Survival disparities in Indigenous and non-Indigenous New Zealanders with colon cancer: the role of patient comorbidity, treatment and health service factors

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ABSTRACT

Background Ethnic disparities in cancer survival have been documented in many populations and cancer types. The causes of these inequalities are not well understood but may include disease and patient characteristics, treatment differences and health service factors. Survival was compared in a cohort of Maori (Indigenous) and non-Maori New Zealanders with colon cancer, and the contribution of demographics, disease characteristics, patient comorbidity, treatment and healthcare factors to survival disparities was assessed.

Methods Maori patients diagnosed as having colon cancer between 1996 and 2003 were identified from the New Zealand Cancer Registry and compared with a randomly selected sample of non-Maori patients. Clinical and outcome data were obtained from medical records, pathology reports and the national mortality database. Cancer-specific survival was examined using Kaplan—Meier survival curves and Cox hazards modelling with multivariable adjustment.

Results 301 Maori and 328 non-Maori patients with colon cancer were compared. Maori had a significantly poorer cancer survival than non-Maori (hazard ratio (HR)=1.33, 95% CI 1.03 to 1.71) that was not explained by demographic or disease characteristics. The most important factors contributing to poorer survival in Maori were patient comorbidity and markers of healthcare access, each of which accounted for around a third of the survival disparity. The final model accounted for almost all the survival disparity between Maori and non-Maori patients (HR=1.07, 95% CI 0.77 to 1.47).

Conclusion Higher patient comorbidity and poorer access and quality of cancer care are both important explanations for worse survival in Maori compared with non-Maori New Zealanders with colon cancer.

INTRODUCTION

Ethnic disparities in cancer survival have been described in many populations and cancer types. Survival disparities are found between Indigenous and non-Indigenous peoples in New Zealand,[1–3] Australia[4 5] and the USA,[6–8] and between ethnic minority and majority populations in many countries, particularly the USA.[6 9 10] Survival differences are seen across a range of cancer sites including malignancies of the breast, prostate, lung and colon.[6 9 10]

Cancer is an important and growing contributor to the 8—9-year difference in life expectancy between Maori and non-Maori New Zealanders.[11 12] Maori are the Indigenous peoples of New Zealand and make up 15% of the 4 million population; the non-Indigenous population is predominantly European in origin with significant Pacific (7%) and Asian (9%) groupings.[13] As with many kinds of cancer, Maori patients have a poorer survival from colon cancer compared with non-Maori.[1 2] New Zealand has a particularly high incidence and mortality from colorectal cancer.[14] Age-adjusted incidence is lower in Maori compared with non-Maori populations (nine compared with 15 per 100 000),[2] but mortalities are now similar, having decreased in non-Maori and increased in Maori over time.[11 15 16]

The causes of ethnic disparities in cancer survival are poorly understood but are likely to include factors at the level of individual patients, healthcare processes and health systems overall.[9 17 18] The existence of ethnic survival disparities in many populations and cancer types suggests that these factors are at work across a range of different contexts and ethnic groupings.

Patient-level factors that may affect survival include tumour characteristics (grade and stage at diagnosis) and comorbid conditions. Late-stage diagnosis contributes to cancer survival disparities between Maori and non-Maori New Zealanders,[1 2] Indigenous and non-Indigenous Australians,[4] and Black and White Americans[19–21] but is unlikely to explain the majority of ethnic survival disparities. Fewer studies have assessed the impact of patient cormorbidity (which is difficult to measure accurately from administrative data), although a higher comorbidity contributes to survival disparities between Indigenous and non-Indigenous Australians.[4] There is some evidence that biological factors play a role in survival disparities for breast cancer[22] but not for colon cancer.[20 23]

