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Ethnic differences in cardiovascular risk profiles among 475,241 adults in primary care in Aotearoa, New Zealand

Vanessa Selak, Katrina Poppe, Corina Grey, Suneela Mehta, Julie Winter-Smith, Rod Jackson, Sue Wells, Daniel Exeter, Andrew Kerr, Tania Riddell, Matire Harwood

The aim of this study was to describe CVD risk profiles by ethnicity using data collected in primary care clinics between 2004 and 2016. 475,241 people (43% women) were included of whom 14% percent identified as Māori, 13% Pacific, 8% Indian, 10% Other Asian and 55% European. Māori and Pacific people experience the most significant inequities in exposure to CVD risk factors compared with other ethnic groups. Indian people have a high prevalence of diabetes and coronary heart disease. We recommend strong political commitment and cross-sectoral action to implement interventions that address these inequities.

Estimated inequities in COVID-19 infection fatality rates by ethnicity for Aotearoa New Zealand

Nicholas Steyn, Rachelle N Binny, Kate Hannah, Shaun C Hendy, Alex James, Tahu Kukutai, Audrey Lustig, Melissa McLeod, Michael J Plank, Kannan Ridings, Andrew Sporle

We estimate the risk of fatality from COVID-19 for different ethnicities in New Zealand. We find that Māori have at least a 50% higher risk of fatality than non-Māori. Pacific people may also be at a higher risk of fatality. These results show the need for good data collection systems and measures that pro-tect at-risk groups, communities and regions.

Inequity in one-year mortality after first myocardial infarction in Māori and Pacific patients: how much is associated with differences in modifiable clinical risk factors? (ANZACS-QI 49)

Janine Mazengarb, Corina Grey, Mildred Lee, Katrina Poppe, Suneela Mehta, Matire Harwood, Wil Harrison, Nicki Earle, Rod Jackson, Andrew Kerr

We studied the reasons for differences in survival in people from different ethnic groups experiencing their first heart attack in New Zealand. We found that mortality in the first year was nearly three times higher for Pacific and Māori people than European people of similar age and sex. At least half of these worse outcomes for Māori, and three-quarters for Pacific people, were found to be related to differences in potentially preventable or modifiable clinical factors present at, or prior to, the heart attack. It follows that much of the poorer survival in Māori and Pacific people relative to European people could be reduced by improvements in prevention in the community (eg, diet, exercise, stopping smoking), in primary (General practice) care (eg, blood pressure, cholesterol and diabetes control) and at the community-to-hospital interface.

Racism and health in Aotearoa New Zealand: a systematic review of quantitative studies

Natalie Talamaivao, Ricci Harris, Donna Cormack, Sarah-Jane Paine, Paula King

This paper reviews the body of quantitative research in New Zealand that examines self-reported experiences of racial discrimination and associations with a range of health outcomes (eg, mental health, physical health, self-rated health, wellbeing, individual level health risks and healthcare indicators). The review found 24 studies that report associations between health outcomes and experience of racial discrimination—finding that experience of racism is linked with poorer health outcomes. Indigenous and minoritised ethnic groups are more likely to experience racial discrimination and therefore are disproportionately affected by the impacts of racial discrimination on health outcomes. There is an urgent need to identify and implement policy initiatives and interventions to address the negative impact of racism on health.

Inequalities between Māori and non-Māori men with prostate cancer in Aotearoa New Zealand

Richard Egan, Jacquie Kidd, Ross Lawrenson, Shemana Cassim, Stella Black, Rawiri Blundell, Jerram Bateman, John Broughton

Māori experience poorer health statistics in terms of cancer incidence and mortality compared to non-Māori. For prostate cancer, Māori men are less likely than non-Māori men to be diagnosed with prostate cancer, but those that are diagnosed are much more likely to die of the disease than non-Māori men resulting in an excess mortality rate in Māori men compared with non-Māori. A review of the literature included a review of the epidemiology of prostate cancer; of screening; of access to healthcare and of treatment modalities. Our conclusion was that there are a number of reasons for the disparity in outcomes for Māori including differences in staging and characteristics at diagnosis; differences in screening and treatment offered to Māori men; and general barriers to healthcare that exist for Māori men in New Zealand. We conclude that there is a need for more culturally appropriate care to be available to Māori men.

The most commonly diagnosed and most common causes of cancer death for Māori New Zealanders

Jason K Gurney, Bridget Robson, Jonathan Koea, Nina Scott, James Stanley, Diana Sarfati

Cancer is an important cause of health burden and death for Māori, with a quarter of all Māori deaths attributable to this disease. There are also unfair differences between Māori and non-Māori New Zealanders in terms of who gets cancer, who dies from cancer and who survives it. In this paper we show the most important cancers for Māori—the top-10 most commonly diagnosed, the top-10 most common causes of cancer death, and how survival differs between Māori and non-Māori for these cancers. We finish by talking about the things that need to happen to reduce the cancer burden for Māori.

Acknowledging and acting on racism in the health sector in Aotearoa New Zealand

Vanessa Selak, Jamie-Lee Rahiri, Rod Jackson, Matire Harwood

"I think New Zealand is the best place on the planet, but it's a racist place."

Taika Waititi (9 April, 2018)[1]

"I grew up believing that New Zealand was a country with limitless opportunities. Success was there for those who worked hard, and was therefore deserved by those who achieved it. And if certain groups in New Zealand weren't achieving, it was most likely because they weren't working hard enough. After all, my parents had achieved financial success in my lifetime despite arriving in New Zealand without any money or the ability to speak or write English. This belief persisted and was reinforced as I succeeded academically at school before earning and, I thought, deserving, a place in medical school. Once I got to medical school, this belief extended to health. After all, so many of the conditions I was learning about were caused, and/or significantly exacerbated, by modifiable 'lifestyle factors'. Throughout this time, I believed myself to be a good person. My aim in medicine was to 'help people' and I considered myself to be 'colour blind': I would treat everyone the same, irrespective of their ethnicity. But if they chose not to attend or follow my instructions, that was their choice and outside of my responsibility as a clinician. It wasn't until I embarked upon training in public health medicine that I began to realise (and am continuing to realise) how mistaken I have been about many of my core beliefs, and about how, through my ignorance, I have contributed to racism in New Zealand."

Vanessa Selak (personal reflection, 9 August 2020)

The purpose of our editorial is twofold. First we will highlight some of the false beliefs that persist, and contribute to, ongoing racism within the health sector in Aotearoa New Zealand. Such racism, albeit often unconscious, has been identified in recent studies of New Zealand medical students.[2,3] We will use examples of false beliefs we have encountered through the academic peer review process, as Māori (MH) and Pākehā (VS, RJ) researchers exploring and addressing differences in cardiovascular disease (CVD) risk factors and outcomes by ethnicity. This work builds on a paper by Reid, Robson and Jones, that explored and debunked common myths regarding disparities in health 20 years ago,[4] and draws on the excellent articles highlighting ethnic inequities in this issue of the *NZMJ*. Second we recommend some appropriate ways for the *NZMJ* and Pākehā health professionals/researchers to contribute to research and peer review that will support culturally safe research and equitable outcomes for Māori and other groups experiencing inequities.

Before we begin, it is important to define racism, and understand how it affects health. Racism is often thought of as the belief that certain races of people are by birth, or nature, superior to others. Professor Camara Jones' definition provides a much more useful definition of the complex nature of racism, conceptualising three types of racism: (1) institutionalised ("*differential access to the goods, services and opportunities of society by race*"), (2) interpersonal ("*prejudice and discrimination*", which can be intentional or unintentional) and (3) internalised ("*acceptance by members of the stigmatised races of negative messages about their own abilities and intrinsic worth*").[5] Jones highlights the importance of using such a framework to help to understand the reasons for differences in health outcomes by race, rather than simply adjusting for or ignoring these, and to thereby inform appropriate action to address the differences.[5] She notes that "*Ignoring the etiologic clues embedded in group differences impedes the advance of*

scientific knowledge, limits efforts at primary prevention and perpetuates ideas of biologically determined differences between the races." Action to address racism in the health sector should consider the three main pathways by which racism affects health: (1) differential access to the determinants of health or exposures (which leads to differences in disease incidence), (2) differential access to healthcare and (3) differences in the quality of care received.[6] Reid and Robson, and a more recent publication by the Health Quality and Safety Commission (HQSC), have provided evidence of each of these pathways operating in the New Zealand health system.[6,7]

In this issue of the *NZMJ*, Talamaivao and colleagues report their findings of a systematic review that investigated the quantitative association between experiencing racism and health in New Zealand.[8] Consistent with international literature, the reviewers found that experiencing racism is associated with worse primary healthcare experience, lower healthcare utilisation and poorer health (particularly mental health) outcomes in studies that were predominately cross-sectional. The reviewers conclude that, because racism is experienced more frequently by Māori, Asian and Pacific groups than Europeans, these groups are disproportionately affected by the effects of racism on health outcomes. The reviewers note that there is a need for research into the longitudinal effects of racism on health outcomes as well interventions to combat racism and its adverse health consequences.

However, there is also a need for a healthy and safe 'article review' process in order for such research to be published. Some examples of incorrect and racist beliefs we have encountered through peer review of our research are discussed below.

Māori are immigrants, like other immigrant groups, as there are no Indigenous people of New Zealand

The ancestors of Māori were the first human inhabitants of New Zealand, settling here by 1,300, having travelled here from East Polynesia.[9] They began to identify as tangata māori (meaning the ordinary or usual people) in the 1800s, in part to differentiate themselves from immigrants to New Zealand, who they were referring to as Pākehā by 1815.[10] Māori, as tangata whenua, are the Indigenous people of New Zealand.[11] While there is no internationally adopted definition of Indigenous peoples,[12] the 2007 United Nations Declaration of the Rights of Indigenous Peoples[13] recognises the right of Indigenous peoples to self-determination, including their right to self-identify as and be recognised as Indigenous.[11] Māori clearly meet all definitions of Indigenous people provided in an overview by the United Nations.[12] For example, the Martinez Cobo Study has proposed a working definition of "*communities, peoples and nations … which, having a historical continuity with pre-invasion and pre-colonial societies that developed on their territories, consider themselves distinct from other sectors of the societies now prevailing in those territories, or parts of them*".[12] Another definition notes four key factors of importance when defining indigeneity: (1) "*priority in time, with respect to the occupation and use of a specific territory*", (2) "*the voluntary perpetuation of cultural distinctiveness*", (3) "*self-identification … as a distinct collectivity*" and (4) "*an experience of subjugation, marginalisation, dispossession, exclusion or discrimination, whether or not these conditions persist*".[12]

The distinction between Indigenous and other ethnic (immigrant) groups in New Zealand must be acknowledged and honoured.[11] In addition to UN covenants which endorse the right of Māori as tangata whenua to determine their individual and collective identities, Māori status as tangata whenu is affirmed by te Tiriti o Waitangi.[11]

Māori experienced development, not colonisation

Development and colonisation, though related, are different concepts that need to be considered separately. First, in terms of development, while the way in which development occurred was strongly influenced by colonisation, any assumption that without colonisation development would not have occurred in New Zealand is incorrect. Development had occurred in New Zealand prior to the arrival of Europeans, and continued to occur after the arrival of Europeans and prior to colonisation. Had New Zealand not been colonised, Māori would no doubt have continued to trade and share ideas/advances with non-Māori as they had prior to colonisation. Colonisation changed the way in

which development occurred for Māori. Post-colonial theory argues that the "*colonial practices of progressive developmentalism*" contributed to the trauma of colonisation itself by undermining the value and role of Indigenous identity and structures through the development process.[14] According to subalternism, another relevant theory, Indigenous people of a colonised state are subalternate because they are "*politically, economically and socially excluded from the power structure*".[14] Through subalternatism the process of colonisation is seen to cause Indigenous people to "*shift from a state of self-reliance and autonomous personal dignity to dependency and humiliation*".[14] Both theories help to explain how the trauma of colonisation is perpetuated and reinforced as Indigenous peoples "*remain subalternate in the state and even attempts by the state to change this status through development only serve to reinforce this power inequality, by reinforcing the idea that Indigenous people need 'developing'*".[14]

Colonisation was, and continues to be, a traumatic experience for Māori.[6,14–16] This is fundamentally because the process of colonisation results in a forced relocation of power and resources from Indigenous people to the colonisers who, however well intentioned, construct new systems according to their own, not Indigenous, values, and these new systems ultimately redistribute power and resources to the advantage of colonisers.[6] Despite the assertion that such "*new systems provide equal opportunity for all participants*" they cannot, and clearly do not, because they are imbued with the values of the colonisers: "*they promote new ideas about who is normal (and therefore who is not); who is knowing and who is ignorant; who is civilised and who is barbaric; who is deserving and who is undeserving; and who is good and who is bad*".[6] The process of colonisation has therefore resulted in moving Māori, the tangata whenua, from being normal, to being seen by Pākehā as different and classified as outsiders.[6] Further, Māori are then framed as being to blame for their own inferior health outcomes compared with Pākehā, without acknowledgement of the structural bias that is inherent in our health system because it is designed to advantage Pākehā over Māori.[6]

Health differences between Māori and non-Māori are inequalities, not inequities

The New Zealand Ministry of Health definition of equity is "*In Aotearoa New Zealand, people have differences in health that are not only avoidable but unfair and unjust. Equity recognises different people with different levels of advantage require different approaches and resources to get equitable health outcomes*."[17] This definition has been informed by Te Tiriti o Waitangi obligations (which go "*beyond just remedying disadvantage and reducing inequities, enabling Māori to flourish and lead their aspirations for health*"), as well as international literature on equity.[18] The World Health Organization (WHO) defines equity as "*the absence of avoidable or remediable differences among groups of people, whether those groups are defined socially, economically, demographically or geographically*".[19] The purpose of the Ministry of Health developing and providing their definition of equity was to enhance the coordination and hence effectiveness of action to achieve equity in health across the health and disability sector and other government agencies that address the broader socioeconomic determinants of health in New Zealand.[18]

WHO notes that health inequities "*involve more than inequality with respect to health determinants, access to the resources needed to improve and maintain health or health outcomes. They also entail a failure to avoid or overcome inequalities that infringe on fairness and human rights norms*".[19] WHO further notes that "*reducing health inequities is important because health is a fundamental human right and its progressive realisation will eliminate inequalities that result from differences in health status (such as disease or disability) in the opportunity to enjoy life and pursue one's life plans*".[19]

In addition to meeting international legal obligations, addressing inequities between Māori and non-Māori is required to meet New Zealand government obligations under te Tiriti o Waitangi and health sector obligations under the New Zealand Public Health and Disability Act 2000.[7] The ongoing inequities experienced by Māori in their health outcomes is the subject of the Waitangi

Tribunal Health Services and Outcomes Inquiry (Wai 2575), which was initiated in November 2016.

In their viewpoint article in this issue of the *NZMJ*, Gurney and colleagues note that there is considerable evidence of enduring inequities between Māori and non-Māori in cancer incidence and mortality, and that cancer is an important contributor to the life expectancy gap between Māori and non-Māori.[20] In addition to data on the most commonly diagnosed cancers and causes of cancer death among Māori between 2007 and 2016, Gurney et al found higher morbidity and mortality from most of these cancers for Māori compared with non-Māori. They note that, reflecting on the commitment from central Government to close the cancer gap for Māori, the country's new Cancer Action Plan 2019–2029 is focused on achieving equitable cancer outcomes for all New Zealanders by being equity-led and achieving equity by design.

Behavioural risk factors wholly reflect individual choice

Behavioural risk factors are strongly influenced by broad contextual factors, often referred to as the social determinants of health or the causes of the causes. The importance of considering social determinants of health when determining how to intervene effectively to support equitable health outcomes has been extensively investigated internationally over many years by many authors, most notably Professor Michael Marmot[21] and including a very well articulated Position Statement on Health Equity approved by the New Zealand Medical Association in 2011.[22] A lack of understanding of the effect of social determinants of health on health behaviours may lead to healthcare professionals inappropriately blaming individuals for their health behaviours, rather than seeing that behaviour as the consequence of their wider social context and therefore considering what strategies might genuinely assist and support that individual to address the behaviour.

Institutional racism is an opinion, not a fact, in the New Zealand health sector

The fact that there are large and enduring differences between Māori and non-Māori in most health outcomes is evidence of the fact that institutional racism occurs in New Zealand. A recent publication by the HQSC notes: "*The Aotearoa New Zealand health system has generated and continues to reinforce inequities in health outcomes between Māori and non-Māori.*"[7] The HQSC explains that "*institutional racism is a systemic pathway to inequity. It occurs and continues because people at all levels of the system make decisions that disadvantage one group in relation to another*" and that "*such racism encompasses both action and inaction*".[7]

In considering how cancer inequities between Māori and non-Māori should be addressed, Gurney and colleagues note that these inequities are "*driven by disparities in the social determinants of good health, determinants that are structural in nature and not controlled by Māori*".[20] Gurney and colleagues label the inequities as a systems-level problem, that require a system-level solution. They further note that "*there is compelling evidence that Māori have poorer access to timely best-practice treatment compared to non-Māori*" and that therefore "*cancer care services have an important role to play in reducing the cancer burden for Māori*".[20] And finally, noting that the International Agency for Research on Cancer has identified the following key drivers of equitable access to cancer care as the three A's (availability, affordability, acceptability), Gurney and colleagues state "*we can identify our own system within these three A's, and how these are likely to be contributing to poorer cancer outcomes for Māori*".[20]

Egan and colleagues present their narrative review of disparities between Māori and non-Māori men in prostate cancer in New Zealand in this issue of the *NZMJ*.[23] They found that despite being less likely to be diagnosed with prostate cancer, Māori men are more likely than non-Māori men to die from prostate cancer. They note that addressing this inequity for Māori will require a number of systems solutions, including addressing social determinants of health (especially economic disparity), racism within health services and the development of culturally appropriate models of healthcare delivery for Māori.

Institutional racism is also likely to be important in addressing novel health challenges such as COVID-19. Steyn

and colleagues estimated that the likely COVID-19 infection fatality rates for Māori would be up to 2.5 times higher and for Pacific would be almost double that for Europeans.[24] They note that actual inequities could be even higher because, compared with Europeans, Māori and Pacific people are more likely to experience multimorbidity, avoidable hospitalisation ("*reflecting broader and more complex structural disadvantage*"), and "*widely reported racism within the healthcare system*".[24]

Recommendations

In order to support culturally safe research and equitable outcomes for Māori, and other groups experiencing inequities, we make the following recommendations.

New Zealand Medical Journal (NZMJ)

We note that the *NZMJ* is the official journal of the New Zealand Medical Association and New Zealand's "*leading online scientific information journal for medical practitioners and health professionals*" (http://www.medconnect.co.nz)". The NZMA published an excellent Position Statement on Health Equity nearly a decade ago[22] and "*welcomes the Waitangi Tribunal's Report on stage one of the Health Services and Outcomes Kaupapa Inquiry*".[25] The NZMA notes that "*To achieve that necessary equity NZMA supports the two broad recommendations made in the report – namely amendments to the New Zealand Public Health and Disability Act to include a Treaty of Waitangi clause and enshrining in that Act the Crown's and sector's commitment to achievement of equitable outcomes for Māori*".[25] We consider that in order to support these objectives of the NZMA, the pervasiveness of racism within the health sector and to fully realise the role of the *NZMJ* in Aotearoa New Zealand, the NZMJ should:

1. Assign a Māori co-editor
2. Develop a code of practice on racism in their peer review process
3. Commit to an open peer review process so that peer reviewers are no longer anonymous.

Pākehā health professionals and health researchers

In addition to drawing on the experiences and expertise of our numerous Māori and Pacific colleagues, we recommend the following, based on the advice of Randy Vince[26] in his recent article in *JAMA*:

1. Review and understand the history of race and racism within this country
2. Undertake and mandate antiracism/implicit bias training (eg, Project Implicit https://implicit.harvard.edu/implicit/)
3. Do not accept differences in health outcomes on the basis of ethnicity because most of these differences are avoidable and unjust (ie, they are inequities not inequalities)
4. Support and encourage the development of our Māori and Pacific colleagues throughout their careers as we need health services to be designed, delivered and researched by Māori and Pacific to ensure that the needs of Māori and Pacific people are optimally addressed and equity is achieved.
5. Undertake and facilitate the implementation of culturally aware mentorship training for all health professionals and researchers, to ensure that we all have the opportunity to reflect on our identities and, *"using the thoughts from this reflection to examine (our) biases toward people from other cultural identities"*.[26]

Editor's note

The NZMA publishes the *NZMJ*, however the *NZMJ* has full editorial independence via its Editor-in-Chief.

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Nil.

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Ethnic differences in cardiovascular risk profiles among 475,241 adults in primary care in Aotearoa, New Zealand

Vanessa Selak, Katrina Poppe, Corina Grey, Suneela Mehta, Julie Winter-Smith, Rod Jackson, Sue Wells, Daniel Exeter, Andrew Kerr, Tania Riddell, Matire Harwood

ABSTRACT

AIM: In Aotearoa, New Zealand, cardiovascular disease (CVD) burden is greatest among Indigenous Māori, Pacific and Indian people. The aim of this study was to describe CVD risk profiles by ethnicity.

METHODS: We conducted a cross-sectional analysis of a cohort of people aged 35–74 years who had a CVD risk assessment in primary care between 2004 and 2016. Primary care data were supplemented with linked data from regional/national databases. Comparisons between ethnic groups were made using age-adjusted summaries of continuous or categorical data.

RESULTS: 475,241 people (43% women) were included. Fourteen percent were Māori, 13% Pacific, 8% Indian, 10% Other Asian and 55% European. Māori and Pacific people had a much higher prevalence of smoking, obesity, heart failure, atrial fibrillation and prior CVD compared with other ethnic groups. Pacific and Indian peoples, and to a lesser extent Māori and Other Asian people, had markedly elevated diabetes prevalence compared with Europeans. Indian men had the highest prevalence of prior coronary heart disease.

CONCLUSIONS: Māori and Pacific people experience the most significant inequities in exposure to CVD risk factors compared with other ethnic groups. Indians have a high prevalence of diabetes and coronary heart disease. Strong political commitment and cross-sectoral action to implement effective interventions are urgently needed.

Cardiovascular diseases (including diabetes) account for 17% of health loss among people living in Aotearoa, New Zealand.[1] There have been considerable reductions in the incidence and mortality of cardiovascular disease (CVD) in New Zealand over the past 20 years through good prevention and access to treatment.[1–3] For ischaemic heart disease (IHD) and stroke, hospitalisation and mortality rates have fallen in all demographic 'groupings' including gender and ethnicity, with a smaller decline in IHD rates in Pacific peoples than other ethnicities[6] and a larger decline in stroke rates for Indian people than other ethnic groups (unpublished). However, the burden of CVD is unevenly distributed across New Zealand by ethnicity, with Māori, Pacific and Indian people experiencing a greater burden of this condition than other groups.[4–8] This increased burden of CVD is one of the main drivers behind the 6–7 year lower life expectancy experienced by Māori and Pacific people at birth compared with other groups in this country.[4,5] There is a need for continued focus on CVD given the magnitude of health loss associated with CVD and the potential avoidability of this burden through prevention and treatment.[1]

The Ministry of Health states that "In Aotearoa New Zealand, people have differences in health that are not only avoidable but unfair and unjust. Equity recognises different people with different levels of advantage require different approaches and resources to get equitable health outcomes."[9] More research to understand CVD disparities, including the contribution of inequities[9] in healthcare access and quality, is required to develop and implement effective equity-promoting interventions and ultimately reduce CVD burden across ethnic groups. Although self-reported data on CVD risk factor levels by ethnicity are available through national surveys, the numbers in non-European ethnic groups are generally small, and the availability of data from primary care is limited. The PREDICT-CVD cohort is a very large, contemporary, representative and ethnically diverse cohort recruited at the time of CVD risk assessment in routine primary care practice in New Zealand.[10] The aim of this study was to describe and compare the baseline CVD risk profiles of people in the PREDICT-CVD cohort by ethnicity. The study does not directly document access to, or quality of, CVD care.

