

Low but increasing rates of inflammatory bowel disease in Māori: a report from Lakes District Health Board IBD

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ABSTRACT

Inflammatory bowel diseases (IBD) are chronic, inflammatory diseases that are increasingly prevalent in New Zealand. Previous regional studies describe significantly lower rates of IBD in Māori compared to non-Māori. This article reports the prevalence and incidence of IBD at Lakes District Health Board, and discusses potential contributing factors to the observed increasing incidence rates in Māori. Although the rates are still less than non-Māori, colonisation with increased urbanisation and changes in diet and hygiene suggest that IBD rates may continue to increase in Māori.

Inflammatory bowel diseases (IBD) are chronic, inflammatory diseases comprising ulcerative colitis (UC) and Crohn's disease (CD). New Zealand has one of the highest incidence rates of IBD worldwide, with an estimated 20,792 patients with IBD (1 in 227) increasing by 5.6% per year.¹

It has been observed that Māori are less often diagnosed with IBD. Wigley & Maclaurin² reviewed all hospital admissions in New Zealand between 1954 and 1958, and found a 14-fold difference between NZ European and Māori colitis admission rates (0.030% vs 0.0022%, respectively). Although this is likely to have been influenced by factors such as disease severity, compliance to treatment and healthcare accessibility, the considerable difference in hospital admissions suggested a substantial difference in UC prevalence between Māori and NZ European. Similarly, between 1969 and 1978, Māori accounted for 15% of the Auckland population, but only 2/456 (0.4%) of UC admissions and 0/137 (0%) of CD admissions.³ Subsequently, a study conducted between 1983 and 2013 at Dunedin Hospital found that, although Māori comprised 7.5% of the Otago population, they comprised only 1.7% of total IBD cases.⁴

In the only New Zealand population-based IBD epidemiology study, the Canterbury IBD Study, identified 1,420 patients with IBD from both the public sector and private clinics. Once again, a low Māori IBD rate was confirmed (Māori comprising 1% of IBD patients but 7.3% of the general population).⁵

These studies have shown low rates of IBD in Māori, despite high and increasing rates of IBD in New Zealand overall. However, there are no data on the rates of IBD from regions of New Zealand where Māori comprise a larger proportion of the population.

IBD at Lakes DHB

Lakes District Health Board (DHB) is located at the northeast of the North Island, covering Rotorua, Taupo and the surrounding rural areas. The population of Lakes DHB is 116,370 in 2020/2021,⁶ with 36.9% self-identified as Māori.⁷

All new IBD diagnoses in Lakes DHB are made by gastroenterologists in the Rotorua Hospital, and followed up by gastro nurse specialists. We screened the Lakes DHB computer database and reviewed all patients with a definitive or suspected diagnosis of IBD. Patients' relevant medical records such as clinical letters, endoscope reports, previous discharge summaries and demographic information were screened, and data were collected including age, sex, year of diagnosis, IBD subtype, clinical presentation, smoking status, previous surgery, and medication use. Prevalence and incidence of IBD were calculated based on population estimates from New Zealand censuses.⁸ It was hard to determine the exact year of diagnosis for patients diagnosed over 10 years ago, as often the medical records were incomplete. Therefore, five-year average incidences

were calculated instead of annual incidences. After excluding one deceased patient, a total of 197 patients were included in our study (Table 1).

We studied 197 IBD patients living in the Lakes DHB region. This was undertaken via the IBD patient database used to manage IBD patients attending Lakes DHB. Demographic and clinical data were extracted. The incidence and prevalence of IBD were calculated based on population estimates from past Stats NZ censuses. The characteristics of these patients are shown in Table 1.

Māori IBD patients were more likely to be female (80% vs 47%, $p=0.01$) and there was a trend to younger age compared with non-Māori (41 years vs 49 years, $p=0.07$). Although not significantly, Māori experienced more hospital admissions than non-Māori (1.80 vs 1.11, $p=0.21$). No significant differences in IBD phenotypes and

clinical presentations were found between Māori and non-Māori.