Health system factors may impact both at the level of treatment decisions and processes and at more structural levels such as the location, resourcing and accessibility of healthcare facilities. Lower rates of cancer treatment (including surgery and chemotherapy) contribute to Indigenous/non-Indigenous survival disparities in Australia[4] and ethnic survival disparities in the USA.[9 19 24–26] Differential healthcare access and institutional factors receive particular emphasis in the USA context[27 28] but do not fully explain survival inequalities, since these are found even in equal-access healthcare settings.[9 19]

New Zealand has a publicly funded national health system that provides specialist and hospital care to all residents without patient charges. There are no existing New Zealand data on the role of health systems factors in cancer survival disparities, but Maori/non-Maori inequalities are found in management of other diseases with Maori receiving fewer health services relative to expected need[29–31] and lower quality care in some contexts.[32] Maori are more likely to self-report experiences of being discriminated against by a health professional due to their ethnicity,[33] which may contribute to suboptimal treatment. Higher rates of socio-economic disadvantage in the Maori population make it harder for many Maori patients to access services requiring copayments such as primary healthcare and prescription medication.[31 34]

Our study examined survival disparities between Maori and non-Maori patients with a first-time diagnosis of colon cancer. We assessed the relative contribution of patient, treatment and health systems factors to survival disparities, adjusting for patient-level factors first in order to assess the role of treatment and health service differences independent of clinical factors. Our study cohort was drawn from the entire country and included individual review of medical notes from both public and private health facilities, allowing a comprehensive comparison of factors contributing to colon cancer survival disparities at a national level.

METHODS

New Zealand residents diagnosed as having colon cancer between 1996 and 2003 were eligible for study inclusion. Cases came from patients notified to the New Zealand Cancer Registry with a primary tumour in the colon (ICD-10-AM site codes C18.0 to 19.0 excluding 18.1) and morphology consistent with adenocarcinoma. (New Zealand has mandatory registration of all primary cancers except non-melanoma skin cancers and carcinoma in situ.) Patients were ineligible if they were less than 25 years at diagnosis, were normally resident outside New Zealand, had a previous diagnosis of colon cancer, had no histological diagnosis or were diagnosed after death.

All Maori patients meeting the above criteria were included along with an approximately equal number of randomly sampled non-Maori patients. Patients were classified as Maori if their ethnicity was recorded as such in any of the three cancer registry ethnicity fields. (These fields are based on self-identified ethnicity data from hospital admission and registration sheets.) Patients whose ethnicity was not recorded in the cancer registry were classified as non-Maori.[35]

Clinical data were abstracted from patients' medical records, including public and private healthcare providers. Pathology reports were obtained for all patients from their healthcare records, the cancer registry or directly from the reporting laboratory. Data were recorded on a standardised form by a physician (SH) and double-entered into an electronic database. Data included details of patients' presentation, investigation for diagnosis of colon cancer, comorbid conditions present at the time of diagnosis, smoking status, tumour characteristics (including location, histological features and stage at diagnosis), surgical treatment and adjuvant treatment (including chemotherapy and radiotherapy). Small area deprivation and rurality were assigned according to each patient's domicile (census area) code at the time of diagnosis. Small area deprivation was classified by the New Zealand deprivation index, an area-based index calculated from aggregated census data on residents' socio-economic characteristics (such as car access, housing tenure and benefit receipt).[36] Outcome data (vital status

and cause of death) were obtained by linking study patients to the national mortality database, with follow-up to the end of 2005. Patients whose deaths were not recorded in the mortality database were assumed to be still alive at the end of follow-up, while those who died from causes other than colon cancer were censored at the date of death.

Maori and non-Maori cohorts were compared for demographics, tumour characteristics, patient comorbidity and smoking, treatment and markers of health service access. Maori/non-Maori prevalence ratios were adjusted for age, sex and year of diagnosis using log Poisson regression with robust variance estimation.[37] Cancer-specific survival curves for Maori and non-Maori were estimated using the Kaplan–Meier method and compared by logrank test. Mortality hazards were compared using Cox regression modelling.