Methods

Design, setting and entry

This was a cross sectional study. People were entered into the cohort the first time their CVD risk was assessed by their primary care clinician (physician or nurse) entering data into PREDICT, a web-based decision support programme integrated with electronic primary care practice management systems in New Zealand.[10] The programme enables available clinical data in the electronic medical record to auto-populate fields, and the data template has a number of compulsory fields and built-in range and validity checks at the point of data entry. These factors facilitated accurate and nearly complete (>99%) data collection for variables required in the CVD risk prediction equation used in New Zealand at the time.[10] This equation was based on a Framingham risk equation with adjustments for groups whose CVD risk may be underestimated by that equation (eg, family history of premature CVD; Māori, Pacific or Indian ethnicity).[11]

Since 2003, New Zealand CVD risk management guidelines have recommended that men aged over 45 years and women aged over 55 years (or 10 years earlier for subpopulations at increased risk: those of Māori, Pacific or Indian ethnicity and individuals with known CVD risk factors) have a regular CVD risk assessment.[11] Whether a person visiting the primary care clinic is risk assessed, and therefore whether they enter the cohort, is at the discretion of the primary care clinician. Most primary care physicians receive alerts through their electronic practice management system (PMS) advising them of individual patient eligibility for CVD risk assessment.[10]

Data up to 2015 indicate that approximately 90% of people eligible for CVD risk assessment (according to national guidelines[12]) in practices using the PREDICT programme had their CVD risk assessed using this software.[10] National coverage data show Māori, Pacific and Indian ethnicities had slower increases in coverage compared to other ethnicities and at the end of 2015 there was a reported 4% gap in coverage between Māori and non-Māori.[13] This programme is implemented in approximately 35% of New Zealand primary care practices, which serve approximately 1.6 million people (around 35% of the New Zealand resident population).[10] The practices include all Northland primary care practices, approximately 80% of all practices in the Auckland region and some further practices in both the North and South Islands. The decision to participate in PREDICT was made at the PHO level, not the individual practice level, so the probability of selection biases at the practice level is low. The practices participating in PREDICT include large rural and urban areas and include the largest Māori, Pacific, Indian, Chinese and other Asian populations in New Zealand. It is unlikely that significant population subgroups are not represented in this study. The only exclusion criterion for the PREDICT programme is current pregnancy, and no decision support is given for those under 18 years of age.[10]

Study entry occurred between 20 October 2004 and 31 December 2016.

Participants and exclusion criteria

All people who received CVD risk assessment in primary care using the PREDICT programme were considered for inclusion in this study. People were excluded from this analysis if they were outside an age group in which CVD risk assessment is recommended (ie, aged less than 35 years or 75 years or more).

Data sources and linkage

Data on cardiovascular risk factors (including age, sex, smoking status, diabetes status, blood pressure, body mass index [BMI] and cholesterol levels) and medical history (including CVD and atrial fibrillation [AF]) were obtained during CVD risk assessment in primary care. These data were automatically stored both in the PMS and anonymously on a central database. With the permission of clinicians, the central database risk profile was regularly linked to an encrypted National Health Index number, a unique personal identifier which was used to anonymously link individual risk profiles to national and regional health databases.

National health databases were used to obtain or update participant data on demography (age, sex, ethnicity and socioeconomic deprivation),[14] publicly funded hospitalisations (from 1988 onwards),[15] and subsidised pharmaceutical dispensing (from 2005 onwards).[16]

Ethnicity was self-reported within the PMS and triangulated with PHO enrolment and hospitalisation databases. For those in whom more than one ethnic group was recorded, a prioritisation output method was used to assign each individual to one ethnic group. This prioritisation method was modified from that outlined in national ethnicity data protocols and prioritised groups in the following order: Māori > Pacific > Indian (including Fijian Indian) > Other Asian (including Chinese) > European > Middle Eastern/Latin American/African (MELAA) > Other > Unknown.[17] National ethnicity data protocols in use during the study period enabled identification of Indians (who comprise 90% of South Asians in New Zealand) but non-Indian South Asians were unable to be differentiated from the rest of the Other Asian group. People whose ethnicity was MELAA, Other and Unknown were excluded because of the small numbers (<1,000) within each group.

The definitions of risk factors are provided in the Appendix.

Statistical methods

Continuous variables were summarised as means with standard deviations, and categorical data as frequencies and percentages. Summaries for continuous and categorical data were age-adjusted (unless otherwise specified) using the WHO world standard population.[18] Potential differences between ethnic groups in means or proportions were tested using ANOVA or the chi-squared test, respectively, with the level of statistical significance set at p=0.05. Data were extracted using R version 3.5.0 (http://cran.r-project.org/), age-adjusted summaries were obtained using Microsoft Excel Version 2016, the chi-squared test was calculated using the online calculator Social Science Statistics[19] and the ANOVA test was calculated using the online calculator at http://statpages.info/anova1sm.html.

Ethics approval

The PREDICT study (under which this research was conducted) was approved by the Northern Region Ethics Committee Y in 2003 (AKY/03/12/314) with subsequent annual approval by the National Multi Region Ethics Committee since 2007 (MEC07/19/EXP). Participant informed consent was not obtained, consistent with a waiver granted by the Ethics Committee, as the study involved secondary use of patient data that was anonymised prior to being received by the research team.

Results

A total of 206,508 women (mean age 57 years, SD 8.7 years, Table 1) and 268,733 men (mean age 53 years, SD 10.1 years, Table 2) were risk assessed between 20 October 2004 and 31 December 2016. The cohort comprised Māori (14%), Pacific (13%), Indian (8%), other Asian (10%) and European (55%) people. Mean age among Europeans was higher than that for Māori, Pacific and Indian women and men, due to age-sex-ethnicity differences in national risk assessment criteria. Māori and Pacific women (46%, 57%) and men (44%, 58%) were much more likely to be living in areas

Table 1: Cohort description by ethnicity using age-standardised values, women.

	Māori	Pacific	Indian	Other Asian	European	Total	p-value*
Number (% of all women)	30,012 (15)	27,026 (13)	16,329 (8)	22,045 (11)	111,096 (54)	206,508	
Age, years, mean (SD)	53 (8.4)	52 (8.8)	53 (8.7)	57 (7.8)	60 (7.9)	57 (8.7)	<0.001
NZ Deprivation index, 5th quintile, %	46	57	22	15	11	21	<0.001
Smoking, %							<0.001
Current	39	16	1	3	14	15	
Ex-smoker	22	11	1	3	16	12	
Never	39	73	98	94	70	73	
BMI, kg/m^2, %							<0.001
Obese (30+)	53	72	27	10	30	39	
Overweight (25–29.9)	21	14	35	26	25	23	
Normal (18.5–24.9)	13	5	23	47	24	21	
Underweight (<18.5)	1	0	1	2	1	1	
Missing	12	9	14	16	20	16	
Systolic blood pressure, mm Hg, mean (SD)	130 (17.3)	129 (16.3)	125 (15.4)	123 (15.7)	127 (15.2)	127 (16.0)	<0.001
Diastolic blood pressure, mm Hg, mean (SD)	81 (10.5)	80 (10.1)	77 (9.0)	76 (9.5)	78 (9.1)	82 (9.7)	<0.001
Total:HDL cholesterol, mean (SD)	4.1 (1.23)	3.9 (1.09)	4.0 (1.05)	3.7 (1.02)	3.8 (1.18)	3.9 (1.14)	<0.001
Diabetes, %	23	38	33	21	12	17	<0.001
Atrial fibrillation, %	3.7	2.4	0.7	0.7	1.3	1.6	<0.001
Heart failure, %	5.4	4.2	2.0	0.4	1.7	2.3	<0.001
Haemorrhagic stroke, %	0.6	0.4	0.2	0.2	0.3	0.3	0.508
CVD, %							
Any type	8.1	5.6	3.9	1.6	3.5	3.9	<0.001
Coronary heart disease (including procedures)	5.6	3.4	3.0	1.0	2.3	2.6	<0.001
Cerebrovascular disease	2.6	2.0	0.8	0.6	1.0	1.2	<0.001
Peripheral vascular disease (including procedures)	1.6	0.9	0.4	0.1	0.7	0.7	0.001

All values age-standardised (except for Number (%)) using the WHO world standard population.[17]
Fewer than 1% of values missing unless otherwise specified.
*p for difference between ethnic groups.
SD=standard deviation.

of the highest quintile of socioeconomic deprivation than Indians (22%) and Europeans (11%).

Smoking was most common among Māori women (39%) and Māori and Pacific men (35% and 27%, respectively), and least common among Indian people (1% women, 10% men). Obesity was most common among Pacific (72% women, 62% men) and Māori (53% women, 50% men) and least common among Other Asian people (10% women, 9% men). Mean blood pressure (BP) was highest for Māori (130/81 [SD 17.3/10.5] mmHg in women, 131/82 [15.7/10.2] mmHg in men) and lowest for Other Asian people (123/76 [15.7/9.5] mmHg in women, 125/79 [14.4/9.4] mmHg in men). Similarly, the mean ratio of total cholesterol to high density lipoprotein cholesterol (TC:HDL) among women was highest in Māori (4.1

Table 2: Cohort description by ethnicity using age-standardised values, men.

	Māori	Pacific	Indian	Other Asian	European	Total	p-value*
Number (% of all men)	34,187 (13)	33,425 (12)	23,909 (9)	25,278 (9)	151,934 (57)	268,733	
Age, years, mean (SD)	49 (9.8)	48 (10.1)	47 (10.5)	53 (9.3)	56 (9.1)	53 (10.1)	<0.001
NZ Deprivation index, 5th quintile, %	44	58	23	14	11	20	<0.001
Smoking, %							<0.001
Current	35	27	10	15	15	16	
Ex-smoker	21	16	8	15	19	14	
Never	44	57	82	70	66	70	
BMI, kg/m², %							<0.001
Obese (30+)	50	62	15	9	27	32	
Overweight (25–29.9)	27	22	41	37	37	33	
Normal (18.5–24.9)	9	5	30	38	16	17	
Underweight (<18.5)	0	0	1	1	0	0	
Missing	14	10	13	16	19	17	
Systolic blood pressure, mm Hg, mean (SD)	131 (15.7)	129 (14.9)	127 (14.4)	125 (14.4)	129 (14.0)	128 (14.5)	<0.001
Diastolic blood pressure, mm Hg, mean (SD)	82 (10.2)	81 (9.9)	79 (9.1)	79 (9.4)	80 (8.9)	80 (9.4)	<0.001
Total:HDL cholesterol, mean (SD)	4.5 (1.34)	4.4 (1.26)	4.5 (1.20)	4.3 (1.13)	4.4 (1.27)	4.4 (1.25)	<0.001
Diabetes, %	18	23	24	15	9	11	0.036
Atrial fibrillation, %	5.8	3.2	1.1	1.1	2.9	2.7	<0.001
Heart failure, %	6.1	4.3	2.1	0.6	1.5	2.1	<0.001
Haemorrhagic stroke, %	0.5	0.6	0.2	0.3	0.2	0.3	0.451
CVD, %							
Any type	9.5	7.6	8.7	2.8	6.4	5.9	<0.001
Coronary heart disease (including procedures)	7.2	5.7	7.8	2.1	4.9	4.6	<0.001
Cerebrovascular disease	2.2	1.9	1.2	0.7	1.3	1.3	0.044
Peripheral vascular disease (including procedures)	1.9	1.2	0.8	0.3	1.1	1.0	0.011

All values age-standardised (except for Number (%)) using the WHO world standard population.[17]
Fewer than 1% of values missing unless otherwise specified.
*p for difference between ethnic groups.
SD=standard deviation.

[1.23]) and lowest for Other Asians (3.7 [1.02]). Among men, mean TC:HDL was also highest among Māori (4.5, SD 1.34), as well as Indian people (4.5, 1.20), and lowest among Other Asian people (4.3, 1.13). Diabetes was most common for Pacific (38% women, 23% men) and Indian people (33% women, 24% men), and elevated for Māori (23% women, 18% men) and Other Asian people (21% women, 15% men) compared to European people (12% women, 9% men). Among all ethnic groups, diabetes was more prevalent in women than men. A history of atrial fibrillation, heart failure and all CVD were most common among Māori (women 3.7%, 5.4% and 8.1%, respectively; men 5.8%, 6.1% and 9.5%, respectively) and generally least common among Other Asian people (women 0.7%, 0.4% and 1.6%, respectively; men 1.1%, 0.6% and 2.8%,

respectively). A similar pattern was evident for prior coronary heart disease among women (ranging from 5.6% for Māori to 1.0% for Other Asian people), whereas Indian men had the highest prevalence of previous coronary heart disease (7.8%) followed by Māori (7.2%), with Other Asian men also having the lowest prevalence (2.1%). Among Indian people, the burden of CVD was much more pronounced among men than women as compared to other ethnic groups.

Discussion

We have demonstrated major differences in CVD risk factors by ethnicity in a large, primary care cohort. Overall, Māori and Pacific people, followed by Indian people, have the greatest burden of CVD and its risk factors in Aotearoa New Zealand. Māori have the highest BP and rates of smoking, atrial fibrillation, heart failure and prior CVD, while Pacific people have the highest rates of obesity and, together with Indian people, the highest prevalence of diabetes. Indian men have the highest rate of previous coronary heart disease of all ethnic groups. In addition to having the greatest burden of CVD and its risk factors, Māori and Pacific people are also much more likely to be living in areas of the highest socioeconomic deprivation than other ethnic groups.

The proportions of the ethnic groups in this study are broadly similar to those from the 2018 national census among adults aged 30–64 years.[20] The main exception to this is that our study had a higher proportion of Pacific people (approximately double) compared with the national Census. This is because the cohort was predominately drawn from the northern region of New Zealand in which the majority (65%) of Pacific people in New Zealand live.[21] Second, national CVD risk assessment guidelines criteria recommend CVD risk assessment a decade earlier for Pacific (as well as for Māori and Indian) people than for other ethnic groups (and for men compared with women), because of their increased burden of CVD.[11] This will have also contributed to the lower mean age of Māori, Pacific and Indian people and the greater number of, and lower mean age of, men compared with women in our cohort.

Strengths of this study were that: data were based on CVD risk assessments undertaken by primary care clinicians and supplemented with information from regional and national databases; the cohort was large, contemporary and ethnically diverse; and less than 1% of data were missing for all variables except BMI, which was unavailable in 19% of people. The cohort is likely to be representative of people in New Zealand in whom CVD risk is recommended as approximately 90% of eligible patients in the study practices were included and about 35% of all primary care practices in New Zealand were included in the study.[11] In the future it should be possible to obtain data required for CVD risk assessment directly from electronic health records for the whole country. This will be feasible because electronic data across primary care are increasingly available and nearly 80% of adults attend their primary care physician in a year,[22] although the level of missing data is likely to be greater than what has been achieved in this prospectively designed cohort study.

Similar overall rates of smoking and obesity among women and men were observed in this study to those from an ongoing national health survey among adults, based on self-report. Diabetes rates were much lower in the national survey than in those observed in our study among women (5.3% vs 17%) and men (6.5% vs 11%). This difference is likely to at least partially reflect the higher proportions of Pacific people in our study (as noted above), the inclusion of younger people (from age 15 years) and lack of age adjustment in the national survey, and under-reporting through self-report in surveys. As with our study, Māori and Pacific people experienced increased levels of all three risk factors (smoking, obesity and diabetes),[22] and Indian people experienced increased levels of diabetes[23] compared with other ethnic groups.

Internationally, inequities in exposure to CVD risk factors and consequent CVD outcomes are evident among Indigenous peoples, similar to the experience of Māori highlighted here. A review of CVD risk factors among Indigenous populations (including from Australia and the US, as well as New Zealand) found that Indigenous peoples experienced a greater

burden of CVD events and risk factors (including smoking, obesity and diabetes) at much younger ages than non-Indigenous groups.[24,25] A call for action to address CVD inequities for Māori in 2004,[26] including the prevention and management of CVD risk factors, has yet to be fully realised. A 2010 review of interventions focused on reducing inequities between Indigenous and non-Indigenous people across a wide range of conditions only identified 19 intervention studies meeting Cochrane Effective Practice and Organization Criteria for methodologically adequate research design[27] and only six of those studies focused on preventing or managing CVD risk factors.

Since that review, the evidence base has improved and interventions have been identified that are likely to improve CVD outcomes for Indigenous peoples (eg, health literacy programmes[28]), reduce inequities in CVD outcomes between ethnic groups (eg, fixed dose combination therapy[29]) or are currently being investigated to determine their likely effect on such inequities (eg, Indigenous health worker support[30]). There is an ongoing need to ensure adequate funding to support more high-quality Indigenous-led research into the effectiveness of interventions that achieve CVD equity for Indigenous Māori.

Similarly, research and implementation of effective interventions for Pacific people is also urgently needed, given their high burden of CVD risk factors, particularly diabetes, and the CVD inequities that are evident. Pacific people have migrated over many generations from island nations that have a special relationship and historical ties to this country. Our research group recently received a three-year research grant to investigate and improve access to CVD care that achieves equity for Māori and Pacific people, confirming the importance of this work. Research questions will address current gap in knowledge such as ethnic differences in first-response care and in drug prescribing proportionate to recommended indications.

The Indian population in New Zealand is predominantly comprised of relatively recent immigrants; the 2013 census indicated that 75% of Indians in New Zealand are overseas born, 90% of whom have been in New Zealand for less than 20 years. Despite the supposed health advantages associated with being migrants (the "healthy migrant effect"[31]), a history of CVD (mostly from coronary heart disease) was common and significantly high diabetes prevalence was observed in keeping with overseas studies among South Asians.[32] The cardiovascular risk profile of Indian people is very different to that of Māori and Pacific people. BP, smoking levels and history of atrial fibrillation and heart failure are not currently increased compared with other ethnic groups in New Zealand, but this may change over time with acculturation. Using standard BMI cut-offs, the total proportion of overweight/obese Indian men was not marked and was somewhat higher among Indian women than the proportions observed among Europeans. However, at a given BMI, compared to European counterparts, Indians have a greater percentage fat mass, greater abdominal subcutaneous fat and greater visceral fat deposition.[32] Together with other South Asian groups, they develop vascular-metabolic diseases at lower BMIs than the traditional thresholds for overweight and obesity. Hence, the American Diabetes Association and NICE in UK both suggest lower BMI thresholds for Indians than those used in this study.[33] An analysis of New Zealand Health Survey data from 2011–2013 found that 56% of South Asian adults were obese and 17% overweight using ethnic-specific lower cut-offs, implying that our results may have under-captured the proportion of Indians (and also Other Asians) that are overweight and obese.[23]

Although guidelines recommend Māori, Pacific and South Asian people have their CVD risk assessed at the same, younger, age than other ethnicities, we found very different risk profiles between these groups, as noted above. Further research to understand the impact of immigration, country of birth and length of stay in New Zealand on the incidence and prevalence of CVD and diabetes for different ethnic populations is recommended, including for non-Indian South Asian peoples who can now be differentiated from the Other Asian group using recent data resources like the Statistics New Zealand Integrated Data Infrastructure. Such research will enable a more tailored response to the health needs of high-risk ethnic groups comprised of large numbers

of migrants and can inform the nature and timing of preventive strategies such as CVD risk assessment, diabetes screening and health promotion.

The reasons for CVD inequities by ethnicity are complex and multifactorial.[34] The differences in socioeconomic status are stark and highlight the importance of the social determinants of health in the creation and perpetuation of inequities for Māori and Pacific. Opportunities and privileges provided by income, housing and education are not evenly distributed in New Zealand. Due to the effects of colonisation, both historical and contemporary, non-Māori non-Pacific groups have higher median incomes and educational achievement than Māori and Pacific people.[35,36] Institutional or structural racism[37] is now recognised as a 'social determinant' that can adversely impact on health-promoting activities including health literacy and access to and through excellent and timely healthcare. While health providers generally have limited ability to change the social determinants of health, it is well within our remit to ensure that people's rights to receive high-quality, responsive and culturally safe healthcare services are being met.[33]

Our study is the largest and most recent review of CVD risk profiles in Aotearoa New Zealand, and we have demonstrated significant ethnic inequities. In our experience, generic interventions to reduce smoking and obesity or manage diabetes across the 'whole of population' are prioritised over ethnic-specific programs. A 'one-size fits all' approach will simply not work to achieve equity of CVD and its risk factors in New Zealand. In addition to current generic population health and risk-based approaches, there is a need for the continuing development of interventions from the perspective of those experiencing inequities.[26,38] We believe that more support, including strong political commitment, is required to ensure such interventions are resourced appropriately so that they are implemented, evaluated and, if effective, scaled up in order to increase their reach, and achieve equity.

Appendix

Appendix Table 1: Definitions of risk factors.

Variable	Source	Definition
Sex	NHI database	Sex recorded on NHI database
Age	NHI database	Age at index PREDICT assessment
Ethnicity	NHI database	Self-reported ethnicity was categorised using the prioritised output method according to national ethnicity data protocols (http://www.health.govt.nz/system/files/documents/publications/ethnicitydataprotocols.pdf). The South Asian population is known to have an elevated risk of CVD; Indians, who comprise 90% of the South Asian population in New Zealand, were able to be identified but non-Indian South Asians cannot currently be differentiated: NZ Māori > Pacific > Indian > Other Asian (Chinese and other East Asian) > European > MELAA > Other > Unknown/not answered/not identifiable (No_not_stated) People with ethnicity in the last three categories (MELAA [Middle Eastern/Latin American/African], Other and Unknown) were excluded from the analysis due to small numbers
Deprivation quintile	NHI database	We used the New Zealand Index of Deprivation (NZDep) (2006) as a measure of socioeconomic position. The NZDep was constructed from nine census-derived variables representing eight dimensions of deprivation. In this study, deprivation quintiles (1=least deprived, 5=most deprived) rather than the conventional NZDep2006 deciles were used, ie, Deprivation quintile 1 (least deprived) = NZ Dep decile 1 or 2 Deprivation quintile 2 = NZ Dep decile 3 or 4 Deprivation quintile 3 = NZ Dep decile 5 or 6 Deprivation quintile 4 = NZ Dep decile 7 or 8 Deprivation quintile 5 (most deprived) = NZ Dep decile 9 or 10
Smoking status	PREDICT	Smoker = current smoker or ex-smoker Current smoker = smokes up to 10 cigarettes/day, 11–19 cigs/day or 20+ cigs/day at index assessment Ex-smoker = quit over 12 months ago or quit within 12 months at index assessment Never smoker = never smoker at index assessment
BMI	PREDICT	BMI obtained at index assessment
Systolic BP	PREDICT	Mean of two systolic BP measurements obtained at index assessment
TC:HDL	PREDICT	Result entered into PREDICT index assessment. One measure, fasting or non-fasting
Diabetes mellitus	Multiple	- History of diabetes (PREDICT) AND/OR - Prior hospitalisation in which diabetes or associated condition noted (ICD-10-AM E10-14 or ICD-9-CM-A 250) AND/OR - 1+ dispensing of diabetes medication of any of the following in the last six months: Insulin; Acarbose; Chlorpropramide; Glibenclamide; Gliclazide; Glipizide; Metformin; Pioglitazone; Rosiglitazone; Tolazamide; Tolbutamide
Atrial fibrillation	Multiple	-History of AF (PREDICT) AND/OR -Prior hospitalisation in which AF diagnosis noted (ICD-10-AM I48)

Appendix Table 1: Definitions of risk factors.

Heart failure	Multiple	-Prior hospitalisation in which CHF diagnosis noted (any of ICD-10-AM I50, I110, I130, I132) AND/OR -Dispensing of 1+ loop diuretic (frusemide or bumetanide) on 3+ occasions in the last five years AND/OR - Any dispensing of metolazone in the last six months
Haemor-rhagic stroke	NMDS database	Subarachnoid haemorrhage: I60[b], Intracerebral haemorrhage: I61[b], Sequelae of subarachnoid haemorrhage: I690, Sequelae of intracerebral haemorrhage: I691
Coronary heart disease	Multiple	-History of angina OR MI or IHD or PTCA or CABG (PREDICT) AND/OR -Prior hospitalisation[a] in which atherosclerotic CHD diagnosis (incl angina) or procedure noted
Cerebro-vascular disease	Multiple	-History of ischaemic stroke or TIA (PREDICT) AND/OR -Prior hospitalisation[a] in which atherosclerotic CeVD diagnosis (incl ischaemic stroke and TIA) noted
Peripheral vascular disease	Multiple	-History of PVD (PREDICT) AND/OR -Prior hospitalisation[a] in which atherosclerotic PVD diagnosis or procedure noted
Athero-sclerotic CVD	Multiple	Criteria for coronary heart disease, cerebrovascular disease or peripheral vascular disease met

CABG=coronary artery bypass graft, CeVD = cerebrovascular disease, CVD=cardiovascular disease, CHD=coronary heart disease, IHD=ischaemic heart disease, MI=myocardial infarction, NHI=National Health Index, PTCA=percutaneous transluminal coronary angioplasty, PVD=peripheral vascular disease, TIA=transient ischaemic attack.
[a]See Table 6 for ICD (International Statistical Classification of Diseases and Related Health Problems) codes used to identify relevant hospitalisations (principal and secondary diagnoses considered).