The incidence rates of IBD at Lakes DHB is compatible with data of Auckland and Otago regions, but markedly lower than that of Canterbury (Table 2). One reason is the incomplete recruitment of cases, as only patients known to the public healthcare system were included in Lakes, Auckland, and Otago studies, whereas a Canterbury study recruited patients from both public and private sectors. Another crucial reason is the disparity in medical resources and socio-economic status between DHBs, which gives rise to barriers to healthcare, and delay in diagnosis. The ratio of doctors to population is 303 per 100,000 at Lakes DHB, which is markedly lower than 363 per 100,000 at Canterbury.⁹ Regarding socio-economic status, 54.3% people

Table 1: Inflammatory bowel disease at Lakes District Health Board

| | Māori | non-Māori | Total |
|---|------------------|--------------------|------------|
| Number of patients | 15 (7.6%) | 182 (92.4%) | 197 |
| Sex (%) | | | |
| Male | 3 (20%) | 97 (53%) | 100 (51%) |
| Female | 12 (80%) | 85 (47%) | 97 (49%) |
| Age (mean ± standard deviation) | 41±14 | 49±17 | 48±17 |
| Smoker/ex-smoker (%) ¹ | 4 (31%) | 47 (30%) | 51 (30%) |
| Inflammatory bowel disease phenotype (%) | | | |
| Ulcerative colitis | 5 (33%) | 72 (40%) | 77 (39%) |
| Crohn's disease | 9 (60%) | 91 (50%) | 100 (51%) |
| Inflammatory bowel disease unclassified | 1 (7%) | 19 (10%) | 20 (10%) |
| Median age of diagnosis ² | 33±15 | 37±17 | 37±17 |
| Median number of hospital admission since 2010 | 1.80 | 1.11 | 1.16 |
| Current biologic drug use (%) | 6 (40%) | 73 (40%) | 79 (40%) |
| Clinical remission (%) ³ | 9 (60%) | 81 (46%) | 90 (47%) |
| Surgical intervention for IBD (%) | 3 (20%) | 47 (26%) | 50 (25%) |

¹Missing data on 29 patients (2 Māori, 27 Non-Māori)

²Missing data on 22 patients (1 Māori, 21 Non-Māori)

³Missing data on 7 non-Māori CD (100 patients, 51%) was more common than UC (77 patients, 39%) and IBD-U (20 patients, 10%). Fifteen (7.6%) patients were Māori and 182 (92.4%) NZ European/Other, leading to IBD ethnic-specific prevalence rates of 34.9 and 256.8/100,000 in Māori and non-Māori, respectively.

at Lakes DHB are ranked in quantile 4 or 5,¹⁰ the most deprived groups, as compared to 25.6% in the Canterbury DHB.¹¹ Therefore, it is highly likely that the incidence and prevalence rates of IBD at Lakes DHB is underestimated.

Lakes data have illustrated a steep increase in IBD incidence over time (Figure 1). This is consistent with a systematic review of IBD incidence rates globally between 1930 and 2010. Including epidemiological studies of IBD with a follow-up of at least 10 years, a statistically significant increase was reported in 60% of UC and 75% of CD studies.

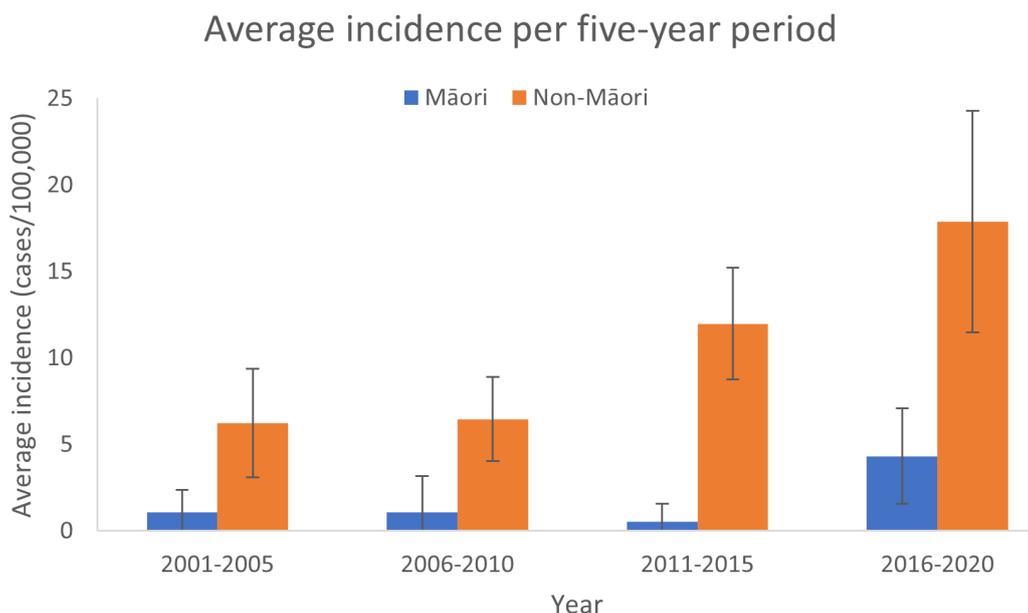
Low but increasing rates of IBD in Māori

The rates of IBD in Māori are lower than those in non-Māori at Lakes DHB, as Māori accounts for 36.9% of Lakes population, but only 7.6% of the cases. However, Figure 1 demonstrates an 8-fold increase in IBD diagnoses in Māori between the 2011–2015 and 2016–2020 periods. This is the first time that Māori IBD incidence rates have been shown to be increasing. Furthermore, the Lakes DHB data demonstrate the highest rate of IBD in Māori described to date, albeit at rates less than non-Māori living in the same region.

