Cancers in Australia in 2010 attributable to inadequate consumption of fruit, non-starchy vegetables and dietary fibre

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Abstract

Objectives: To estimate the number and proportion of cancers occurring in Australia in 2010 attributable to consumption deficits in fruit, non-starchy vegetables and dietary fibre.

Methods: We estimated the population attributable fraction (PAF) for cancers causally associated with inadequate intake of fruit and non-starchy vegetables (oral cavity, pharynx, oesophageal squamous cell carcinoma, stomach, larynx); inadequate intake of fruit (lung); and insufficient intake of fibre (colorectum). We used standard formulae incorporating prevalence of exposure (1995 National Nutrition Survey) and relative risks from independent studies.

Results: Overall, 1,555 (1.4% of all) and 311 (0.3% of all) cancers were attributable to inadequate intakes of fruit and non-starchy vegetables, respectively. A further 2,609 colorectal cancers (18% of colorectal) were attributable to insufficient fibre intake. If Australians increased their fibre intake by eating the recommended daily intakes of fruit and vegetables, an estimated 1,293 (8.8%) colorectal cancers could be prevented.

Conclusions: One in six colorectal cancer cases was attributable to inadequate intake of dietary fibre and about 1,800 cancers at other sites were attributable to insufficient fruit and non-starchy vegetable consumption.

Implications: Increasing the proportion of Australians who consume the recommended intake of fruit, vegetables and fibre could prevent up to 4% of all cancers.

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

Research has shown that many cancers are caused by lifestyle choices that are potentially modifiable, such as diet. In the Second Expert Report on Food, Nutrition, Physical Activity and the Prevention of Cancer, the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) concluded there was a probable causal relationship between consumption of fruit (eaten as fruits, not fruit juices) and non-starchy vegetables and a reduced risk of cancers of the oral cavity and pharynx, oesophagus, stomach and larynx; and between consumption of fruit and reduced risk of lung cancer.[1] The WCRF also concluded that the consumption of dietary fibre, defined as foods naturally containing fibre (fruit, vegetables, legumes and whole grains), as well as foods that have had fibre added to them, probably decreased the risk of colorectal cancer. In the more recent Continuous Update Project (CUP) Report for colorectal cancer, the WCRF concluded that there was now a convincing level of evidence for this being a causal relationship.[2] For the primary prevention of cancer, it is of interest to determine the magnitude of the potential reduction in cancer incidence if everyone were to consume the recommended amounts of fruit, vegetables and fibre.

The *Australian Dietary Guidelines* released in 2013[3] recommend that adults eat two serves (equivalent to 300 g) of fruit and five serves (equivalent to 375 g) of vegetables including potatoes (no more than one serving per day and not fried) and legumes, per day. These guidelines also encourage the consumption of foods high in dietary fibre such as vegetables, fruit and wholegrain foods. The National Health and Medical Research Council (NHMRC) has declared an 'Adequate Intake' (AI) level of dietary fibre based on median daily intakes in the 1995 National Nutrition Survey of Australia and the 1997 National Nutrition Survey of New Zealand.[4] For men and women aged 19 years and over, the AIs are 30 g/day and 25 g/day, respectively.[4] No upper level of daily intake for dietary fibre has been set, as the NHMRC considered that high fibre intakes imposed no deleterious effects when part of a healthy diet.[4]

Using the *Australian Dietary Guidelines* and NHMRC levels to define an adequate intake, we estimated the fraction of cancers diagnosed in the Australian population in 2010 that were attributable to inadequate intake of fruit, non-starchy vegetables and fibre.

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Methods

Based on the WCRF/AICR causality assessments, we assessed the fraction of cancers of the oral cavity and pharynx, oesophagus, stomach and larynx attributable to inadequate consumption of fruits and non-starchy vegetables, the fraction of lung cancer attributable to inadequate fruit consumption, and the fraction of colorectal cancer attributable to inadequate fibre consumption. Because the WCRF concluded that evidence for a protective effect of fruit and vegetables on colorectal cancer was limited, we did not estimate colorectal cancer PAFs for these exposures specifically. However, given that fruits and vegetables are a primary source of dietary fibre, we performed additional calculations estimating the effects of increasing dietary fibre through those sources.

