RESEARCH COMMUNICATION

A Comparison of Risk and Protective Factors for Colorectal Cancer in the Diet of New Zealand Maori and non-Maori.

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Abstract

By international standards New Zealand (population 3.8 x 10^6) has a high rate of colorectal cancer, with approximately 2000 new cases occurring and approximately 1000 deaths each year. But within the New Zealand population, a lower incidence of colorectal cancer is reported for Maori than for non-Maori New Zealanders (22.2 and 43.7 per 100,000 respectively). Information from the New Zealand National Nutrition Survey 1997 shows that in comparison to non-Maori, Maori eat more in total, eat more red meat, drink more alcohol, consume more saturated fat, have a higher prevalence of obesity and have a lower proportion of individuals consuming a given level of fruit and vegetables per day. All these factors would be expected to increase colorectal cancer risk. Puha (sow thistle; *Sonchus sp.*) and watercress (*Nasturtium officinale, N.aquaticum*) are foods with plausible cancer protective properties which are components of the Maori, but not the non-Maori diet.

Key Words: Colorectal cancer - risk factors - diet - Maori/non-Maori - New Zealand

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Introduction

The New Zealand population comprises European (75%), Maori (8%), Pacific Islands (4%) and multiple ethnicity (9%) based on self-identification (Statistics NZ, 2001). The Maori people are descended from ancestors who arrived from Polynesia approximately 1000 years ago. Europeans, particularly from the UK and Ireland have lived in New Zealand for approximately 160 years. New Zealand has a high rate of colorectal cancer (CRC) by international standards but interestingly, within the New Zealand population, a lower incidence of CRC is reported for Maori than for non-Maori.

Evidence for the role of environmental factors in cancer incidence date back to 1700 when Ramazzini noted the high rate of breast cancer among nuns, which he attributed to their celibate life. This was followed in 1775 by Pott, who postulated that occupational exposure of chimney sweeps was causally related to cancer of the scrotum. Perhaps the first diet related risk factor for cancer may be attributed to Dungal (1961) who suggested that the widespread consumption of smoked food might be an important factor in the high prevalence of gastric cancer among people in Iceland. Cancer epidemiology during the last century has clearly shown that cancer incidence and mortality rates vary dramatically across the globe (Armstrong and Doll, 1975, IARC, 2001) and within countries according to differential exposure to cancer initiators and promotors. In addition, rates of cancer among populations migrating from low- to high-incidence countries change markedly, approximating the rates in the new region within 1 to 3 generations (Doll and Peto, 1981, Zeigler et al,1993). For example, oesophageal cancer in Japanese people living in Japan associated with high consumption levels of smoked food reduced significantly in the Japanese population who migrated to the USA following World War II. This change in cancer rates is evidence that the primary determinants of cancer rates are not necessarily genetic factors, but rather environmental and lifestyle factors that could, in principle, be modified to reduce cancer rates in high-risk areas. For CRC, Doll and Peto (1981) estimated that 90% of deaths were potentially avoidable by dietary changes. This has been revised subsequently to around 70% (Willett, 1995). Fruits and vegetables are clearly protective against cancer risk, most likely by inhibiting the initiation or promotion of

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carcinogenesis (WCRF, 1997, Johnson, 2002). For example, free radical scavenging antioxidants (eg carotenoids) in food are likely to be important in deactivating highly reactive carcinogenic radicals (Thompson et al,1999). The Ames assay for mutagenicity has been used to explore the protective role of fruits and vegetables on the basis that many anticarcinogenic factors are also antimutagenic (Botting et al, 1999).

Because of the potential role of diet in the etiology of colorectal cancer, and because dietary change is a possibility, a comparison of dietary risk and beneficial factors for CRC between New Zealand Maori and non-Maori was undertaken with an ultimate goal to reduce the incidence of this illness in New Zealand.

Methodology

Cancer rates

Information on worldwide and New Zealand cancer incidence and mortality rates was obtained from data compiled by IARC (2001) and the New Zealand Health Information Service (NZHIS, 2002). A cancer registration scheme has operated in New Zealand since 1948 with mandatory reporting of all cancer cases since 1994. Where rates are given, these have been age standardised against Segi's world population (Waterhouse et al, 1976) to adjust for differences in age distribution of the populations being compared. Ethnicity for cancer registrations is based on the concept of self-identification. Where ethnicity was not specified, these registrations were not included in Maori versus non-Maori comparisons. Hence CRC rates are understated for both Maori and non-Maori populations. CRC subsite (right and left colon, rectum) cancer rates were aggregated for 1996-1998 to increase count numbers.

