Public Health Nutrition -- NUTD 9113

Assessor:

Fruit and Vegetable Consumption & Colorectal Cancer Risk

Farrah TATE, 2001

INTRODUCTION

Colorectal cancer features as a significant public health concern; statistics reveal that it is one of the most commonly diagnosed cancers nation wide, and features highly as a leading cause of both mortality and morbidity, with significant health care system burden. Hence, colorectal cancer has been listed as one of the seven national health priority area cancers, for which approximately 50% of cases are attributable to dietary factors (NHMRC, 1999). This paper considers the evidence for an association between fruit and vegetable consumption and risk for colorectal cancer, discusses current national fruit and vegetable intake, and provides information regarding various appropriate strategies and supportive food and nutrition related policy required to increase population intake.

Significance of Colorectal Cancer in Australia

Incidence & mortality

Australian colorectal cancer incidence and mortality rates for both males and females (as summarised in Table 1) are ranked amongst the countries with the highest rates (AIHW & AACR, 2000). Approximately one in twenty Australians are likely to develop the disease before 75 years of age and colorectal cancer was the most commonly diagnosed cancer in 1997, representing 14% of total new cancer cases (AIHW & ACCR, 2000; NHMRC, 1999). Additionally, colorectal cancer was the second most common cause of cancer death, (14% of the total 4678 deaths due to cancer) behind only lung cancer (20%), contributing in total 31 573 Person Years of Life Lost (PYLL) (AIHW & ACCR, 2000).

Table 1: Summary Statistics for Colorectal Cancer

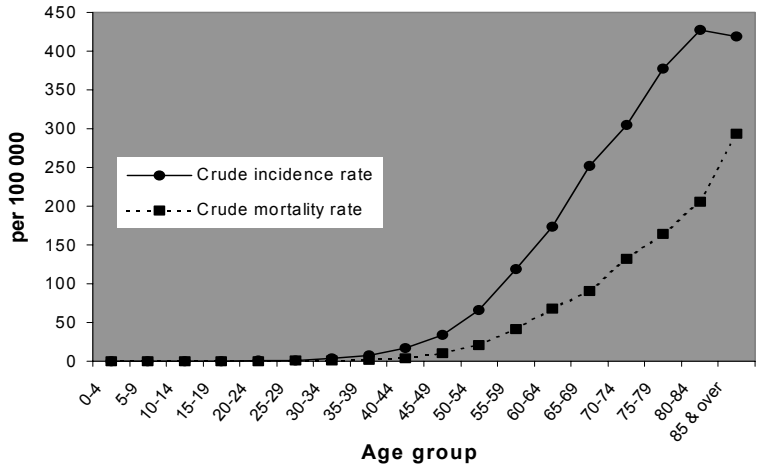
	Males	Females	Persons
Australia 1997*			
Incidence			
– Number	6139	5106	11,245
– †CR (95% CI)	66.6 (65.0-68.3)	54.8 (53.3-56.3)	60.7 (59.6-61.8)
– ‡ASR (95% CI)	68.4 (66.7-70.1)	46.6 (45.3-47.9)	56.6 (55.5-57.6)
– Life-time risk (0-74)	1 in 17	1 in 26	1 in 21
– Per cent of all cancers	14.2	14.3	14.1
Mortality			
– Number	2544	2134	4678
– †CR (95% CI)	27.6 (26.5-28.7)	22.9 (21.9-23.9)	25.3 (24.5-26.0)
– ‡ASR (95% CI)	28.8 (27.7-30.0)	18.7 (17.9-19.5)	23.2 (22.6-23.9)
– Life-time risk (0-74)	1 in 44	1 in 70	1 in 55
– Per cent of all cancers	13.3	14.3	13.8
– PYLL	18,500	13,073	31,573
Australia 1996			
DALYs (%)	252,118 (19.0)	226,461(19.2)	478,579 (19.1)

**Data: AIHW & ACCR, 2000; **Data: Mathers et al, 1999; †CR = crude rate (per 100,000); ‡ASR = age-standardised rate (per 100,000 considering the 1991 Australian population statistics); PYLL = person years of life lost; DALYs = Disability Adjusted Life Years, as explained in text.*

Generally, colorectal cancer risk increases with age (Figure 1). Most affected individuals will be over the age of 40, with incidence then rising sharply and progressively from the age of 50 (NHMRC, 1999). As the population in Australia continues to age, we can expect the number of new cases (the crude incidence rate) of colorectal cancer to increase (CCA, 2001). Correspondingly, a significant proportion of the increase in crude incidence rate depicted in Figure 2, is due to an aging population. However, upon adjusting for age there still has been some increase in incidence, particularly for males, who continue to experience higher incidence and mortality rates than females (Figure 2, Table 1). Of note, the male incidence rate rose only marginally between 1992 and 1997 (an average annual rise of 0.9%), while the female rate fell slightly (0.3%) (Figure 2). The colorectal cancer population mortality rates also fell slightly between 1993 and 1997, most likely due to earlier detection and more advanced treatment technology (AIHW & AACR, 2000).

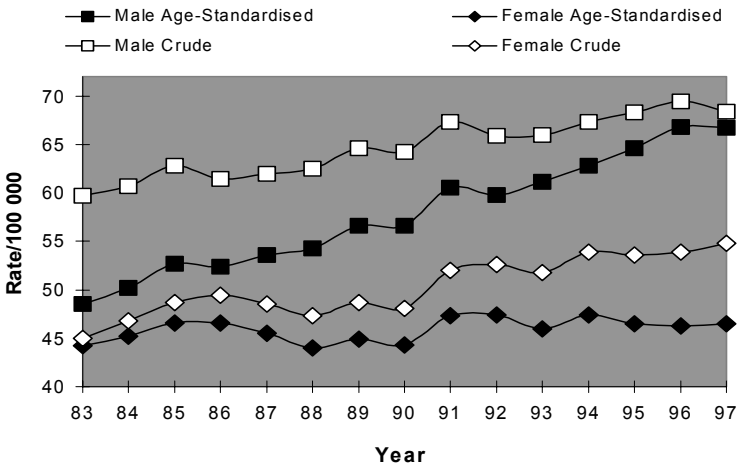
A comparison of state and territory incidence shows similar incidence and mortality rates (Figure 3), however it is difficult to compare with validity the incidence rates in States and Territories with small populations (eg NT, Tas, ACT). Implementation of a nationally delivered screening program, as widely recommended (CCA, 2001) and discussed subsequently, would likely see incidence rates increase, as has previously been observed with female breast cancer incidence rates.