Relative risk estimates

Fruit and non-starchy vegetables

The relative risks (RR) for the association between fruit and non-starchy vegetable consumption and stomach cancer, and between fruit consumption and lung cancer, were sourced from the WCRF[1] because these estimates were based on meta-analyses of data (principally collected using food frequency questionnaires) from multiple cohort studies (Table 1). WCRF also published RRs for the association between fruit and vegetable consumption and risk of cancers of the oral cavity and pharynx, larynx and oesophagus; however, those estimates were based on data from case-control studies, widely acknowledged to be susceptible to numerous biases (selection and recall),[5-9] which are less problematic in prospective studies. Therefore, in our primary analysis, we used RR estimates from the NIH-AARP Diet and Health Study[10,11] for the association between fruit and non-starchy vegetable consumption and cancers of the oral cavity and pharynx, larynx and oesophagus (Table 1). The NIH-AARP Study is a large cohort study (490,802 participants) with a mean follow-up of 4.5 years and includes analysis of vegetable intake that excluded potatoes.[10,11] Dietary intake was assessed using a food frequency questionnaire, calibrated using two non-consecutive 24-hour recalls in a sample of participants. The RR estimates reported in the NIH-AARP cohort were converted from estimates per serve/1,000 calories/day to estimates based on g/day using the WCRF methodology for converting dose–response results to a common scale.[12] Information about the average serving size (80 g) and the mean daily intake (1,693 calories/day) in the NIH-AARP cohort study gave a conversion of 1 serving = 80 g x 1.693 = 135 g/day (personal communication, C. Abnet via email dated 25 April, 2014).

We performed sensitivity analyses to estimate PAFs for cancers of the oral cavity, pharynx, larynx and oesophagus by using relative risk estimates from the WCRF[1] and two additional cohort studies (see supplementary file: Table S1, available with the online version of this article).

Fibre

The RR for the association between dietary fibre intake and colorectal cancer used in the primary analysis was sourced from a case-control study nested within seven UK cohort studies[13] (Table 1). This RR was selected over those published by the WCRF because fibre intake was measured using food diaries, rather than food frequency questionnaires (FFQs), and food diaries have been shown to have less measurement error than FFQs.[14,15] In addition, the prevalence data for fibre consumption used in this analysis were measured through a single 24-hour dietary recall.[16] However, we also conducted a sensitivity analysis using the relative risk from the WCRF CUP 2010 (0.90 per 10 g/day of dietary fibre, equivalent to an increase in risk of colorectal cancer of 1.054×10^{-2} per g/day fibre deficit).[12] The WCRF estimate was substantially closer to the null than the one used in our primary analysis.

Exposure measurement – mean daily intake

The latent period between dietary intake and cancer diagnosis is uncertain and may vary by cancer site. We assumed a latent period of more than 10 years, and so used data from the 1995 National Nutrition Survey (NNS)[17] to obtain prevalence estimates of fruit, vegetable and fibre intake. The prevalence data for fruit, vegetables and fibre consumption were obtained primarily from the daily food consumption (24-hour recall) method. Ten per cent of the original sample completed a second 24-hour recall interview to provide adjustment for within-person variation.[16] That particular survey had the additional benefit of allowing us to exclude potatoes from the estimates of vegetable consumption. The mean daily intake (g/day) of vegetables was adjusted to exclude potatoes by subtracting the mean daily intake of potatoes. Deficits in consumption of fruit and non-starchy vegetables, respectively, were calculated by subtracting the mean

Table 1: Summary risk estimates for the associations between fruit and vegetable consumption and dietary fibre intake and site-specific cancers.