Appendix Table 2: ICD-10-AM codes used to identify history of CVD from hospital records.

Category		**ICD-10-AM codes**[a]
Coronary heart disease	Cardiac arrest	I46[b]
	IHD	Angina pectoris: I20[b], Acute MI: I21[b] Subsequent MI: I22[b], Complications of acute MI: I23[b], Other IHD: I24[b] (except I241 – Dressler's syndrome), Chronic IHD: I25[b]
	Coronary procedures	Angioplasty/stent(s): 3530400-3530401, 3530500-3530501, 3530906-3530909, 3531000-3531005, Bypass: 3849700-3849707, 3850000-3850004, 3850300-3850304, 9020100-9020103, Other: 3845619, 3850500, 3850700, 3850800, 3850900, 3863700, Presence of coronary procedure: Z951, Z955, Z958, Z959
Cerebrovascular disease	Ischaemic stroke	Cerebral infarction: I63[b], Stroke, not specified as haemorrhage or infarction (as these are usually ischaemic): I64 (no subcategories), Sequelae of cerebral infarction: I693, Sequelae of stroke, not specified as haemorrhage or infarction: I694
	Other CeVD	TIA: G45[b] (except G454 – transient global amnesia), G46[b] Occlusion and stenosis of precerebral arteries, not resulting in cerebral infarction: I65[b], Occlusion and stenosis of cerebral arteries, not resulting in cerebral infarction: I66[b], Dissection of cerebral arteries, nonruptured: I670, Cerebral atherosclerosis: I672, Sequelae of other and unspecified CeVD: I698

Appendix Table 2: ICD-10-AM codes used to identify history of CVD from hospital records.

Peripheral vascular disease	PVD	Atherosclerosis with symptoms: I702[b], Atherosclerosis (other): I700, I701, I7020, I708, I709, Aortic aneurysm and dissection: I71[b], PVD, unspecified: I739, Arterial embolism and thrombosis: I74[b], DM with peripheral circulatory complications DM with other circulatory complications: E105[b], E115[b], E145[b]
	PVD procedures	The following procedures: aneurysm excisions, repairs and replacements, bypasses, endarterectomies and patch grafts, resections and re-anastomoses Involving the following arteries: carotid: 327000-3271011, 3270300, 3310000, 3350000 aorta: 3270800-3270803, 3311200, 3311500, 3311800, 3312100, 3315100, 3315400, 3315700, 3316000, 3350900, 3351200, 3351500 femoral: 3271200-3271201, 3271500-3271503, 3271800-3271801, 3273900, 3274200, 3274500, 3274800, 3275100-3275103, 3275400-3275402, 3275700-3275701, 3351501, 3352100, 3354200 mesenteric : 3273000-3273001, 3273300-3273301, 3273600, 3353001, 3353300, 3353600 other: 3276300-3276303, 3276305-3276314, 3276316-3276319, 3305000, 3305500, 3307500, 3308000, 3312400, 3312700, 3313000, 3316300, 3317800, 3318100, 3350600-3350601, 3351800, 3352400, 3352700, 3353000, 3353900, 3354800-3354803, 3355100, 3355400, 3530306-3530307, 3531200-3531201,3531500-3531501, , 9022900, 902300

Hospital records from 1 January 1988 to 31 December 2016.
CVD=cardiovascular disease, CeVD=cerebrovascular disease, CHF=congestive heart failure, DM=diabetes mellitus, ICD-10-AM= International Statistical Classification of Diseases and Related Health Problems, Australian Modification, IHD=ischaemic heart disease, MI=myocardial infarction, PVD=peripheral vascular disease, TIA=transient (cerebral) ischaemic attack.
[a]These are the codes used by the Vascular Informatics Using Epidemiology and the Web (VIEW) team, Department of Epidemiology and Biostatistics, University of Auckland (at March 2016) to identify people with CVD from hospital records. Only ICD-10-AM codes were used because diagnoses and procedures were mapped by the Ministry of Health to ICD-10-AM 2nd edition (where mappings existed), as well as the original submitted ICD-9-CM-A /ICD-10-AM version.
[b]Includes any subcategories that come after the last number, unless specified as excluded.

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Estimated inequities in COVID-19 infection fatality rates by ethnicity for Aotearoa New Zealand

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ABSTRACT

AIMS: There is limited evidence as to how clinical outcomes of COVID-19 including fatality rates may vary by ethnicity. We aim to estimate inequities in infection fatality rates (IFR) in New Zealand by ethnicity.

METHODS: We combine existing demographic and health data for ethnic groups in New Zealand with international data on COVID-19 IFR for different age groups. We adjust age-specific IFRs for differences in unmet healthcare need, and comorbidities by ethnicity. We also adjust for life expectancy reflecting evidence that COVID-19 amplifies the existing mortality risk of different groups.

RESULTS: The IFR for Māori is estimated to be 50% higher than that of non-Māori, and could be even higher depending on the relative contributions of age and underlying health conditions to mortality risk.

CONCLUSIONS: There are likely to be significant inequities in the health burden from COVID-19 in New Zealand by ethnicity. These will be exacerbated by racism within the healthcare system and other inequities not reflected in official data. Highest risk communities include those with elderly populations, and Māori and Pacific communities. These factors should be included in future disease incidence and impact modelling.

The COVID-19 outbreak originated in Wuhan, China before spreading globally to become a pandemic in March 2020. While as of early June 2020, the virus is likely to be eliminated in New Zealand,[1] it is still widespread globally and there is very low domestic immunity. There is an ongoing risk of reincursions into New Zealand and planning for these is important. Understanding the potential consequences of future outbreaks with widespread community transmission is crucial to designing and justifying effective measures to prevent this, including border controls, surveillance strategies and social distancing restrictions. Furthermore, better understanding the differential impacts of COVID-19 for high-risk groups within New Zealand, particularly Māori and Pasifika communities, is essential if New Zealand is to appropriately meet the needs of those communities, and mitigate against the effects of existing health inequities.

Obtaining accurate estimates of the risk of fatality is difficult, particularly in the early stages of an epidemic. One reason for this is the difficulty in ascertaining the true number of infections. Testing during an epidemic tends to focus on clinically severe cases, which may bias estimates of fatality rates upwards. Conversely, there is a lag time between onset of symptoms and clinical outcome, which may lead to under-reporting of fatalities.[2] Fatality rates also depend on factors such as age, pre-existing health conditions and access to healthcare. The case fatality rate (CFR) is the ratio of the number of fatalities to the number of diagnosed cases, whereas the infection fatality rate (IFR) is the ratio of the number of fatalities to the total number of infections. Note although some authors argue that these quantities are ratios and not rates, we use the term fatality rate because it is more commonly used in epidemiology. The CFR

is easier to calculate but often less useful than the IFR, which is independent of testing regimes and case definitions.

In this study, we estimate potential inequities in COVID-19 IFRs in New Zealand by ethnicity in the event that a future reincursion of COVID-19 leads to widespread community transmission. Fortunately, the number of cases in New Zealand to date has been too small to provide a sufficient sample size to stratify by ethnicity and age. Therefore, we project international age-stratified data on COVID-19 IFR[2] onto New Zealand's population, accounting for age structure and the effect of major comorbidities by ethnicity. The international IFR data was derived using a robust statistical approach, accounting for case under-ascertainment and right censoring.[2] It is consistent with more recent evidence from international studies[3] and serological surveys[4,5] which point to a population-level IFR between 0.5% and 1%. Using this data avoids the need to make assumptions around case ascertainment rates or total number of infections in New Zealand. Nevertheless, as the age-stratified IFR can vary between populations, our results should be viewed in a relative sense for comparing ethnicities rather than a precise prediction of the absolute value of IFR. The methodology we present could also be useful in the future if similar novel infectious diseases arrive in New Zealand and cannot be contained.

We adjust our estimates to account for the fact that, although Māori and Pacific populations are structurally younger than other ethnic groups, they have shorter life expectancy and higher rates of premature death at all ages. Mortality rates for older Māori are shaped by their life course, which includes increased exposure to infectious disease and conditions affecting respiratory function.[6] We also adjust for inequity in unmet healthcare need, which captures some of the structural biases and racism within the healthcare system.[7,8] We discuss other factors, not reflected in official data, which could further increase IFR for high-risk communities. These increased risk factors, and the adjustments made to model them, critically acknowledge the historic and contemporary differential experiences of exposure to, infection with, transmission of, and treatment for infectious and chronic disease for Māori.[9] During the 1918 influenza pandemic, Māori death rates were seven times higher than those for New Zealand European/Pākehā. As recently as 2009, during the H1N1 influenza pandemic, rates of infection for Māori were twice that of Pākehā, with increased severity.[10] The prevailing impacts of colonisation, resulting in historically under-served communities, provide key contexts for the need to understand IFR by ethnicity for New Zealand.

IFR is only one aspect of the epidemiology of COVID-19 and other factors, such as COVID-19 incidence and reduced access to healthcare services during a pandemic, could also contribute to inequities in overall health burden. We focus on IFR because it provides a key indication of how the severity of COVID-19 could vary by ethnicity, which will help identify high-risk communities. In addition, IFR is an important input for models of COVID-19 spread and mortality.[11] However, it will be important to refine these models to account for ethnicity-specific differences in other factors, including incidence and access to healthcare.

To date, there has been little quantitative analysis on the effects of ethnicity for COVID-19 in New Zealand. Given the speed at which COVID-19 can spread, there is an urgent need to prepare healthcare services and establish measures to protect at-risk groups. To address this, we use a simplified and approximate methodology, which contains numerous limitations (see Discussion). There are also shortcomings in the data on which our estimates are based, which make it difficult to disentangle the effects of age and comorbidity. Our results are an initial guide to the potential scale of COVID-19 inequity in New Zealand rather than a prediction of absolute IFR.

Methods

Data

Tables 1–2 show data on the age structure (2018 census, usual resident population[13]) for Māori, Pacific and New Zealand European/other, life expectancy for Māori, Pacific and non-Māori,[12] and international data on age-specific COVID-19 IFRs.[2] We chose to use this IFR data because it was stratified by age and included robust

Table 1: International data on age-specific COVID-19 IFR[2] and age distribution of Māori Pacific and New Zealand European/other ethnicity groups in New Zealand.[12]

Age group	0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80+
IFR	0.0016%	0.007%	0.031%	0.084%	0.16%	0.60%	1.90%	4.30%	7.80%
Age distribution									
Māori	21.79%	19.44%	15.73%	11.66%	11.42%	10.18%	6.19%	2.69%	0.90%
Pacific	23.00%	20.60%	17.16%	12.14%	10.51%	8.46%	4.94%	2.33%	0.85%
NZ Euro	12.59%	12.51%	12.39%	11.42%	13.07%	13.80%	11.63%	8.07%	4.53%

controls for under-ascertainment of cases and right-censoring. New Zealand European/other population statistics were estimated by subtracting the sum of Māori and Pacific populations from the total. Table 3 shows data on the prevalence of diabetes, heart disease, asthma, cancer and smoking by ethnicity in New Zealand.[14–18] The health data uses a mixture of prioritised ethnicity and total response classifications, we do not expect this to have a significant effect on the final results. Table 4 shows data on relative case fatality rate (CFR) for these conditions from China CDC.[19] Hypertension has not been classified by New Zealand district heath boards as a high-risk condition[20] so we did not include hypertension in our analysis. This is supported by a recent study from the UK, which found that hypertension was not associated with higher risk of fatality after controlling for other comorbidities.[21] Other chronic conditions such as renal disease may also have a significant effect,[21] but these were not included in the China CDC study.[19] In the absence of data on these health conditions collected using a consistent study design, we therefore excluded these from our study.

Adjusting for life expectancy

Māori typically experience adverse health outcomes at an earlier age than non-Māori.[22] To reflect this, we adjusted the age-specific IFR estimates[2] by the most recent (2012–14) estimates of life expectancy for each ethnicity. This approach is consistent with international evidence that COVID-19 mortality is approximately proportional to total mortality, meaning that COVID-19 amplifies existing mortality risk evenly for different groups.[23] The gap in life expectancy is different for male and female and for different age groups. For simplicity, we used an average of the male and female life expectancy gap for the youngest age cohort. We calculated the IFR for age group , adjusted for the life expectancy of ethnicity group *j*, as

$$IFR^{0}_{j,A} = \sum_{a \in A} q_{ja}\, IFR_{\text{data}}(r_j a),$$

where q_{ja} is the proportion of ethnicity j within age group *A* that is age *a*, $IFR_{data}(a)$ is the IFR at age in the reference population (in which the IFR data were measured), and r_j is the ratio of the life expectancy of the reference population to the life expectancy of group *j*. We used 20-year age brackets to match the New Zealand health data, but Eq. (1) accounts for the distribution of ages within each age bracket for each ethnicity.

Table 2: Life expectancy at birth (in years) of Māori, Pacific and non-Māori ethnicities.[12]

	Female	**Male**	**Average**
Māori	77.1	73.0	75.1
Pacific	78.7	74.5	76.6
Non-Māori	83.9	80.3	82.1

Table 3: Data on prevalence by ethnicity and age of four health conditions and smoking.[14–18]

Age group	Prevalence		
	Māori	Pacific	NZ European/other
Diabetes			
0–19	-	-	-
20–39	5.50%	10.70%	2.80%
40–59	20.80%	32.90%	6.90%
60–79	34.70%	34.20%	13.20%
80+	40.10%	55.80%	20.30%
Heart disease			
0–19	-	-	-
20–39	1.17%	1.10%	0.52%
40–59	7.46%	6.99%	3.78%
60–79	25.12%	22.26%	17.06%
80+	46.80%	38.68%	40.75%
Asthma (medicated)			
0–19	17.80%	15.80%	14.80%
20–39	16.40%	11.60%	12.70%
40–59	16.40%	11.60%	12.70%
60–79	16.40%	11.60%	12.70%
80+	16.40%	11.60%	12.70%
Cancer			
0–19	0.01%	0.02%	0.02%
20–39	0.08%	0.10%	0.09%
40–59	0.58%	0.54%	0.50%
60–79	1.97%	1.76%	1.69%
80+	3.15%	2.19%	2.78%
Smoking			
0–19	4.83%	3.14%	2.34%
20–39	39.47%	29.71%	19.52%
40–59	34.99%	24.12%	15.51%
60–79	19.01%	12.98%	8.49%
80+	12.18%	7.71%	6.52%

IFR_{data} was evaluated at ages $r_j a$ by linearly interpolating between the midpoints of the age brackets.[2] The midpoint for the 80+ age group was set at 85, with the IFR for all ages >85 fixed at this rate.

Adjusting for unmet healthcare need

There is evidence from the UK that groups with greater socioeconomic deprivation and black, Asian and minority ethnic groups, after controlling for age and comorbidities, have higher fatality risk.[21] These effects are difficult to quantify for New Zealand and no direct data is available. To capture some of this effect, we used data on unmet healthcare needs as a rough proxy for under-reporting of comorbid conditions and other inequities (see Discussion). The proportion of people who reported being unable to see a GP when needed (u_j) was 41.4% for Māori, 35.9% for Pacific and 30.1%[16] for New Zealand European/other. We weighted IFRs for each ethnicity by these values.

Adjusting for comorbidity

We calculated relative risk factors C_k (Table 4) for each comorbid condition as:

$$C_k = \frac{CFR \text{ with condition } k}{CFR \text{ without condition } k} = \frac{D_k/N_k}{(D_T - D_M - D_k)/(N_T - N_M - N_k)}$$

where D_k is the number of deaths in patients with condition k and N_k is the number patients with condition k.[19] Subscripts T and M respectively represent the same quantities for the total sample and for those with missing data.

To account for effect of comorbidity, we made several simplifying assumptions:

1. The overall population IFR in New Zealand across all ethnicities is approximately equal to the overall average IFR estimates from China[2] (see Discussion).
2. Conditions are independent so P(condition 1 and condition 2) = P(condition 1)*P(condition 2).
3. Individuals with multiple conditions experience the product of the risk factors of each condition, ie, there are no interaction effects between conditions.
4. The relative effect of comorbidities on IFR is the same as the measured effect on CFR[19] and is not age specific.

This allowed us to define a comorbidity weighting factor for ethnicity j and age group A as:

$$w_{j,A} = \sum_k P_{j,A,k} C_k$$

where $P_{j,A,k}$ is the proportion of ethnicity j and age with condition k.

Accounting for the combined effects of age and comorbidity is not straightforward, as we only had data on the overall effect of each comorbidity rather than age-specific effects. Prevalence of comorbid conditions, such as heart disease, will be higher in groups with older populations. This is already reflected, to some extent, in the age distribution of IFR (Table 1). Therefore, taking an age-structured IFR and adjusting for comorbidity will over-account for the effects of age-related health conditions. Similarly, adjusting for differences in life expectancy and prevalence of comorbid conditions will also result in some over-accounting. Conversely, ignoring age structure and only adjusting for selected comorbidities may ignore some age-related effects, for example from conditions that are not in the dataset or age effects that are not linked to a specific health condition (see Discussion).

We therefore calculated IFRs using two different methods: (i) starting with an age-specific baseline IFR; and (ii) starting with the same population-wide baseline IFR. For each method, we then adjusted the baseline IFR by ethnicity for life expectancy, unmet healthcare need, and comorbidity. Reality lies somewhere between (i) and (ii), so this gives an indicative range for the scale of relative differences in IFR by ethnicity. For method (i), we calculated IFR for age group A and ethnicity j as:

$$IFR_{j,A} = IFR_A^0 \frac{IFR_{j,A}^0 u_j w_{j,A}}{\sum_j IFR_{j,A}^0 u_j w_{j,A} \left(p_{j,A}/p_A\right)}$$

where $IFR^o{}_A$ is the population average IFR of age group A, $IFR^o{}_{j,A}$ is the life-expectancy-adjusted IFR from Eq. (1), and $p_{j,A}$ is the proportion of the population that is in age group A and ethnicity j. The denominator of Eq. (3) normalises so that the overall average IFR in the age group is $IFR^o{}_A$. For method (ii), we calculated the overall IFR for ethnicity j as:

Table 4: Data on COVID-19 case fatality rates for four comorbidities[27] and calculated relative risk factors (C_k). Data were unavailable on the effect of smoking on CFR so we used the incidence of severe cases as a proxy.[28]

Condition	Confirmed cases	Fatalities	CFR	Relative risk factor
Diabetes	1,102	80	7.26%	3.34
Heart disease	873	92	10.54%	5.10
Chronic resp. disease	511	32	6.26%	2.69
Cancer (any)	107	6	5.61%	2.33
None	15,536	133	0.86%	
Missing	23,690	617	2.60%	
Total	44,502	1121	2.52%	
Condition	**Confirmed cases**	**Severe cases**	**Severity rate**	**Relative risk factor**
Smoking	137	29	21.17%	1.41
Total	1,099	173	15.74%	

$$IFR_j = IFR^0 \frac{u_j \sum_A L_{j,A} w_{j,A} p_{j,A}}{p_j \sum_j u_j \sum_A L_{j,A} w_{j,A} p_{j,A}}$$

where $L_{j,A}$ is a factor adjusting for the effect of life expectancy on the IFR for ethnicity j and age group A. The denominator of Eq. (4) normalises so that the overall population average IFR is fixed at IFR^O.

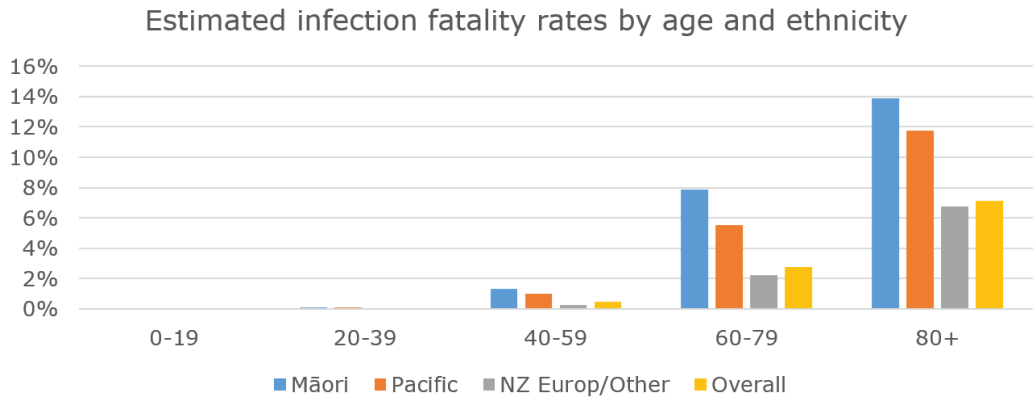
Results

The estimated overall population IFR is 0.81%, which is consistent with results from international studies placing the population-level IFR between 0.5% and 1%.[3–5] This overall rate could be influenced by numerous factors not accounted for here (see Discussion). The observed case fatality rate (CFR) may be substantially higher than the infection fatality ratio due to asymptomatic infections and case under-ascertainment.[2] As of early June 2020, New Zealand's CFR is around 1.5%. To be consistent with an IFR of 0.81% would imply that 46% of all infections were either asymptomatic or otherwise undiagnosed. This is plausible in light of studies pointing to high rates of asymptomatic infection.[24–26] It is also consistent with CFRs and case under-ascertainment rates in the international data.[2] Nevertheless, the results shown here should

Table 5: Estimated infection fatality rates for each ethnicity group. If age itself is the primary factor, then the results from method (i) are likely to be more accurate. If the age effect is driven by the increase in comorbidity rates with age, the results from method (ii) are likely to be more accurate.

Method (i)	Māori	Pacific	NZ Euro./ other	Overall
0–19 years	0.01%	0.01%	0.00%	0.01%
20–39 years	0.12%	0.09%	0.04%	0.06%
40–59 years	1.33%	1.00%	0.28%	0.45%
60–79 years	7.88%	5.52%	2.22%	2.78%
80+ years	13.87%	11.75%	6.76%	7.14%
Overall	1.15%	0.72%	0.75%	0.81%
Method (ii)	Māori	Pacific	NZ Euro./ other	Overall
Overall	1.66%	1.17%	0.62%	0.81%

Figure 1: Estimated infection fatality rates by age and ethnicity using method (i). These estimates are adjusted for age structure, relative life expectancy, unmet healthcare need and comorbidity (first section of Table 5).



be interpreted primarily as indicating relative differences in IFR across ethnicities and age groups, rather than exact predictions of absolute IFR.

The New Zealand European/other population is structurally old, but has relatively high life expectancy and low unmet healthcare need. Māori and Pacific populations are structurally younger, but have lower life expectancy, higher unmet healthcare need and higher prevalence of comorbid conditions, such as diabetes and asthma. These factors have opposing effects on the IFR and it is difficult to predict whether age, or other covarying factors, is more important. There is little direct evidence to distinguish these effects for COVID-19. We therefore used two methods: method (i) in which IFRs were pre-adjusted for age and method (ii) where they were not. Regardless of which method is used, Māori have a higher IFR than non-Māori (Table 5 and Figure 1). If age is the dominant variable, the estimated IFR for Māori is about 50% higher than for New Zealand European/other. If underlying health conditions (which correlate with age) are more important than age per se, the estimated IFR for Māori is more than 2.5 times higher than New Zealand European/other, and the IFR for Pacific people is almost double that of New Zealand European/other. Recent evidence suggests that age is the dominant factor with comorbidities having smaller though still statistically significant effects.[21] This suggests that IFRs are likely to the results from method (i) than to method (ii).

These prevalence data were standardised to 20-year age brackets by making the following approximations. In cases where data were more finely stratified (cancer, smoking), we calculated a weighted average for the prevalence in 20-year bands. The diabetes data were assigned to the closest age bracket (eg, 25–44-year-old diabetes rates were assigned to the 20–39 age bracket). The asthma data were reported in two age brackets: under 15 and 15+; the former was applied to the 0–19 age group and the latter to the others. There were no data on smoking rates for under 15-year olds so the rate was assumed to be zero. This is clearly an underestimate but this will little impact as IFR for COVID-19 is very low in this age bracket.

We performed a sensitivity analysis on two model assumptions: the magnitude of the difference in age-specific health outcomes between Māori, Pacific and New Zealand European/other; and the magnitude of the disparity in unmet healthcare need. The estimates we have used for these effects are based on indirect or proxy data (life expectancy and GP access respectively), which are likely to be underestimates. Table 6 shows three scenarios: (1) the impact of the difference in life expectancy (r_j in Eq. (1)) between New Zealand European/other and Māori/Pacific people is doubled from 8.6% to 17.2%; (2) the discrepancy in unmet healthcare need between New Zealand European/other and Māori/Pacific people is doubled; and (3) both adjustments. These scenarios reflect a plausible additional level of inequity that may be present. This additional inequity may result in Māori people experiencing fatality rates up to four times greater than New Zealand European/other.