Table 2: Comparison of New Zealand inflammatory bowel disease incidence rates (cases/100,000)

| Studies | IBD | CD | UC | IBDU |
|---------------------------------|-------------|------------|------------|------------|
| Auckland 1969–1978 ³ | 7.2 | 1.8 | 5.4 | N/A |
| Otago 1996–2013 ⁴ | 14.1 | 7.6 | 5.4 | 1.1 |
| Canterbury 2004 ⁵ | 24.9 | 16.3 | 7.5 | 0.3 |
| Canterbury 2014 ¹² | 39.5 | 26.4 | 12.6 | 0.2 |
| Lakes 2016–2020 | | | | |
| Total population | 12.5 | 6.0 | 4.1 | 2.3 |
| Māori | 4.3 | 2.9 | 0.9 | 0.5 |
| non-Māori | 17.9 | 8.2 | 6.2 | 3.5 |

Figure 1: Comparison of IBD incidence in Māori and non-Māori at Lakes DHB.



It is not clear why Māori have lower rates of IBD than non-Māori, or why Māori IBD rates are now increasing dramatically. IBD are complex diseases with genetic, environmental and immunological contributions to their aetiology. It is possible that rates of IBD in Māori are underestimated or that they experience delay in diagnosis, given known inequities throughout health services, including delays to see a GP,¹³ lack of accessibility to healthcare services¹⁴ and lower rates for referral to and having a colonoscopy¹⁵ compared with non-Māori. However, it remains unlikely that IBD rates are similar to that of non-Māori. Awareness of bowel health for Māori through the national bowel screening programme,¹⁶ increasing iwi based healthcare initiatives¹⁷ and proactive prioritisation by some GPs and DHBs,¹⁸ are non-specific for IBD diagnosis but may be contributing to recent higher rates of IBD diagnosis in Māori.

Although, the Lakes DHB data does not suggest inequities in receiving biological treatment, with Māori and non-Māori having similar use of advanced therapies, there is no data describing the challenges faced by Māori navigating the IBD diagnostic and treatment pathways.

The genetic risk factors for IBD have been extensively studied in Caucasian populations. Polymorphisms in *CARD15*, which encodes for the NOD2 protein that regulates interactions between the mucosal immune system and gut microbiome, have been strongly associated with the risk of developing CD in multiple populations including in New Zealand.¹⁹ However, *CARD15* polymorphisms are less common in Māori.²⁰ Other non-Caucasian populations have also demonstrated low rates of *CARD15* polymorphisms but higher rates of other polymorphisms associated with the risk of IBD.²¹ More in-depth genetic studies of IBD in Māori are not available.

Increasing IBD incidence rates worldwide suggest that environmental factors are most likely implicated. Many environmental risk factors for IBD have been studied across multiple populations, including New Zealand. Among them, the strongest associations that have been identified with an increased risk of IBD are cigarette smoking and an increased risk of CD, and cessation of cigarette smoking and an increased risk of UC.²² However, there are a range of other environmental exposures associated with colonisation and subsequent urbanisation that may impact the microbiome and its subsequent interaction with the mucosal immune system, increasing the risk of IBD. These include high saturated fat,

high refined sugar, low fibre diet, antibiotic use, and markers of hygiene (such as less crowded living conditions, more bathroom amenities and reduced *Helicobacter pylori* and other infections).

There is an ethnic gradient in daily smoking prevalence in New Zealand adults, with smoking rates almost three times higher in Māori, and one and a half times higher in Pacific island peoples than non-Māori/non-Pacific peoples.²³ Yet the rates of both CD and UC are lower in Māori and Pacific Island peoples. Smoking rates are among the highest in young Māori women²³ who were also more likely to develop CD in the Lakes DHB cohort. However, smoking rates in female patients from Lakes DHB were no higher in Māori than non-Māori. The observation of relatively low CD rates in a population with high daily smoking prevalence remains unexplained, but suggests that other environmental factors may be more important risk factors than smoking in Māori and Pacific Island people.