Cancer (ICD-10 code)	Reference	Study	Non-starchy Vegetables		Fruit		Dietary Fibre	
			RR (95% CI)	Risk per g/day deficit[a]	RR (95% CI)	Risk per g/day deficit[a]	RR (95% CI)	Risk per g/day deficit[a]
Stomach (C16)	World Cancer Research Fund (2007)[1]	Meta-analysis of 7 cohort studies (vegetables) and 8 cohort studies (fruit)	0.98 (0.91-1.06) per 100g/day (I^2 44%, Q 14.4, df 8, p 0.07)	0.2×10^{-3}	0.95 (0.89-1.02) per 100g/day (I^2 30%, Q 11.5, df 8, p 0.2)	0.51×10^{-3}		n/a
Lung (C34)	World Cancer Research Fund (2007)[1]	Meta-analysis of 14 cohort studies	n/a		0.94 (0.90-0.97) per 80g/day (I^2: 34%)	0.77×10^{-3}		n/a
Oral cavity & pharynx (C00-C06, C09, C12-C14), larynx (C32)	Freedman et al (2008)[11]	NIH-AARP Diet and Health Study (1995-96); 490,802 participants, 787 cancers (follow-up to 2000)	0.89 (0.82-0.97) per 135 g/day	0.86×10^{-3}	0.94 (0.85-1.04) per 135 g/day	0.46×10^{-3}		n/a
Oesophagus (SCC) (C15[b])	Freedman et al (2007)[10]	NIH-AARP Diet and Health Study (1995-96); 490,802 participants, 103 oesophagus SCCs (follow-up to 2000)	0.84 (0.66-1.07) per 135 g/day	1.29×10^{-3}	0.56 (0.38-0.82) per 135 g/day	4.29×10^{-3}		n/a
Colorectal (C18-C20)	Dahm et al (2010)[13]	Nested case-control study using data from seven UK cohort studies. 579 colorectal cancers (380 colon, 199 rectal)	n/a		n/a		0.84 (0.70-1.00) per 6g/day	2.9×10^{-2}

a: For formula see text

b: Squamous cell carcinoma of the oesophagus (histology codes 8050 – 8082)

daily intake of fruit/non-starchy vegetables in each age and sex category from the recommended daily intake of 300 g (2 serves) of fruit and 260 g of non-starchy vegetables (3.5 serves reduced from 5 serves (375 g/day) to adjust for an estimated 1 serving per day of potatoes and 0.5 of legumes), see online supplementary file: Table S2.[3]

The principal sources of dietary fibre for the adult population in 1995 were cereals and cereal-based products (45% men, 43% women); followed by fruit and vegetable products and dishes (37% men; 41% women).[18] The prevalence of different levels of dietary fibre intake by age and sex in Australia in 1995 was derived using the percentile distribution of adjusted daily dietary fibre intake (g) by age and sex[18] (online supplementary file: Table S3).

To account for population ageing with time since exposure, and to accommodate the assumed latent period of at least 10 years, 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 29–34 year age group in 2010 was attributed to inadequate fruit/vegetable/fibre intake in the 19–24 year age group in 1995).

Statistical analysis

Relative risks for the cancers of interest associated with increasing intake of fruits, non-starchy vegetables and dietary fibre are less than unity (i.e. protective). We modelled the impact of insufficient intake of these dietary items in the diet. The increase in risk associated with a *deficit* per g in daily consumption of fruit, non-starchy vegetables or dietary fibre was calculated, assuming a log-linear relationship between exposure and risk relationship (i.e. that the natural logarithm of the relative risk exhibits a linear relationship with intake):

$$Increase\ in\ risk\ per\ g/day\ deficit = (\ln(1/RR_x))/x$$

where RR_x is the relative risk associated with a fruit, vegetable or fibre intake of x g/day for the specified cancer.

We assumed a log-linear relationship because this is the model used by the studies that reported the RRs we used to calculate the PAFs and it is the method recommended by the WCRF.[19]

The RR for a deficit in consumption for each age and sex category of fruit, vegetable or fibre intake (RR_y) was then calculated as:

*RR_y = exp(Increase in risk * deficit in consumption by age, sex, and fruit, vegetable or fibre intake category)*

where the deficit in g in consumption was determined by subtracting the mean fruit/non-starchy vegetable intake for each age and sex group from the recommended daily intake (260 g for non-starchy vegetables and 300 g for fruit); and the mid-point of each dietary fibre intake category (in g per day) from the recommended adequate intake levels set by the NHMRC (30 g for adult males and 25 g for adult females), see online supplementary file: Table S2 and Table S3).