Table 6: Results of sensitivity analysis of the estimated infection fatality rates on assumptions about inequities in healthcare outcomes at a given age. IFRs are pre-adjusted for age (method (i)). Darker colours indicate higher rates. For scenario (1), the change in impact of life expectancy is assumed to redistribute the rates without changing the overall IFR. In scenario (2) and (3), the increase in unmet healthcare needs is assumed to increase the overall IFR.

Scenario				
(1) Increase in impact of differences in life expectancy	Māori	Pacific	Other	Overall
0–19 years	0.01%	0.01%	0.00%	0.01%
20–39 years	0.14%	0.10%	0.04%	0.06%
40–59 years	1.71%	1.15%	0.21%	0.45%
60–79 years	10.12%	6.77%	1.98%	2.78%
80+ years	14.16%	12.01%	6.74%	7.14%
Total population	1.44%	0.84%	0.69%	0.81%
(2) Increase in impact of unmet healthcare need	Māori	Pacific	Other	Overall
0–19 years	0.02%	0.01%	0.00%	0.01%
20–39 years	0.24%	0.18%	0.04%	0.08%
40–59 years	2.66%	2.01%	0.28%	0.67%
60–79 years	15.76%	11.05%	2.22%	3.59%
80+ years	27.74%	23.51%	6.76%	7.91%
Total population	2.30%	1.44%	0.75%	1.03%
(3) Both of the above combined	Māori	Pacific	Other	Overall
0–19 years	0.02%	0.01%	0.00%	0.01%
20–39 years	0.27%	0.19%	0.04%	0.09%
40–59 years	3.41%	2.30%	0.21%	0.73%
60–79 years	20.25%	13.54%	1.98%	3.81%
80+ years	28.33%	24.01%	6.74%	7.93%
Total population	2.87%	1.69%	0.69%	1.08%

Discussion

Disentangling the effects of age structure and comorbidity on COVID-19 infection fatality rates is difficult because most studies have been limited to univariate analysis. Estimates from China of the impacts of comorbid conditions are not stratified by age.[19] The list of health conditions impacting on COVID-19 infections continues to be expanded as the pandemic develops. The data from which the baseline IFRs used in the current analysis were calculated were adjusted for under-reporting, bias towards more severe cases, and lag time from onset to clinical outcome,[2] but may be affected by other biases. The baseline IFRs in our analysis are based on data from China, but there will be country-specific variations in IFR, and potentially higher IFRs in countries with large ethnic minority or Indigenous populations. It is also possible that the IFR may decrease over time as we develop improved treatments. The results discussed here should be treated as a preliminary estimate of relative inequity by ethnicity, rather than predictions of the absolute IFR.

We calculated IFRs using two different methods, giving an indicative range for the scale of potential inequity in IFRs between

ethnicities. We adjusted IFRs for differences in life expectancy, unmet healthcare need and prevalence of comorbid conditions. This methodology should be refined over time, particularly as more data become available on outcomes from COVID-19 cases in New Zealand. An alternative approach would be to use standardised metrics such as disability-adjusted life year (DALY) and years lost due to disability (YLD) to infer IFRs by age and ethnicity in New Zealand from the Chinese data. This approach should be investigated, although it is possible that the true magnitude of inequities are not captured in these metrics and the data from which they are derived, so there is a risk that this will underestimate the health burden for Māori and Pacific people.

There are multiple reasons why inequities could end up being larger than estimated here. Hospitalisation and fatality rates for Māori and Pacific people from pandemic H1N1 influenza in 2009 were significantly higher than for New Zealand European.[10,29] Māori are more likely to experience multi-morbidity and if the effect of multiple underlying health conditions is worse than simply multiplicative as assumed here, this will increase the IFR for Māori. These disparities could be wider still if differences in age-specific health outcomes and unmet healthcare need are larger than captured in official data. Data on prevalence of comorbid conditions among Māori and Pacific people (Table 3) may be influenced by underreporting, which would make their IFRs higher than calculated here. Avoidable hospitalisations are higher for Māori and Pacific populations,[22,30] reflecting broader and more complex structural disadvantage. There exists other widely reported racism within the healthcare system[22,31,32] that is not reflected in the available data.

Some of these factors may be less important while COVID-19 case numbers are low, the goal is elimination or containment, and surveillance and contact tracing capacity is adequate. However, if rapid community transmission of COVID-19 takes hold, as has happened elsewhere, it will place unprecedented stress on the healthcare system. This will make access to healthcare increasingly difficult and necessitate decisions by practitioners about who gets access to care. This will almost certainly amplify existing racism in the healthcare system. For example, if triage decisions are based on existence of underlying health conditions, this will automatically disadvantage Māori further. Similar concerns about the inequitable impacts of prioritisation tools have been raised elsewhere.[9] Transparency is needed in the risk factors and weightings used to guide decision-making about healthcare service provision, and independent oversight by at-risk groups likely to be disparately impacted by these.

COVID-19 is likely to be more severe in regions or communities with a relatively old population, which is one of the biggest factors affecting hospitalisation and fatality rates. Rural Māori communities have an older age distribution than Māori as a whole[33] and have higher unmet healthcare need, so this is a particularly high-risk group. Reported COVID-19 fatalities do not capture indirect impacts, for example deaths attributed to underlying conditions, but precipitated or hastened by COVID-19 infection. These indirect impacts are also likely to fall disproportionately on Māori and Pacific peoples due to higher prevalence of comorbid conditions.

A report from the UK suggests black, Asian and minority ethnic groups are at higher risk from COVID-19 than white majority groups.[34] Reports from the US suggest similar trends, where African-American communities are bearing a disproportionate health burden from COVID-19.[35] These at-risk communities typically have higher prevalence of underlying health conditions, are more likely to live in overcrowded and multi-generational households, and have relatively young populations.[36] Similar factors apply to Māori in New Zealand[37] and this reinforces the need to account for the multitude of factors behind inequity, rather than crudely using age structure alone to estimate IFR. The methodology we have used is a first attempt at addressing this. Data on COVID-19 incidence and outcomes in the context of ethnic minority or Indigenous populations that experience inequities in health and healthcare is currently scarce. Making robust comparisons and informing interventions to eliminate inequitable outcomes requires not only more data, but data that is accessible

to decision makers in a timely fashion. This reinforces the importance of systematic, comprehensive and timely data collection in New Zealand in order to manage this and any future epidemics.

This study has focused on the infection fatality rate, which does not account for potential differences in transmission and incidence by ethnicity. Risk factors for accelerated transmission include crowded housing, which affects approximately 25% of Māori and 45% of Pacific people.[38,39] In addition, multi-generational households increase the risk of transmission to older groups. These compounding factors mean that Māori and Pacific peoples are at risk of bearing a disproportionate health burden from COVID-19. A comprehensive analysis of these factors is outside the scope of this work. It will be critical to incorporate these into disease transmission models that are used to inform New Zealand's COVID-19 response.[40] This will enable the combined effect of incidence and IFR to be more accurately measured and to inform effective strategies that recognise the diversity of higher-risk groups, communities and regions.

Competing interests:
Dr Binny, Ms Hannah, Dr James, Dr Plank, Mr Steyn, Dr Hendy and Dr Lustig report grants from Te Pūnaha Matatini during the conduct of the study. Dr Kukutai is a member of the Chief Science Advisor Forum and incoming Director of Ngā Pae o Te Māramatanga (pending successful bid).

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Inequity in one-year mortality after first myocardial infarction in Māori and Pacific patients: how much is associated with differences in modifiable clinical risk factors? (ANZACS-QI 49)

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ABSTRACT

AIMS: Ischaemic heart disease (IHD) mortality rates after myocardial infarction (MI) are higher in Māori and Pacific compared to European people. The reasons for these differences are complex and incompletely understood. Our aim was to use a contemporary real-world national cohort of patients presenting with their first MI to better understand the extent to which differences in the clinical presentation, cardiovascular (CVD) risk factors, comorbidity and in-hospital treatment explain the mortality outcomes for Māori and Pacific peoples.

METHODS: New Zealand residents (≥20 years old) hospitalised with their first MI (2014–2017), and who underwent coronary angiography, were identified from the All New Zealand Acute Coronary Syndrome Quality Improvement (ANZACS-QI) registry. All-cause mortality up to one year after the index admission date was obtained by linkage to the national mortality database.

RESULTS: There were 17,404 patients with a first ever MI. European/other comprised 76% of the population, Māori 11.5%, Pacific 5.1%, Indian 4.3% and Other Asian 2.9%. Over half (55%) of Māori, Pacific and Indian patients were admitted with their first MI before age 60 years, compared with 29% of European/other patients. Māori and Pacific patients had a higher burden of traditional and non-traditional cardiovascular risk factors, and despite being younger, were more likely to present with heart failure and, together with Indian peoples, advanced coronary disease at presentation with first MI. After adjustment for age and sex, Māori and Pacific, but not Indian or Other Asian patients had significantly higher all-cause mortality at one year compared with the European/other reference group (HR 2.55 (95% CI 2.12–3.07), HR 2.98 (95% CI 2.34–3.81) for Māori and Pacific respectively). When further adjusted for differences in clinical presentation, clinical history and cardiovascular risk factors, the excess mortality risk for Māori and Pacific patients was reduced substantially, but a differential persisted (HR 1.77 (95% CI 1.44–2.19), HR 1.42 (95% CI 1.07–1.83)) which was not further reduced by adjustment for differences in in-hospital management and discharge medications.

CONCLUSION: In New Zealand patients after their first MI there is a three-fold variation in one-year mortality based on ethnicity. At least half of the inequity in outcomes for Māori, and three-quarters for Pacific people, is associated with differences in preventable or modifiable clinical factors present at, or prior to, presentation.

In New Zealand, age standardised hospitalisation rates for ischaemic heart disease (IHD) and its most important clinical manifestation, myocardial infarction (MI), have steadily decreased over the last 10 years.[1,2] However, despite this good news, Māori, Pacific and South Asian peoples continue to have higher IHD hospitalisation rates, and Māori and Pacific peoples have higher IHD mortality rates compared to European.[3,4] The reasons for these differences are complex and incompletely understood. The All New Zealand Acute Coronary Syndrome Quality Improvement (ANZACS-QI) registry collects a comprehensive dataset for all patients presenting to New Zealand public hospitals with an ACS (acute coronary syndrome) who undergo investigation with a coronary angiogram. The ANZACS-QI registry is linked via an encrypted identifier to national administrative datasets to augment data and to track patient outcomes.[5] Our aim was to use this contemporary real-world national cohort, in patients presenting with their first MI, to better understand the extent to which differences in the clinical presentation, cardiovascular disease (CVD) risk factors, comorbidity and in-hospital treatment explain the divergence in outcomes between ethnic groups.

Methods

Cohort

New Zealand residents aged ≥20 years hospitalised with their first MI between 1 January 2014 and 31 December 2017 and who underwent coronary angiography were identified from the ANZACS-QI registry.

The ANZACS-QI registry is a web-based electronic database that captures a mandatory dataset which includes patient demographics, admission ACS risk stratification, cardiovascular risk factors, investigations and management, inpatient outcomes and medications prescribed at discharge. The patients captured in the registry are linked via an encrypted unique National Health Identifier (NHI) to national hospitalisation, mortality and pharmaceutical dispensing national datasets. Details regarding the ANZACS-QI programme, registry data collection and linkage to national datasets have been previously reported.[5] Data collected in ANZACS-QI has been previously described in detail.[5,6] The registry is subject to monthly auditing to ensure capture of >95% of all patients admitted with suspected ACS who are investigated with coronary angiography, and annual audit to check the accuracy of data entry.

Data and definitions

MI was defined according to the contemporary universal definition.[7] Socio-demographic variables and residency status were derived from the linked national dataset. For patients in whom more than one ethnic group was recorded, ethnicity was prioritised, in accordance with health sector protocols, in the following order: indigenous Māori, Pacific, Indian, Other Asian and European/other.[8] European /other included all those identifying as European as well as a small number of people from the Middle East, Africa and Latin America. Fijian Indian people are categorised as 'Indian', as opposed to 'Pacific'. Socioeconomic deprivation was assessed by the NZDep13 score, a census-based small area 10-point index of deprivation based on the person's domicile.[9] Clinical presentation variables from the ANZACS-QI registry included type of MI (ST-elevation MI (STEMI) or non ST-elevation MI (NSTEMI)), known prior congestive heart failure (CHF), components of the Global Registry of Acute Coronary Events (GRACE) in-hospital mortality risk score (including Killip class, admission heart rate and blood pressure, cardiac arrest on admission, electrocardiogram findings, troponin level, admission creatinine),[10] left ventricular ejection fraction (LVEF) assessed by transthoracic echo or left ventriculogram, coronary artery disease extent on angiography. Killip class was divided into those without (Class I) and with acute heart failure (Classes II–IV).[11] Left ventricular ejection fraction (LVEF) assessment was classified into normal (≥50%), mild impairment (40–49%), moderate to severe impairment (<40%), and not quantified further. Coronary artery disease (CAD) extent was defined by the findings at angiography and were grouped into one of the following: (i) no significant CAD, defined as the absence of any stenosis with ≥50% diameter loss in the epicardial vessels, (ii) significant (≥50% stenosis) single vessel coronary disease, (iii) significant (≥50% stenosis) double vessel coronary artery disease, (iv) significant three-vessel disease and/or left main

stem (LMS) disease ≥50%. Due to the use of multiple different Troponin assays across New Zealand, the peak troponin values for each patient were stratified into quintiles for each separate assay. eGFR at admission was calculated using the CKD-EPI equation in ml/min and reported in CKD stages one (>90ml/min), two (60–90ml/min), three (30–60ml/min), four (15–30ml/min) and five (<15ml/min).[12] Cardiovascular disease risk factors, history and comorbidity variables included: smoking status defined as current, ex-smoker or never smoker, diabetes mellitus, hypertension (HT), low-density lipoprotein (LDL) and total cholesterol (TC) to high-density lipoprotein (HDL) ratio, body mass index (BMI), history of chronic obstructive pulmonary disease (COPD), history of congestive heart failure (CHF) and prior atherosclerotic CVD—defined as a prior diagnosis or history of transient ischaemic attack or ischaemic stroke, peripheral vascular disease or radiological evidence of vascular disease.

Investigation and management variables were coronary revascularisation by percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG). Discharge medications were: aspirin, P2Y12 inhibitor (clopidogrel or ticagrelor), statins, angiotensin converting enzyme inhibitors (ACEIs) and beta-blockers. Dual anti-platelet therapy (DAPT) was aspirin plus a P2Y12 inhibitor.

Outcomes

All-cause mortality up to one year after the index admission date was obtained by linkage to the national mortality database.

Statistical analysis

Categorical variables were summarised as frequency and percentage and continuous variables as mean and standard deviation (SD). Comparisons across ethnic groups were made using Pearson's chi-square test, one-way ANOVA or a Kruskall Wallis test, as appropriate. Multivariable Cox regression models were used to estimate the hazard ratio of each ethnicity compared to European/other for all-cause mortality and 30-day post admission all-cause mortality outcomes in four models: Model One—unadjusted ; Model Two—adjusted by age and sex ; Model Three—adjusted by age, sex, worst Killip class, EF categories, CAD extent, troponin quintile, cardiac arrest, MI sub-type, prior CHF, prior CVD, smoking, diabetes, TC:HDL, hypertension, eGFR, COPD; Model Four—Model Three variables plus coronary revascularisation (PCI or CABG), medications (statin, ACEI/ARB, beta blocker, DAPT) at discharge. Cumulative mortality plots stratify time to death by ethnic group.

The proportional hazards assumptions were tested by plotting the standardised score residuals over time. The assumptions were met. All tests of statistical significance were two tailed and a p-value <0.05 was considered statistically significant. Data were analysed using SAS version 9.4 (SAS Institute, Cary, NC), and cumulative mortality plots were created using RStudio version 1.2.1335.

Ethical approval

This research was performed as part of the VIEW-ANZACS-QI research programme. Ethics approval was obtained from the Northern Region Ethics Committee (AKY/03/12/314) and Multi-Region Ethics Committee (MEC/01/19/EXP and MEC/11/EXP/078).

Results

There were 17,404 patients with a first ever MI. European/other comprised 76% of the population, Māori 11.5%, Pacific 5.1%, Indian 4.3% and other Asian 2.9%. Patient demographics are shown in Table 1. Two thirds of patients were men and the mean age was 64 years. Māori, Pacific and Indian patients presented at a younger age (mean age 58–59 years) compared with other Asian and European/other patients (mean age 61 and 66 years respectively). Over half (55%) of Māori, Pacific and Indian patients were admitted with their first MI before age 60 years, compared with 35% of other Asian and 29% of European/other patients.

Clinical presentation (Table 2)

Māori patients were the most likely to present with cardiac arrest. Māori and Pacific patients were 1.5 to 2 times more likely to have acute heart failure than European/others (17%, 19.5%, 11.5%, respectively), while 26% of Pacific patients and 21% of Māori patients had moderate-severe LV impairment, but only 15% of European/others. This was despite a similar proportion of each ethnic group having

Table 1: Baseline demographics.

		Ethnicity					P value
	Overall (n=17,404)	**Māori (n=2,003) 11.5%**	**Pacific (n=880) 5.1%**	**Indian (n=740) 4.3%**	**Other Asian (n=505) 2.9%**	**NZ European/other (n=13,276) 76%**	
Age, years							<.001
<50	2,057 (11.8)	417 (20.8)	214 (24.3)	180 (24.3)	90 (17.8)	1,156 (8.7)	
50–<60	4,028 (23.1)	685 (34.2)	277 (31.5)	225 (30.4)	139 (27.5)	2,702 (20.4)	
60–<70	5,023 (28.9)	563 (28.1)	228 (25.9)	186 (25.1)	138 (27.3)	3,908 (29.4)	
70–<80	4,367 (25.1)	298 (14.9)	148 (16.8)	121 (16.4)	103 (20.4)	3,697 (27.9)	
80+	1,929 (11.1)	40 (2.0)	13 (1.5)	28 (3.8)	35 (6.9)	1,813 (13.7)	
Mean (SD)	64.4 (12.1)	58.4 (10.9)	57.9 (11.4)	58.6 (12.1)	61.2 (12.0)	66.2 (11.7)	<.001
Gender							<.001
Male	11,874 (68.2)	1,215 (60.7)	579 (65.8)	580 (78.4)	369 (73.1)	9,131 (68.8)	
Female	5,530 (31.8)	788 (39.3)	301 (34.2)	160 (21.6)	136 (26.9)	4,145 (31.2)	

All values are number of patients and frequency (%) unless otherwise specified.

myocardial necrosis in the highest quintile (based on troponin levels). At coronary angiography nearly half of all patients had obstructive disease in more than one coronary artery. Pacific and Indian patients had more severe three-vessel disease and/or left main coronary artery disease (38% and 33%) than Māori, Other Asian or European/other patients (25%, 26.5% and 26%). The proportion of STEMIs was similar for Māori, Indian and European/other groups but lower for Pacific patients and slightly higher for Other Asian patients. While only 3.1% of the overall cohort had advanced (Stage 4 or 5) CKD, Pacific (11.4%) and Māori (5.8%) were markedly over-represented, with 4.2% of Indian people also affected. In contrast, only 2% of European/other patients had advanced CKD.

Atherosclerotic CVD risk factors and medical history (Table 3)

Nearly half of Pacific and Indian patients had diabetes, 30% of Māori, 29% of Other Asian and 16% of European/others. Of those with BMI recorded, 33.8% of Maori and 44.2% of Pacific compared to 23.7% of European/others had a BMI in the obese range (Indian 18.4% and Other Asian 9.5%). Mean TC:HDL was highest in Māori and lowest in Other Asian patients with intermediate levels in other groups. Nearly half of Māori and a third of Pacific patients were current smokers compared with less than a quarter of other ethnic groups. Māori patients, correspondingly, were more likely to have COPD. Māori and Pacific patients were twice as likely to have a diagnosis of prior CHF compared with non-Māori/non-Pacific.

Treatment (Table 4)

Overall, 74.5 % of patients underwent coronary revascularisation: 61.6% with PCI and 12.9% by CABG. Revascularisation was highest for Other Asian patients (77%) followed by European/others (75%), Indian (72%), Pacific (70.5%) and Māori (67%). Compared to European/other patients, Māori, Pacific and Indian patients had higher rates of CABG and lower rates of PCI consistent with their higher prevalence of diabetes and among Pacific and Indian patients, diffuse coronary artery disease. Indian people were equally likely to receive PCI as European/other patients. However, Māori and Pacific patients were less likely to receive PCI and less likely overall to receive coronary intervention. There was a high level of prescription of aspirin and statin medication at discharge with only minor ethnic differences. Use of a P2Y12 anti-platelet agent was higher in European/other than other groups and use of ACEI/ARB was highest in Māori, Pacific and Indian patients.

Table 2: Clinical presentation.

	Overall (N=17,404)	Māori (n=2,003)	Pacific (n=880)	Indian (n=740)	Other Asian (n=505)	NZ European/other (n=13,276)	P-value
Cardiac arrest	821 (4.7)	135 (6.7)	40 (4.5)	29 (3.9)	25 (5.0)	592 (4.5)	<.001
Worst Killip class							<.001
I	15,186 (87.3)	1,656 (82.7)	708 (80.5)	634 (85.7)	437 (86.5)	11,751 (88.5)	
II, III, IV	2,218 (12.7)	347 (17.3)	172 (19.5)	106 (14.3)	68 (13.5)	1,525 (11.5)	
LV EF							<.001
Normal (≥ 50%)	8,253 (61.2)	844 (56.2)	401 (54.2)	402 (64.0)	253 (62.2)	6,353 (62.3)	
Mild (40 to 49%)	2,723 (20.2)	306 (20.4)	127 (17.2)	121 (19.3)	69 (17.0)	2,100 (20.6)	
Moderate or severe (<40%)	2,210 (16.4)	314 (20.9)	191 (25.8)	86 (13.7)	75 (18.4)	1,544 (15.1)	
Not quantified further	291 (2.2)	38 (2.5)	21 (2.8)	19 (3.0)	10 (2.5)	203 (2.0)	
Peak troponin level in hospital, quintiles							0.040
1 (lowest peak troponin)	3,480 (20.0)	433 (21.6)	189 (21.5)	132 (17.8)	112 (22.2)	2,614 (19.7)	
2	3,454 (19.9)	425 (21.2)	172 (19.5)	147 (19.9)	88 (17.4)	2,622 (19.7)	
3	3,471 (19.9)	381 (19.0)	188 (21.4)	145 (19.6)	81 (16.0)	2,676 (20.2)	
4	3,471 (19.9)	376 (18.8)	165 (18.8)	158 (21.4)	100 (19.8)	2,672 (20.1)	
5 (highest peak troponin)	3,463 (19.9)	378 (18.9)	162 (18.4)	156 (21.1)	123 (24.4)	2,644 (19.9)	
Missing	65 (0.4)	10 (0.5)	4 (0.5)	2 (0.3)	1 (0.2)	48 (0.4)	
Anatomical extent of CAD							<.001
No significant disease	2,367 (13.6)	392 (19.6)	138 (15.7)	58 (7.8)	74 (14.7)	1,705 (12.8)	
Single vessel disease	6,571 (37.8)	716 (35.8)	234 (26.6)	240 (32.4)	185 (36.6)	5,196 (39.1)	
Double vessel disease	3,796 (21.8)	386 (19.3)	178 (20.2)	199 (26.9)	112 (22.2)	2,921 (22.0)	
Three vessel disease and/or LMS>50% and/or graft	4,670 (26.8)	509 (25.4)	330 (37.5)	243 (32.8)	134 (26.5)	3,454 (26.0)	
Type of ACS							0.001
NSTEMI	11,646 (66.9)	1,356 (67.7)	643 (73.1)	494 (66.8)	320 (63.4)	8,833 (66.5)	
STEMI	5,758 (33.1)	647 (32.3)	237 (26.9)	246 (33.2)	185 (36.6)	4,443 (33.5)	
Heart rate							<.001
Mean (SD)	77.3 (19.8)	79.5 (22.6)	81.3 (21.8)	81.3 (19.6)	76.7 (18.6)	76.5 (19.2)	
eGFR stages (ml/min/1.73m^2)							<.001
>90	3,602 (20.7)	561 (28.0)	191 (21.7)	250 (33.8)	153 (30.3)	2,447 (18.4)	
60–90	9,315 (53.5)	939 (46.9)	393 (44.7)	339 (45.8)	247 (48.9)	7,397 (55.7)	
30–60	3,957 (22.7)	386 (19.3)	196 (22.3)	120 (16.2)	88 (17.4)	3,167 (23.9)	
15–30	275 (1.6)	47 (2.3)	26 (3.0)	18 (2.4)	7 (1.4)	177 (1.3)	
<15	254 (1.5)	70 (3.5)	74 (8.4)	13 (1.8)	10 (2.0)	87 (0.7)	
Missing	1 (0.01)	0 (0)	0 (0)	0 (0)	0 (0)	1 (0.01)	
GRACE in-hospital mortality risk score							<.001
<1%	3,657 (21.0)	578 (28.9)	251 (28.5)	211 (28.5)	123 (24.4)	2,494 (18.8)	
1–<3%	6,538 (37.6)	719 (35.9)	320 (36.4)	279 (37.7)	183 (36.2)	5,037 (37.9)	
≥3%	7,205 (41.4)	705 (35.2)	309 (35.1)	250 (33.8)	199 (39.4)	5,742 (43.3)	
Missing	4 (0.02)	1 (0.05)	0 (0)	0 (0)	0 (0)	3 (0.02)	

Values are number of patients and frequency (%) unless otherwise specified. CAD, coronary artery disease; NSTEMI, non-ST segment elevation myocardial infarction; STEMI, ST segment elevation myocardial infarction; eGFR, estimated glomerular filtration rate.