Figure 1: 1997 Colorectal Cancer Crude Incidence & Mortality Rates, by Age*



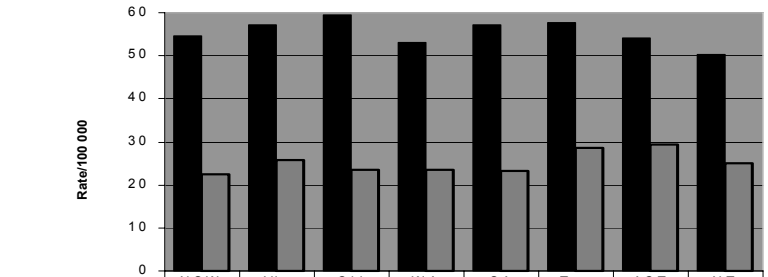
* *Data source:* AIHW & AACR, 2000

Figure 2: Crude & Age-Standardised Colorectal Cancer Incidence Rates, 1983-1997*



* *Data source:* AIHW & AACR, 2000; Age standardised to the Australian Population (1991)

Figure 3: State & Territory Age-standardised (Aust 1991) Colorectal Cancer Incidence & Mortality Rates



	NSW	Vic	Qld	WA	SA	Tas	ACT	NT
Incidence Rate	54.7	57.2	59.6	53.1	57.1	57.8	54.1	50.2
Mortality Rate	22.4	25.7	23.5	23.5	23.3	28.7	29.3	25.1

* *Data source:* AIHW & AACR, 2000; Age standardised to the Australian Population (1991)

Burden and cost estimates associated with colorectal cancer

Colorectal cancer has a mortality to incidence ratio of 0.42, suggesting a survival rate of approximately 60% on average (Mathers *et al*, 1999). Cost to the community, the individual and their family is accrued through medical treatment, and subsequent rehabilitation, disability or mortality. Disability-Adjusted Life Years (DALYs) provide the best approximation of the total burden contributed by an illness, by taking into account both cost associated with premature mortality (YLL) and/or disability (YLD), and are the equivalent of one year of lost 'healthy' life. Based on 1996 population data, Mathers and colleagues (1999) estimated colorectal cancer to be second only to cardiovascular diseases in leading causes of burden (19% & 20% of DALYs respectively). In 1993-94 dollars, health system costs attributed to colorectal cancer sum to estimates of $205 million, representing 10.8% of the total costs associated with cancer, second only to skin cancer ($298 million, at 15.6% of total costs) (Mathers *et al*, 1999).

Evidently, colorectal cancer represents a major public health problem. Hence its inclusion as one of the seven national health priority areas (NHMRC, 1999), and the need to address population prevention and screening strategies, as discussed later.

On Over-view of the Epidemiological Evidence: Is There an Assocation between Dietary Intake of Fruit and Vegetables, and Risk for Colorectal Cancer?

Most of the early epidemiological studies undertaken to examine this relationship have been retrospective, case-control studies; in comparison cohort studies and intervention/clinical trials have been so far limited (with respect to quantity but not quality of evidence). Studies considered are summarised in Table 2. Those considered in detail in the text have been chosen based on size and methodological superiority.

Evidence from retrospective Case-Control studies

A recent review of case-control studies found a statistically significant, inverse association for one or more vegetable and/or fruit categories in 79% (28/31) of colon cancer and 80% (8/13) of rectum cancer studies. However, when considered as total fruit or vegetables, or specific categories thereof, significance of results were equivocal (Steinmetz & Potter, 1996). More recently, Baghurst and colleagues (1999) reported similar findings. An extensive review undertaken by The (UK) Committee on Medical Aspects of the Food Supply (COMA, 1998) found that the relative risks for highest consumption versus lowest consumption were generally between 0.5 and 0.9. The relative risks for the smaller number of studies considered in Table 2 are more variable.

In a well-designed and controlled, large Italian study of 1953 men and women with colorectal cancer and 5155 controls, Franceschi & colleagues (1998) reported a significant but weak, inverse association between 80^{th} compared with 20^{th} percentiled raw vegetable intake and incident colon cancer (OR = 0.7; 95% CI = 0.7-0.8). A similar result was obtained for cooked vegetable consumption and incidence of rectum cancer (OR = 0.7; 95% CI = 0.6-0.8). How globally consistent and representative these findings are is debatable, and the authors acknowledge that Italians consume considerable amounts of oil (and particularly olive oil) with vegetables and salads, which *may* confer an additional, if not synergistic protective

Table 2: Selected Epidemiological Studies: Fruit/ Vegetable Intake, and Risk for Colorectal Cancer

Study	Location	Subject Description	Dietary Assessment	Summary Findings	Comments
Retrospective case-control studies					
Deneo-Pellegrini *et al*, 1996	Uruguay 1992-4	160 colorectal cancer cases, ≤ 84 years of age. 287 hospital controls.	FFQ, 61 items ? validation	RR (95% CI) for uppermost vs lowest quartile of intake: V: 0.39 (0.21-0.75); F: 0.46 (0.25-0.82); V&F: 0.38 (0.20-0.71)	Small no. cases → low statistical power No apparent validation of dietary intake data Adjusted for potential confounders: age, residence, education, family history, BMI, total energy, alcohol intake, tobacco smoking.
Franceschi *et al*, 1998	Northern & Southern Italy 1991-6	1225 colon (M: W = 1.3) & 728 rectal (M: W = 1.5) cancer cases. Median age 62 years. Cases histologically confirmed. 5155 hospital controls (M: W = 0.7). Median age 52 years.	Validated FFQ (12 vege, 11 fruit), delivered by trained interviewers. Dietary habits 2 years prior diagnosis	OR (95% CI) for 80th vs 20th percentiles of intake: Colon: RV: 0.7 (0.7-0.8); CV: 0.7 (0.8-0.8); F: 1.0 (0.9-1.0) Rectal: RV: 0.8 (0.7-1.0); CV: 0.7 (0.7-0.8); F: 0.9 (0.9-1.0)	Adjusted for potential confounders: age, sex, center, year of interview, education, physical activity, alcohol and energy intake. Estimated weekly consumption subjects: 20th percentile: 4 serves (s's) RV, 2.3 s's CV, 9 s's F 80th percentile: 12 s's RV, 6.2 s's CV, 42 s's F Note: consumption of vegetable oil with raw vegetables in salads. Well-designed/controlled study.
Kampman *et al*, 1995	Netherlands 1989-93	232 colon cancer cases, 259 population controls	Validated FFQ delivered by trained interviewers.	OR (95% CI) uppermost vs lowest quartile of intake: V: 0.4 (0.23-0.69); F: 0.82 (0.84 -1.41)	Adjusted for potential confounders: age, gender, urbanisation level, total energy intake, alcohol use, cholecystectomy & family history.
Steinmetz & Potter, 1993	Australia 1979-81	220 colon cancer cases (M: W= 1.2) 438 population based controls (M: W = 1.2)	Self-administered, validated FFQ (48 vegetables, 14 fruits)	OR (95% CI) for uppermost vs lowest quartile of intake: Men: V: 1.29 (0.67-2.51); RV: 1.27 (0.62-2.60); F: 1.74 (0.88-3.46) Women: V: 1.11 (0.50-2.45); RV: 1.34 (0.67-2.72); F: 0.90 (0.38-2.11)	Adjustment for potential confounders: occupation, family history, weight, BMI, intakes of energy, protein, fat, saturated fat & alcohol + additional factors specific to female subjects Well designed/controlled study Lowest quartile intake: V: <15 s's/week; RV: ≤0.2 s's/week; F: ≤8 s's/week Uppermost quartile intake: V: ≥32 s's/week; RV: ≥3.6 s's/week; F: ≥28 s's/week
Zaridze *et al*, 1993	Russia	217 colorectal cancer cases & community matched controls	FFQ, ? validation	OR (95% CI) for uppermost vs lowest quartile of intake consumption: V: 0.27 (0.13-0.57); F: 0.47 (0.26-0.86)	Adjusted for potential confounders: educational status & total energy intake. No apparent validation of dietary assessment
Prospective, cohort studies					
Jansen *et al*, 1999 Seven Countries Study Cohort (Ecological)	Finland, Italy, Croatia/ Serbia, Greece, Japan, United States, The Netherlands	12 763 men, aged 40 to 59 in 1960, 25 years follow-up→ 162 colorectal cancer mortalities.	7-day dietary record for sub-cohorts (1959-64)	RR (95% CI) for 10% increase in consumption above mean intake: V: 1.00 (0.89-1.12) F: 0.99 (0.96-1.02)	Mortality outcome → cannot differentiate factors that influence survival from those that affect incidence. Adjusted for potential confounders: age, smoking physical activity, BMI and intakes of energy, fat and alcohol No validation of dietary intake data.