Because detailed information was not available describing the proportion of the population consuming different amounts of fruits and vegetables, we used mean daily intakes to estimate the PAF for each cancer. To do this, we first estimated the numbers of cancers attributable to deficits in consumption of fruits and non-starchy vegetables, separately, in each age and sex category, as follows:

Excess Attributable cases = Observed cases – Observed cases/RR

Then, we calculated PAFs for each cancer site by dividing the number of cases in excess in each age and sex category by the total number of cases observed in each category:

PAF = Excess Attrib. cases/Observed cases

The total number of excess cases for fruit and non-starchy vegetables for each cancer site was then summed and expressed as a proportion of the total number of all incident cancers (excluding basal cell and squamous cell carcinoma of the skin) in Australian adults in 2010.

For dietary fibre, we calculated the population attributable fraction (PAF) using the standard formula,[20] as:

$$PAF = \frac{\Sigma(p_x\ x\ ERR_x)}{1 + \Sigma(p_x\ x\ ERR_x)}$$

where p_x is the prevalence of dietary fibre consumption by age, sex and intake category and ERR_x is the excess relative risk (RR–1) associated with each dietary fibre consumption category.

To obtain the number of cancers attributable to inadequate dietary fibre consumption, the PAF was multiplied by the number of incident colorectal cancers in 2010[21] for each age and sex category. This number 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 29 and over in 2010.

Potential impact of increasing fruit and vegetable intake on cancers attributable to dietary fibre

We also estimated the number of colorectal cancers in 2010 that would have been prevented in a hypothetical situation where everyone in the adult population met the recommended number of daily servings for fruit and non-starchy vegetables, thereby increasing daily dietary fibre intake. We used the proportion of fibre obtained from fruit and non-starchy vegetables and the mean daily intake of fibre for the adult male and female population (19+ years) from the 1995 NNS[18] to estimate the average fibre intake (g/day) through fruit/non-starchy vegetables. On the assumption that fruit and vegetable intake increased to the recommended 2 serves/day of fruit and 3.5 serves/day of non-starchy vegetables (excluding an estimated 1 serve of potatoes and 0.5 serves of legumes), we calculated the average increase in fibre consumption resulting from the increase in fruit and vegetable consumption. We then used the relative risk per gram deficit in dietary fibre to estimate the new relative risks across all intake categories and calculated the potential impact fraction (PIF) using the formula of Barendregt and Veerman[22] as:

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

where p_x is the prevalence of dietary fibre consumption by age, sex and intake category, RR_x is the relative risk for that category compared to no fibre intake deficit at the observed level of fibre intake and RR^*_x is the new relative risk assuming fruit and vegetable intake increased to the recommended levels.

We calculated the number of colorectal cancer cases that would have occurred in Australia in 2010 assuming that the alternative scenario of all Australian adults consuming the recommended daily serves of fruit and vegetables had prevailed. The PIF is then the proportional difference between the observed number of cancers and the number expected under the alternative scenario.

Results

Fruit and non-starchy vegetable intake

The mean daily intake of non-starchy vegetables was higher for men than for

women across all age groups (online supplementary file: Table S2). Men aged 45–64 years had the highest mean daily intake (194 g) while women aged 19–24 years had the lowest (149 g). The mean daily deficit of non-starchy vegetable intake (against the recommended 260 g) was 83 g for men and 98 g for women. In 1995, Australians were consuming, on average, one serve of fruit per day (150 g) – about half the recommended amount. For both men and women, mean daily fruit intake was highest in the 65+ years age category (179 g for men, 176 g for women), and lowest for men and women aged 19–24 years (89 and 92 g respectively) (online supplementary file: Table S2). The mean daily deficit in fruit intake (recommended 300 g) was 159 g for men and 154 g for women.