Table 3: Cardiovascular risk factors.

	Overall (N=17,404)	Māori (n=2,003)	Pacific (n=880)	Indian (n=740)	Other Asian (n=505)	NZ European/other (n=13,276)	p-value
Diabetes	3,647 (21.0)	598 (29.9)	427 (48.5)	350 (47.3)	147 (29.1)	2,125 (16.0)	<.001
BMI, kg/m^2							
BMI available (n)	13,745	1,538	745	645	415	10,402	<.001
<20 Underweight	316 (1.8)	25 (1.2)	2 (0.2)	12 (1.6)	22 (4.4)	255 (1.9)	
20–<25 Normal	2,957 (17.0)	185 (9.2)	54 (6.1)	182 (24.6)	175 (34.7)	2,361 (17.8)	
25–<30 Overweight	5,342 (30.7)	429 (21.4)	172 (19.6)	305 (41.2)	165 (32.7)	4,271 (32.2)	
30–<35 Mildly obese	3,178 (18.3)	445 (22.2)	234 (26.6)	113 (15.3)	46 (9.1)	2,340 (17.6)	
35–<40 Moderately obese	1,238 (7.1)	253 (12.6)	155 (17.6)	23 (3.1)	2 (0.4)	805 (6.1)	
40+ Morbidly obese	714 (4.1)	201 (10.0)	128 (14.5)	10 (1.4)	5 (1.0)	370 (2.8)	
Missing	3,659 (21.0)	465 (23.2)	135 (15.3)	95 (12.8)	90 (17.8)	2,874 (21.6)	
Mean (SD)	29.1 (5.9)	32.1 (6.9)	33.9 (7.3)	27.4 (4.6)	25.7 (4.0)	28.6 (5.5)	<.001
TC:HDL							<.001
n	16,395	1,898	850	723	487	12,437	
Mean (SD)	4.42 (1.99)	4.76 (2.59)	4.47 (1.84)	4.43 (1.65)	4.05 (1.43)	4.37 (1.92)	
Smoking status							<.001
Non-smoker	7,562 (43.4)	447 (22.3)	345 (39.2)	485 (65.5)	307 (60.8)	5,978 (45.0)	
Ex-smoker	5,380 (30.9)	562 (28.1)	231 (26.3)	115 (15.5)	114 (22.6)	4,358 (32.8)	
Current smoker	4,462 (25.6)	994 (49.6)	304 (34.5)	140 (18.9)	84 (16.6)	2,940 (22.2)	
COPD	1,476 (8.5)	268 (13.4)	79 (9.0)	38 (5.1)	30 (5.9)	1,061 (8.0)	<.001
Prior CVD	2,788 (16.0)	331 (16.5)	120 (13.6)	99 (13.4)	57 (11.3)	2,181 (16.4)	0.001
History of CHF	452 (2.6)	85 (4.2)	37 (4.2)	12 (1.6)	13 (2.6)	305 (2.3)	<.001
Hypertension	9,595 (55.1)	1,125 (56.2)	505 (57.4)	445 (60.1)	267 (52.9)	7,253 (54.6)	0.014

Values are number of patients and frequency (%) unless otherwise specified. BMI, body mass index; TC:HDL, total cholesterol to high-density lipoprotein ratio; COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease; CHF, congestive heart failure.

Outcomes (Table 5, Figure 1)

The unadjusted one-year cumulative mortality was highest for Pacific (8.8%) followed by Māori (7.6%), European/others (4.9%), Other Asians (4.8%) and lowest in Indian (3.5%). The age and sex adjusted cumulative mortality is shown in Figure 1. There is a steep early hazard for all ethnic groups which is greatest in Māori, Pacific and Indian patients. Beyond this early phase, there is only minimal incremental increase in mortality in European/other, Indian and Other Asian patients but a steady incremental increase in Māori and Pacific mortality, leading to progressive divergence of the mortality curves. After adjusting for age-group and sex, Māori and Pacific, but not Indian or Other Asian patients, had significantly higher all-cause mortality at one year compared with the European/other reference group. (HR 2.55, (95% CI 2.12–3.07), HR 2.98 (95% CI 2.34–3.81), for Māori and Pacific respectively). When further adjusted for differences in clinical presentation, clinical history and cardiovascular risk factors the excess mortality risk for Māori and Pacific patients compared with European/others was reduced but a substantial differential persisted (HR 1.77, (95% CI 1.44–2.19), HR 1.42, (95% CI 1.07–1.83). Further adjustment for differences in mode of revascularisation and discharge medications made little difference (HR 1.72, (95% CI 1.39–2.12), HR 1.35 (95% CI 1.01–1.80).

Table 4: Post MI management.

	Overall (N=17,404)	Māori (n=2,003)	Pacific (n=880)	Indian (n=740)	Other Asian (n=505)	NZ European/other (n=13,276)	p-value
PCI	10,727 (61.6)	1,047 (52.3)	437 (49.7)	471 (63.6)	315 (62.4)	8,457 (63.7)	<.001
CABG	2,243 (12.9)	297 (14.8)	183 (20.8)	133 (18.0)	74 (14.7)	1,556 (11.7)	<.001
Total revascularisation	12,809 (73.6)	1,327 (66.3)	602 (68.4)	591 (79.9)	383 (75.8)	9,906 (74.6)	<.001
Discharged medication (of those discharged alive)							
Aspirin	16,298 (95.6)	1,831 (93.7)	822 (96.6)	707 (97.7)	471 (95.9)	12,467 (95.7)	<.001
P2Y12 inhibitor	13,937 (81.7)	1,509 (77.2)	615 (72.3)	567 (78.3)	368 (74.9)	10,878 (83.5)	<.001
Statin	16,053 (94.2)	1,836 (93.9)	807 (94.8)	711 (98.2)	461 (93.9)	12,238 (93.9)	<.001
Beta-blocker	14,198 (83.3)	1,585 (81.1)	700 (82.3)	625 (86.3)	399 (81.3)	10,889 (83.6)	0.006
ACEI/ARB	12,334 (72.3)	1,439 (73.6)	660 (77.6)	568 (78.5)	332 (67.7)	9,335 (71.7)	<.001

Values are number of patients and frequency (%) unless otherwise specified. PCI: percutaneous intervention. CABG: coronary artery bypass graft.

Table 5: All-cause mortality in the year after a first MI: multivariable models.

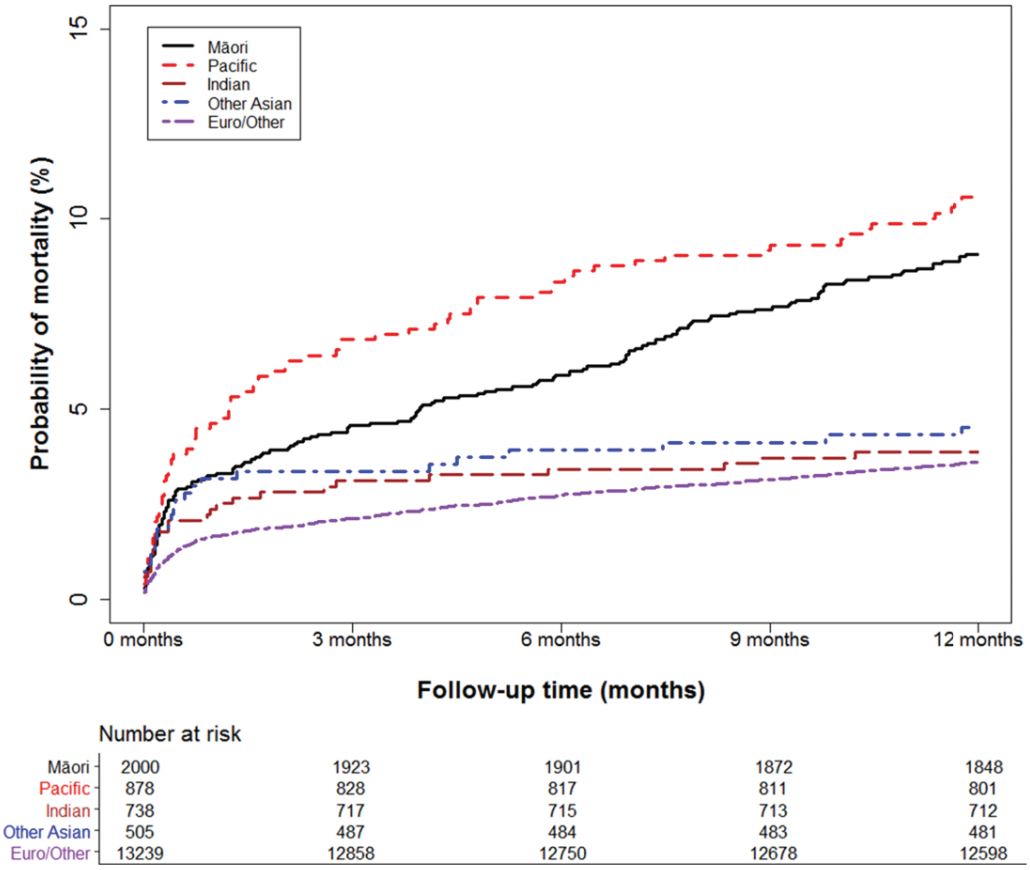
	Event/N (%)	Model 1 HR (95% CI)	p-value	Model 2 HR (95% CI)	p-value	Model 3 HR (95% CI)	p-value	Model 4 HR (95% CI)	p-value
Ethnic group									
Māori	153/2,003 (7.6)	1.57 (1.32–1.87)	<.001	2.55 (2.12–3.07)	<.001	1.77 (1.44–2.19)	<.001	1.72 (1.39–2.12)	<.001
Pacific	77/880 (8.8)	1.82 (1.44–2.30)	<.001	2.98 (2.34–3.81)	<.001	1.42 (1.07–1.83)	0.016	1.35 (1.01–1.80)	0.041
Indian	26/740 (3.5)	0.71 (0.48–1.06)	0.091	1.07 (0.72–1.59)	0.730	0.78 (0.50–1.22)	0.284	0.77 (0.49–1.20)	0.250
Other Asian	24/505 (4.8)	0.97 (0.65–1.46)	0.893	1.25 (0.83–1.88)	0.280	0.99 (0.64–1.52)	0.986	0.93 (0.60–1.43)	0.728
Euro/other	652/13,276 (4.9)	Reference	-	Reference	-	Reference	-	Reference	-
Age group, years									
<50				Reference	-	Reference	-	Reference	-
50–<60				1.49 (1.05–2.12)	0.026	1.27 (0.85–1.88)	0.244	1.33 (0.90–1.98)	0.157
60–<70				2.67 (1.92–3.71)	0.001	1.96 (1.33–2.87)	0.001	1.91 (1.30–2.80)	0.001
70–<80				4.57 (3.31–6.33)	<.001	2.72 (1.84–4.03)	<.001	2.61 (1.76–3.86)	<.001
80+				9.64 (6.89–13.50)	<.001	4.92 (3.24–7.48)	<.001	4.01 (2.64–6.10)	<.001
Gender									
Male				1.15 (0.999–1.31)	0.052	1.10 (0.95–1.29)	0.205	1.10 (0.95–1.29)	0.211
Female				Reference	-	Reference	-	Reference	-
Killip Class									
I						Reference	-	Reference	-
II–IV						2.38 (2.02–2.80)	<.001	2.08 (1.76–2.46)	<.001
Troponin quintile									
1						0.97 (0.76–1.24)	0.825	0.80 (0.62–1.02)	0.066
2						0.81 (0.64–1.04)	0.095	0.70 (0.55–0.89)	0.004
3						0.76 (0.61–0.96)	0.020	0.71 (0.56–0.89)	0.004
4						0.99 (0.81–1.21)	0.915	0.91 (0.75–1.12)	0.375
5						Reference	-	Reference	-
CAD									
No significant CAD						Reference	-	Reference	-
Single VD						0.87 (0.66–1.14)	0.305	2.07 (1.52–2.82)	<.001
Double VD						1.03 (0.77–1.37)	0.858	2.27 (1.66–3.11)	<.001
Three VD and/or Graft						1.60 (1.23–2.08)	0.001	3.23 (2.41–4.33)	<.001
Cardiac arrest									
Yes						2.38 (1.89–2.99)	<.001	1.85 (1.46–2.34)	<.001
No						Reference	-	Reference	-

Table 5: All-cause mortality in the year after a first MI: multivariable models (continued).

ACS Type									
NSTEMI						Reference	-	Reference	-
STEMI						1.83 (1.54–2.17)	<.001	1.96 (1.65–2.33)	<.001
Prior CVD									
Yes						1.12 (0.95–1.33)	0.183	1.06 (0.89–1.25)	0.515
No						Reference	-	Reference	-
History of CHF									
Yes						1.68 (1.31–2.16)	<.001	1.60 (1.25–2.05)	<.001
No						Reference	-	Reference	-
Current smoker									
Yes						1.17 (0.97–1.42)	0.097	1.16 (0.96–1.40)	0.126
No						Reference	-	Reference	-
Diabetes									
Yes						1.38 (1.17–1.63)	<.001	1.42 (1.20–1.69)	<.001
No						Reference	-	Reference	-
TC:HDL						0.92 (0.87–0.96)	0.001	0.91 (0.86–0.95)	<.001
Hypertension									
Yes						0.89 (0.77–1.02)	0.097	1.02 (0.88–1.17)	0.838
No						Reference	-	Reference	-
eGFR stages, ml/ min/1.73m²									
>90						Reference	-	Reference	-
60–90						1.37 (1.01–1.86)	0.041	1.43 (1.05–1.93)	0.022
30–16						2.44 (1.78–3.34)	<.001	2.38 (1.73–3.26)	<.001
15–30						4.26 (2.80–6.48)	<.001	3.18 (2.09–4.85)	<.001
<15						9.00 (6.11–13.25)	<.001	6.97 (4.70–10.33)	<.001
COPD									
Yes						1.38 (1.13–1.69)	0.002	1.22 (0.997–1.49)	0.053
No						Reference	-	Reference	-
Revascularisation									
Yes								0.67 (0.56–0.79)	<.001
No								Reference	-
Statin									
Yes								0.31 (0.26–0.37)	<.001
No								Reference	-
ACEI/ARB									
Yes								0.58 (0.50–0.67)	<.001
No								Reference	-
Beta Blocker									
Yes								0.46 (0.40–0.54)	<.001
No								Reference	-
DAPT									
Yes								0.64 (0.54–0.75)	<.001
No								Reference	-

CAD, Coronary artery disease; VD, vessel disease; ACS, Acute coronary syndrome; CVD, Cardiovascular Disease; CHF, Congestive heart failure; TC:HDL, total cholesterol to high density lipoprotein ratio; eGFR, Estimated glomerular filtration rate; COPD, Chronic obstructive pulmonary disease; DAPT, Dual antiplatelet therapy. For detailed description of the multi-variable regression models please refer to the statistical analysis section above.

Figure 1: Age and sex adjusted cumulative mortality.



Discussion

In this real-world nationwide study, Māori and Pacific people presenting with their first myocardial infarction were younger, but had more advanced cardiac disease, were more acutely unwell, and had higher case fatality rates at one year compared with New Zealand European/other patients. In particular, Māori and Pacific patients were more likely to have acute heart failure and LV dysfunction, and Māori were more likely to present with a cardiac arrest, despite a similar ratio of STEMI to NSTEMIs. Indian and Other Asian patients presenting with their first MI were also younger than New Zealand European/other patients. Pacific peoples, followed by Indian, were more likely to have severe obstructive coronary artery disease at their first admission than other ethnic groups. There were differences in the burden of modifiable cardiovascular risk factors with smoking and associated COPD most frequent in Māori and Pacific patients. Pacific and Indian peoples had the highest prevalence of diabetes, with higher prevalence also observed among Māori and Other Asian people compared to the European/other group. Despite having similarly high prevalence of diabetes and hypertension, fewer Indian patients had advanced CKD than Pacific patients. Indian patients also had less advanced CKD than Māori patients despite diabetes being less frequent in Māori.

The age and sex adjusted one-year mortality was 2.5 times and 3 times higher for Māori and Pacific patients, respectively, compared to European/others, while Indian and Other Asian patients had outcomes similar to European/others. After adjustment for differences in the clinical presentation, risk factors and comorbidity, the excess mortality associated with Māori or Pacific ethnicity was significantly attenuated, although remained at about 1.8 times and 1.4 times higher risk, respectively. Adjustment for differences in in-hospital treatment did not modify this further. These findings suggest that at least half of the inequity in outcomes for Māori, and three

quarters for Pacific people, is associated with differences in preventable or modifiable clinical factors, and could therefore be reduced by improvements in healthcare delivery in primary care and in the acute, community-to-secondary care interface. The remaining—unaccounted for—differences in mortality require further study, but may include differences in other modifiable factors including medical comorbidities and inequities in healthcare access and delivery both before hospitalisation and post-discharge.

What is already known

In New Zealand, despite ongoing reductions in IHD mortality, there are persisting major ethnic inequalities in IHD mortality, with Māori and Pacific patients having approximately double the European age standardised mortality rate.[4] Ischaemic heart disease accounts for 40.2% of Māori deaths in those aged less than 65 years, compared to 10.5% of non-Māori deaths.[13] Furthermore, we have previously reported similarly disproportionate rates of IHD mortality both in Māori and Pacific patients who die before they can be hospitalised, as well as those who die after a hospitalisation.[3,14] The causes of inequity in IHD outcomes will be multifactorial, including differential exposure to CVD risk factors, socioeconomic deprivation, and unequal access to healthcare, utilisation of primary prevention and treatment. The majority of premature IHD incidence is attributable to uncontrolled but potentially modifiable risk factors.[15,16]

In this study we have shown, at a national level, that there are ethnic differences in potentially modifiable risk factors which can be identified across the care continuum, from primordial prevention in primary care through to post-hospital discharge. These potentially modifiable factors explain at least half of the inequity in outcomes observed.

Ethnic differences in clinical presentation

Māori patients were more likely to present with a cardiac arrest. Cardiac arrest is associated with worse outcome post-MI but its impact can be ameliorated by effective CPR and early cardioversion.[17,18] The most effective way to improve access to defibrillation is to reduce the time between symptom onset and the call for medical help with subsequent ambulance attendance or utilisation of community automated external defibrillators. Despite community programmes in New Zealand aimed at increasing recognition of MI symptoms and encouraging people to call for help there remain long delays in making the call. In the ANZACS-QI national cohort of patients with STEMI the median delay to call for help was 45 minutes for ambulance-transported patients and 97 minutes for those self-transported to hospital. That delay was more common in older people, Māori and Indian peoples and those self-transported to hospital.[19]

Both Māori and Pacific peoples were more likely to present with acute heart failure and worse LV systolic function. Both of these are associated with more adverse outcomes after MI.[20,21] They may be presenting with larger heart attacks, although the similar proportions of all ethnic groups in the highest quintile of peak Troponin T, a measure of MI size,[22] argues against this. An alternative explanation is that Māori and Pacific patients have more pre-existing cardiac disease, making them more susceptible to developing acute heart failure despite similar amounts of acute myocardial necrosis. Both of these explanations point to possible interventions. Delayed presentation reduces the opportunity for early medical treatment and revascularisation of both culprit and non-culprit coronary lesions. Remarkably, over a quarter of patients in all ethnic groups and over a third of Pacific and Indian patients had severe three vessel or left main stem coronary artery disease when they presented with their first MI. Because an acute MI is usually due to sudden occlusion of one coronary artery these patients must have had pre-existing but unrecognised obstructive CAD prior to their first MI. Earlier identification of both asymptomatic coronary artery disease and heart failure/LV dysfunction, and their determinants, are an opportunity to modify the disease course using well established lifestyle and pharmacological interventions.

The comparatively higher rate of MI without obstructive coronary artery disease in Maori, and to a lesser extent in Pacific patients also requires further investigation—in particular whether these patients have atherosclerotic plaque rupture or,

alternatively, have other cardiac disease (cardiomyopathy and arrhythmia) for which other management is required.

Ethnic differences in modifiable longer-term determinants of risk

The most clinically important ethnic differences in CVD risk factors included excess smoking and associated COPD in Māori, excess diabetes in Pacific, Indian and Māori patients, and excess CKD in Pacific and to a lesser extent in Māori and Indian patients. Our group has described and discussed the differences in traditional risk factors in an earlier, smaller ANZACS-QI cohort.[23] Both smoking and diabetes mellitus and their clinical sequelae are potentially preventable risk factors.[24,25] Although CKD is not a traditional risk factor it is predominantly caused by poorly controlled diabetes and high blood pressure, and is in that sense, a surrogate for those more traditional, treatable risk factors. In our multivariable analysis and a prior ANZACS-QI report, advanced CKD conferred a 5–10-fold excess mortality risk compared with patients with normal renal function.[26] Of importance, despite having similar high rates of diabetes, Indian patients had less advanced CKD than Pacific patients. Indian patients were also less likely to have advanced CKD than Maori patients, despite having more frequent diabetes.

The triad of diabetes, high blood pressure and CKD are important determinants of the LV dysfunction and pre-clinical CAD discussed above. The causative pathway is complex—diabetes and high blood pressure are in part determined, and potentially modifiable, by lifestyle factors including physical activity, diet and weight which are in turn related to the wider determinants of health including poverty, education, housing and institutionalised and interpersonal racism.[27,28] Of note, nearly half of the Māori and Pacific patients in this cohort lived in the poorest geographical quintile in New Zealand and a higher burden of cardiovascular risk factors and multi-morbidity is associated with higher levels of deprivation.[29,30]

Ethnic differences in treatment

Adjustment for differences in treatment in the multivariable analysis had only a minor impact on the risk estimates of each variable. All patients in this cohort underwent coronary angiography, but there were small differences in overall revascularisation rates and type of revascularisation. In particular revascularisation occurred among 75% of European/others, 70.5% in Pacific and 67% in Māori patients. This difference has been reported and investigated in depth in prior reports. At one large metropolitan hospital where this difference was studied this was largely explained by differences in the nature of the CAD, with more non-obstructive disease in Māori and Pacific which does not require revascularisation, combined with more diffuse small vessel coronary artery disease in some patients with diabetes, which is not suitable for revascularisation and is more appropriately treated medically.[6] However, in another hospital anatomic and clinical factors did not explain all the differences in revascularisation between ethnic groups.[31] Each cardiology unit in the country should audit their practice and review processes to ensure that institutional racism does not contribute to the observed lower rate of invasive coronary investigation and management in Maori and Pacific.[32] Māori and Pacific patients had lower rates of dual anti-platelet therapy prescription on discharge in this study, likely due to the more frequent finding of non-obstructive CAD and the higher rates of CABG. In New Zealand, contrary to guidelines, the use of DAPT for these two indications is known to be low,[33,34] and an opportunity for improvement.

Ethnic differences in outcomes

This study has established that at least half of the excess in ACS mortality between European/other and Māori and three-quarters of that between Pacific and European /other patients, is related to potentially modifiable or preventable clinical factors and could therefore be reduced markedly. Modification of these factors span the continuum of care and life course from primordial prevention of risk factors to primary prevention management of risk factors in the community, to acute pre-hospital and in-hospital care, and then to post-discharge secondary prevention in primary care. This and prior studies referenced above have documented ethnic differences in risk factors and clinical management at each stage in this continuum. In many cases the differences

are small but cumulatively may add up to a large impact on outcome. The implication of this finding is that improvement initiatives are required to identify and address barriers to appropriate care for Maori and Pacific people at every stage in the continuum. It is likely that disparities in cardiovascular risk are perpetuated by the wider determinants of health including poverty, education, housing and institutionalised and interpersonal racism, and that addressing these determinants will be required to achieve equitable health outcomes. Further research is needed to determine the relative importance of these various factors.