Dietary factors

Protective and risk factors

Protective dietary factors particularly relevant to colorectal cancer include fruits, vegetables, fibre, folic acid, vitamin E, vitamin C, tea, beer and dietary supplements. Possible diet and diet-related risk factors include energy intake, alcohol, meat, meat cooking practices, fat (total and saturated animal), iron, cadmium, pesticides and supplements (Potter, 1999, WCRF, 1997, COMA, 1998). Obesity was included as a useful measure of the difference between energy intake and energy expenditure.

Food consumption

Information on food and nutrient intakes, dietary habits and nutrition-related clinical measures were obtained from the 1997 New Zealand National Nutrition Survey (NNS97) (Russell et al, 1999). This survey includes data from 4,636 New Zealanders, including an oversampling of NZ Maori, collected over the 12-month period December 1996 to November 1997. The survey included a computer assisted, 24 hour diet recall interview and a self-administered, questionnaire which estimated the frequency of intake of foods over the preceding 12 months. The survey achieved a response rate of 84.7%.

Antimutagenic Foods

New Zealand foods which showed antimutagenic properties in a modified Ames test were identified from the work of Botting et al (1999). The percentage of Maori and non-Maori consuming these antimutagenic foods were taken from the NNS97 (Russell et al, 1999).

Results

New Zealand (population 3.8 x 10^6) has a high rate of colorectal cancer (CRC), with 2433 new cases occurring and 1123 deaths in 1998 (NZHIS, 2002). The projected incidence of CRC in New Zealand in 2000 is exceeded only by two out of 30 OECD countries (Figure 1), with projected age standardised incidence rates of 55.3 and 43.4 per 100,000 for males and females respectively (IARC, 2001). This rate is also the highest of a selection of Asian countries (IARC, 2001), although it is noted that the projections for China and Samoa are based on limited data sets (Figure 2).

A markedly lower rate of colorectal cancer for Maori compared with non-Maori New Zealanders is observed and

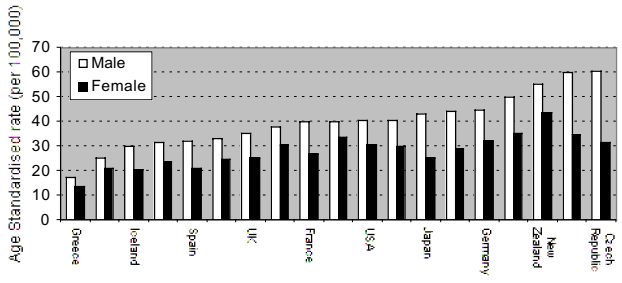

Figure 1: Projected rates of colorectal cancer for selected OECD countries (from IARC, 2001)

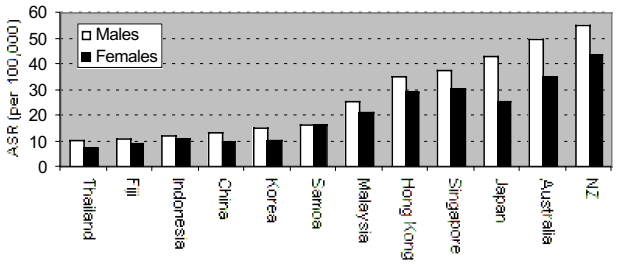

Figure 2: Projected rates of colorectal cancer for Asia-Pacific countries (from IARC, 2001)

has been maintained over the period 1980 to 1998 (Table 1). The difference in incidence rate is seen at each of the colorectal cancer subsites (Table 1) with the most marked difference being for right-sided colonic cancers.

Dietary factors which have been implicated as either protective or risk factors for CRC are shown in Table 2 with intake or food frequency data for males and females of each population group. In comparison to non-Maori, Maori eat more in total. However on a per body weight basis food consumption is surprisingly similar between the two population groups for both males and females. Maori eat more red meat, drink more alcohol, consume more saturated fat, have a higher prevalence of obesity and have a lower proportion of individuals consuming more than two servings of fruit or 3 servings of vegetables per day than non-Maori. All these factors would be expected to increase rather than decrease colorectal cancer risk. Maori have a higher total intake of vitamin C relative to non-Maori. Vitamin C is plausibly cancer protective via an antioxidant mechanism. A higher vitamin C intake may also be indicative of fresh fruit and vegetable intake, since these are the predominant source of vitamin C.