An estimated 311 cancers (207 in men and 104 in women) diagnosed in Australian adults in 2010 could be attributed to inadequate intake of non-starchy vegetables (Table 2), corresponding to 0.3% of all cancer cases in people aged 29 years and over (excluding basal cell carcinoma and squamous cell carcinoma of the skin). Cancer sites with the highest proportion of cases attributable to inadequate vegetable intake were oesophagus (9.8% of squamous cell carcinoma (SCC) cases; 3.5% of all oesophageal cancer cases) and oral cavity and pharynx (6.6%). An estimated 1,555 cancers (968 in men and 587 in women) diagnosed in Australian adults (aged 29 years and over) in 2010 could be attributed to inadequate fruit intake (Table 2), corresponding to 1.4% of all cancer cases (excluding basal cell carcinoma and squamous cell carcinoma of the skin). For inadequate fruit intake, the cancer sites with the highest PAFs were oesophagus (43% of oesophageal SCC cases; 15% of all oesophageal cancer cases) and lung (9.6%) (Table 2).

Sensitivity analyses using the higher relative risk estimates from WCRF (derived from case-control studies) led to substantially higher estimates of the numbers and proportions of all cancers attributable to deficits in fruit and vegetable consumption (oral cavity and pharynx: fruit 37%, vegetables 41%; larynx: fruit 36%, vegetables 39%; oesophagus: fruit 19%, vegetables 7%) see online supplementary file: Table S4.

Dietary fibre intake

The mean daily intake of dietary fibre was higher for men than women across all age groups. Overall, the mean daily intake for men was 26 g of fibre/day, while for women the mean daily intake was 20 g/day. The proportion of men and women with an adequate daily intake of dietary fibre was 30% and 18%, respectively (online supplementary file: Table S3).

An estimated 2,609 (18%) of colorectal cancer cases diagnosed in adults aged 29 years and over in 2010 were attributable to a deficit in dietary fibre consumption, corresponding to 2.3% of all cancer cases at these ages excluding basal cell carcinoma and squamous cell carcinoma of the skin (Table 3). In sensitivity analyses using the RR from WCRF,[12] an estimated 959 (6.5%) colorectal cancer cases diagnosed in 2010 were attributable to deficits in dietary fibre consumption; 0.8% of all cancer cases.

Potential impact of increasing fruit and vegetable intake on cancers attributable to dietary fibre

As noted above, fruits and vegetables are the second highest source of fibre in the Australian diet after cereals. If Australians had consumed fruits and vegetables at recommended levels in 1995, then the average daily intake of dietary fibre would have increased by 5.1 g for men and 5.3 g for women. This would have brought the total average daily dietary fibre intake to 31 g for men and 25.6 g for women – just over the recommended adequate daily intake amounts for both sexes. We estimate that increasing fibre intake solely through increasing fruit and vegetable intake would have reduced the total number of colorectal cancers in 2010 by 1,293 cases from 14,776 to 13,463 (PIF 8.8%).

Discussion

These analyses suggest that more than 300 and 1,500 cancer cases diagnosed in Australia in 2010 were attributable to inadequate intakes of non-starchy vegetables and fruits, respectively, and that at least 1,000 (and more likely up to 2,600) colorectal cancers were attributable to inadequate levels of dietary fibre. The site with the greatest proportional burden of cancer due to inadequate fruit and vegetables was the oesophagus (SCC only: 9.8% for non-starchy vegetables and 43% for fruit). In absolute terms, most of the cancers attributable to an inadequate intake of fruit were lung cancers (989).

Other published studies have reported considerably higher PAFs for cancers associated with deficits in consumption of fruits and vegetables. The UK PAF project used different relative risk estimates, predominantly from meta-analyses that included case-control studies.[23] When we repeated our analyses for the Australian population using the same risk estimates, we generated markedly higher PAFs, similar in magnitude to those reported by the UK PAF project. We contend that the PAFs derived using the case-control risk estimates are likely to be inflated, since risk estimates from methodologically superior prospective studies are consistently and considerably lower. Results from the WCRF Preventability Report cannot be compared directly with our findings, since the baseline category (the theoretical optimal consumption level) varied by cancer site (ranging from ≥57.1 g of fruit per day for stomach and oral cavity and pharyngeal cancers to ≥160 g of fruit per day for lung cancer).[24]