M= men, W= women; V = vegetables, F = fruit, R = raw, C = cooked; RR = relative risk, OR = odds ratio; FFQ = food frequency questionnaire; BMI = Body Mass Index (weight/height^2); SD = standard deviation.

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Table 2 (cont.) Selected Epidemiological Studies: Fruit/ Vegetable Intake, and Risk for Colorectal Cancer

Study	Location	Subject Description	Dietary Assessment	Summary Findings	Comments
Michels *et al*, 2000. Nurses' Health Study & Health Professionals' Follow-up Study Cohorts	USA 1986-96	88 764 women & 47 325 men, 1 743 654 person-years follow-up → 937 cases colon cancer & 244 cases of rectal cancer.	Cumulative dietary intake from repeated, staggered, validated self-administered semiquantitative FFQs	RR (95% CI) for one additional serving/day: Colon Men: V: 1.01 (0.90-1.14); F: 1.08 (1.00-1.16) Women: V: 1.03 (0.97-1.10); F: 0.96 (0.89-1.03) Rectal Men: V: 1.01 (0.80-1.27); F: 1.09 (0.94-1.26) Women: V: 1.03 (0.91-1.17); F: 0.96 (0.83-1.11)	Adjusted for potential confounders: age, family history, prior sigmoidoscopy, height, BMI, physical activity, aspirin use, smoking, vitamin supplement use, alcohol consumption, total caloric intake, red meat consumption (and menopausal status and post-menopausal hormone use). FFQ differed across years Essentially a well designed/controlled study Low uppermost intake & narrow range of intakes could dilute effect: Lowest quintile: 1 serving per day Uppermost quintile: ≥5 servings per day
Steinmetz K *et al*, 1994. Iowa Women's Health Study cohort	USA 1986-91	41 837 women, aged 55-69 years, 5 years of follow-up → 167 447 person-years & 212 colon cancer cases (pathologically confirmed)	Self-administered, 127-item semi-quantitative, validated FFQ.	RR (95% CI) for uppermost vs lowest quartile of intake: V&F: 0.89 (0.57-1.40); V: 0.73 (0.47-1.13); F: 0.86 (0.58-1.29)	Adjusted for potential confounders: age, BMI, parity, age at first live birth, physical activity, smoking, education, history of polyps or colitis, and intakes of energy, fat, protein and alcohol. Well designed/controlled study. Narrow intake range could dilute effect. Mean no. servings per week: Cases: V&F: 36.5; V: 23.9; F: 12.6 Noncases: V&F: 38.0; V: 24.6; F: 13.4
Thun *et al*, 1992. Cancer Prevention Study II cohort	USA 1982-8	764 343 adults, average age 57 years, six years of follow-up → 1150 colon cancer mortalities.	Self-administered, validated FFQ.	RR (95% CI) for uppermost vs lowest quintile of intake: Men: V: 0.76 (0.57-1.02) Women: V: 0.62 (0.45-0.86)	Adjusted for potential confounders: family history, physical activity, total fat consumption, BMI & aspirin use.
Voorrips *et al*, 2000	The Netherlands 1986-92	62 573 women & 58 279 men, aged 55-69 years. 6.3 years follow-up → 659 colon & 375 rectal cancer cases.	Self-administered, validated 150-item FFQ	RR (95% CI) for uppermost compared with lowest quintiled intake: Colon: Men: V: 0.85 (0.57-1.27); F: 1.33 (0.90-1.97); Women: V: 0.83 (0.54-1.26); F: 0.73 (0.48-1.11) Rectal: Men: V: 0.88 (0.55-1.41); F: 0.85 (0.55-1.32); Women: V: 1.78 (0.94-3.38); F: 0.67(0.34-1.33)	Potatoes and mature beans were not included in vegetable consumption. Adjusted for potential confounders: family history, alcohol intake, age. Intakes similar for cases/noncases), i.e. Mean Intake (±SD): Men: Colon Cancer: V: 184.7 ± 77.6; F:160.4 ± 104.2; Rectal Cancer: V: 186.5 ± 74.5; F: 154.8 ± 122.5; Subcohort: V: 187.1 ± 76.3; F: 154.4 ± 111.8 Women: Colon Cancer: V: 182.4 ± 69.6 F: 187.0 ± 121.9; Rectal Cancer: V: 200.2 ± 77.6 F: 184.9 ± 101.5; Subcohort: V: 191.0 ± 74.5 F: 196.1 ± 118.9
Randomised Controlled Trial					
Schatzkin *et al*, 2000	USA (4 years)	2079 men & women, ≥ 35 years, who had one or more histologically confirmed colorectal adenomas removed within 6 months before randomisation	Intervention: intensive counselling, assigned to follow a diet low in fat (20% total calories) & high in fruits/ vegetables (3.5 servings per 1000 kcal). Control: standard brochure healthy eating, assigned to follow usual diet.	RR (recurrence adenomas) (95% CI) intervention vs control grp: 0.96 (0.71-1.31)	92% of intervention grp & 91% of control grp completed the trial. Consumption significantly greater in intervention grp: Servings/1000 kcal: Intervention grp: At randomiation 2.05±0.03; At year 4 3.41±0.04 Control group: At randomiation 2.00±0.03; At year 4 2.23±0.03 Absolute difference in change b/w groups (95%CI) = 1.13 (1.04-1.21).

M= men, W= women; V = vegetables, F = fruit, R = raw, C = cooked; RR = relative risk, OR = odds ratio; FFQ = food frequency questionnaire; BMI = Body Mass Index (weight/height^2); SD = standard deviation.

effect. It is likewise questionable whether case-control studies showing significant protective association of fruit and vegetable consumption and risk for colorectal cancer, and undertaken in Uruguay (Deneo-Pellegrini *et al*, 1996), and even Russia (Zaride, 1993) and The Netherlands (Kampman *et al*, 1995), are representative of consumption in most Western countries (both in type, cooking method and total amount that is used to compare upper and lower intake levels). Certainly, in discrepancy with the above cited studies, in a similarly well-controlled and designed but smaller study, considering 220 colorectal cancer cases and 438 controls, Steinmetz and Potter (1993) found no association between fruit and vegetable intake and incident colon cancer in either Australian males or females (Table 2).

