

ORIGINAL ARTICLE

Regional differences in incidence of gastric and colonic cancer in the Maori of New Zealand

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Postgrad Med J 2002;78:419–421

Background: It is known that there are ethnic differences in cancer in New Zealand between Maori (the indigenous people) and non-Maori, however, until now no regional comparisons have been made.

Study design: A retrospective study of patients diagnosed at Whangarei Hospital, New Zealand between 1995 and 1997 with gastric or colonic cancer was combined with population data from the 1996 census for Whangarei District to calculate incidence figures. The incidence of cancer was compared to national rates.

Results: Between 1995 and 1997, 19 Maori and 24 non-Maori were diagnosed with gastric cancer, and 10 Maori and 125 non-Maori with colonic cancer. The age standardised rates (per 100 000) for Maori and non-Maori with gastric cancer were 68.3 and 7.9 respectively. Gastric cancer is known to be increased in the Maori, but in Whangarei was significantly higher than the national Maori rates (20.5). There was no difference in the rate of colonic cancer in the Maori and non-Maori in Whangarei, again this differs from the national trends, in which the Maori are protected against cancer.

Conclusion: This study highlights that there is still much more to be learnt in understanding the aetiology of gastrointestinal cancers, to explain such strong regional differences.

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Submitted
7 December 2001
Accepted 7 May 2002

Cancer is the second commonest cause of deaths in New Zealand, with gastrointestinal cancer accounting for a quarter of these.

Gastric cancer is thought to develop from a sequence of events, starting with chronic gastritis, passing through atrophy and intestinal metaplasia to dysplasia—a precursor to cancer development. There is strong evidence that *Helicobacter pylori* infection, which results in chronic gastritis, is a gastric carcinogen. Dietary factors, including alcohol, salted or pickled foods, and nitrates may act as initiators or promoters of carcinogenesis. Pernicious anaemia and gastrectomy increase the risk of developing cancer as achlorohydrria allows bacterial colonisation and metaplasia. Genetic factors are likely to be involved as there is a higher incidence of gastric cancer in blood group A, and certain families have a high incidence of gastric cancers at young ages.

The genetic basis of colonic cancer is better understood, with multiple genetic events (involving the APC gene, Ras oncogene, p53, FAP genes) implicated in the histogenesis of colonic cancer. However epidemiological evidence suggests the environment is still the most important aetiological factor, especially the diet. The amount of fat consumed in the New Zealand diet is higher than that recorded for virtually any other country and it is known that certain kinds of bacteria—the nuclear dehydrogenating clostridia—can act on bile acids to produce carcinogens. Similarly bacterial transformation of amino acids may result in carcinogen (or co-carcinogen) production. Thus a high fat diet (increases bile acid production) and a high protein diet (increases the possible transformation of amino acids by bacteria) are believed to favour carcinogenesis and colonic cancer development. Other possible risk factors are alcohol, inflammatory bowel disease (especially ulcerative colitis), cigarette smoking, and a sedentary occupation.

The Maori have a twofold to fourfold increased incidence of gastric cancer, and less than half the rates of colonic cancer compared with the non-Maori.^{1,2} The evidence from migration studies suggests these differences are likely to be due to environmental factors rather than genetic factors. The increased rates of gastric cancer in the Maori may be partially explained

by increased *H pylori* infection, but the reason why they are protected from colonic cancer remains elusive as their diet puts them at a similar if not higher risk of developing colonic cancer, according to proposed aetiological factors.¹

This study examined the clinical profile of Maori and non-Maori patients diagnosed with gastric and colonic cancer at Whangarei Hospital in order to identify differences in the presentation of these two groups. The use of regional data also allowed the regional incidence rate to be estimated and compared to national data.

METHODS

Patients diagnosed with gastric cancer (*International Classification of Diseases*, ninth revision (ICD9) 151) or colonic cancer (ICD9 153) from Whangarei Hospital between 1 January 1995 and 31 December 1998 were identified by computer discharge records. Patients with a recurrence of cancer were excluded, and 10 patients whose records were not located were excluded. The patient's date of diagnosis was the date of histological confirmation and a clinicopathological method of staging was used, combining histological findings and the surgeon's assessment (as described by Fielding *et al*).³ This took into account tumour penetration (through submucosa, muscularis propria, and serosa) and lymph node or distant metastases. For colonic cancer, the Dukes's staging classification was used. The Maori and non-Maori patients in Whangarei were compared using χ^2 and a χ^2 test for trends (linear by linear association).