Worldwide, there are many studies of populations migrating from regions of low IBD prevalence to high IBD prevalence. Usually these studies have focussed on non-Caucasian populations moving to Western countries, such as migrants from the Indian subcontinent moving to cities in the United Kingdom²⁴ or Canada.²⁵ Invariably, slightly increased rates of IBD (compared to the regions that they had migrated from) are seen in the migrants, with significantly higher rates of IBD (sometimes surpassing those of non-migrants) seen in subsequent generations.

Intercontinental migration involves a sudden change of environment which may impact a wide range of environmental exposures leading to changes in risk of disease. Similar, albeit slower, changes can also occur with urbanisation in a population within a country or over time, leading to changes in diet, hygiene, antibiotic use and infection rates. Family size also often reduces with urbanisation as seen in fertility rates for Māori reducing from 6.2 in 1962 to 2.2 in 1986.²⁶ For Māori, colonisation, land confiscation and the increased job opportunity perpetuated urbanisation, which has been dramatic since 1946 when 26% of Māori lived in towns or cities²⁶ compared to 82% in 2018.²⁷ For non-Māori, urbanisation occurred earlier which was associated with an earlier increase in the incidence of IBD.

Changes in dietary habit prompted by colonisation and urbanisation may be another explanation for the increased IBD incidence in Māori. With increasing availability and accessibility of

packaged food, the unaffordability of healthy meals with inequitable distribution of wealth, and high levels of poverty among Māori households, a traditional diet rich in fresh vegetables, fruits and meat has transitioned to a diet high in ultra-processed food containing additives, artificial colours and flavourings, or other chemical ingredients.²⁸ Recently, a strong positive correlation was identified between consumption of ultra-processed food and IBD risk by a large, prospective cohort study that reviewed 116,087 participants from 21 countries.³⁰ A clear dose-dependent relationship was described for UC, with consumption of less than one serving of ultra-processed food per day as a reference standard, the relative risk (RR) for daily consumption of 1–4 servings was 2.93, and 4.90 for 5 or more servings. These findings were corroborated by a systematic review and meta-analysis involving 54,580 participants from nine studies, which concluded that western dietary pattern doubled the risk of developing IBD (RR 1.92), especially UC (RR 2.15).³¹ The pathophysiological mechanism between ultra-processed food and IBD remains elusive, yet some suggest it could be related to a poor nutrient profile and resultant gut microflora dysbiosis, which triggers an excessive inflammatory response.²⁹ Studies of Thai migrants to the United States have demonstrated that major changes in diet (reduction in fibre and increases in saturated fat, protein and sugar consumption) are associated with significant reductions in gut microbial diversity and a pro-inflammatory phenotype.³²

The 2002 National Children's Nutrition Survey demonstrates that, on average, Māori children consume higher amounts of ultra-processed foods than NZ European/Others and, with estimates of child poverty at 23.3% leading to food unaffordability, consumption is unlikely to have improved.³³ However, this has not translated into higher rates of IBD than NZ European/Others at present. It is possible that the effects of environmental risk factors have been mitigated in Māori for unknown reasons, although the rapid increase

in Māori IBD incidence over the last five years reported at Lakes DHB suggests that IBD may continue to become more prevalent in Māori.

Markers of hygiene have been associated with an increased risk of developing IBD, particularly when the overall prevalence in a population is low. The hygiene hypothesis, states that exposure to enteric pathogens in early childhood may be beneficial in the establishment of healthy and diverse gut microbiota, which reduces risks of inappropriate immune response later in life.³⁴ *H. pylori* infection may be a sign of increased childhood exposure to enteric pathogens and subsequently a more diverse gut microbiome. Māori have traditionally had higher rates of *H. pylori* infection than non-Māori. Both have reduced dramatically since *H. pylori* was associated with upper gastrointestinal ulceration and cancer through testing and antibiotic eradication.³⁵ Although the absence of *H. pylori* infection is weakly associated with IBD, it is likely that the increased use of antibiotics, increasing sanitation and other markers of hygiene may be contributing to increasing IBD incidence in Māori.

Conclusion

We have reported the highest rates of Māori with IBD ever described. We have also shown a dramatic increase in IBD incidence for Māori over the last five years. Although the rates are still less than non-Māori, colonisation, with increased urbanisation and changes in diet and hygiene, suggests that IBD rates may continue to increase in Māori. Prospective studies should be undertaken to understand changes in disease incidence to ensure that appropriate healthcare resources are allocated to diagnose and treat IBD early, alongside reviewing whether our current IBD pathways take into account Māori perspectives and ideologies. Furthermore, increasing rates of IBD in Māori may provide an opportunity to understand the unique impact of the environment as a risk factor for IBD development.

COMPETING INTERESTS

Nil.

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