Evidence from prospective Cohort studies

In a well-controlled study, Thun *et al* (1992) followed 764 343 men and women with an average age of 57 years for six years, finding an association between uppermost compared with lowest quintiled vegetable intake and colon cancer mortality, in women (RR = 0.62; 95% CI = 0.45-0.86) but not men (RR = 0.76; 95% CI = 0.52-1.02). However, The Seven Countries Study, a prospective ecological analysis of 16 cohorts in 7 countries, examined colorectal cancer mortality in 12 763 men aged 40 to 59, followed-up over a much longer period of 25 years. No statistically association was found between colon cancer mortality risk for either vegetables (RR = 1.00; 95% CI = 0.89-1.12) or fruit (RR = 0.99; 95% CI = 0.96-1.02), at an intake 10% above the mean (Jansen *et al,*1999). However, only a small sub-group of each of these cohorts had a 7-day dietary record taken, and food consumption may have varied significantly during the subsequent 25 years of follow-up.

Consistent with the above findings, an essentially well-designed study by Michels *et al* (2000), found no association between fruit and vegetable consumption and incidence of colon or rectal cancer in either males or females (Table 2). The very large and well-studied Nurses' Health Study and Health Professionals' Follow-up Study cohorts, accounted for

1 743 645 person-years of follow-up, contributing 937 cases of colon cancer and 244 cases of rectal cancer. Importantly, dietary intake was analysed several times over the follow-up period, and the results were adjusted for potential confounders of supplemental vitamin use and aspirin use, both of which many earlier studies had not considered.

Two of the more recently reported null result studies with significantly large cohorts and adequate methodology include the Iowa Women's Health Study (Steinmetz *et al*, 1994) and the Netherlands Cohort Study (Voorrips *et al*, 2000). Recall bias may account for the differences between case-control and cohort studies. With retrospective recall, cancer patients may under-report their intake due to the influence of the disease experience on past dietary estimations. Additionally, selection bias may distort case-control results as those controls who participate are more likely to be health-conscious and thus, to consume, or report consuming, more fruits and vegetables. (Michels *et al*, 2000).

However, there are valid criticisms of the Michels *et al* study and similar other prospective studies undertaken in North America and Northern Europe, which have reported relatively low high-end intakes, and an overall narrow range of lowest and uppermost intakes (Table 2). Such results could attenuate relative risks (Clark, 2000; Flood & Schatzkin, 2000). Certainly, the accuracy of dietary habits assessed in a large cohort by self-administered food frequency questionnaires is debatable. Vegetable intakes are most difficult to assess via self-administered food frequency questionnaires, particularly when portion sizes must be estimated (Clark, 2000). Previous validation studies have found the correlation coefficient for total vegetable consumption to be only 0.4 (Voorrips *et al*, 2000). This is comparable with the figures reported for most prospective studies (Michels *et al,* 1999; Voorrips *et al*, 2000).

Furthermore, most Americans receive plenty of the nutrients believed to protect against colon cancer even if they eat few fruits and vegetables, predominantly through the consumption of processed, fortified foods (Boyles, 2000). Correspondingly, Michels and colleagues (2000) acknowledge results do not exclude the possibility that, in populations for

which fruits and vegetables are important determinants of nutrient status, these may be associated with lower risk of colorectal cancer.

Clinical trials & Animal studies

A recently published randomised controlled trial (Schatzkin *et al*, 2000) provides further substantial evidence for the null association observed between fruit and vegetable intake and risk of recurrent colorectal adenomas (RR = 0.96; 95% CI = 0.71-1.31). Subjects were 2079 American men and women aged 35 years or older, who had had one or more histologically confirmed colorectal adenomas removed within six months before randomisation into low fat, high fruit and vegetable intervention or the unmodified diet control group (details provided in Table 2). Both intervention and control subjects completed an annual four-day food record followed by a food frequency questionnaire, and were followed up for a total period of four years, over which time the number of servings of fruit and vegetables per 1000 kcal increased by about two thirds in the intervention group whilst subjects in the control group raised their intake only slightly (Table 2). However, there has been much debate regarding the applicability of results from adenomatous polyps, as an intermediate marker for carcinoma outcome, particularly since it is unknown whether a fruit and vegetable protective benefit is conferred in the later stages of adenoma growth and/or transformation (Byers, 2000). Additionally, as *blinded* randomisation is impossible, systematic over-reporting of fruit and vegetable intake *could* have occurred in the intervention subjects (Schatzkin *et al,* 2000).

In 22 animal studies in which cancer was experimentally induced, the majority unequivocally found that animals fed vegetables or fruit experienced either fewer tumours, smaller tumours, less DNA damage, higher levels of enzymes involved in the detoxification of carcinogens, or other outcomes indicative of lower cancer risk (Steinmetz & Potter, 1996). However, extrapolation of results from animal studies to humans is difficult given species

differences, the use of administered carcinogens, the use of genetically susceptible animals, and relative doses of vegetables well above those typically consumed by human beings (Steinmetz & Potter, 1996). Further clinical trials in humans will help to resolve the debate regarding the applicability of early animal study findings to the human situation.

Summary: is there an association fruit and vegetable intake and risk for colorectal cancer?

Various difficulties associated with researching this relationship have been extensively discussed above and include the long latency period for carcinoma development and validity of using adenomatous polyps as intermediate markers of outcome. Related to these points, we still don't know when (if at all) it is in the life-time exposure that the protective benefits are conferred (Michels *et al* 2000; Voorrips *et al* 2000). Most of the cohort studies and trials have been limited in their relatively short follow-up/intervention time, and have considered only subjects of middle age (at the study commencement). There are also significant limitations associated with self-administered with food frequency questionnaires, and issues of "social desirability" over-reporting (Steinmetz & Potter, 1993), for which identifying unbiased biomarkers of fruit and vegetable consumption would of great value (Flood & Schatzkin, 2000). Additionally, there is a lack of definition in epidemiological literature as to what constitutes fruit and vegetable groups, with the differential inclusion and exclusion of potatoes, legumes, fruit-juice, canned, tinned, frozen or other packaged fruit and vegetables, thereby limiting meaningful comparison of study results (Baghurst *et al,* 1999).

Findings of prospective studies and a controlled trial have largely been inconsistent with those from case-control studies and animal studies, with the majority of the former finding no association between level of vegetable and fruit consumption and risk for colorectal cancer. After undertaking an extensive review of relevant literature, The Committee On Medical Aspects of the Food Supply (COMA, 1998) report concluded that: "There is only limited and inconsistent evidence of an effect of fruit consumption" and "Overall there is a

moderate risk to conclude that higher intakes of vegetables would reduce risk of colorectal cancer". However, a diet rich in these foods remains advisable, because it conveys protection against other diseases, such as cardiovascular disease and possibly other cancers (Michels *et al,* 2000; Baghurst *et al*, 1999; Steinmetz & Potter, 1996).