National cancer registration details for 1996 were obtained from *Cancer: New Registrations and Deaths 1996*, published by New Zealand Health Information Services.⁴ Non-Maori incidence rates were calculated by subtracting Maori cases from the total number of cases, and directly age standardised to Segi's world population, with the maximum age at 65+ years, in order to compare to Whangarei data.

To calculate the Whangarei age standardised rates for 1996 an average number of cases for 1996 was taken (using 1995, 1996, and 1997) and combining with population data for Whangarei District obtained from the 1996 census of populations and dwellings.⁵ The incidence rate included nine patients

Table 1 Demographics of 1995–98 cohort

	Gastric		Colonic	
	Maori (n=21)	Non-Maori (n=26)	Maori (n=13)	Non-Maori (n=154)
No of males	10	18	6	67
Mean (SD) age	61 (13.2)	70 (13.4)	62 (9.4)	68 (9.8)
No of females	11	8	7	87
Mean (SD) age	52 (19.3)	77 (5.0)	56 (12.0)	73 (10.1)
M:F ratio	1 : 1.1	1 : 0.8	1 : 1.2	1 : 1.3

identified by computer records, whose records were not located (that is, their ethnicity was known, but no information on staging was available). Both the 1996 census and national cancer registration details include as Maoris anyone who registers at least one of their ethnic groups as Maori. They were directly age standardised to Segi's world population, and 95% confidence intervals calculated using the method described by Jensen *et al*⁶ on an Excel database. The rates were compared using Student's *t* test.

RESULTS

Between 1995 and 1998, 51 patients were discharged from Whangarei Hospital with gastric cancer and 173 with colonic cancer. Ten patient records could not be located (four gastric and six colonic). The cohort demographics are shown in table 1.

Between 1995 and 1997, 43 patients were diagnosed with gastric cancer (19 Maori, 24 non-Maori) and 135 patients with colonic cancer (10 Maori, 125 non-Maori). Age standardised incidence rates are shown in table 2. The incidence of gastric cancer was three times higher in the Maori population of New Zealand compared to the non-Maori population. In Whangarei it was eight times higher. The incidence of colonic cancer in the Maori is less than half that of the non-Maori in New Zealand. However in Whangarei, no differences were found.

DISCUSSION

A major limitation in the calculation of the regional incidence rates is the assumption that Whangarei District census population covers the same population as the hospital's catchment area. The geographical catchment area of Whangarei Hospital allocated by Northland Health Limited is similar to the census area, but the surrounding hospitals are small, so Whangarei may include referrals from a larger area. If the actual population served by the hospital is larger than that of the census, the incidence of cancer in Whangarei will be overestimated, and if more Maori live outside the census area than non-Maori, or vice versa direct comparisons between the two groups will be distorted.

Studies of different ethnic groups are often biased by notification differences within the groups. As both the census and cancer registration details allow respondents to self identify ethnic status, the numerator and denominator for incidence calculations will be the same and reduce this bias. A change in 1996 census classifications now includes anyone who selects Maori as one of their three ethnicity options as Maori, and has increased the Maori population and incidence rates.

This study found the incidence of gastric cancer in the Maori of Whangarei was three times greater than that in the Maori of New Zealand. The incidence of gastric cancer in the non-Maori was unchanged. There is nothing to suggest that Maori patients will be referred differently to non-Maori patients, and if the rates in Whangarei are due to incomplete population data for the hospital's catchment area they would be expected to increase both the Maori and non-Maori incidence rates in Whangarei.

The Maori have increased *H pylori* infection, and increased intestinal metaplasia, occurring at a younger age.¹ It is also suggested that early exposure to aetiological factors is significant, and *H pylori* is more prevalent in the Maori by the age of 11–12 years.⁷ The risk of *H pylori* infection then increases with age, lower socioeconomic status, and lower household income (groups in which the Maori are over represented). A study by Dockerty *et al* showed that the association between increased gastric cancer and the Maori remained, after adjusting for socioeconomic status and smoking.⁸

The reason why the Maori in Whangarei may have a higher incidence is unclear. They may have an increased prevalence of *H pylori* infection, and although the data are not standardised for socioeconomic status, it is not likely that this can account for increased *H pylori*. Altogether 75.1% of Maori people aged 15 years and over in Whangarei District have an annual income of \$20 000 or over compared with 67.8% for the Maori as a whole.⁹ Alternatively there may be an increased prevalence of familial gastric cancer in the Whangarei Maori (which normally accounts for a very small proportion of cases, but could affect a small study in one region).