Hazard ratios were sequentially adjusted for five domains of covariates to assess the relative contribution of each domain to Maori/non-Maori survival disparities. These domains were: patient demographics (age, sex and year of diagnosis), disease characteristics (stage, grade and site of tumour, and emergency presentation), patient comorbidity (specific comorbid conditions and smoking), treatment (definitive surgery, surgeon type, delay to surgery and adjuvant chemotherapy) and markers of healthcare access (treatment facility type, small area deprivation and rurality). We used the Hausman test to assess the significance of a change in hazard ratio with adjustment for each domain.[38 39]

Specific comorbid conditions were included as covariates in survival analyses if they were found to be independently associated with colon cancer survival in the study cohort.[40] These conditions were: previous myocardial infarction, previous or current heart failure, current respiratory disease, diabetes mellitus, cerebrovascular disease, renal disease and neurological disorders. For the purpose of survival analyses, small area deprivation was conceptualised as a marker of healthcare access (rather than an individual sociodemographic variable). This reflects the influence of deprivation on cancer survival independent of individual characteristics such as stage at diagnosis, comorbidity or smoking (which were adjusted for prior to including deprivation in the model).[41 42]

Approval for this study was granted by the New Zealand Multi-Region Ethics Committee (MEC/05/06/069). All analyses were carried out in SAS (version 9.1, SAS Institute, Cary, North Carolina).

RESULTS

A total of 376 Maori patients met the study criteria based on cancer registry records, and a further 400 non-Maori patients were randomly selected from the registry as a comparison cohort. Ninety one (12%) of those sampled were later excluded because further information showed they were ineligible for study inclusion (65 had miscoded data (primarily cancer site) in the cancer registry, and a further 26 had no histological diagnosis), giving 329 Maori and 356 non-Maori patients. Full data were obtained for 301 Maori and 328 non-Maori patients (92% of the eligible sample). Based on cancer registry records, 93% of the non-Maori cohort were of European ethnicity.

The Maori cohort was significantly younger than the non-Maori cohort (Table 1) in keeping with the younger age structure of the total Maori population in New Zealand.[12] Maori patients had a higher prevalence of comorbid conditions, with around two and a half times the rates of diabetes, heart failure, respiratory disease and renal disease seen in non-Maori. Maori patients were about 50% more likely to be smokers.

Table 1 Demographics, tumour characteristics, comorbid conditions and smoking status in Maori and non-Maori cohorts

	Maori (n=301)	Non-Maori (n=356)	Prevalence ratio (95% CI)*	p Value
Age at diagnosis (mean)	61.3 years	70.6 years		<0.0001
Female	43.9%	52.4%		0.03
Tumour stage				
Stage I and II	40.9%	44.8%	1.01 (0.83 to 1.23)	0.9
Stage III (+ve nodes)	28.9%	34.2%	0.84 (0.65 to 1.08)	0.2
Stage IV (metastases)	28.9%	20.1%	1.20 (0.89 to 1.62)	0.2
Unstaged	1.3%	0.9%	2.33 (0.91 to 5.97)	0.08
Tumour site				
Right colon	35.9%	46.7%	0.81 (0.66 to 0.99)	0.04
Left colon	44.9%	29.6%	1.37 (1.09 to 1.72)	0.007
Rectosigmoid	16.0%	16.2%	1.09 (0.74 to 1.61)	0.7
Synchronous	3.3%	7.6%	0.46 (0.20 to 1.05)	0.06
Tumour grade				
Well differentiated	12.0%	7.6%	1.97 (1.17 to 3.33)	0.01
Moderately differentiated	71.1%	73.5%	0.93 (0.84 to 1.04)	0.2
Poorly differentiated	16.9%	18.9%	0.91 (0.63 to 1.32)	0.6
Emergency presentation	38.2%	26.5%	1.44 (1.13 to 1.84)	0.004
Comorbid conditions				
Previous myocardial infarction	8.0%	8.2%	1.22 (0.68 to 2.19)	0.5
Heart failure	11.6%	9.2%	2.65 (1.63 to 4.32)	<0.0001
Diabetes	20.9%	10.7%	2.46 (1.66 to 3.65)	<0.0001
Respiratory disease	7.0%	3.7%	2.42 (1.18 to 5.00)	0.02
Cerebrovascular disease	6.6%	9.2%	1.25 (0.69 to 2.26)	0.5
Renal disease	6.6%	4.0%	2.60 (1.27 to 5.32)	0.01
Neurological disorder†	5.3%	7.6%	0.71 (0.36 to 1.40)	0.3
Smoking status				
Current smoker	27.9%	12.2%	1.54 (1.07 to 2.23)	0.02
Ex-smoker	38.5%	36.0%	1.20 (0.97 to 1.49)	0.09
Non-smoker	29.6%	45.4%	0.72 (0.57 to 0.90)	0.005
Missing	4.0%	6.4%	0.64 (0.31 to 1.33)	0.2