National recommendations for and estimated consumption of fruit and vegetables

The Dietary Guidelines for Australians (NHMRC, 1992) advise the public to 'eat plenty of breads and cereals (preferably wholegrain), vegetables (including legumes) and fruit'. This guideline ranks second in importance only to 'eat a wide variety of nutritious foods', to which it is directly related. However, the latest available intake data suggests Australians are not meeting this guideline with respect to fruit and vegetable consumption.

Proportion Consuming Fruit & Vegetables

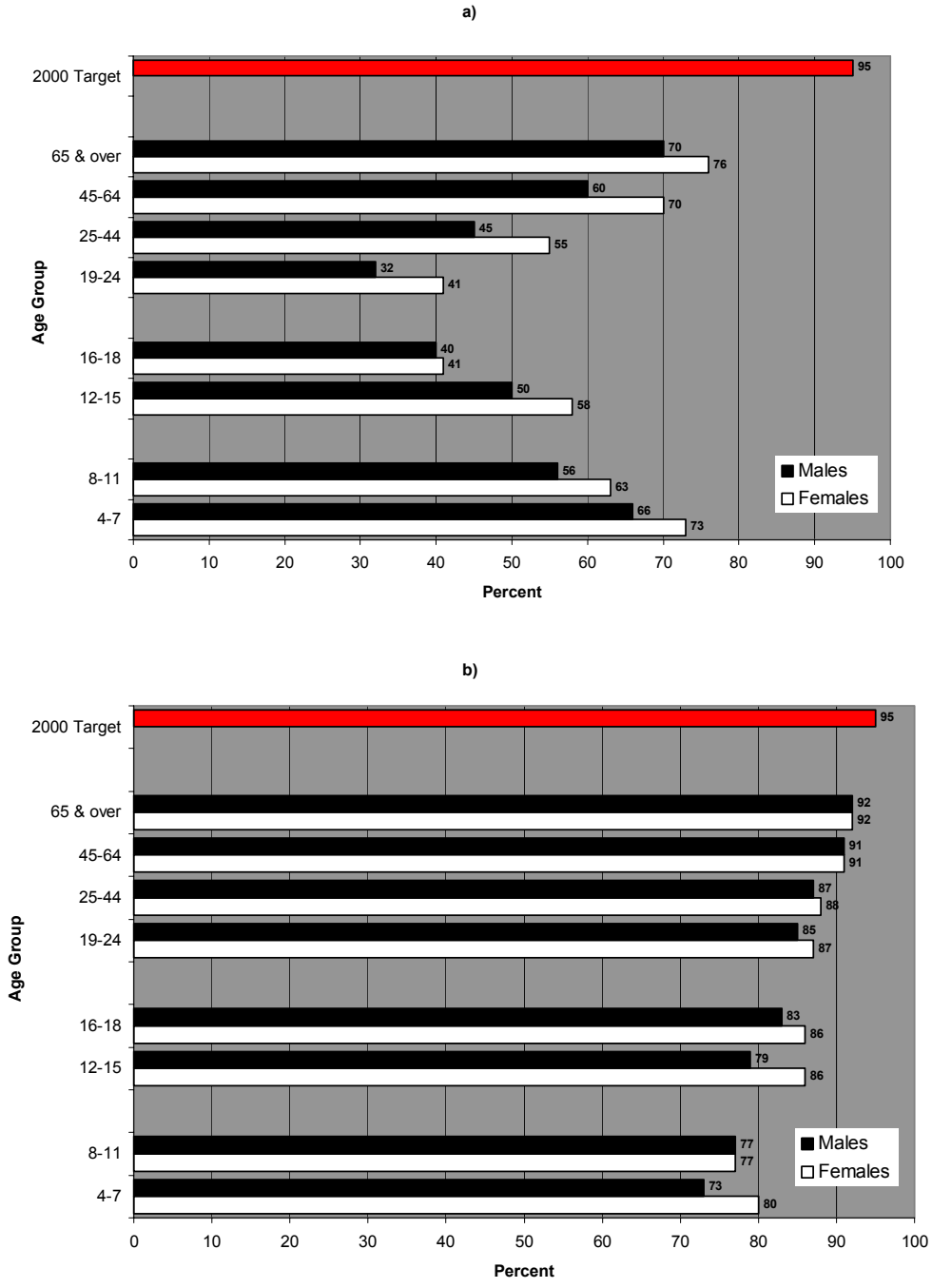
In the 1995 National Nutrition Survey (NNS), approximately one half of Australian adults surveyed reported *not* consuming fruit, and approximately one tenth reported *not* consuming vegetables on the day before the survey (McLennan & Podger, 1999). Additionally, one quarter of children and adolescents did not eat fruit on the day of the survey and one fifth did not eat vegetables (Magarey *et al*, 2001). Frequency of consumption appears relatively unchanged over a 10 year period for adults (Table 3), and similarly over a 12 year period for children (Magarey *et al*, 2001). The groups that remain furthest from reaching the 2000 target of 95% consuming fruit and vegetables on a daily basis (Nutbeam *et al*, 1993), are adolescents and young adults, particularly with respect to fruit intake (Figure 4).

Table 3: Comparison of recommended daily intake and estimated national adult Australian consumption of fruit and vegetables, by sex and year (1983 & 1995).

	Recommended serves*	Recommended intake (g)**	Estimated mean intake (g) (% recommended)		% consuming food item	
			1983†	1995††	1983†	1995††
Vegetables						
♦ *Men*	5-8	375-600	298 (61‡)	295 (60‡)	91.5	96
♦ *Women*	5-7	375-525	239 (53‡)	242 (54‡)	92.5	96.2
Fruit						
♦ *Men*	2-4	300-600	174 (38‡)	141 (31‡)	58.9	51.4
♦ *Women*	2-3	300-450	182 (49‡)	146 (39‡)	69.8	61.1

**Australian Guide to Healthy Eating (Smith et al, 1998) ** one serve is 75g of vegetables & 150g of fruit (Smith et al, 1998*); ‡*Mid-point considered for calculations of % recommended intake;* †*Ages 25-64; includes legumes (Cashel et al, 1986);* ††*Ages 19+; includes legumes (McLennan & Podger, 1999)*

Figure 4: Percentage of National Nutrition Survey* (1995) participants by age and gender who consumed any a) fruit & b) vegetables (including legumes) on the day prior to the interview.