Limitations

Only patients who received a coronary angiogram in the public health system of New Zealand were included in this study. However, given other data showing excess IHD mortality in Māori and Pacific patients out of hospital it is likely that similar conclusions apply to those patients. With further variable refinement, some differences in risk between ethnic groups might have been more marked. For example we have previously reported more suboptimal glycaemic control and proteinuria in Pacific compared with European patients.[6] Those variables are not available at a national level for inclusion in this study. We did not adjust for post-discharge medication adherence but have previously reported differences by ethnicity.[6,35,36] We had no access to other variables which might be very important including physical activity, family support, health literacy level and health beliefs.

Conclusion

In New Zealand, at first presentation with MI, Māori, Pacific and Indian patients are younger, have more advanced cardiac disease and a greater burden of CVD risk factors compared with European/others, and there is a three-fold variation in one-year mortality based on ethnicity. Over half of this inequity in outcomes is associated with differences in potentially preventable or modifiable factors and could therefore be reduced by improvements in primordial and primary prevention in the community, and in healthcare delivery in primary and secondary care and at the community-to-secondary care interface.

Competing interests:
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Racism and health in Aotearoa New Zealand: a systematic review of quantitative studies

Natalie Talamaivao, Ricci Harris, Donna Cormack, Sarah-Jane Paine, Paula King

ABSTRACT

BACKGROUND: Racism is an underlying cause of ethnic health inequities both in Aotearoa New Zealand and internationally. It is timely to synthesise racism and health research within New Zealand particularly given the current policy environment and shift towards addressing the health effects of racism.

AIM: To review quantitative research examining self-reported experiences of racial discrimination and associations with measures of health (health conditions, health risk, health status and healthcare) in New Zealand.

METHODS: MEDLINE, PsycINFO, Web of Science and CINAHL databases were searched for studies reporting on associations between experiences of racism and health.

RESULTS: The systematic review identified 24 quantitative studies reporting associations between self-reported racial discrimination across a wide range of health measures including mental health, physical health, self-rated health, wellbeing, individual level health risks, and healthcare indicators.

CONCLUSIONS: Quantitative racism and health research in New Zealand consistently finds that self-reported racial discrimination is associated with a range of poorer health outcomes and reduced access to and quality of healthcare. This review confirms that experience of racial discrimination is an important determinant of health in New Zealand, as it is internationally. There is a pressing need for effectively designed interventions to address the impacts of racism on health.

Racism has been firmly established as an important determinant of health and an underlying cause of ethnic health inequities in Aotearoa New Zealand and internationally.[1–3] Racism is an organised system of oppression involving the social construction and valuing of racial/ethnic groups based on ideologies of superiority (and inferiority), which serves to privilege some groups over others.[4–6] In its institutional and cultural forms, racism has been, and continues to be, a major contributor to the creation and sustaining of racial/ethnic inequities across a range of societal outcomes combining to create inequities in health.[1,2]

Racism operates at multiple levels with various pathways to health.[7] These levels have been conceptualised by some scholars as internalised (or intrapersonal), interpersonal (personally-mediated) and systemic (structural or institutional).[2,6,7] Internalised racism involves attitudes, beliefs or ideologies often founded on understandings of supposedly innate superiority and inferiority that may be held by members of dominant social groups and/or oppressed ones.[2] Interpersonal racism refers to racism between people, with varying degrees of frequency and intensity, including manifestations from racially motivated assault to verbal abuse, ostracism and exclusion.[2] Systemic, structural or institutional racism involves the production, control and access to material, informational and symbolic resources within societal institutions, laws, policies and practices.[2]

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It has been posited that structural racism is the most powerful way that racism impacts on population health.[7] This view acknowledges how deeply embedded social structures in society are and how they ultimately determine the inequities arising from unequal access to the wide range of factors that drive health.[5] Interpersonal racism reflects the direct experience of individuals within the organised system of racism that operates within a colonised society. Interpersonal racism can impact negatively on health in multiple ways such as a chronic stressor, and experience of unfair treatment.[5,6,8]

In New Zealand there is a long standing body of qualitative research[8–11] as well as a large and growing research base examining peoples' experiences of racism and potential impacts on health by ethnicity, particularly in the last decade. The majority of this evidence has centred around measures of self-reported experiences of racial/ethnic discrimination. Research shows a consistent link between experience of racism and a range of negative health measures (such as mental and physical health, and individual level factors such as smoking) that may impact on racial/ethnic health inequities[1,12] and negatively impact on access to healthcare and experiences of healthcare interactions.[13] Unsurprisingly, research consistently shows that non-European ethnic groups, including Māori, Asian and Pacific, have higher prevalence of reporting experience of racism than European ethnic groups.[1,12] A strength of the New Zealand-based studies has been the focus on inequity analyses, which centre Māori and often conceptualise racism as a determinant of health within a context of the enduring and harmful impacts of colonisation.[14]

International systematic reviews show strong and consistent associations when examining the links between racial/ethnic discrimination and health, particularly for mental health indicators.[3,5,15] Systematic reviews have examined experience of racism and dimensions of the health and disability system such as service utilisation[16] and specific population groups such as children and young people.[17] However, these reviews are largely dominated by studies undertaken in the US.

Recently, the New Zealand policy environment has begun to acknowledge the impacts of racism and relationships to health, particularly the role of institutional racism.[18–22] Given the current environment and the opportunity to inform emerging policy and intervention development, it is timely to collate and synthesise the available body of quantitative racism and health research within New Zealand.

This paper seeks to investigate and report on the quantitative association between self-reported experience of racial/ethnic discrimination and health within a New Zealand setting. It draws upon systematic review methods used in previous reviews set within an international scope[3,16] in order to provide an overview for New Zealand on the range of health indicators that racism has been linked to and the consistency and strength of these associations.

Methods

This systematic literature review followed the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guideline. The Medline, Web of Science, CINAHL and PsycINFO databases were searched systematically to identify articles published before May 2019 (no lower date limit was specified). Search terms were based on terms from previous international systematic reviews and the thesaurus for each database, utilising MeSH terms where possible. A copy of the Medline search strategy is provided in Appendix Table 1. An additional step to the PRISMA process was the addition of three articles sourced via database alerts (between May and October 2019) and assessed as meeting the pre-set criteria and, therefore, included in the final dataset.

Study selection

Articles were included if they were a) undertaken in New Zealand, b) reported empirical, quantitative study findings **and** c) reported an association between self-reported experience of racial/ethnic discrimination and one or more health-related measures. Self-reported experience of racial/ethnic discrimination included experience of racism on the basis of race, ethnicity and/or skin colour. Studies were excluded if

Table 1: Summary of characteristics of studies of self-reported experiences of racial/ethnic discrimination and health in New Zealand.

	Number of studies	% of total studies n=24
Publication year		
2005–2007	1	4.2%
2008–2010	0	
2011–2013	5	20.8%
2014–2016	6	25.0%
2017–2019	12	50.0%
Study location		
National	19	79.2%
Regional	4	16.7%
Local (eg, study clinic)	1	4.2%
Study type*		
Cross-sectional	21	87.5%
Longitudinal	5	20.8%
Sample size		
<100	1	4.2%
100–199	0	
200–499	1	4.2%
500–999	2	8.3%
1,000–4,999	5	20.8%
5,000–9,999	7	29.1%
>10,000	8	33.3%
Study populations		
Ethnicity		
Māori	22	91.7%
Pacific	17	70.8%
Asian	15	62.5%
NZ European	8	33.3%
NZ European/Other	9	37.5%
Middle Eastern, Latin American and African	1	4.2%
Other	1	4.2%
Age of study populations		
Infants (first 12 months)	3	12.5%
Children (0–14 years)	2	8.3%
Adolescents (13–18 years)	5	20.8%
Adults (15 years or over)	18	75.0%
Aged 80+	1	4.2%
Gender of participants		
Female and male	21	87.5%
Female only	3	12.5%
Male only	0	
Specific population groups		
Caregivers	2	8.3%
Secondary school students	5	20.8%
Immigrant	1	4.2%

Table 1: Summary of characteristics of studies of self-reported experiences of racial/ethnic discrimination and health in New Zealand (continued).

Racism exposure measurement		
Exposure scales		
NZ Health Survey racism module questions (five items)	11	45.8%
Growing up in New Zealand study questions	3	12.5%
General social survey two-step questions	2	8.3%
Youth survey (bulling, unfair treatment measure)	5	20.8%
Perceived discrimination single item (NZAVS)	4	16.7%
Everyday discrimination scale	1	4.2%
Timeframe of exposure		
Last 12 months	2	8.3%
Ever	7	29.2%
Mixed (last 12 months and ever)	6	25.0%
Not reported/specified	9	37.5%
Single-item or multi-item measures		
Single-item measure	6	25.0%
Multi-item measure	18	75.0%
Exposure setting		
Healthcare setting only	2	8.3%
General and healthcare	17	70.8%
General only	5	20.8%
Method of administration		
Self-administered	9	37.5%
Interviewer-administered	15	62.5%
Missing racism data identified/discussed		
Yes	4	16.7%
No	20	83.3%

*Thayer[23] and Stronge[24] incorporated both cross-sectional and longitudinal study elements and are included in both categories.

they were not published quantitative studies and/or did not report on a direct association between experience of racial/ethnic discrimination and a health outcome.

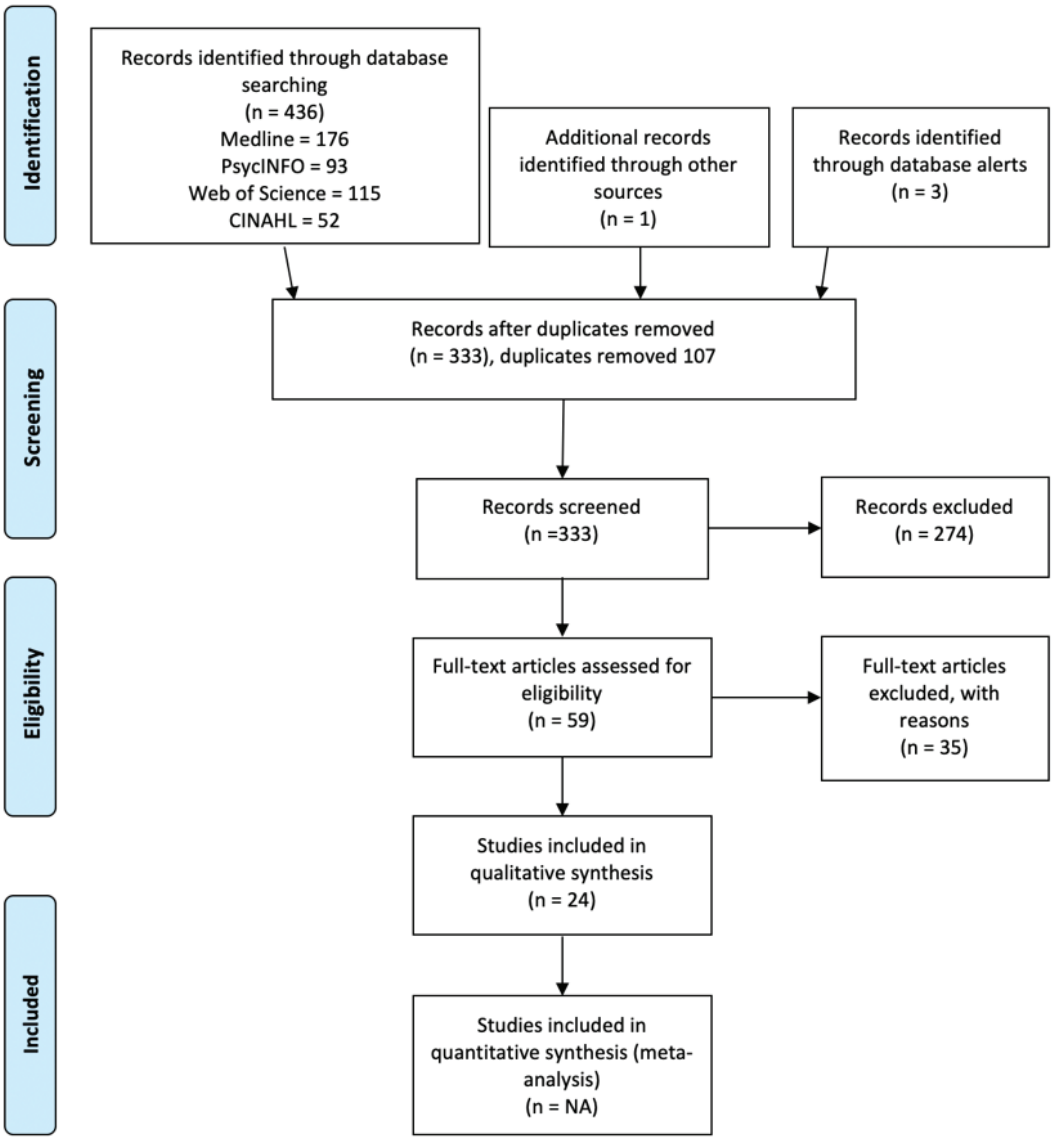
In total, 436 articles were initially identified across the four databases, with a number of duplicates (n=107). A further article was located through other sources as it was known to the investigators due to their involvement in the study. Three articles were sourced via database alerts (from the same search terms) in the four months following the initial database search. After removing duplicates and reading through titles and abstracts, 59 articles were identified for retrieval. Retrieved articles were reviewed via full text screening to ascertain relevance and fit with inclusion criteria, and a further 35 articles excluded. One investigator initially screened all the abstracts retrieved from database searching where exclusions were clear. Article abstracts that were unclear were then reviewed with two other investigators and consensus obtained on exclusions and inclusions. Full text screening review included discussion and decision making with two other investigators regarding the inclusion of particular studies. These studies were independently screened by the two other investigators and a consensus agreement made on the final dataset.

Data extraction and appraisal

The final dataset was made up of 24 studies (Figure 1).

Each paper in the final dataset was reviewed and associations between racism and health assessed via the strength of evidence presented in analysis. Relevant information was entered into Microsoft Excel™ based on pre-determined categories (eg, sample size, study approach) by one investigator. A meta-analysis was not undertaken for this systematic review due to the

Figure 1: PRISMA guideline flow diagram.



broad range of outcome measures across the studies. Of note, however, is that some individual studies used meta-analytic methods to pool data across multiple surveys of the same survey to increase precision of estimates.[1,12]

Associations between self-reported experience of racial/ethnic discrimination and health were grouped into broad health indicator categories (eg, mental health, physical health) based on the studies in this review and informed by previous systematic reviews.[3,16] Study characteristics are presented in the findings, using a qualitative approach to synthesis.

Results

Characteristics of included studies

Table 1 displays the characteristics of the 24 studies included. The volume of published studies has increased steadily since 2010, with most studies being published from 2014 (n=18). Over half of the studies had sample sizes of over 5,000 people (15, 63% of studies). The majority of studies were conducted at a national level (19, 79%) and cross-sectional in study design (21, 88%). Three studies were longitudinal in design using the Growing up in New Zealand (GUINZ) study data.[25–27]

Two studies[23,24] utilised both longitudinal and cross-sectional analyses and therefore feature in both categories.

Studies undertaken with adult populations generally included all genders. A few studies focused on maternal health.[23,27,28] Some focused on a specific population group, eg, infants,[28] caregiver's experiences of racism linked with children's health,[29,30] adults aged 80 years and over,[31] and immigrant status among secondary school students.[32]

Measurement of racial/ethnic discrimination

An analysis of the exposure scales used in the studies showed that 11 studies (46% of all studies) used the New Zealand Health Survey (NZHS) racism questions. The NZHS questions, which feature multiple times in the NZHS since 2002/03, have provided the ability to explore experience of racism (using five items) and health for various health conditions, outcomes and settings, including two studies exploring the association of racism and health over time.[1,12] The questions informed development of racism questions in other studies such as GUINZ and The National Youth Health and Wellbeing Survey (Youth 2000).

The majority of studies presented experience of racism data that was self-reported and represented a direct experience for participants. The exception to this was the investigation into vicarious racism experience for children, where caregivers' experience of racism and resulting impact on their children's health was examined.[29,30]

The exposure setting for the majority of the studies were 'both general and healthcare related' reflecting the multi-item nature of many of the measures.

The timeframe within which exposure to racism was examined varied across studies. Excluding those studies (n=9) which did not report or specify a timeframe, most studies used an 'ever' or 'mixed' (last 12 months and ever) timeframe (n=13). Two studies used a timeframe of last 12 months.[1,33]

Most studies used a multi-item measure to assess experience of racial/ethnic discrimination (eg, examination across a number of items and settings). Six studies used a single-item measure to assess racial/ethnic discrimination either by examining one dimension of experience of racism, eg, unfair treatment by a health professional,[26,34] or a broader question exploring participants' response to a single question of feeling discriminated against because of ethnicity.[24,35,36]

Over half (n=15, 63%) of the studies used interviewer-administered data sources, largely reflecting the sizeable body of work that utilises the NZHS racism question set, as well as GUINZ studies,[25,27,28] Life and living in advanced age: a cohort study (LiLACS) data[31] and a small-scale study conducted in a health clinic.[23] Self-administered data was sourced via surveys that were either postal (eg, New Zealand Attitudes and Values Study, NZAVS) or via electronic data collections (eg, Youth 2000).

Categorisation of racism variables

Many studies used data from multi-item experience of racism questions and created dichotomous or composite variables for analysis.[31,37,38] A number of studies explored experience of racial/ethnic discrimination from multiple items, examining a dose-response relationship with health measures.[25,29,30,39,40]

Ethnicity

Ethnicity is a key variable in understanding the risk of experiencing racial/ethnic discrimination. Most studies provided detail on ethnicity data and are categorised using the Statistics New Zealand Census question and/or classification that allows for self-identification of ethnicity. Six studies focused on the Māori population only.[24,31,35,36,38,41] Two studies presented analysis focusing specifically on Pacific.[34,42] Remaining studies included analysis across the major ethnicity population groupings in New Zealand (eg, Māori, Pacific, Asian, New Zealand European) with nearly all studies including Māori as a study population (22, 92%). Of note, with the exception of Māori, ethnic groupings were an aggregate of multiple ethnic groups. When measured within the body of racism and health research in New Zealand the prevalence of experience of racism was consistently highest among Māori, Pacific and Asian populations.

Missing racism data

A qualitative assessment of missing racism data was undertaken to obtain a sense of the quality of the major exposure

being tested. Only a few studies examined missing racism data in any detail. Harris[12,43] identified missing data numbers of 116 (0.93% of participants) in the 2002/03 NZHS, eight (0.06% of participants) in the 2006/07 NZHS and 165 (1.3% of participants) in the 2011/12 NZHS.

Covariates

Many of the studies adjusted for age, gender and socioeconomic variables when examining associations between racism and health. Some of these studies explicitly considered socioeconomic position (SEP) as a marker of institutional racism given entrenched ethnic inequities by SEP in New Zealand.

Associations between self-reported experience of racial/ethnic discrimination and health

Table 2 summarises the associations between self-reported experience of racial/ethnic discrimination and health measures reported in the studies.

Associations are grouped into broad health outcome categories (studies can feature multiple times).

Mental health

An association between experience of racism and negative mental health was found across six studies that examined depression (including pre-natal/post-natal), stress, psychological distress and diagnosed mental health conditions.[12,25,35,37,38,42] Using longitudinal data, Bécares[25] found strong associations between experiences of ethnically-motivated interpersonal attacks and unfair treatment on maternal mental health among Māori, Pacific and Asian women. Studies also found a negative association for lower scoring on SF12/36 mental health scales,[1,12,33,39] feelings of control[35] and previous suicide attempt.[38]

Wellbeing/overall health

A number of studies explored experience of racial/ethnic discrimination and resulting impact on self-rated health—with associations with poorer health confirmed in all studies.[1,12,23,33,35,37,39] Experience of racism was associated with negative life satisfaction in five studies,[1,24,33,35,42] with one study using longitudinal data.[24] Other indicators of wellbeing that also showed an association between experience of racism and negative outcomes were overall wellbeing, quality of life measures, happiness, self-esteem, subjective evaluation of health and body satisfaction.[30,31,35,38,42,44]

Physical health

A few studies explored the association between racism and physical health measures. Harris[12,39] found a negative health association for CVD and SF36 physical health scale scoring. Hobbs,[28] using longitudinal data linked to national hospitalisations, found that maternal experience of healthcare-based racism was associated with increased infectious disease hospitalisations for Māori infants. In a study of caregiver experiences of racism and child health outcomes, no association was found between experience of vicarious racism and medicated asthma for children.[30]

Health related behaviours

The association of experience of racism was less clear when examining health-related behaviours. Associations between experience of racism and factors linked to increased health risk were found in four studies examining cigarette smoking,[12,32,37,39] and four studies that analysed hazardous or binge drinking.[12,32,37,41] Muriwai,[36] in an examination of perceived appearance and smoking status, found an unexpected lack of association between perceived ethnic discrimination and smoking status among Māori. Ethnic discrimination was considered a covariate in this analysis and it is possible that the perceived appearance variable also partly captured experience of racism, potentially over-adjusting for racism. No relationship was demonstrated between experience of racism and hours of watching TV, exercise[35] and body size.[12]

Healthcare

Five studies examined healthcare measures and relationships to experience of racial/ethnic discrimination.[1,13,29,34,35] Healthcare measures included unmet need, access to services, patient satisfaction and patient experience with healthcare services. In all but one study, experience of racism was associated with negative healthcare measures, however the association with lower cancer screening (breast and cervical) was only significant for Māori women.[13] No association was found between vicarious racism and children not having a usual healthcare provider.[29]

Table 2: Associations between experience of racial/ethnic discrimination and health.

Health indicators	Association with negative health	Association with positive health	No association	Studies
Mental health				
Pre-natal/post-natal depression	1			25
Depressive symptoms	2			37,38
Pre-natal perceived stress	1			25
Psychological distress	3			12,35,42
Diagnosed mental health condition	1			12
Suicide attempts	1			38
SF 12/36 mental health	4			1,12,33,39
Feelings of control over life	1			35
Wellbeing/overall health				
Self-rated health	7			1,12,23,33,35,37,39
Life satisfaction	5			1,24,33,25,42
Overall wellbeing	2			38,42
Quality of life	2			30,31
Happiness	1			44
Self esteem	2			35,42
Subjective evaluation of health	1			42
Body satisfaction	1			35
Physical health				
Childhood asthma (medicated)			1	30
Cardiovascular disease	2			12,39
SF36 physical health	2			12,39
Infectious disease hospitalisations (first year of life)	1			28
Health related behaviours				
Cigarette smoking	4		1	12,32,36,37,39
Marijuana use	1			32
Alcohol use (hazardous or binge)	4			12,32,37,41
Watching TV			1	35
Exercise			1	35
Body size (BMI, waist circumference)			1	12
Healthcare				
Patient experience	1			13
Unmet need	2			29,43
Satisfaction with health care service	2			29,43
Access to healthcare service	2		1	29,34,35
Cancer screening (breast and cervical)	1*		1	13
Maternal health				
Morning maternal cortisol levels	1		1	23
Evening maternal cortisol levels				23
Maternal diurnal cortisol decline	1		1	23
Infants higher cortisol reactivity at six weeks				23
Birth weight	1*	1#	1^	27
Gestation length	1*			27
Other health indicators				
Feeling safe in neighbourhood	1			37
Sleep disturbance/poorer sleep	2			35,40
Fatigue	1			35
Relationship dissatisfaction/conflict			1	35

* association found for Māori women only.
\# association found for Asian women only.
^ association found for Pacific women only.

Maternal health indicators

Thayer & Kuzawa,[23] using cross-sectional and longitudinal methods, focused on cortisol levels (as an indicator of stress in pregnancy and infants soon after birth) with a small sample of pregnant women (n=55) and found associations with higher maternal evening cortisol and higher cortisol reactivity for a sub-set of infants at six weeks of age (n=19). Experience of racism had no association with maternal levels of morning cortisol and diurnal cortisol. A study using GUINZ longitudinal data explored maternal experiences of ethnic discrimination and birth outcomes and found that for Māori women there was an association with lower birth weight.[27] For Pacific women, no association was found and for Asian women experience of racism was associated with higher birth weight (often categorised as a positive health). Discussing the unexpected finding, the authors noted that Asian women had the lowest birth weights out of all ethnic groups and postulated that it was possible that higher birth weight may actually represent a less healthy birth weight.[27]

Other health indicators

Experience of racism was associated with negative health measures in two studies that examined racism and sleep disturbance[40] or poorer sleep.[35] Houkamau[35] also found that experience of racism was associated with fatigue, however experience of racial/ethnic discrimination had no association with relationship dissatisfaction and conflict. In a study of secondary school students, Crengle[37] found that experience of racism had a negative effect on feeling safe in your neighbourhood.

