The prevalence of colorectal adenomas in Māori and New Zealand Europeans parallels colorectal cancer rates

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Abstract

**Background** New Zealand (NZ) has a high incidence of colorectal cancer (CRC). Māori have a documented incidence that is approximately half that found in NZ Europeans, possibly the result of under-reporting.

**Aim** To determine and compare the prevalence of colorectal adenomas in Māori and NZ European patients.

**Methods** Colonoscopy records from the Middlemore Colonoscopy Audit Database between 1 July 2001 and 31 December 2005 were reviewed. Studies performed for indications associated with an increased risk of colorectal polyps were excluded from the analysis. Patient demographics, including self-identified ethnicity, and number and location of colonic polyps were recorded. All polyp histology was reviewed.

**Results** Data was analysed from 2842 colonoscopies—2523 were NZ Europeans (mean age 67 yrs) and 319 were Māori patients (mean age 60.6 yrs). To adjust for age, a comparison of data between 40 and 59 years was undertaken. In 643 (81.2%) NZ Europeans, polyps were identified in 213 (33.1%). In the 149 (18.8%) Māori patients, polyps were identified in 35 (23.5%) p=0.029. The comparative rates of adenomas in NZ Europeans and Māori were 16.7% and 8.7% respectively (p=0.019; 8% difference, CI=2.3-13.9%).

**Conclusion** The prevalence of colorectal adenomas in Māori is approximately half that found in NZ Europeans. This mirrors the reported difference in CRC incidence between these groups and lends support to this being a real finding and not a bias in the manner in which the data has been collected.

New Zealand has a high incidence of colorectal cancer with an age standardised incidence (non-Māori, 2005) of 51.9 per 100,000.1,3 However, the rate amongst the Māori population appears to be approximately half that of the non-Māori population.3 Despite this difference the CRC mortality rate for Māori is no better than non-Māori.2,3,17 This is partially explained by a greater proportion of Māori presenting at a more advanced stage.2 Correspondingly there is poorer survival from diagnosis for Māori. The documented lower CRC incidence rate for Māori remains unexplained. One potential explanation is incomplete recording of ethnicity at the time of diagnosis.

To minimise the influence of any confounding factors we decided to study the prevalence of adenomatous polyps which are the precursors of colorectal cancer.4 The differences in prevalence between ethnicities should reflect the differences in cancer incidence.5
Methods

We performed a retrospective review of all colonoscopies recorded on the Middlemore Colonoscopy Audit Database between 1/7/01 and 31/12/05. Demographics, patient self-identified ethnicity, polyp details and indication for colonoscopy were recorded. Indications which were associated with a low prevalence of polyps (e.g. inflammatory bowel disease) or a high prevalence (e.g. previous polyps, family history of cancer or abnormal radiology) were excluded. The endoscopy database, Endoscribe, was used to determine polyp location, number and size. Polyp histology was checked from case notes and presence of high risk features (high grade dysplasia, tubulovillous, size>1cm or >3 adenomas) documented.

Statistical analysis: Continuous data was expressed as the mean ± standard deviation. A p value<0.05 was considered statistically significant. Statistical analysis was carried out using Graphpad. Proportions were analysed using Fisher’s exact test and continuous variables with an unpaired t-test.

Results

2842 (2842) colonoscopies were performed for accepted indications (2523 European, 319 Māori). The average age of the European group (64.7±21.1) was significantly older than the Māori group (55.8±14.2; p=0.0001). Accordingly to ensure parity we selected and analysed only the 40-59 years age bracket as the majority of CRC occurs after the age of 50 years, with precursor adenomas developing 5-10 years previously. This age bracket also had the highest number of patients identified as Māori.

Average ages were not significantly different (Māori 50.2±6.3, European 50.4±6.3; p=0.72) and the sex ratio was also comparable (Māori 44% male, Europeans 40.6%; p=0.46). At colonoscopy, polyps were found in 213 Europeans (33.1%) but only 35 Māori (23.5%; p=0.029). At histology, the adenomatous polyp rate in Europeans was 16.7% compared to 8.7% in Māori (p=0.019; 8% difference, CI=2.3-13.9%).