*Data source: *McLennan & Podger, 1999.*
Year 2000 target defined in *Nutbeam et al, 1993.*

Comparison with recommended servings of the Australian Guide to Healthy Eating

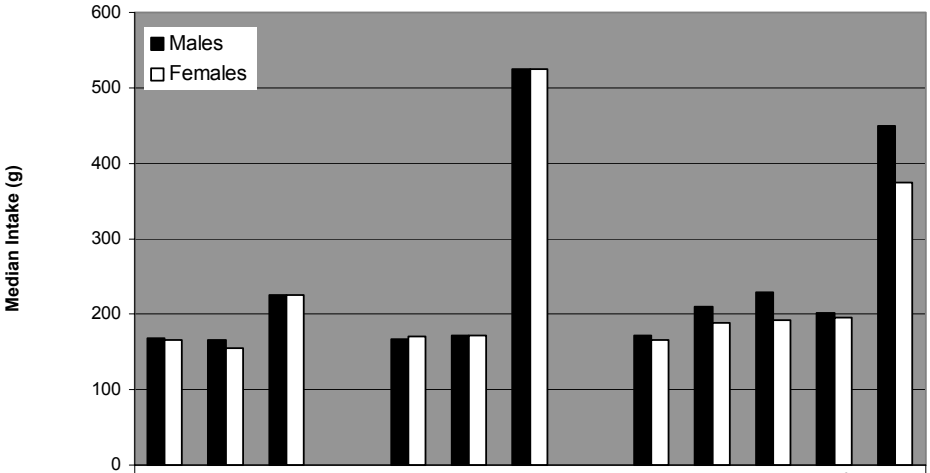
The Australian Guide to Healthy Eating (AGHE) recommends adults consume a *minimum* of five serves of vegetables and two serves of fruit per day (with no upper limit) (Smith *et al,* 1998). The 1995 NNS revealed intakes in those consuming vegetables and fruit, which were significantly below recommended levels, particularly with respect to fruit intake, and most significantly for adolescents and young adults (Figure 5). Thorough evaluation of the 1995 data has revealed that only 1 in 3 people surveyed met or exceeded the recommended *minimum* intake for vegetables and 1 in 5 for fruit, (CCA, 2001). Additionally, Magarey *et al,* 2001, found that less than 50% of all children and adolescents (and less than 25% of adolescents) participating in the 1995 NNS had an adequate fruit intake, and only one third met the vegetable intake recommendations. Recommendations were said to be met when consumption levels were within 25% of the *lower* serve recommendations of the AGHE. Both adult intakes (Table 3), and child and adolescent intakes (Magarey *et al*, 2001) have remained relatively unchanged, if not slightly declined over the past 10 and 12 years, respectively.

Contribution of vegetables and fruit to intake by type

There is international expert debate as to which foods comprise the "fruits and vegetables" category. The World Cancer Research Fund group recommendations (1997) exclude potatoes and legumes, for which the former would make a significant difference in the Australian context, as it constitutes between 35-50% of the intake of vegetables (Figure 6). By percentage weighted intake, no other vegetable sub-category contributed nearly as significantly (Figure 6). Potato intake as assessed by the 1995 NNS includes fried potato. However, fried potato is not included in the NGHE vegetable category, but rather in the "extras" category (along with oil, margarine, biscuits and other non-essential condiments), for which the *total* intake should not exceed 2-3 serves (Smith *et al*, 1998). However, Magarey *et al*, 2001 found that 32% of boys and 26% of girls consumed potato chips or wedges on the

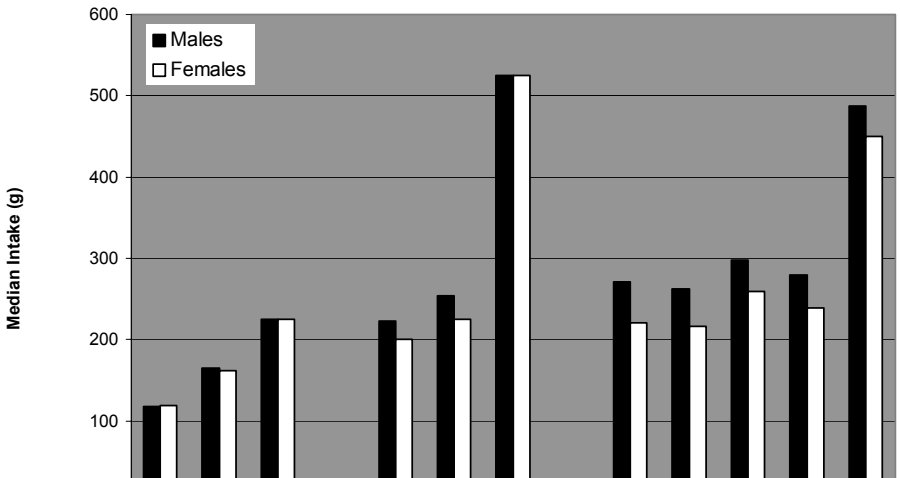
Figure 5: Recommended* & estimated median a) fruit b) & vegetable (including legumes) intake of National Nutrition Survey (1995) participants consuming these foods, by gender and age-group.**

a)



	4-7	8-11	CR	12-15	16-18	AntR	19-24	25-44	45-64	65 & over	AR
Males	168	166	225	167	172	525	172	210	229	202	450
Females	166	155	225	170	172	525	166	188	192	196	375

b)



	4-7	8-11	CR	12-15	16-18	AntR	19-24	25-44	45-64	65 & over	AR
Males	118	165	225	223	254	525	271	263	298	280	488
Females	119	162	225	200	225	525	221	216	259	239	450

**Data source: National Nutrition Survey:* McLennan & Podger, 1999
***Data source: The Australian Guide to Healthy Eating:* Smith et al, 1998. CR= child recommendations, AntR = adolescent recommendations, AR = adult recommendations. Recommendations are the mid-point of upper and lower recommendations of the Australian Guide to Healthy Eating.