The PAF for inadequate dietary fibre intake estimated in our primary analysis (18%) was higher than that reported for the UK (12%).[25] While we used the same dose–response relative risks as in the UK report,[13] the deficit of dietary fibre intake in the UK study was measured against a lower threshold of 23 g of intake per day (for both men and women) compared to the Australian recommended levels of 30 g/day for men and 25 g/day for women. The WCRF preventability estimates[24] (11% US, 12% UK) used categorical relative risks (three categories), with consumption of ≥30 g/day as the reference category and so are again not directly comparable to our estimates.

The methodological challenges of estimating dietary intake should be acknowledged. We obtained prevalence data from the 1995 National Nutrition Survey, which has the advantage of being a nationally representative survey. Moreover, that survey presented consumption data in a manner that allowed dose–response analyses of fruit and vegetable intake by age and sex groups. Although the survey relied on a single 24-hour food recall, which may not accurately represent usual diet at the individual level, the fact that it was conducted across a national sample suggests that it should provide a reasonable estimate of intake at the population level. Complete results from the 2011-12 Australian Health Survey (which included a dietary component), were

Table 2: Population attributable fraction (PAF) and estimated number of cancers diagnosed in Australia in 2010 attributable to inadequate intake of fruit and non-starchy vegetables.

Age at Outcome[a]	Oral Cavity and Pharynx (C00-C06,C09,C12-C14)[b]					Oesophagus SCC (C15)[b,c]					Stomach (C16)[b]					Larynx (C32)[b]					Lung (C34)[b]					All Cancers[d]				
	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit	Obs.	Exc. Veg	Exc. Fruit	PAF Veg	PAF Fruit
Males																													
29-34 yrs	23	2	2	8.9	9.2	0	0	0	0.0	0.0	4	0	0	2.2	10.3	0	0	0	8.9	9.2	10	-	1	-	15.1	595	2	3	0.4	0.7
35-54 yrs	611	44	47	7.2	7.6	34	4	18	10.6	52.5	150	3	13	1.7	8.5	69	5	5	7.2	7.6	474	-	59	-	12.5	8,845	56	142	0.6	1.6
55-74 yrs	1061	59	62	5.5	5.9	154	13	66	8.2	43.2	643	9	42	1.3	6.5	316	17	19	5.5	5.9	3,255	-	315	-	9.7	35,792	98	504	0.3	1.4
75+ yrs	349	24	19	6.8	5.4	92	9	37	10.0	40.6	512	8	31	1.6	6.0	155	10	8	6.8	5.4	2,498	-	224	-	8.9	19,488	51	319	0.3	1.6
Total	2,044	129	130			280	26	121			1,309	20	86			540	32	32			6,237	-	599			64,720	207	968		
PAF_{aw}				6.3	6.4				9.1[e]	43.4[e]				1.5	6.6				6.1	6.0					9.6				0.3	1.5
									2.7[f]	13.0[f]																				
Females																													
29-34 yrs	15	1	1	9.2	9.1	0	0	0	13.4	0.0	3	0	0	2.2	10.1	1	0	0	9.2	9.1	5	-	1	-	14.8	833	1	2	0.2	0.3
35-54 yrs	175	16	13	8.9	7.4	12	2	6	13.1	51.4	86	2	7	2.2	8.2	10	1	1	8.9	7.4	395	-	48	-	12.2	11,449	21	75	0.2	0.7
55-74 yrs	358	24	21	6.6	5.8	93	9	40	9.8	42.8	248	4	16	1.6	6.5	48	3	3	6.6	5.8	2,108	-	202	-	9.6	22,115	40	281	0.2	1.3
75+ yrs	265	20	15	7.7	5.5	125	14	52	11.3	41.2	342	6	21	1.9	6.2	24	2	1	7.7	5.5	1,526	-	139	-	9.1	14,986	42	228	0.3	1.5
Total	813	61	50			230	25	98			679	12	44			83	6	5			4,034	-	390			49,383	104	587		
PAF_{aw}				7.5	6.1				10.8[e]	42.4[e]				1.8	6.5				7.2	5.9					9.7				0.2	1.2
									6.0[f]	23.5[f]																				
Persons																													
29-34 yrs	38	3	3	9.0	9.2	0	0	0	6.7	0.0	7	0	0	2.2	10.2	1	0	0	9.1	9.1	15	-	2	-	15.0	1,428	3	5	0.3	0.5
35-54 yrs	786	60	60	7.6	7.6	46	6	24	11.3	52.2	236	5	20	1.9	8.4	79	6	6	7.4	7.6	869	-	107	-	12.4	20,294	77	217	0.4	1.1
55-74 yrs	1,419	83	83	5.8	5.8	247	22	106	8.8	43.1	891	13	58	1.4	6.5	364	20	22	5.7	5.9	5,363	-	517	-	9.6	57,907	138	786	0.2	1.4
75+ yrs	614	44	34	7.2	5.5	217	23	89	10.7	41.0	854	14	52	1.7	6.1	179	12	9	6.9	5.4	4,024	-	363	-	9.0	34,474	93	547	0.3	1.6
Total	2,857	190	180			510	51	219			1,988	32	130			623	38	37			10,271	-	989			114,103	311	1,555		
PAF_{aw}				6.6	6.3				9.8[e]	43.0[e]				1.6	6.6				6.3	6.0					9.6				0.3	1.4
									3.5[f]	15.4[f]																				

