not represent a 'safe' or 'no-risk' drinking level. At the recommended level, the lifetime risk of death from an alcohol-related disease is around 0.4 in 100 people. Above this level, the risk increases with the number of drinks per day and is higher than 1 in 100 at three drinks per day. Above three drinks per day, the risk increases more sharply for women than for men.[8]

The Cancer Council of Australia[9] recommends that "to reduce the risk of cancer, people limit their consumption of alcohol, or better still avoid alcohol altogether. For individuals who choose to drink alcohol, consumption should occur within the NHMRC guidelines". The World Cancer Research Fund/American Institute for Cancer Research[2] also notes that there is no evidence for a "safe limit" of alcohol intake.

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Earlier studies have estimated the proportion of cancers in Australia attributable to alcohol consumption. Some are now out of date;[10] others[11] have used approaches that do not permit comparison with recent international efforts.[12] 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.[13] 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.[2] Hence, for cancers of the oral cavity, pharynx, larynx and oesophagus, we used relative risk estimates from recent meta-analyses (oral cavity,[14] pharynx[14] and larynx[15]) and pooled analyses (oesophageal SCC[16]) (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 and strength of each drink are unknown.[2] In addition, the WCRF/AICR[2] 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:

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

We performed sensitivity analyses using the same relative risks as used by the UK PAF Project.[12] Thus, for most cancers we used relative risks published in an earlier meta-analysis.[17] For breast cancer, relative risks were sourced from a meta-analysis of 53 studies by the Collaborative Group on Hormonal Factors in Breast Cancer;[18] while relative risks for colorectal cancer were sourced from four meta-analyses[19-21] (including WCRF[2]).

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

Cancer Site (ICD-10 Codes)	Level of Evidence – IARC[1]	Level of Evidence – WCRF[2,4,5,13]
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

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)[22] 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

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[a]
Oral cavity (C01-C06) and pharynx (C09-C14)	Meta-analysis of 43 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.[14]	0 g/day Ref 10 g/day 1.29 (1.25-1.32) 25 g/day 1.85 (1.74-1.96) 50 g/day 3.24 (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)	1.021 per g/day
Oesophagus (SCC) (C15)[b]	Pooled analysis of nine population-based case-control studies and two cohort studies (BEACON Consortium). 1,106 cases of ESCC and 9,253 controls.[16]	0 g/day Ref >0-<7 g/day 0.8 (0.56-1.14) 7-<14 g/day 1.23 (0.55-2.75) 14-<42 g/day 2.56 (1.10-5.96) 42-<70 g/day 4.56 (2.32-8.96) 70-<98 g/day 7.17 (2.98-17.25) ≥98 g/day 9.62 (4.26-21.71)	1.022 per g/day
Colon (C18, C19)	Meta analysis of 12 studies (10 cohort, 1 case-cohort, 1 nested case-control). Moderate heterogeneity explained largely by region of study.[13]	1.08 (1.04-1.13) per 10g/day	1.008 per g/day
Rectal (C20)	Meta analysis of 11 studies (10 cohort, 1 nested case-control). Low heterogeneity.[13]	1.10 (1.07-1.12) per 10g/day	1.009 per g/day
Liver (C22)	Meta-analysis of six cohort studies. No heterogeneity.[2]	1.10 (1.02-1.17) per 10g/day	1.009 per g/day
Larynx (C32)	Meta-analysis of 38 case-control and 2 cohort studies.[15]	0 g/day Ref 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)	1.013 per g/day
Breast (C50)	Meta-analysis of nine cohort studies. High heterogeneity, partly explained by differential adjustment for age and reproductive history.[2]	1.10 (1.06-1.14) per 10g/day	1.009 per g/day

a: assuming a log-linear relationship

b: Oesophageal cancer (squamous cell carcinoma) with histology codes 8050-8082.

their consumption in that week was more, about the same, or less than usual.[23] 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/7).[23] 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[24]). 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:[25]

$$\mathrm{PAF} = \frac{\Sigma(p_x \times \mathrm{ERR}_x)}{1 + \Sigma(p_x \times \mathrm{ERR}_x)}$$

where p_x is the proportion of the population and ERR_x is the excess relative risk (RR_x-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:

$$ERR_x = \exp(R_g \times G_x) - 1$$

where R_g 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 product of age-, sex- and cancer site-specific PAFs and the corresponding number of incident cancers in 2010.[26] Because histology-specific incidence data for oesophageal cancers were not available for 2010, we applied the average 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*,[8] 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:[27]

$$PIF = \frac{\sum_{x=1}^{n} p_x RR_x - \sum_{x=1}^{n} p_x RR^*_x}{\sum_{x=1}^{n} p_x RR_x}$$