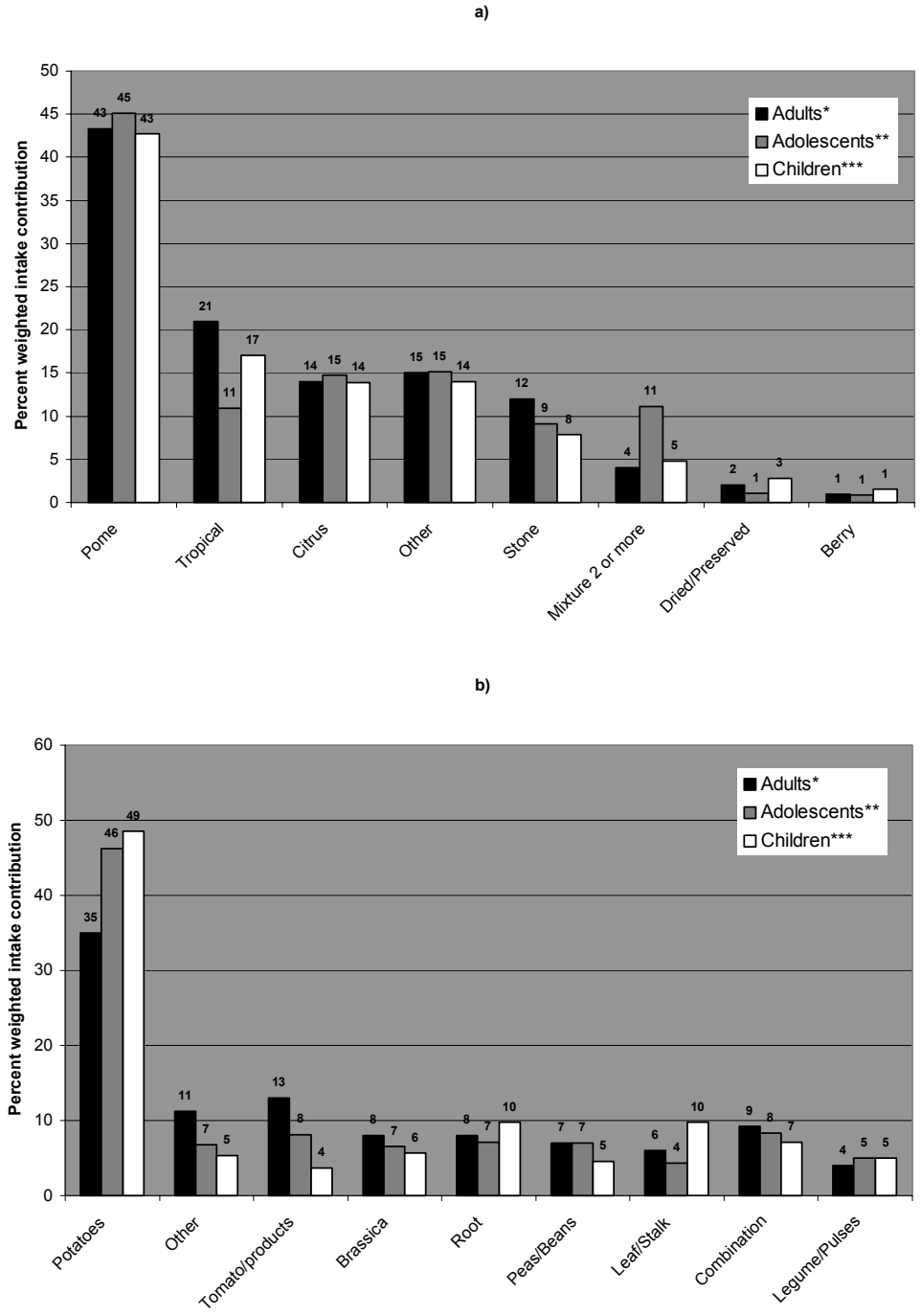
day before the interview, and for 16% of boys and 11% of girls fried potato was the only vegetable consumed.

Similarly, fruit juice was found to be the only source of fruit on the day of recall for 22% of boys and 18% of girls, and for 16-18 year olds the proportion was as high as one-third (Magarey *et al*, 2001). Excluding fruit juice from analysis, pome fruit contributed the most significant proportion of fruit intake for children/ adolescents (43%) and adults (45%), with other fruit sub-groups contributing at maximum only approximately one half of this amount (Figure 6). Such data presented above, suggest that variety in intake may be lacking, as further evidenced for children and adolescents by the extensive analyses of Magarey *et al,* 2001.

Summary

Comparisons of the 1983 and 1995 National Nutrition Surveys are only approximate, due to differential classification of food groups, with the 1985 survey not including fruit dishes or vegetable dishes containing cereal (Rutishauser, 2000). Additionally, age categories and response rates differed for the two surveys (Cashel *et al* 1986; McLennan & Podger 1999). However, it is apparent that average fruit and vegetable intake and frequency of consumption have remained relatively unchanged if not slightly decreased. This is consistent with indications from previous CSIRO surveys carried out at five-year intervals (CSIRO, 1996, cited in CCA, 2001). Lack of experimental data indicating optimal level of consumption to prevent chronic non-communicable disease, including cardiovascular disease and possibly cancers has prevented revision of current population recommendations, (Baghurst *et al,* 1999). However, regardless of methodological limitations and questions regarding the current recommended level of intake, it is more than apparent that adult Australian estimated intakes of fruit and vegetables were inadequate in 1995, with respect to estimated amount, frequency and variety of consumption.

Figure 6: Per cent contribution by a) fruit & b) vegetable sub-categories to weighted mean intake, of National Nutrition Survey† (1995) participants.



*Ages 19+ **Ages 12-18 *** Ages 4-11

†Data source: *McLennan & Podger, 1999.*

Note: will not sum to 100% as not all sub-categories included in analyses are presented in the above figure.

Food and nutrition policies and related strategies for colorectal cancer prevention, incorporating fruit and vegetable consumption

Why is action needed*?*

Although review of the evidence for fruit and vegetable consumption in the aetiology of colorectal cancer specifically remains equivocal, according to 1996 data, inadequate fruit and vegetable intake (specified as less than 5 serves of fruit and vegetables per day), is responsible for around 3% of total disease and 11% of cancer burden (Mathers *et al*, 1999). Moreover, inadequate fruit and vegetable consumption ranks a close second to tobacco control as the most important preventable health measure (SIGNAL, 2001a), and certainly, as previously elaborated upon, current population intake is inadequate.

What action is recommended*?*

A national colorectal cancer screening program based on biennial Faecal Occult Blood Testing has been recommended by the NHMRC (1999), and is endorsed by The Australian Cancer Council (2001). The 1999 NHMRC economic analysis suggests an estimated 40% reduction in mortality could be achieved through such an initiative, by establishing early detection and therefore curative treatment. Whilst emphasis in this section has been placed predominantly on nutrition related preventative measures, it is acknowledged that both dietary prevention and early detection together represent a major strategy towards reducing the burden of colorectal cancer.

'Eat Well Australia', the latest strategic framework for public health nutrition 2000-2010, developed by the Strategic Inter-Governmental Nutrition Alliance (SIGNAL, 2001b), defines promoting vegetables and fruit as one of six major strategic directions to focus health gain. Their proposed national action plan (outlined in Figure 7), constitutes a comprehensive, intersectoral approach towards reaching the goal of increased consumption of vegetables and fruit throughout Australia by at least one serve per day, over the next five years (SIGNAL, 2001c).