Abbreviations: yrs = years; Obs. = observed cancers in 2010; Exc. = excess cancers in 2010 attributable to inadequate intake of fruit and vegetables; 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.

b: International Classification of Diseases Code (ICD-10 code).

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 all oesophageal squamous cell carcinomas (in adults 29+ years).

f: % of all oesophageal carcinomas (in adults 29+ years).

not available at the time we conducted our analyses, and no national nutrition survey was conducted in the intervening period.

The dose–response analyses we employed took account of scaled effects for a range of intakes of fibre. Moreover, the prevalence data allowed us to perform these analyses within strata of age and sex. The effect estimates we used were derived from studies using food diaries, a measurement technique comparatively less prone to error than FFQs.[14,15] The NNS employed a 24-hour food recall to capture fibre intake, ensuring reasonable comparability with the methods used to generate the relative risk estimates.

Arguably more problematic are the estimates of relative risks to which the PAF is very sensitive. In our primary analyses of inadequate fruit and non-starchy vegetable intake we elected to use relative risks from a very large cohort study for cancers of the oral cavity and pharynx, larynx and oesophagus because of the inherent concerns regarding relative risks from case-control studies. As a result, our PAF estimates for these dietary exposures are notably lower than those from other studies.[23,24] We did not use relative risks from Boeing and colleagues[26] in the primary analysis, as they were based on a smaller number of cancer cases, did not exclude potatoes, and did not calculate separate relative risks for oesophageal SCC. Steevens and colleagues[27] considered only oesophageal SCC (and not the other head and neck cancers) using data from the Netherlands Cohort Study. Estimates from NIH-AARP Diet and Health cohort were preferred for our primary analyses as they allowed us to use relative risks for all head and neck cancers that were calculated using the same methodology (Table 1). Moreover, dietary patterns in the American cohort arguably approximate Australian patterns of intake to a greater extent than those in the Netherlands.[10,11]

As additional large, prospective studies analyse and publish their data on the associations between dietary factors and cancer, it is anticipated that risk estimates will converge on new values that better reflect the true associations. In particular, it will be important to control for the confounding effects of other factors that are known or suspected to cause cancer and that are correlated with poor diets. This particularly applies to lung cancer, where very precise measures of smoking exposures are required to minimise residual confounding.

Table 3: Population attributable fraction (PAF) and estimated number of colorectal cancers diagnosed in Australia in 2010 attributable to a deficit in dietary fibre intake.