No difference was found in the incidence of colonic cancer in the Maori and non-Maori in Whangarei; however, it is well known that the Maori in New Zealand have a decreased incidence of colonic cancer. This is despite having a similar diet or increased risk factors for colonic cancer compared with European New Zealanders (saturated fats, meat intake, fibre intake, and alcohol consumption.)^{10 11} Just as the reason for this difference remains elusive, it is not clear why the Maori in Whangarei in the study do not benefit from this protection. The increased incidence in Whangarei in both the Maori and the non-Maori, may therefore be due to incomplete population data for the catchment area.

Gastric cancer in the non-Maori of Whangarei is twice as common in males as in females, which is consistent with national trends and almost all parts of the world, where the

Table 2 Age standardised incidence rates

	Incidence per 100000 (95% CI)		
	Maori	Non-Maori	SRR (95% CI)
Gastric cancer			
New Zealand (1996)	20.5 (15.1 to 25.8)	7.0 (6.2 to 7.8)	3.0 (1.9 to 4.5)*
Whangarei (1996)	68.3 (36.6 to 100.1)	7.9 (4.6 to 11.1)	8.7 (2.8 to 27.3)*
SRR (95% CI)	3.3 (1.4 to 7.5)*	1.1 (0.7 to 1.8)	
Colonic cancer			
New Zealand (1996)	12.5 (8.2 to 16.9)	33.7 (32.0 to 35.4)	0.4 (0.3 to 0.5)*
Whangarei (1996)	36.0 (13.3 to 58.7)	42.8 (35.0 to 50.6)	0.8 (0.5 to 1.5)
SRR (95% CI)	2.9 (1.02 to 8.12)*	1.3 (1.02 to 1.56)*	

*Significant difference $p < 0.05$ (across: between ethnic groups in New Zealand or Whangarei; down: between Whangarei and New Zealand within each ethnic group). CI, confidence interval; SRR, standardised rate ratio.

ratio of male:female is between 1.5–3:1.¹² There were no differences in the number of male and female Maori's diagnosed with cancer—the reason for this discrepancy is not known, and may be an artefact of the small numbers involved.

There were no differences in the stage at which the Maori and non-Maori patients presented with their cancers (gastric or colonic). This is of interest, because many studies have highlighted differences in the equity of access to the medical system by the Maori. (A study by Sutton *et al* found that colorectal cancer in the Maoris between 1970 and 1984 was diagnosed at a more advanced stage, and suggested this was due to social or economic factors.³) As the Maori are diagnosed at a similar stage, it suggests the barriers preventing the Maori from access are being reduced, or having little effect on the cancer stage. It also suggests that the aetiology or pathology of the cancer development is similar. There may have been no delay in presentation because of increased attendance at hospital (instead of general practice), or because of increased surveillance by doctors who are treating them for other conditions. (The Maori have increased comorbidity, but there is no evidence to suggest the Maori in Whangarei will have more surveillance than other Maori groups.)

One of the most important factors to come from the study is the suggestion that there may be significant regional differences in gastric and colonic cancer development within New Zealand. It will be useful to verify this, and extend it to other regions, which can be done by linking patient details to their postal address, and thereby use appropriately matched regional population data. Confirmation of regional differences would allow further investigations, into the reasons for such differences, and facilitate the targeting of resources. For example, if the Maori in Whangarei do have such high rates of gastric cancer, it may become cost effective to set up screening programmes, with endoscopy or *H pylori* detection, which are used with some success in Japan.

While the understanding of the aetiology of cancers has developed a great deal, these unexplained differences imply that we still have more to learn, and offer tantalising evidence that cancer rates can be reduced still further.

ACKNOWLEDGEMENTS

I would like to acknowledge the assistance of the helpful and friendly staff at Whangarei Hospital.

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