*Prevalence ratios are adjusted for age, sex and year of diagnosis using log Poisson regression with robust convergence estimation.
†Significant neurological and psychiatric disorders other than cerebrovascular disease—that is, bipolar disorder, blindness, dementia, epilepsy, idiopathic peripheral neuropathy, intellectual impairment, multiple sclerosis, Parkinson disease, polio, previous head injury, schizophrenia and spinal stenosis.

Table 2 Treatment and markers of health service access in Maori and non-Maori cohorts

	Maori (n=301)	Non-Maori (n=356)	Prevalence ratio (95% CI)*	p Value
Definitive surgery†	87.7	93.6	0.93 (0.88 to 0.98)	0.01
Surgeon type				
Colorectal surgeon	14.3	15.6	0.70 (0.47 to 1.05)	0.09
General surgeon	72.1	72.9	1.02 (0.92 to 1.13)	0.7
Surgical trainee	8.6	6.7	1.37 (0.76 to 2.49)	0.3
Delay to surgery (>28 days)	14.3	11.9	1.40 (0.90 to 2.20)	0.1
Adjuvant chemotherapy	18.9	19.8	0.61 (0.42 to 0.86)	0.006
Treatment facility				
Public secondary	61.1	46.3	1.40 (1.20 to 1.65)	<0.0001
Public tertiary	29.2	33.2	0.89 (0.69 to 1.14)	0.4
Private	5.0	17.7	0.19 (0.11 to 0.32)	<0.0001
Small-area deprivation				
1 (least deprived)	6.3	14.3	0.37 (0.22 to 0.62)	0.0002
2	7.3	19.2	0.34 (0.21 to 0.56)	<0.0001
3	16.0	22.0	0.81 (0.57 to 1.16)	0.2
4	27.2	26.5	1.06 (0.80 to 1.41)	0.7
5 (most deprived)	43.2	18.0	2.41 (1.82 to 3.19)	<0.0001
Rurality				
Urban	77.4	90.0	0.86 (0.79 to 0.93)	0.0001
Semirural	6.6	4.9	1.20 (0.59 to 2.41)	0.6
Rural	16.0	4.3	3.82 (2.00 to 7.29)	<0.0001

*Prevalence ratios are adjusted for age, sex and year of diagnosis using log Poisson regression with robust convergence estimation.
†Definitive surgery: surgical removal of tumour (including complete excision during colonoscopy).

between diagnosis and treatment. Maori patients were significantly less likely to receive adjuvant chemotherapy.

The cohorts differed significantly in indicators of health service access. Maori patients were more likely to be treated in secondary (smaller) public healthcare facilities and less likely to be treated in private facilities. They were also more likely to live in high-deprivation areas and were almost four times as likely to live in rural areas compared with non-Maori patients.