Age at outcome[a]	Colorectal (C18-C20)[b]			All Cancers[c]	
	PAF	Obs.	Exc.	Obs.	Exc.
Males					
29-34 yrs	16.4	28	5	521	5
35-54 yrs	18.2	977	178	8,845	178
55-74 yrs	16.7	4,457	746	35,792	746
75+ years	22.1	2,752	608	19,488	608
Total		8,214	1,537	64,646	1537
PAF_{aw}	18.7			PAFaw =	2.4
Females					
29-34 yrs	18.2	28	5	701	5
35-54 yrs	15.5	818	126	11,449	126
55-74 yrs	15.4	2,921	451	22,115	451
75+ years	17.5	2,794	490	14,986	490
Total		6,561	1,072	49,251	1,072
PAF_{aw}	16.3			PAFaw =	2.2
Persons					
29-34 yrs		56	10	1,222	10
35-54 yrs		1,795	304	20,294	304
55-74 yrs		7,378	1,197	57,907	1,197
75+ years		5,546	1,098	34,474	1,098
Total		14,775	2,609	113,897	2,609
PAF_{aw}	17.6			PAFaw =	2.3

Abbreviations: Obs. = observed cancers in 2010; Exc. = excess cancers in 2010 attributable to inadequate intake of fibre; PAF = population attributable fraction (expressed as a percentage); PAF_{aw} = 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.

b: International Classification of Diseases Code (ICD-10 code).

c: Excluding basal cell carcinoma and squamous cell carcinoma of the skin.

At other cancer sites, better control for the confounding effects of alcohol intake, physical inactivity and obesity will be similarly important.

Both WCRF and IARC concluded that the evidence of a protective effect of fruit and vegetable intake on cancer risk is not strong. Although the WCRF did not classify the relationship between fruit and vegetable intake and cancer as 'convincing' and some of the pooled RRs were not statistically significant (e.g. stomach cancer), the WCRF deemed the relationship 'probable', which they define as "strong enough to support a judgement of probable causal relationship that would generally justify goals and recommendations designed to reduce the incidence of cancer".[1(p60)]

There are a number of mechanisms through which dietary factors are postulated to reduce the risks of various cancers. For example, fruit and vegetables contain substances that are potentially anticarcinogenic, such as vitamins, carotenoids and flavonoids. Consideration of the potential biologic effects of these various constituents of fruit and vegetables suggests plausible mechanisms for protective effects including reducing oxidative damage to DNA, increasing cancer cell apoptosis or increasing the activity of enzymes able to detoxify carcinogens.[28] Several biologic mechanisms have also been proposed to explain the inverse association between fibre intake and colon cancer development.[29] Dietary fibre resists digestion in the small intestine and enters the colon where it is fermented to produce short chain fatty acids (SCFAs) that may enhance the healthy composition of gut microbiota. These SCFAs also have anticancer properties such as promoting cancer cell cycle arrest and apoptosis, and inhibiting chronic inflammatory processes. Dietary fibre also increases faecal bulking and viscosity, reduces the time for proteolytic fermentation that results in harmful substances, binds potential luminal carcinogens (e.g. secondary bile acids), and shortens the contact between potential carcinogens and mucosal cells.

Accepting these methodological and mechanistic limitations, our analyses provide some idea of the scale of the cancer burden that may be amenable to prevention through higher quality diets. Our analyses suggest

that substantial gains in cancer prevention could be achieved through relatively modest increases in fruit and vegetable intake, which would have the added benefit of increasing fibre intake. We also recognise that the benefits of dietary modifications are not restricted to cancer prevention, and indeed are likely to be overshadowed by reductions in many other chronic diseases, particularly those of the cardio-vascular and metabolic systems.[8,30]

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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: Sensitivity Analyses using alternative RR estimates for the association between total vegetables and total fruit consumption and risk of cancers of the oral cavity, pharynx, larynx, and oesophagus.

Supplementary Table 2: Fruit and non-starchy vegetable intake and estimated deficit against Australian dietary guidelines by age and sex.

Supplementary Table 3: Average daily intake of dietary fibre (grams) by age and sex, Australia 1995.

Supplementary Table 4: Summary of results of sensitivity analysis for inadequate fruit and vegetable intake.