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 year age categories (14–15%), see supplementary file: Table S1, 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 outcome[a]	Oral cavity (C01-C06)[2] 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 cancer[d]	
	PAF	Obs.	Exc.	PAF	Obs.	Exc.	PAF	Obs.	Exc.	PAF	Obs.	Exc.	PAF	Obs.	Exc.	PAF	Obs.	Exc.	PAF	Obs.	Exc.	Obs.	Exc.
Males																							
25–34 yrs	31.3	17	5	32.6	0	0	10.0	37	4	12.7	18	2	12.7	6	1	18.5	2	0				1,042	12
35–44 yrs	41.4	85	35	42.8	5	2	14.3	134	19	17.9	79	14	17.9	25	4	25.6	7	2				2,214	76
45–54 yrs	40.1	310	124	41.5	29	12	14.0	469	66	17.5	296	52	17.5	179	31	25.0	62	16				6,632	301
55–64 yrs	41.5	462	192	42.9	65	28	14.7	1,187	174	18.4	709	130	18.4	271	50	26.0	149	39				16,279	613
65–74 yrs	38.8	336	130	40.1	88	35	13.3	1,817	242	16.7	744	124	16.7	253	42	23.9	166	40				19,513	613
75–84 yrs	31.9	146	47	33.1	70	23	10.5	1,557	163	13.2	538	71	13.2	229	30	19.1	118	23				14,520	357
85+ yrs	1.1	48	1	1.2	22	0	0.3	498	2	0.4	159	1	0.4	53	0	0.6	37	0				4,968	4
Total		1,404	534		279	100		5,699	670		2,543	394		1,016	158		541	120				65,168	1,976
PAF_{aw}	38.0			36.1[e]			11.7			15.5			15.7			22.0						PAF_{aw}=	3.0
Females				10.0[f]																			
25–34 yrs	11.0	15	2	11.5	0	0	3.6	40	1	4.6	16	1	4.6	4	0	6.6	1	0	4.6	258	12	1,401	16
35–44 yrs	13.0	44	6	13.5	2	0	4.4	134	6	5.5	65	4	5.5	7	0	7.9	2	0	5.5	1,420	79	3,637	95
45–54 yrs	16.9	100	17	17.6	11	2	5.5	422	23	6.9	198	14	6.9	33	2	10.0	8	1	6.9	3,385	235	7,812	294
55–64 yrs	16.2	145	24	16.9	35	6	5.4	908	49	6.8	317	22	6.8	72	5	9.8	23	2	6.8	3,893	265	11,042	373
65–74 yrs	14.0	128	18	14.6	58	9	4.6	1,346	62	5.8	350	20	5.8	86	5	8.3	26	2	5.8	2,845	164	11,073	280
75–84 yrs	10.9	106	12	11.4	76	9	3.6	1,522	56	4.6	319	15	4.6	101	5	6.6	20	1	4.6	1,617	74	9,819	172
85+ yrs	0.2	54	0	0.3	50	0	0.1	794	1	0.1	159	0	0.1	70	0	0.1	4	0	0.1	756	1	5,166	2
Total		592	79		232	26		5,166	198		1,424	76		373	17		84	6		14,174	830	49,950	1,232
PAF_{aw}	13.1			10.9[e]			3.8			5.2			4.7			8.0			5.8			PAF_{aw}=	2.5
Persons				6.1[f]																			
25–34 yrs		32	7		0	0		77	5		34	3		10	1		3	0		258	12	2,443	28
35–44 yrs		129	41		7	2		268	25		144	18		32	4		9	2		1,420	79	5,851	171
45–54 yrs		410	141		40	14		891	89		494	66		212	33		70	17		3,385	235	14,444	595
55–64 yrs		607	216		100	34		2,095	223		1,026	152		343	55		172	41		3,893	265	27,321	986
65–74 yrs		464	148		146	44		3,163	304		1,094	144		339	47		192	42		2,845	164	30,586	893
65–84 yrs		252	59		146	32		3,079	219		857	86		330	35		138	24		1,617	74	24,339	529
85+ yrs		102	1		72	0		1,292	3		318	1		123	0		41	0		756	1	10,134	6
Total		1,996	613		511	126		10,865	868		3,967	470		1,389	175		625	126		14,174	830	115,118	3,208
PAF_{aw}	30.6			24.7[e]			8.0			11.8			12.7			20.1			5.8			PAF_{aw}=	2.8
				8.9[f]																			

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

e: % of oesophageal squamous cell carcinomas

f: % of oesophageal carcinomas (in adults 25+ yrs)

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 scenarios. 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 cases attributable to alcohol consumption were cancers of the colon (n=868) and female breast (n=830).

Previous assessments of the fraction of cancers in Australia attributable to alcohol consumption have derived qualitatively similar estimates. For example, Begg and colleagues,[10] 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,[9] 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*.[11] 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,[12] 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).[28] 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[29] 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.[7,30,31] 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.[32]

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.[33] We do not have data to address this directly, but additional sensitivity analyses suggested that the cancer burden attributable to alcohol consumption

would increase to 3.4% and 4.0% if the relative risks of low levels of consumption (less than 20 g/day) compared to zero intake were doubled and trebled respectively (data not shown).

In conclusion, we estimate that more than three thousand cancer cases in Australia in 2010 were attributable to alcohol consumption. Over the decade between the 2001 and 2011-12 Australian Heath Surveys, average alcohol consumption has been relatively stable with the most recent Survey (2011-12) reporting 19.4% of adults consuming more than two standard drinks per day, compared to 18.5% in 2000-01.[34] If an upward trend in alcohol consumption should emerge, a rise in the incidence of 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.[35] While total abstinence from alcohol is an unachievable 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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PAF Project

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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 (PIF).

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