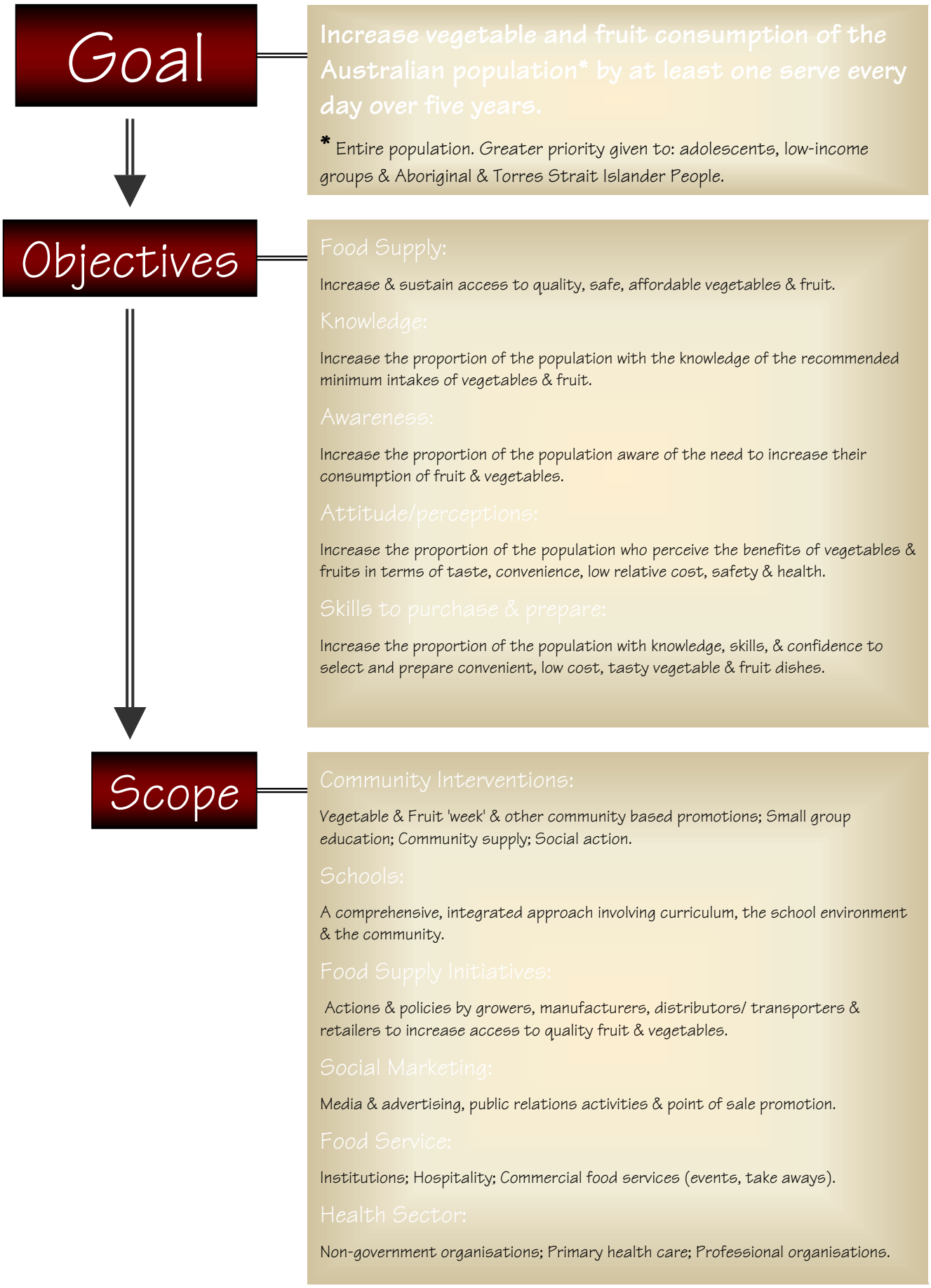
The 'Eat Well' framework features proposed co-operative strategies between government, primary producers, health professionals, non-government organisations, community groups, and stresses the importance of forming meaningful partnerships with industry to achieve a positive influence over healthy food supply. Emphasis is placed on a key setting approach including occupational, schooling, community, and urban commercial and housing environments. The policy acknowledges the need to improve inequities in fresh fruit and vegetable accessibility, for vulnerable groups (including rural and remote dwellers, Aboriginal Torres Strait Islander people, the elderly in institutions, and those with low income), by addressing identified socio-environmental and structural barriers to accessing safe and healthy food (Baghurst *et al,* 1990; Radimer *et al*, 1997). All of these strategies and agenda are consistent with the objectives of the National Food & Nutrition Policy (CDHHCS, 1992).

'Eat Well' complements other previously endorsed initiatives, such as Acting on Australia's Weight: A Strategic Plan for the Prevention of Overweight and Obesity (NHMRC, 1997), the Dietary Guidelines for Australians (NHMRC, 1992), The Australian Guide to Healthy Eating (Smith *et al,* 1998) and the National Clinical Practice Guidelines for colorectal cancer (NHMRC, 1999). Additionally, the initiative complements recommendations by non-government organisations, such as the National Heart Foundation healthy eating recommendations (1997), and The Cancer Strategies group recommendations for 2001-3 (CCA, 2001; CDHAC, 2001; NHMRC, 1999).

Lessons from previous State-based and International Fruit & Vegetable campaigns. What constitutes a successful campaign?

Previous state wide campaigns to increase fruit and vegetable consumption have included, 'Fruit 'n' Veg with every meal' (Western Australia, 1989-93; South Australia, 1990-91), '2 fruit and 5 vegetables every day' (Western Australia, 1989-93; Victoria, 1992-95) and

Figure 7: 'Eat Well' Australia, National Action Plan Outline



Adapted from *SIGNAL, 2001c*

'Eat Well' (Tasmania, 1997). Where available, evaluation results indicate that these campaigns succeeded in promoting increased awareness in targeted adult populations, with those campaigns that were sustained for a number of years (eg Victoria and Western Australia) demonstrating increases in reported fruit and vegetable consumption (Miller *et al*, 1996). Implementation of the State campaigns to increase vegetable and fruit intake was estimated to prevent 3626 DALYs a year, with corresponding cost savings of around $12.5 million a year over the implementation costs (estimated at approximately $2.5 million a year) (CDHAC, 2001).

The overall success of the Victorian and Western Australia campaigns, and the international US National Cancer Institute '5-a-day' program have been attributed to several factors: development of the campaign based on the results of consumer research and evaluation of each strategy; industry consultation and collaboration; and a comprehensive approach involving multi-strategy, multi-setting, multi-level and intersectoral components (Farrell *et al*, 2000; Miller *et al*, 1996). Fruit and vegetable intervention reviews, reveal additional crucial strategies as those which promote structural change and community involvement in identifying priorities and interventions (Ciliska *et al*, 2000; Farrell *et al*, 2000).

These approaches are consistent with Ottawa Charter for Health Promotion (Wass, 2000), and the 'Eat Well' policy framework and proposed National Action Plan (Figure 7) appear to be compatible with all of the above principles and recommendations.

CONCLUSION

There is no doubt that colorectal cancer affects a significant proportion of Australians, contributing significant health care sector burden. What is less certain is the association between fruit and vegetable consumption and risk for colorectal cancer. One of the difficulties

associated with investigating cancer is the long development period and the questions surrounding appropriateness of using adenomatous polyps as intermediate markers of colorectal cancer outcome. Combined with difficulty in accurately estimating vegetable intake, potential for recall and selection bias in case-control studies constituting the greatest volume of evidence, and variation across populations as to what constitutes upper and lower levels of fruit and vegetable intake, it is difficult to draw sound conclusions. Most of the limited number of prospective studies and a clinical controlled trial do not provide support for the association. Despite this, most reviewers conclude that it is important to promote increased fruit and vegetable consumption because of the strong association with risk for other nutrition-related disease. Certainly, Australian fruit and vegetable consumption as assessed on a national basis last in 1995 is significantly below the recommended intake level, particularly for adolescents and younger adults, and all Australians consume a limited variety of fruit and vegetables, with potatoes (including 'fries') contributing the most significant amount of vegetable intake. Fruit and vegetable consumption is thus implicated by the "Eat Well Australia" strategic framework as one of the six strategic directions for health gain over the next ten years.

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