The majority of associations between experience of racism and health outcomes were based on self-reported outcome data. The exceptions to this were indicators reported in three studies including: Hobbs[28] who utilised data linkage from a longitudinal data set with a national database of hospitalisations; Thayer[23] who reported on measured cortisol levels in mothers and infants; and Harris[12] who used BMI and waist circumference measurements.

Overall, the patterning of associations shows a fairly consistent relationship between experience of racism and poorer health outcomes. This is particularly marked for mental health and wellbeing measures and is consistent with international literature.[15] The majority of studies demonstrate the disproportionate exposure to experience of racism for the Māori population and other minoritised ethnic groups (namely, Pacific and Asian peoples). Some studies found experience of racism as a predictor for negative health outcomes for the Māori population only while an association was not found for other population groups.[13,27] Studies examined a broad range of health outcomes (41 indicators across 24 studies) with associations between racism and negative health measures demonstrated for 34 indicators.

Discussion

This systematic review confirms that quantitative research on racism and health in New Zealand generally shows experience of racial/ethnic discrimination to be linked to poorer health outcomes. These findings are consistent with evidence internationally, which also demonstrate relationships between experience of racism and a range of health outcomes, with particularly strong and consistent associations for mental health.[3,15] In alignment with international studies,[16] New Zealand studies also demonstrated how experience of racial/ethnic discrimination was associated with more negative primary healthcare experiences, unmet need or lower healthcare utilisation, signifying the impact that experience of racism has in the context of health system design, functioning and quality of care. The studies included a range of population groups by age, ethnicity, gender and role (eg, caregivers). Of note, however, is the lack of quantitative analysis which further explores what is happening for social groups within the broader ethnic groupings (eg, disability and rainbow communities).

An advantage of this systematic review is that it is not limited or restricted to particular health conditions or outcomes and populations and can be regarded as a comprehensive overview of the available quantitative evidence. A further strength was that most studies provided a clear understanding and definition of their experience of racism measure as well as measurement of ethnicity, which is

important considering that studies demonstrate racial/ethnic discrimination is disproportionally experienced by non-European ethnic groups.

A major strength for New Zealand is that national surveys such as the NZHS and the GSS have included experience of racial/ethnic discrimination measures multiple times since 2002/03, acknowledging the significance of racism as a health determinant and social wellbeing indicator. The NZHS, a health survey which collects information to monitor population health and provide evidence for health policy[45] uses a one-step questionnaire on experience of racial/ethnic discrimination across five items—experience of ethnically-motivated physical and/or verbal attack, unfair treatment because of ethnicity in health, housing or in work. Experience of racism questions can be measured 'in the last 12 months' or 'ever' if more than 12 months ago. The GSS collects data on experience of racism via a two-step question, also allowing for the monitoring of other forms of discrimination. Other significant surveys in New Zealand that collect experience of racism data include the Youth 2000 survey series and the NZAVS. The longitudinal study GUiNZ is an important source of racism and health data in New Zealand and strengthens the research base with regards to longitudinal analyses.

Limitations to this systematic review is that only quantitative research evidence found in published peer reviewed journals have been included. Unpublished research is not included, which may lead to publication bias. There is also the possibility that despite conducting a systematic review process, not all relevant studies were identified. The studies included in this review are focused on individual experiences of racism and do not involve group experience of racism or explore institutional forms of racism.

There are a number of related New Zealand studies that did not meet the inclusion criteria. These include studies that explored experience of racial/ethnic discrimination and associations with deprivation on ethnic inequalities,[46] ethnic consciousness,[47] ethnic density,[48] perceived religious discrimination[49] and how others perceive your ethnicity (socially assigned ethnicity).[50,51] A developing body of work centres on racism among the medical workforce, exploring ethnic bias among medical students.[52,53]

There is a significant body of qualitative health research that has also explored experiences of racial/ethnic discrimination in New Zealand.[8–11] Research focused on institutional racism and health is also emerging within New Zealand-based literature.[54–56] This work is reinforced by recent findings from the Waitangi Tribunal where the need to address institutional racism is clearly laid out.[22]

The majority of studies in New Zealand are cross-sectional in design with a need for further research to incorporate longitudinal design that could add considerably to the strength of evidence on racism and health. Longitudinal research can examine multiple dimensions of racism and mechanisms to understand where and how to intervene, as well as explore dimensions of disease and use life course analysis to assess the impact and exposure of racism across developmental stages and pathways to health and potentially assess key periods of increased risk.

There is also a need for further research that would explore the evidence around anti-racism strategies and interventions. Interventions to address the health impacts of racial/ethnic discrimination need to address racism at all levels. In New Zealand, while the monitoring of racism as a health determinant is included in national level surveys such as the NZHS (although always needing to be advocated for), and the evidence base is robust and growing, there is limited research in New Zealand on effective interventions and system change to address the negative impact of racism on health. There is a pressing need to research and implement effective health interventions which address and dismantle racism.[2]

The evidence is clear that experience of racism is a determinant of health that has a negative impact across a broad range of health outcomes in New Zealand. Additionally, Indigenous and minoritised ethnic groups are significantly more likely to experience racial/ethnic discrimination than the dominant New Zealand European group, and therefore, are disproportionately affected by the impacts of racial/ethnic discrimination on health outcomes. As such, there is a need to continue to understand and explore the relationship of experience of racism, particularly for those groups most

affected. Continued attention needs to focus on how the many manifestations of racism impact health with ongoing and expanded research using multiple methods and within the context and understanding of racism as a system operating at multiple levels. Now is the time for action in identifying and implementing policy initiatives/interventions to address the irrefutable negative impact racism has on health.[2]

Appendix

Appendix Table 1: Example Search Strategy Medline (adapted for other databases as required).

Search statement	Results
1	exp "health care (non mesh)"/ or "delivery of health care"/
2	exp "diseases (non mesh)"/
3	exp "psychiatry and psychology (non mesh)"/
4	(well-being or wellbeing).mp. [mp=title, abstract, original title, name of substance word, subject heading word, floating sub-heading word, keyword heading word, organism supplementary concept word, protocol supplementary concept word, rare disease supplementary concept word, unique identifier, synonyms]
5	1 or 2 or 3 or 4
6	(discrim* or bias or prejud* or hostil* or harrass* or bully* or (unfair and treat*) or oppress* or racis*).ti,ab,kf.
7	(rac* or ethnic* or cultur* or religio* or migr* or immigra* or refugee*).ti,ab,kf.
8	6 and 7
9	racis*.ti,ab,kf.
10	exp racism/
11	9 or 10
12	8 or 11
13	("new zealand" or "NZ").ti,ab,kf.
14	5 and 12 and 13

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URL:
www.nzma.org.nz/journal-articles/racism-and-health-in-aotearoa-new-zealand-a-systematic-review-of-quantitative-studies

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Inequalities between Māori and non-Māori men with prostate cancer in Aotearoa New Zealand

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ABSTRACT

Māori experience poorer health statistics in terms of cancer incidence and mortality compared to non-Māori. For prostate cancer, Māori men are less likely than non-Māori men to be diagnosed with prostate cancer, but those that are diagnosed are much more likely to die of the disease than non-Māori men resulting in an excess mortality rate in Māori men compared with non-Māori. A review of the literature included a review of the epidemiology of prostate cancer; of screening; of access to healthcare and of treatment modalities. Our conclusion was that there are a number of reasons for the disparity in outcomes for Māori including differences in staging and characteristics at diagnosis; differences in screening and treatment offered to Māori men; and general barriers to healthcare that exist for Māori men in New Zealand. We conclude that there is a need for more culturally appropriate care to be available to Māori men.

Prostate cancer is a major health concern globally. In 2015, prostate cancer was the most common incident of cancer in men, with an estimated 1.6 million cases worldwide, and fifth most common cause of cancer deaths for men, with an estimated 366,000 deaths worldwide.[1] Prostate cancer incidence varies more than 25-fold worldwide, with Australia/New Zealand having the highest age-standardised rates at 111.6 per 100,000.[2] In Aotearoa New Zealand, prostate cancer is the most commonly registered cancer, with 3,129 cases in 2013, and third most common cause of cancer death, with 647 deaths in 2013.[3]

Māori are the tangata whenua (Indigenous people) of New Zealand, with 16.5% of the country's population identifying as being Māori, while 64.1% identify as European, 15.1% Asian and 9.1% Pacific peoples.[4] The condition of Māori health is complex. It involves an interplay of social, economic and political factors, preceded by colonial history and land confiscations that resulted in a dispossession of language, identity and self-determination.[5] The contemporary reality of Māori includes societal racism that permeates the health sector at all levels and manifests as inequitable access, treatments and outcomes across most major illnesses.[6,7] Institutional and interpersonal racism and discrimination are significant contributing factors to Māori health inequalities.[8–11] Māori respondents in the New Zealand Health Survey (1996–7 and 2002–3) reported not visiting a GP, making fewer visits per year to see a GP or being sick enough to warrant seeing a GP but failing to do so.[12] A study by Jansen et al in 2011 found that Māori were less likely to see the GP they wanted, when they wanted and are offered fewer appointment options.[13] Even when Māori reported urgently needing to see a GP, Crengle et al found that GPs spent less time with them during the consultation, ordered fewer tests and made fewer referrals in comparison to non-Māori.[14]

Timeliness and access to healthcare are fundamental to improving the health inequities for Māori men. Vulnerable patients, including Māori, have fewer appointment options, further exacerbated if GP practices have rigid rules and restrictive hours of operation.[15,16] 'Access' may include the broader healthcare systems that operate at the structural, clinical and patient levels, which, for cancer patients, is shaped by interaction between and across all of these levels, and is generally facilitated through GP practices.[17] Therefore, the concept of whānaungatanga (rapport and relationship) is crucial to information sharing and care. Māori are less likely to access GP services where the fundamentals of whanaungatanga have not been established.[17] Mastering the fundamentals of cultural engagement with Māori men, particularly for health professionals of other ethnicities, will go some way to achieving equitable health outcomes or reduce the inequities.[18–20]

Consequently, Māori, like many Indigenous peoples around the world, experience the poorest health statistics in terms of cancer incidence and mortality when compared to non-Māori.[21] Prostate cancer is no exception, with Māori men disproportionately impacted in the New Zealand context.

This paper aims to examine the current knowledge as to the nature and cause of the disparities in prostate cancer mortality for Māori and identify opportunities for eliminating the demonstrated inequity.

Epidemiology of prostate cancer in Māori men

Prostate cancer is the second most common cause of cancer death for New Zealand men.[3] Although the incidence rate for Māori men being diagnosed with prostate cancer is lower than non-Māori men (RR 0.80, CI 0.73– 0.88),[3] Māori men had a prostate cancer mortality rate over 1.5 times that of non-Māori men (RR 1.51, CI 1.25–1.83).[22] Māori men newly diagnosed with prostate cancer are significantly more likely to die of the disease compared to non-Māori men.[23–25] In 2011, the age-standardised registration rate of prostate cancer for Māori men was lower than that for non-Māori men (81.4 per 100,000 vs 99.0 per 100,000). In comparison, the age-standardised prostate cancer mortality rate for Māori was higher than that for non-Māori (22.1 per 100,000 vs 16.2 per 100,000 men).[26] In 2013, the gap in the age-standardised registration rate of prostate cancer between Māori men and non-Māori men was smaller than in 2011 (91.8 per 100,000 vs 96.3 per 100,000), but the gap in the age-standardised prostate cancer mortality rate between Māori men and non-Māori men was much wider than 2011 (25.1 per 100,000 vs 17.1 per 100,000 men).[3]

Staging and characteristics at diagnosis

Previous New Zealand studies indicate that information on cancer staging is critical in order to identify reasons for ethnic disparities in survival and to aid decision making for the management of prostate cancer.[25,27–29] However, information on survival based on stage for men diagnosed with prostate cancer is rarely reported at a national level in New Zealand.[29] The data on prostate cancer staging in the New Zealand Cancer Registry (NZCR—a national collection of all cancer registrations in New Zealand) is incomplete, with approximately 75% of prostate cancer registrations having disease extent at diagnosis recorded as 'unknown'.[25]

A study carried out in a single urban population in New Zealand concluded that Māori men seem to present with a higher proportion of palpable disease than non-Māori (67.2% vs 53.3%).[16] On a regional scale, a recent study carried out in the Midland Region of New Zealand indicates that Māori men have a lower proportion of localised prostate cancer, and a higher proportion of metastatic disease than non-Māori (19.1% of Māori men with metastatic prostate cancer vs 9.8% for New Zealand Europeans).[29] In terms of survival, Māori men with locally advanced prostate cancer were more likely to die than non-Māori men; however, in this relatively small study there was no significant difference in survival rate for men with localised or metastatic prostate cancer between the two ethnic groups.[29] In contrast, the analysis of NZCR data by Obertova et al found Māori men with distant metastases at diagnosis were 1.32 times more likely to die of prostate cancer than non-Māori men, irrespective of factors such as age, time of diagnosis and socioeconomic status.[25]Additionally, comorbidities can affect a patient's life expectancy and treatment.[30] In a 2016

study, the proportion of Māori men having at least one comorbidity was higher (70%) than that of New Zealand Europeans (52%).[29] Disparities in survival and the detection of localised versus metastatic disease could also be attributed to differences in screening, referral and treatment between Māori and non-Māori men.[25,29,31]

Screening and treatment

Screening for prostate cancer in New Zealand general practice began in the 1990s and currently almost 30% of men aged over 40 are tested each year and 45% of men aged 65–75 years of age.[32] Prostate cancer screening is well recognised in leading to over diagnosis.[33,34] PSA testing in general practice is principally opportunistic screening initiated by general practitioners with few screening tests initiated by patients.[35] General practitioners (GPs) in New Zealand are half as likely to screen Māori men for prostate cancer compared to non-Māori men.[29,36]

Lower PSA screening rates are a significant factor contributing to lower prostate cancer incidence rates for Māori men.[36] Obertova in 2010 noted Māori men were less likely to be screened compared with non-Māori men (Mantel Haenszel (M-H) age-adjusted risk ratio (RR), 0.52 [95% CI, 0.48, 0.56]).[36] When screened, Māori men were more than twice as likely to have an elevated PSA result compared with non-Māori men (M-H age-adjusted RR, 2.16 [95% CI, 1.42, 3.31]). However, there were no significant differences between Māori and non-Māori men in the rate of follow-up investigations and cancer detection.

Obertova also found that following diagnosis in a cohort of newly diagnosed men a lower proportion of Māori men were treated with prostatectomy and Māori were almost twice as likely to be treated with external beam radiotherapy (EBRT). Māori men were also more likely to be managed expectantly with watchful waiting or active surveillance.[37] These differences in treatment can partly be attributed to higher rates of comorbidities found in Māori men.

Because non-Māori are more likely to be treated with a prostatectomy it was noted that in some cases following surgery the staging of prostate cancer altered from localised (Stage 1 or 2) to locally invasive (Stage 3) based on the post-surgical pathology. This change in staging is likely to lead to additional treatment such as androgen deprivation therapy (ADT) while Māori being treated with watchful waiting or EBRT would not have this additional staging information and may therefore not gain any benefit from adjunct radiotherapy. Researchers also noted that Māori men rarely received low-dose brachytherapy (LDR), compared to non-Māori.[31] LDR is only available privately in New Zealand and it is well recognised Māori are less likely to access private cancer treatment.[38]

Socioeconomic risk factors

Māori experience numerous barriers to healthcare access, diagnosis and treatment in an array of domains, which contribute to the overall disparity in health outcomes. For instance, research suggests that the collateral costs of travel and car parking related to accessing healthcare,[11] alongside the indirect costs of whānau carers (eg, time off work) if care is required,[39] serve as barriers to accessing healthcare. Up to 15% of Māori adults are also unable to access their medication due to cost.[40] For instance EBRT is only available in major centres. Thus, patients undergoing this treatment may have to travel frequently to the treatment facility and/or seek accommodation nearby, which may pose barriers in relation to time, finances and/or travel distance. Such factors hold potential implications for Māori men who may be socioeconomically deprived and living in rural locations.

Generally men living in socioeconomic deprivation have a higher prostate cancer mortality.[27] Further disparities in PSA screening, cancer diagnosis and treatment management are evident for rural men compared to their urban counterparts.[31] Again, Māori men are disproportionately disadvantaged in these contexts as they are more likely than non-Māori men to live in socioeconomic deprivation, and more likely than non-Māori men to be living in small towns and rural areas.[46] Other environmental and biological risk factors such as age, smoking, body weight, diet (and cooking methods), exercise and the higher rate of PSA in Māori men have been posited as reasons for prostate cancer outcome disparities.[9,47,48] The available evidence suggests,

however, that it is more differences in stage at diagnosis and treatment rather than differences in environmental and biological risk factors, that are responsible for the worse survival rates in Māori men.[49]

Furthermore, poor health literacy not only relating to prostate cancer symptoms, but also the cultural health literacy of the health system itself, poses a barrier to Māori men seeking appropriate care.[43,49–51] When health professionals fail to establish rapport, use good communication or share timely and appropriate information, particularly following prostate cancer testing, this may heighten men's experiences of stress and fear.[52] In the context of Māori men and health service provision in particular, issues relating to whakamā (to be ashamed, embarrassed) such as dignity, shyness, not wanting to draw attention to one's self and the perception of receiving value from the provider contribute to poor health outcomes for Māori men.[20]

Overall, research indicates that New Zealand is lagging behind its nearest neighbours in Australia to improving prostate cancer diagnosis, treatment and avoidable deaths.[21,53,54] This is particularly concerning for Māori men, as they are disproportionately impacted in the New Zealand context.

Opportunities for equity

Healthcare service provision generally relies on building health literacy in patients and communities to overcome disengagement from services. However, in order to achieve health literacy and active participation in health decisions for Māori men and their whānau, health services must value the importance of being Māori.[20,55] Health professionals need to endeavour to re-engage Māori men and their whānau by challenging the inherent racism, as outlined in the discussion above, that contributes to the prevalent limitations of health services.[20] A recent study[20] highlighted that when working with Māori men there are gaps in service capacity to understand the Māori view of health, the impacts of generations of economic deprivation and racism, the drivers of health-seeking behaviour, service accessibility, the importance of whānau support and the overall need for Māori-focused services.[20] Such gaps are also well represented in the broader corpus of Māori health literature.[11,56–58] We argue that these factors must shape future research and service initiatives that purport to improve Māori health equity. Addressing the unacceptably high Māori mortality rate in prostate cancer requires targeted anti-racism interventions at specific high-risk points in the treatment pathway. These high-risk points include diagnosis, which requires, for example, an equity-focused review of opportunistic screening to address earlier diagnosis for Māori. Further, many of these equity concerns are raised by the Prostate Cancer Taskforce (2012), where they suggested the need to "Get access to and quality of prostate care right for Māori and we get it right for all. It doesn't work the other way around".[59]

Conclusion

While Māori are less likely to be diagnosed with prostate cancer, they are significantly more likely to die from this disease. Most of the disparity is due to the later stage at diagnosis for Māori men and ethnicity-based differences in treatment. In order to address these disparities and eliminate the inequity for Māori there needs to be several systemic factors addressed. Some include the impact of wider social determinants such generations of economic disadvantage for whānau, meaning that access to general practice is a barrier. The differences in screening rates for Māori men appear to be due to general practitioners being less likely to screen Māori men. While this can be partially addressed through providing Māori men and their whānau with better information on prostate cancer screening to equip them to self-advocate, it is essential to address racism that contributes to inequitable outcomes for Māori men. Finally, further equity-focused research is urgently needed to investigate the relationship between the differences in treatment and outcomes for Māori, and what the impact of comorbidities is on treatment and cancer outcomes. We conclude that there is an urgent need for more culturally appropriate care to be available to Māori men affected by prostate cancer.

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The most commonly diagnosed and most common causes of cancer death for Māori New Zealanders

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ABSTRACT

Cancer is an important cause of morbidity and avoidable mortality for Māori—and substantial disparities exist in cancer incidence, mortality and survival for Māori compared to non-Māori New Zealanders. In this viewpoint, we draw together cancer incidence, mortality and survival data from the previous decade, in order to provide clarity regarding the most important causes of cancer burden for Māori. Covering the decade 2007–2016, our manuscript directly leads on from the landmark Unequal Impact II report (which covered 1996–2006), and provides the most up-to-date record of this burden as is currently possible. While focusing on the absolute burden of cancer for Māori, we also compare this burden to that experienced by non-Māori, and consider how this relative disparity may (or may not) have changed over time. Finally, we discuss how to reduce the occurrence and the overall cancer mortality burden for Māori, with a focus on those cancers that confer the greatest burden.

Cancer is an important cause of morbidity and avoidable mortality for Māori, with more than a quarter of all deaths among Māori attributable to this disease.[1] There is substantial evidence of enduring disparities in cancer incidence, mortality and survival between Māori and non-Māori, with cancer making an important contribution to the life expectancy gap between these groups.[2]

Our country's new Cancer Action Plan for the years 2019–2029 aims to address inequities in the burden of cancer experienced by Māori New Zealanders.[3] In the plan, the Ministry of Health stated that the plan would be equity-led, achieve equity by design, and included as a primary outcome that *"New Zealanders experience equitable cancer outcomes".* These objectives are important and signal a commitment from central Government to closing the cancer gap for Māori.

Given the substantial inequities in cancer outcomes experienced by Māori, the prioritisation of initiatives to close this gap is congruent with the objectives of the new Plan. However, in the presence of finite capital (both fiscal and political), there is a need to carefully set priorities that reflect the reality of the cancer burden faced by Māori. While many initiatives will have pan cancer impact—such as renewed investment in the Māori cancer care workforce—there is value in understanding which cancers cause the largest burden on Māori, before we prioritise and invest in new initiatives that may increase inequities, or only impact one or two cancers (such as screening programmes).

In this manuscript, we present current evidence on the most commonly diagnosed cancers among Māori between 2007–2016, the decade immediately following on from the 1996–2006 period presented in the

landmark Unequal Impact II report.[2] We also present the most common causes of cancer death for Māori over this period. Alongside these absolute cancer death data, we present a relative cancer survival comparison between Māori and non-Māori. Finally, we summarise the factors that link these cancers, and discuss how to reduce their occurrence and the overall cancer mortality burden for Māori.

What are the most commonly diagnosed cancers among Māori?

Using New Zealand Cancer Registry data, we determined the 10 most commonly diagnosed cancers among Māori between 2007–2016, calculated age- and sex-standardised incidence rates (SIR), and also calculated standardised rate differences (SRD) to compare incidence between Māori and non-Māori. We determined incidence rates and rate differences for both the total population (Figure 1) and separately for males and females (Appendix Figure 1).

In terms of absolute numbers of cases, lung cancer was the most commonly diagnosed cancer among Māori with 401 cases/year, followed by breast (373/year), prostate (190/year) and colorectal (170 cases/year). In terms of age- and sex-standardised incidence rates, breast (SIR: 45/100,000 Māori per year) and lung (42/100,000) were highest, followed by prostate (20/100,000) and colorectal (18/100,000). The remainder of the top 10 (stomach, uterine, liver, pancreatic, and the blood cancers leukaemia and non-Hodgkin's lymphoma) clustered around 6–10 cases/100,000 Māori. For Māori females, the most commonly diagnosed cancer was breast (88/100,000 Māori females), followed by lung (46/100,000) and colorectal cancers (17/100,000). For Māori males, the most commonly diagnosed cancer was prostate (46/100,000 Māori males), followed by lung (43/100,000) and colorectal cancers (23/100,000).

In comparison with non-Māori, Māori were more likely to be diagnosed with most of these cancers than non-Māori, particularly lung (SRD: females absolute difference of 34 cases per 100,000, males 28/100,000, total 29/100,000), but less likely to be diagnosed with colorectal (females -4/100,000, males -4/100,000, total -4/100,000) and prostate cancers (males -11/100,000).

What are the most common causes of cancer death for Māori?