Compared with non-Maori patients, Maori were more likely to be diagnosed as having advanced (metastatic) cancer and less likely to be diagnosed as having localised disease, although differences were non-significant after adjustment for age and sex. The stage distribution of the study cohort was not significantly different to that of all registered colon cancers from the corresponding period, except that the study cohort had a lower prevalence of unstaged cancer (4.5% overall compared with 7.4% in the Cancer Registry, p=0.003). Maori patients were more likely to have left-sided tumours, while non-Maori had more right-sided tumours. Cancers in Maori patients tended to be less aggressive with a higher proportion of well-differentiated tumours. Maori patients were significantly more likely to present to hospital services as an emergency case (with bowel obstruction, for example) rather than being electively referred by a primary care physician.

Non-Maori patients were significantly more likely than Maori to undergo definitive surgery (that is, complete removal of the primary tumour either at colonoscopy or at operation) (Table 2). No significant differences were found in the type of surgeon performing the operation, but Maori patients were (non-significantly) more likely to experience a delay of a month or more

Figure 1 Cancer-specific survival for Maori and non-Maori cohorts (unadjusted).

Table 3 Hazard ratios for cancer-specific mortality risk in Maori and non-Maori cohorts with stepwise adjustment for demographics, disease factors, patient factors, healthcare processes and healthcare access

Adjusted for:	Additional variables in model	Hazard ratio	95% CI
0. Unadjusted	—	1.33	1.03 to 1.71
1. Demographics	Age, sex, year of diagnosis	1.30	0.99 to 1.71
2. Disease factors	+Stage	1.29	0.97 to 1.71
	+Grade	1.31	0.99 to 1.74
	+Site	1.36	1.01 to 1.82
	+Emergency presentation	1.33	0.99 to 1.79
3. Patient factors	+Comorbidities*	1.24	0.92 to 1.68
	+Smoking	1.20†	0.89 to 1.63
4. Healthcare processes	+Definitive surgery	1.21	0.89 to 1.64
	+Surgeon type	1.21	0.90 to 1.65
	+Delay to surgery	1.20	0.88 to 1.63
	+Adjuvant chemotherapy	1.17	0.86 to 1.60
5. Healthcare access	+Treatment facility type	1.12	0.82 to 1.53
	+Small-area deprivation	1.10	0.80 to 1.52
	+Rurality	1.07†	0.77 to 1.47

Hazard ratios calculated using Cox proportional hazards regression with imputed data for 33 individuals with missing smoking status (almost identical results obtained with missing variable indicator).
*Comorbidities—that is, previous MI, heart failure, respiratory disease, diabetes, cerebrovascular disease, renal disease and neurological disorders (as outlined in Table 1).
†Significant decrease in hazard ratio compared with previous domain, that is p value <0.05 by Hausman test.

Maori had a lower cancer-specific survival than non-Maori patients (Figure 1). The crude 5-year cancer-specific survival was 61.1% in non-Maori and 52.5% in Maori patients. The crude mortality hazard ratio for Maori compared with non-Maori patients was 1.33 (95% CI 1.03 to 1.71) (Table 3). This disparity persisted with adjustment for demographic factors (hazard ratio=1.30 after adjustment for age, sex and year of diagnosis) and disease factors (hazard ratio=1.33 after further adjustment for stage, grade and site of tumour, and emergency presentation).

Patient comorbidity accounted for around a third of the Maori/non-Maori disparity in cancer survival, with adjustment for specific comorbid conditions and patient smoking reducing the hazard ratio from 1.33 to 1.20 (Table 3). Differences in treatment for Maori and non-Maori patients may have contributed to the survival disparity, with a (non-significant) reduction in the hazard ratio from 1.20 to 1.17 following further adjustment for definitive surgery, surgeon type, delay to surgery and receipt of adjuvant chemotherapy. Differences in indicators of healthcare access contributed significantly to the survival disparity, with the hazard ratio falling from 1.17 to 1.07 with further adjustment for treatment facility type, small area deprivation and rurality of patient's residence.