The prevalence of consumption of New Zealand foods which showed antimutagenic properties are shown in Table 3. Foods which are antimutagenic and which are preferentially consumed by Maori are puha and watercress. The average Maori consumes 6 g/day of puha and 12 g/day of watercress.

Discussion

Sutton et al (1993), reported that the incidence rate of CRC for Maori was 40% of that for the total New Zealand population for the period 1970-1984. The lower CRC incident rate for Maori has persisted for almost 30 years. Given the intermarriage between Maori and non-Maori, that Maori have generally adopted a western lifestyle, including diet, and that both ethnic groups are living in the same environment, this observation is curious, for a cancer strongly linked with dietary factors. Interestingly the fact that interbreeding has not affected incidence strongly suggests that genetic factors are not important determinants.

Total food intake and individual dietary component intakes are greater in Maori than non-Maori. However if they are expressed on a bodyweight basis, there is remarkable similarity in intakes between the two groups. In bioactivity terms the total intake is likely to be a more important parameter, since Maori are more likely to be obese and therefore have a greater proportion of biochemically inert fat. For example, vitamin C is not absorbed by adipose tissue (because of its water solubility) and therefore in Maori who have a greater total intake of vitamin C it is likely that biochemically active concentrations in target tissues are higher than in non-Maori. The converse is true for fat soluble protective agents. Vitamin E is likely to be a less effective (for equivalent intakes) cell protective agent in Maori than non-Maori because it will be sequestered in a biochemically non-available form in adipose tissue.

An ecological approach to dietary cause and effect is the most simplistic epidemiological study design, but if a component of diet is claimed to be a risk or protective factor, this finding should be consistent with observations at a population level. With the exception of vitamin C, dietary risk or protective factors for CRC do not account for the observed differences in CRC between these two ethnic groups. Our assessment has included red meat as a risk factor but does not consider meat cooking methods. It is known that high temperature cooking of meat (and indeed fish and chicken) leads to the formation of precarcinogenic heterocyclic amines and polycyclic aromatic hydrocarbons (Baghurst,1999) so that meat cooking method rather than quantity of meat consumed may be the risk factor for CRC.

Table 1. Estimated Resident Population, Annual and Sub Site Colorectal Cancer Incidence Rates for New Zealand Maori and non-Maori (per 100,000, age standardised)

Population Statistics[1]	Maori	Non-Maori
Males	284 400	1 584 400
Females	289 300	1 634 700
Total	573 700	3 219 100
Overall CRC rates	Maori	Non-Maori
1980[2]	21	44
1998[1]	22.2	43.7
Sub site	Maori	Non-Maori
Colon, right-sided	4.5	13.1
Colon, left-sided	5.8	11.1
Rectum	8.2	14.3

[1]NZHIS (2002). Estimated resident population of New Zealand for year ended 31 December 1998.
[2]Public Health Commission (1993)

Table 2. Exposure to Dietary Protective and Risk Factors for CRC

	Maori		Non-Maori	
Protective factor	males	females	males	females
Folate (μg per day)	280	208	291	222
Fruit ≤1 serving/day (%)	41	26	33	18
>2+ servings/day (%)	31	49	35	57
Average total fruit intake (g/day)*	156		182	
Vegetables ≤1 serving/day (%)	21	14	13	7
>3 servings/day (%)	50	62	65	76
Average total vegetable intake (g/day)	287		296	
Dietary supplements 1+/week (%)	13	25	22	37
Fibre-insoluble (g/day)**	11	9	13	10
Soluble (g/day)	11	9	12	8
Vitamin C (mg/day)	142	109	119	105
Vitamin E (mg/day)	11.8	9.0	11.7	8.8
Beer ≥1 serving /week (%)	51	11	53	10
Tea ≥1 serving /week (%)	50	53	59	66
Risk factors				
Energy intake (kJ/day)	12282	9029	11920	7789
(kJ/kg bw/day)	141	120	151	116
Alcohol (g/day)	25	8	19	9
Red meat (g/day)	98	65	72	42
Total fat (g/day)	122	90	114	73
(g/kg bw/day)	1.4	1.2	1.4	1.1
Anthropometric data				
Mean body weight (kg)	87.3	75.2	78.9	67.1
Mean BMI (kg/m^2)	28.7	28.7	25.6	25.5
Obesity*** (%)	27.0	27.9	12.6	16.7

% = percent of population consuming item
* = excludes fruit juice
**= mean for population group
***= for Maori, BMI ≥ 32 kg/m^2; non-Maori, BMI ≥ 30 kg/m^2.