Table 1 Polyps and colorectal cancer in the 40-59 year old age band

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Number of patients</th>
<th>Number of cancers</th>
<th>Number with polyp at colonoscopy</th>
<th>Number with adenoma at histology</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>643</td>
<td>20</td>
<td>213</td>
<td>107 (16.7%)</td>
</tr>
<tr>
<td>Māori</td>
<td>149</td>
<td>1</td>
<td>35</td>
<td>13 (8.7%)</td>
</tr>
</tbody>
</table>

Amongst patients with adenomas, the percentage showing high risk features was not significantly different (European 39.3%, Māori 38.5%; p=1.0). There was also no difference in the average number of adenomatous polyps per person (European 1.7±1.3, Māori 1.9±1.4; p=0.59) or location in the colon (right colon in 41% European and 52% Māori; p=0.39).

Table 2. Adenomatous polyp characteristics, location and number in the 40–59yr age band

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Number of patients with at least one adenoma</th>
<th>Percentage of these patients with high-risk adenomas</th>
<th>Percentage of total adenomas located in the right colon</th>
<th>Average number of adenomas</th>
</tr>
</thead>
<tbody>
<tr>
<td>European</td>
<td>107</td>
<td>42/107(39.3%)</td>
<td>70/169(41%)</td>
<td>1.7±1.3</td>
</tr>
<tr>
<td>Māori</td>
<td>13</td>
<td>5/13(38.5%)</td>
<td>13/25(52%)</td>
<td>1.9±1.4</td>
</tr>
</tbody>
</table>
Discussion

Differences in the incidence of CRC, between ethnicities, are likely to result from a combination of genetic and environmental factors.

Epidemiology suggests that colorectal cancer is highly dependent on the environment. This dependence is illustrated by the high rates of CRC in the offspring of Japanese immigrants to USA compared to the low rates of CRC for Japanese in Japan. It is unlikely that genetic predisposition to CRC would change so quickly.

Within the United States of America, African Americans have an increased incidence of CRC compared to the White population. The reasons for the higher incidence are not known but environmental factors such as diet, exercise and smoking have been implicated. Some biological differences have also been noted. There is evidence of a more proximal distribution of cancers and adenomatous polyps in African Americans. Also, the rates of microsatellite instability (MSI) in sporadic cancers are much higher in African Americans (45%) than would be expected from the literature (12-17%). MSI-high often implies a better prognosis in CRC patients but its effect on the outcome of African Americans is not clear. Overall, ethnic differences have led to the recommendation that CRC screening starts 5 years earlier in African Americans (45yrs old).

However, recognised environmental risk factors do not appear to explain the differences in incidence of CRC between Māori and NZ Europeans. Factors associated with higher risk of CRC are paradoxically more prevalent in Māori who are more likely to be overweight and have higher intakes of fat, energy and alcohol than NZ Europeans. Although vegetable intake is similar in Māori and NZ Europeans, it has been suggested that the different types of vegetables consumed may be protective. Māori commonly eat watercress, puha (sow thistle), melon, kumara and silverbeet in greater quantities than NZ European. However evidence for the presence of anticarcinogens in these particular foods is lacking.

An explanation for the reported difference in incidence of CRC in Māori and the NZ European remains illusive. Our study contributes usefully by showing that the prevalence of colorectal adenomas, the precursor lesions in CRC, in Māori is approximately half that found in NZ Europeans. This mirrors the reported difference in CRC incidence between these groups. In addition, amongst patient with adenomas there was no significant difference in the number of adenomas or percentage of adenomas displaying high risk features between Māori and European. Accordingly the poorer outcome for Māori with this disease may reflect a lack of timely access to quality diagnosis and treatment, rather than an adverse biologic predilection per se.

Further research comparing the biology and histology of colorectal cancer between these groups would be required to strengthen this conclusion. However, if timely access to quality medical services is a concern the importance of health initiatives to improve outcomes for CRC in Māori would be reinforced.

We recognise that the sample population in this study were a symptomatic group and may not represent the general population. However, there are limited opportunities to study the general (asymptomatic) population due to the low autopsy rate and the
absence of a CRC screening program in New Zealand. Additionally, the number of adenomas in the Māori group was small and could be associated with a type 2 error.

**Conclusion**

The prevalence of colorectal adenomas in Māori is approximately half that found in NZ Europeans. This finding mirrors the reported difference in colorectal cancer incidence and supports this being a real finding. There were no significant differences between Māori and NZ Europeans in the proportion of high risk adenomas, the total number of polyps found and the location in right or left colon. This is potentially a positive health finding for Māori as improvements in diagnosis and management of colorectal cancer could see mortality rates halve.

**Competing interests:** None known.

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