Factors included in this final model (Table 4) together accounted for almost all the Maori/non-Maori disparity in cancer survival, with Maori patients only 7% more likely to die from their colon cancer after adjustment for demographics, tumour characteristics, patient comorbidity, treatment and markers of health service access.

DISCUSSION

In a population-based cohort of New Zealanders with colon cancer, Maori patients had a poorer survival than non-Maori, with around a 30% higher risk of dying from their cancer. This survival disparity was not due to disease characteristics: Maori patients generally had lower-grade tumours and were not significantly more likely to present with advanced disease. Higher rates of pre-existing medical conditions and more limited

Table 4 Hazard ratios for selected variables from final model (cancer-specific mortality risk)

	n	%	HR	95% CI
Indigenous status				
Non-Maori	328	52.2	1.00	
Maori	301	47.9	1.07	0.77 to 1.47
Stage at diagnosis				
Stage I	79	12.6	0.51	0.22 to 1.20
Stage II	191	30.4	1.00	
Stage III	199	31.6	3.81	2.36 to 6.16
Stage IV	153	24.3	19.64	12.36 to 31.20
Unstaged	7	1.1	4.26	1.12 to 16.21
Grade (cell differentiation)				
Well differentiated	61	9.7	0.77	0.44 to 1.35
Moderately differentiated	455	72.3	1.00	
Poorly differentiated	113	18.0	1.45	1.04 to 2.03
Tumour site				
Right colon	261	41.5	1.11	0.80 to 1.55
Left colon	232	36.9	1.00	
Rectosigmoid junction	101	16.1	0.67	0.44 to 1.00
>1 site (multiple tumours)	35	5.6	1.02	0.57 to 1.82
Emergency presentation				
No	427	67.9	1.00	
Yes	202	32.1	1.26	0.94 to 1.69
Comorbid conditions				
Previous myocardial infarction	51	8.1	1.27	0.76 to 2.11
Heart failure	65	10.3	1.53	0.89 to 2.62
Diabetes	98	15.6	0.95	0.64 to 1.42
Respiratory disease	33	5.3	0.68	0.34 to 1.38
Cerebrovascular disease	50	8.0	1.31	0.81 to 2.12
Renal disease	33	5.3	1.00	0.49 to 2.05
Neurological disorder*	41	6.5	2.01	1.16 to 3.48
Smoking status				
Non-smoker	124	19.7	1.00	
Current smoker	234	37.2	1.16	0.78 to 1.72
Ex-smoker	238	37.8	1.50	1.08 to 2.10
Definitive surgery				
No	66	10.5	1.00	
Yes	563	89.5	0.24	0.15 to 0.39
Type of surgeon				
General surgeon	456	72.5	1.00	
Specialist colorectal surgeon	94	14.9	1.22	0.80 to 1.85
Trainee surgeon	48	7.6	1.13	0.66 to 1.92
Delay to treatment				
No	547	87.0	1.00	
Yes	82	13.0	1.10	0.72 to 1.70
Adjuvant chemotherapy				
No	507	80.6	1.00	
Yes	122	19.4	0.55	0.35 to 0.88
Treatment-facility type				
Secondary public hospital	336	53.4	1.34	0.97 to 1.84
Tertiary (teaching) public hospital	197	31.3	1.00	
Private hospital	73	11.6	0.92	0.53 to 1.58
Small-area deprivation (per 10% increase in deprivation score)			1.02	0.96 to 1.08
Rurality				
Urban	531	84.4	1.00	
Semirural	36	5.7	1.24	0.72 to 2.15
Rural	62	9.9	1.21	0.79 to 1.84

Hazard ratios calculated using Cox proportional hazards regression with imputed data for 33 individuals with missing smoking status (almost identical results obtained with a missing variable indicator).
*Significant neurological and psychiatric disorders other than cerebrovascular disease—that is, bipolar disorder, blindness, dementia, epilepsy, idiopathic peripheral neuropathy, intellectual impairment, multiple sclerosis, Parkinson disease, polio, previous head injury, schizophrenia and spinal stenosis.

health service access each appeared to account for around a third of the excess mortality risk in Maori patients, while lower rates of cancer treatment may also have made a modest contribution. Together these factors accounted for almost all the survival disparity between Maori and non-Maori patients.