Comparative meat cooking preferences for New Zealand Maori and non-Maori are not available. Migrant studies indicate that the latent period between exposure and detection of CRC may be 10-20 years (WCRF 1997). Ideally the current assessment would draw on earlier consumption patterns but data of sufficient detail is not available.

Watercress and puha (*Sonchus arvensis*, *S. asper*, *S. oleraceus* and *S.kirkii*) are members of the Brassicaceae and Asteraceae families respectively. Of the two Nasturtium and four Sonchus species only *S. kirkii* is indigenous to New Zealand. The others were introduced by Europeans in the late 19th century and with the exception of the rare *S. kirkii* and *S. arvensis*, are now commonly found growing in the wild throughout New Zealand.

Watercress contains glucosinolates (Fahey et al, 2001) which are converted to isothiocyanates (ITC) by both plant and gut microfloral enzymes. ITCs are likely to be cancer protective via three possible mechanisms. Firstly, they can prevent carcinogen activation through the inhibition of phase I enzymes such as cytochrome P450. Secondly they can enhance the excretion of potential carcinogens by inducing phase II enzymes (including glutathione S-transferase and UDP-glucuronyl transferase), and thirdly, they can induce cell death, thus preventing cell multiplication and tumour development (Hecht, 1999, Rose et al, 2000, Conaway et al, 2002, Johnson, 2002).

There is less evidence of possible chemoprotection from constituents of puha. *Sonchus* species have been reported to contain sesquiterpenes (Helal et al, 2000, Shimizu et al, 1989) and triterpenoids (Shiojima et al, 1997). Various triterpenoids (eg Sporn and Suh, 2000, Wang and Nixon, 2001) and sesquiterpene lactones (Hall et al, 1977, Woerdenbag 1986, Robles et al, 1995) have been implicated in cancer prevention, although these studies are not linked to the specific terpene constituents of puha. *S. oleraceus* has high levels of vitamin C and carotenoids (Guil-Guerrero et al, 1998) which are antioxidants scavenging free radical

Table 3. Percentage* of Population Consuming Foods with Antimutagenic Activity (as determined by a modified Ames test, Botting et al., 1999)

	Maori		Non- Maori**	
	male	female	male	female
Banana -green	7	3	5	5
-yellow	76	77	78	84
Onion	61	64	69	75
Rice	38	38	46	49
Sweetcorn	28	42	30	37
Taro	1	4	0	0
Broccoli	44	54	56	68
Brussels sprouts	7	12	17	24
Carrot	66	78	84	88
Green beans	42	37	47	48
Puha	13	15	0	0
Pumpkin	47	60	50	55
Silverbeet	47	47	40	43
Kumara	30	46	26	31
Tomato	59	68	74	83
Watercress	15	16	1	1

* Expressed as % consuming within the sub-population

** Excludes Pacific Island ethnicities

species. A number of flavonoids (acacetin, aesculetin, apigenin, chrysoeriol, isorhamnetin, kaempferol, luteolin, quercetin, scopoletin and tuteolin) have also been identified in *Sonchus* species (Mansour et al, 1983, Qu et al, 1993, 1995, 1996). Flavonoids exhibit a range of biological activities including both pro- and antioxidant effects, and inhibition of key intracellular enzymes and signalling cascades. In addition many flavonoids are free radical scavenging antioxidants, are absorbed well from the diet, and therefore might represent dietary protection against cooked meat carcinogens for example.

There are other factors that might influence the difference in colorectal cancer rate between Maori and non-Maori New Zealanders; including genetic polymorphisms in the detoxification of carcinogens or perhaps exercise related effects.

Because cancer development is a multistage progression over several years, it is highly unlikely that a single dietary component would explain the differences in CRC cancer incidence between New Zealand Maori and non-Maori. However, we believe the chemopreventive properties of watercress and puha warrant consideration.

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