We did not find any significant Maori/non-Maori differences in stage at diagnosis, although non-significant differences were consistent with previous evidence of more advanced cancer in Maori patients.[2] New Zealand does not currently have a national screening programme for colon cancer, but Maori/non-Maori disparities are evident in access to breast and cervical cancer screening[43 44] and specialist cancer services.[45] Inadequate access to primary and diagnostic health services may also contribute to higher rates of emergency presentation in Maori patients with colon cancer.

Maori patients in our cohort tended to have more favourable tumour characteristics with a higher prevalence of well-differentiated cancers. Black patients in the USA are likewise more likely than White patients to have well-differentiated tumours of the colon,[20 23] arguing against the suggestion that survival disparities reflect less favourable biology in ethnic minority groups. Current evidence does not support a role for genetic factors in ethnic disparities in cancer survival.

Patient comorbidity and smoking were significant mediators of Maori/non-Maori survival disparities. Higher rates of diabetes, cardiovascular and respiratory disease in Maori patients reflect high prevalences in the general Maori population.[12] Valery et al found a similarly high comorbidity in Indigenous Australians with cancer, although the contribution to survival disparities is difficult to assess (the authors controlled for comorbidity only after adjusting for treatment differences).[4] The reasons for higher comorbidity and smoking rates in Indigenous peoples are complex and include greater socio-economic deprivation, poorer access to favourable determinants of health and (ultimately) historical disadvantage through the processes of colonisation.[12 46]

Differences in healthcare access and quality are important mediators of survival disparities between Maori and non-Maori patients with cancer. Similar disparities exist in cardiac care, with Maori patients more commonly admitted to hospitals lacking cardiac intervention services[47] contributing to lower rates of revascularisation compared with non-Maori.[29 30] Our study found several markers of poorer healthcare access in Maori patients with cancer, who were more likely to live in rural and economically deprived areas and less likely to receive treatment in specialist cancer centres or private hospitals. These markers almost certainly overlap with healthcare quality, which was not directly assessed. Differential healthcare access has been shown to contribute to health disparities in other countries.[27 28 48] US hospitals serving predominantly African—American communities have a more limited capacity and struggle to meet treatment standards—a de facto segregation of health services that contributes to poorer health outcomes in the Black population.[49] Even if individual facilities in New Zealand provide equitable care, the structure of the health system as a whole may result in unequal care for Maori and non-Maori patients, a form of institutional racism and an important cause of survival disparities.

New Zealand has a public health system that aims to provide equal-access care to all residents, although individuals may purchase private health insurance or pay directly to access some services (including specialist assessment and surgery) through private health providers. Health insurance coverage is much lower in the Maori population (Stillman S and Cumming J, personal communication) as reflected here by low rates of private hospital treatment among Maori patients. Patients with access to private healthcare may gain a survival benefit from shorter waiting times and easier access to diagnostic and treatment services.

In the New Zealand context, socio-economic position is strongly correlated with ethnicity and is an important mediator in the relationship between ethnicity and health.[11 12] Our only socio-economic measure was residential area deprivation at the time of diagnosis. In this study, we view area deprivation as a marker of health service access more than individual patient demographics. Many studies show that socio-economic deprivation is a predictor of poorer cancer survival primarily through its effect on stage at diagnosis and cancer treatment.[21 41 42] Our multivariable models examined the effects of individual-level factors (such as stage at diagnosis and comorbidity) before contextual factors (such as healthcare access). Having already adjusted for stage and treatment differences, the remaining effect of area deprivation is likely to occur primarily through its influence on healthcare access (including both contextual effects and patients' ability to reach and navigate cancer services).

Potential limitations of our study include modest sample size, misclassified deaths and possible selection effects. The relatively low occurrence of colon cancer in the Maori population during an 8-year window limits our power to demonstrate small Maori/non-Maori differences and changes in the hazard ratio with covariate adjustment. Misclassification of the fact of death is likely to be very small, since all study members were New Zealand residents, and deaths occurring in New Zealand are recorded in the national mortality database. A more likely source of bias is misclassification of non-cancer deaths as due to colon cancer; this would tend to bias Maori/non-Maori hazard ratios towards the null, since a greater proportion of non-Maori deaths are due to causes other than colon cancer. Our sample may represent a slightly selected group of patients, since inclusion required histological evidence of adenocarcinoma, excluding just under 7% of all registered cases. This restriction increased the internal validity of our study, however, and allowed us to assess the role of tumour biology in survival disparities.

Strengths of our study include its population-based sample frame, near-complete data ascertainment (92% of eligible cases) and comprehensive data collection including detailed comorbidity and pathology assessment from review of individual medical records. Since cohort members and data were drawn from throughout New Zealand, these findings inform our understanding of Indigenous/non-Indigenous disparities at a national level, including the role played by health service access and quality. This is highly relevant given the substantial Maori/non-Maori differences that exist in geographical and socio-economic distribution, private health insurance and access to tertiary hospitals.

What is already known on this subject

▶ Ethnic disparities in cancer survival are observed in many populations and cancer types.
▶ Maori (Indigenous) New Zealanders with colon cancer have poorer survival than patients from other ethnic groups, even after adjustment for stage at diagnosis.
▶ Ethnic disparities in cancer survival may reflect differences in healthcare access and quality.

What this study adds

▶ Maori/non-Maori disparities in colon cancer survival are largely accounted for by higher comorbidity levels and poorer access to quality cancer services in Maori patients.
▶ Markers of health service access and quality accounted for over a third of the survival disparity between Maori and non-Maori patients with colon cancer.
▶ Attention to healthcare delivery is important for addressing ethnic inequalities in cancer outcomes.

Attention to health services is a key step in improving cancer care and decreasing disparities in cancer outcomes. Potential strategies to improve access for Maori patients include development and support of Maori health providers, improving the cultural accessibility and competence of mainstream providers and ensuring adequate resources for health services serving area with large Maori populations.[44 45] Finally, ongoing monitoring of treatment and outcomes by ethnicity provides important feedback to improve services and help ensure Indigenous and non-Indigenous New Zealanders receive an equal standard of care.

Contributors All authors listed in this paper fulfil the criteria of authorship, and there is no one else who fulfils these criteria who is not listed here as an author.
Please note: we have included a large number of authors because all these people made substantial contributions to the study design or data analysis and interpretation. This reflects the breadth of disciplines included in this study—that is, public health, epidemiology, cancer management, indigenous health research and sociology. Contributions were as follows: SH contributed to study design, collected the data, led data analysis and interpretation, and wrote the first draft of the paper; DS initiated and led the study design, contributed to data analysis and interpretation, and contributed to draft revisions; TB contributed to study conception and design, data analysis and interpretation and draft revisions; BR contributed to study design, data interpretation and draft revisions; GP contributed to study design, data analysis and interpretation, and draft revisions; JC contributed to data analysis and interpretation, and contributed to draft revisions; ED contributed to study design, data interpretation and draft revisions; DC contributed to study design, data interpretation and draft revisions; RC contributed to data collection, data analysis and interpretation, and draft revisions; KD contributed to study design, data interpretation and draft revisions; TM contributed to study design, data interpretation and draft revisions; IK contributed to data analysis and interpretation, and draft revisions; G Datta provided advice on data analysis; B Cox provided advice on study design; D Kenwright provided pathology advice and reviewed some pathology records; A O'Donnell provided oncology advice; E Britton assisted with data collection, particularly obtaining pathology records.

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