Mortality

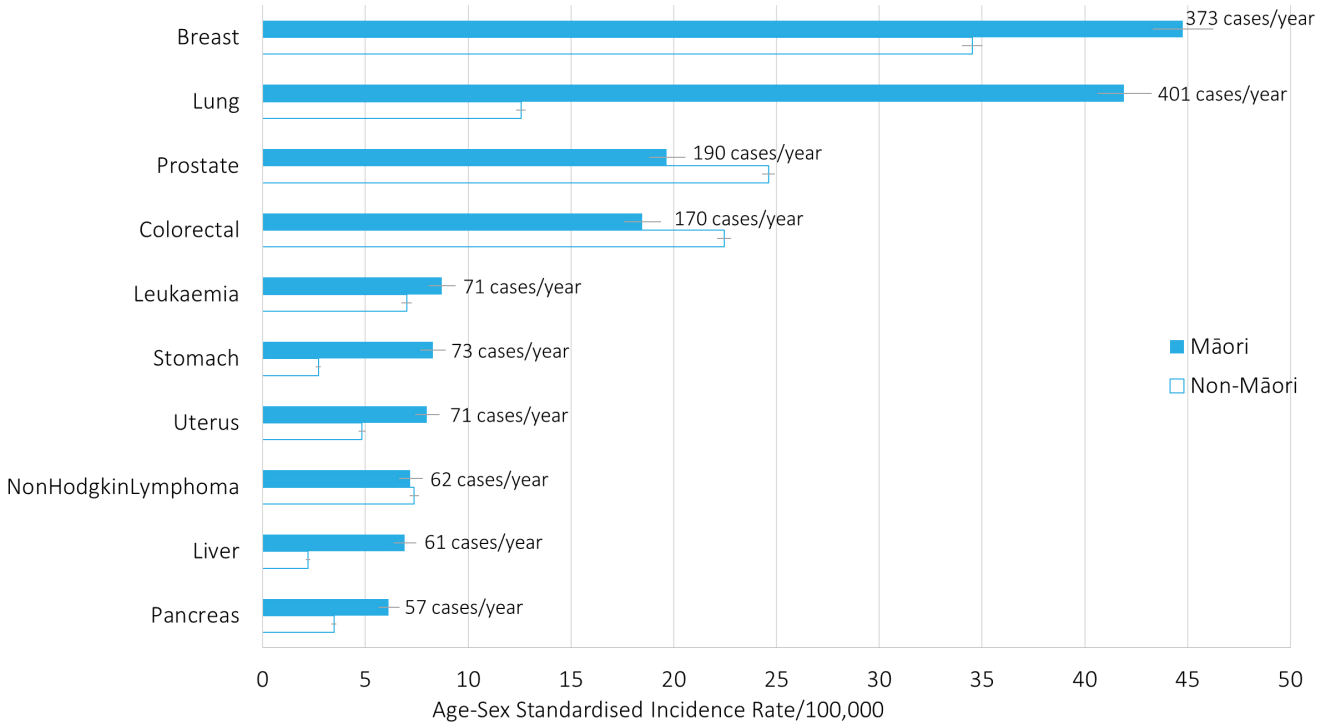
Using New Zealand Mortality Collection data, we determined the 10 most common causes of cancer death among Māori between 2007–2016, calculated age- and sex-standardised mortality rates (SMR), and also calculated standardised rate differences to compare mortality between Māori and non-Māori (SRD). Like incidence, we determined mortality rates and rate differences for both the total population (Figure 2) and separately for males and females (Appendix Figure 2).

Lung cancer was the most common cause of cancer death among Māori, with 311 deaths/year (SMR: 32/100,000). This was followed by breast (77 deaths/year, 9/100,000 Māori) and colorectal cancers (68 deaths/year, 7/100,000). The remainder of the top 10 (stomach, liver, pancreatic, ill-defined, prostate, leukaemia and non-Hodgkin's lymphoma) caused between 20–50 deaths per year (2–6/100,000). The most common cause of cancer death among Māori females was lung (34/100,000 females), followed by breast (17/100,000) and colorectal cancers (6/100,000). The most common cause of cancer death among Māori males was lung (34/100,000 Māori males), followed by colorectal (10/100,000) and prostate cancers (9/100,000).

In comparison with non-Māori, Māori were more likely to die from most of these cancers than non-Māori, particularly lung (SRD: absolute difference in rates for females 25/100,000, males 22/100,000, total 23/100,000), although differences for colorectal cancer were negligible (females -2/100,000, males 0/100,000, total -1/100,000).

There is obvious crossover between the incidence and mortality figures presented above, with nine of the top 10 cancers occurring on both lists. Cancer mortality is of course intrinsically linked to cancer incidence: the more common a cancer is, the more common that death from that cancer will be. As such, many cancer deaths are avoidable via prevention of cancer incidence; but many cancer deaths are also avoidable via improvements in the likelihood of survival following a cancer

Figure 1: Age- and sex-standardised incidence rate (SIR) and absolute numbers of annual cases for the top 10 most commonly diagnosed cancers for Māori between 2007–2016 (top), along with the age- and sex-standardised rate difference (SRD) between Māori and Non-Māori (bottom). Analysis methods are described in the Appendix. Data is direct age- and sex-standardised to the 2001 total Māori population.



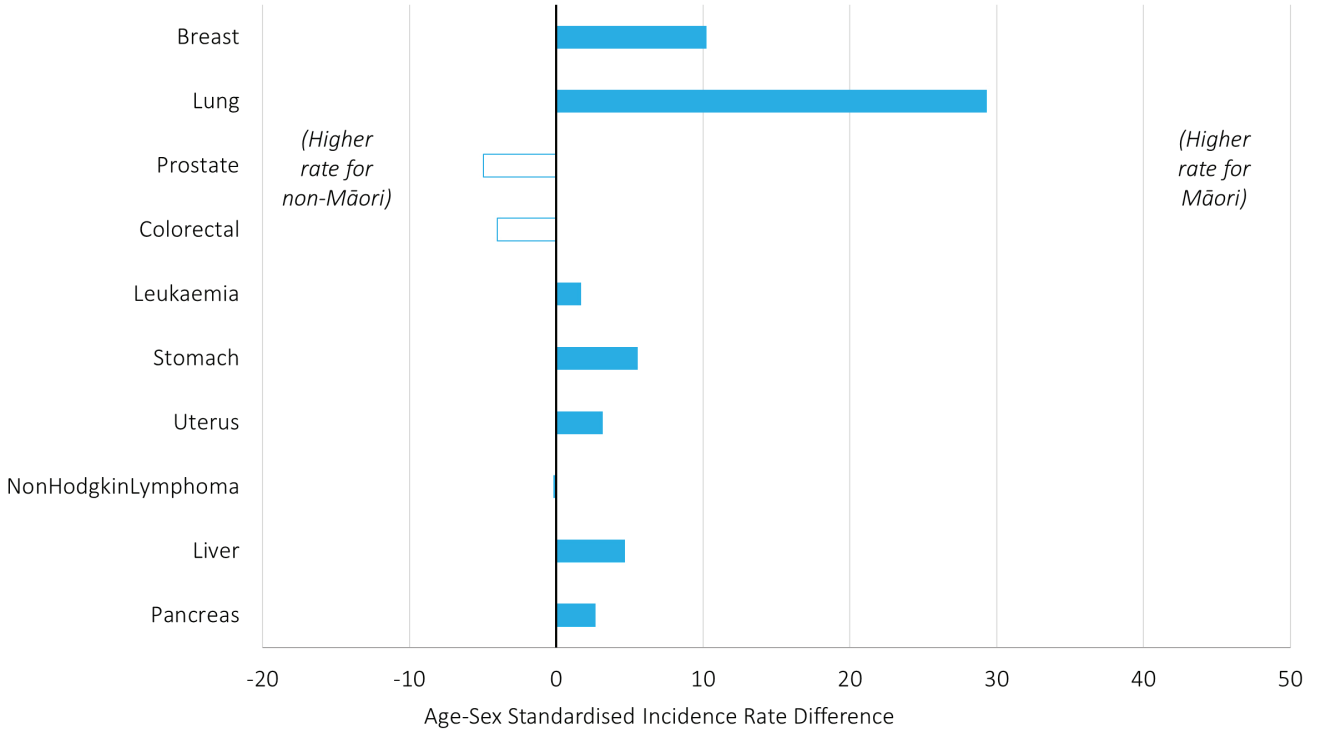


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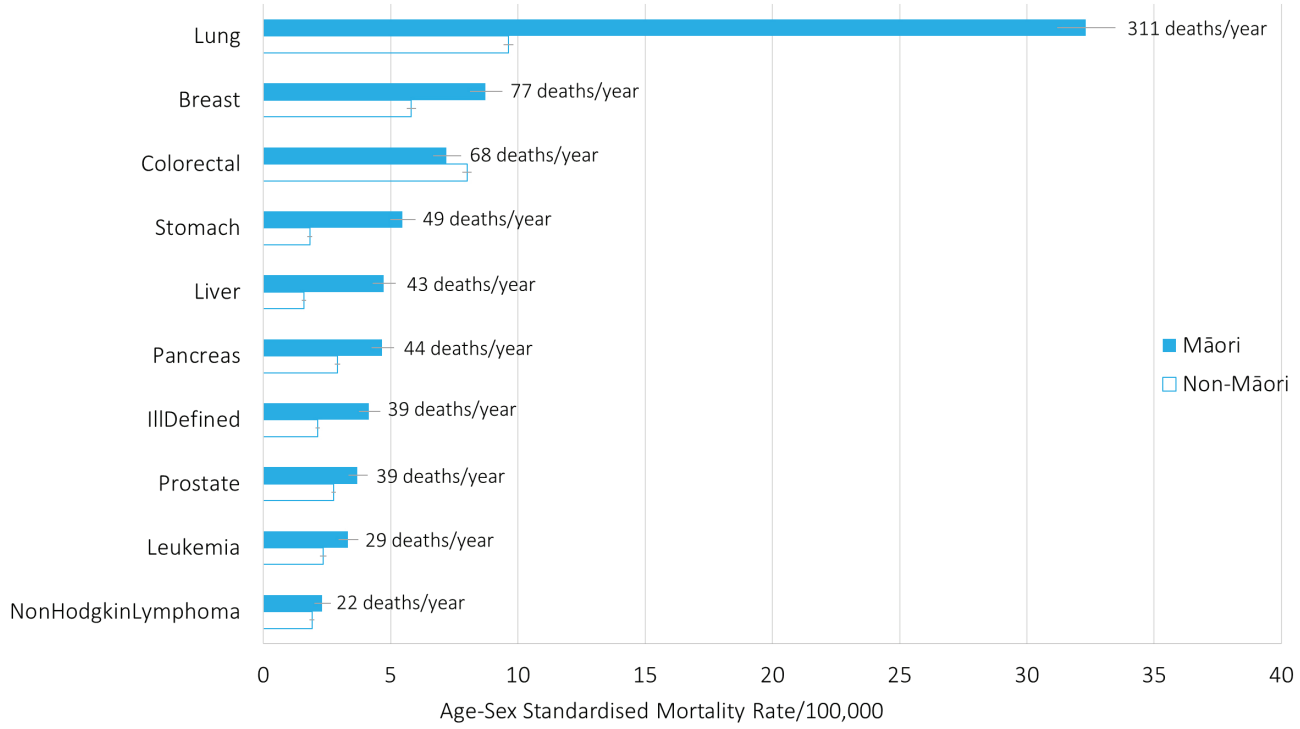
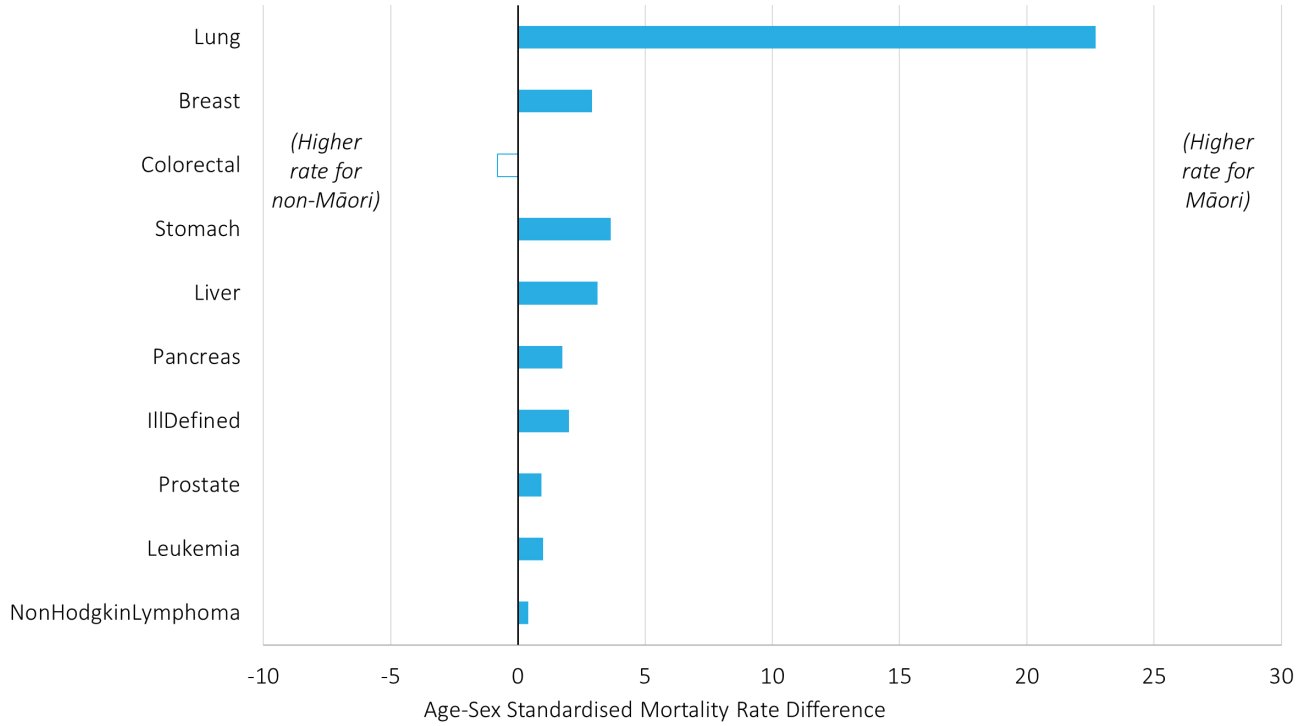
Figure 2: Age- and sex-standardised mortality rate (SMR) and absolute numbers of cases for the top 10 most common causes of cancer death for Māori between 2007–2016 (top), along with the age- and sex-standardised rate difference (SRD) between Māori and Non-Māori (bottom). Analysis methods are described in the Appendix. Data is direct age- and sex-standardised to the 2001 total Māori population.





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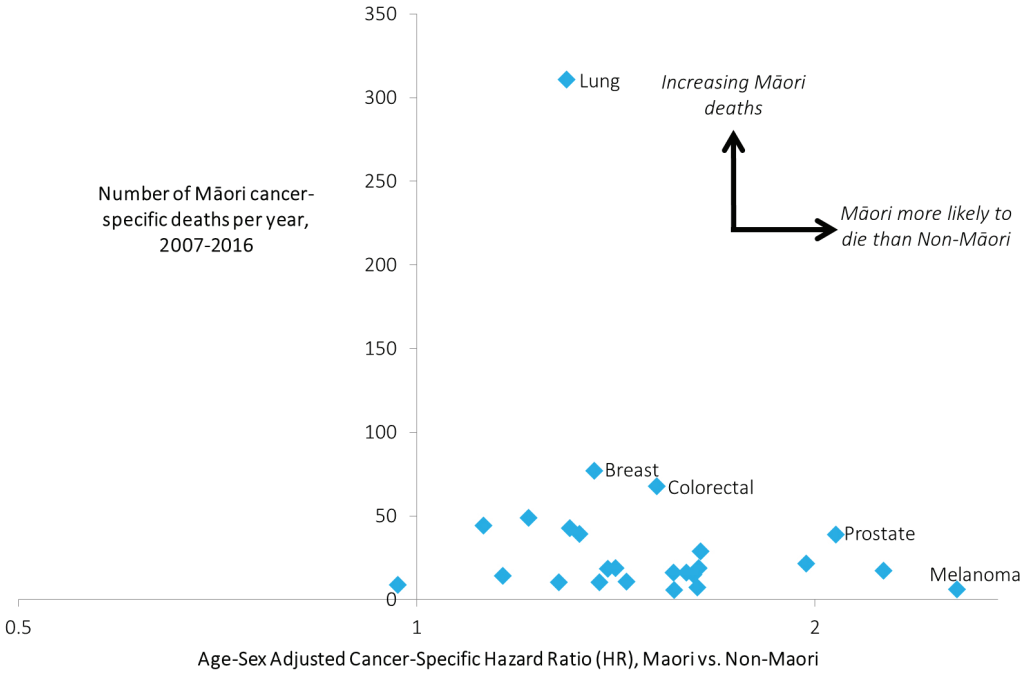
diagnosis. Like incidence, cancer survival is a factor which is not equal between Māori and non-Māori.[2]

Survival

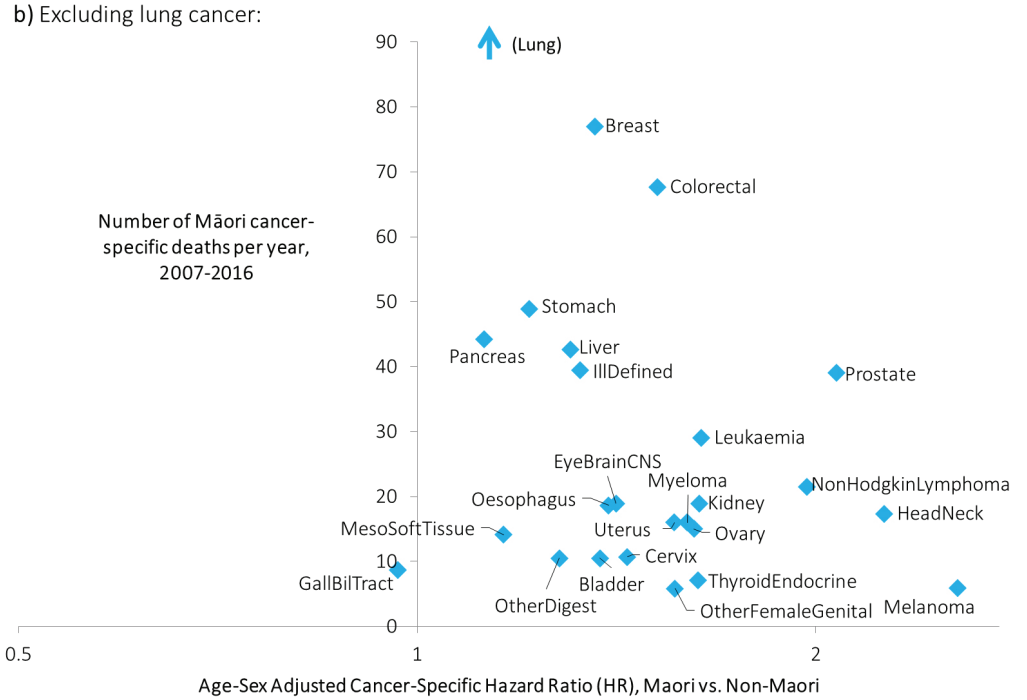
In Figure 3, we have plotted the absolute number of cancer deaths among Māori per year (*y*-axis) against the relative disparity in cancer survival (ie, post-diagnosis survival) between Māori and non-Māori (*x*-axis). This figure helps us to contextualise relative disparities in cancer survival against the actual absolute mortality burden experienced by Māori. For example, the strongest

Figure 3: Scatterplot showing the average annual cancer-specific deaths among Māori (2007–2016) versus cancer-specific mortality hazard ratios (Māori vs Non-Māori), for a) all cancers including lung cancer; and b) all cancers excluding lung cancer. Only cancers where at least five cancer deaths occurred per year are plotted. Analysis methods are described in the Appendix. HRs are presented on the logarithmic scale.

a) Including lung cancer:



b) Excluding lung cancer:



observed survival disparity is found for melanoma (age-sex-adjusted hazard ratio [HR]: 2.6, 95% CI 2.0–3.3), but fewer than six Māori died of this cancer per year over the study period (5.9 deaths/year). Contrast this with lung cancer, for which the survival disparity is smaller (HR: 1.3, 95% CI 1.2–1.4) but the mortality burden is much higher (311 Māori deaths/year). Importantly, survival is poorer for Māori compared to non-Māori for each of the top 10 causes of Māori cancer death, with adjusted hazard ratios ranging from 1.1 (pancreatic cancer) to 2.1 (prostate cancer). A complete list of hazard ratios for all cancers, for both the total combined population and stratified by sex, are presented in Appendix Table 2.

What is happening over time?

Investigating temporal trends in incidence, mortality and survival can help us to understand whether (or not) we are making progress in achieving better cancer outcomes for Māori. In Figure 4, we present the age-sex-standardised incidence, mortality and rate differences for each year between 2007–2016. We have focused on lung cancer as both the most commonly diagnosed and most common cause of cancer death for Māori men and women.

From Figure 4 we observe that, for the most common cause of cancer and cancer death for Māori (lung cancer), both incidence and mortality appear to be reducing over time—along with the disparities between Māori and non-Māori for these measures. However, disparities in lung cancer survival for Māori compared to non-Māori have remained relatively unchanged over the previous decade, ranging between 20–40% excess mortality with no clear temporal trend in either direction (Figure 4).

How do we reduce the cancer burden for Māori?

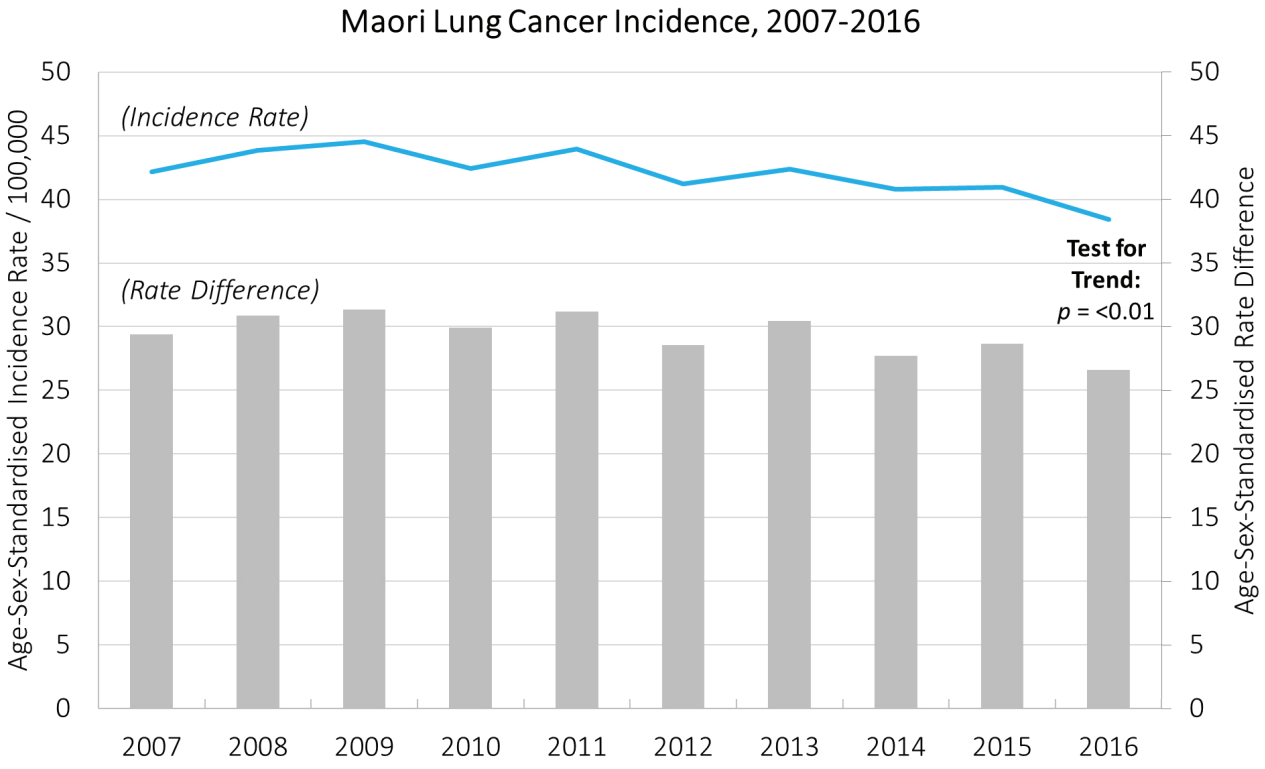
The factors underpinning overall worse cancer incidence, mortality and survival for Māori are systemic. These outcomes are driven by inequities in the social determinants of good health, determinants that are structural in nature and not controlled by Māori (just as they are not controlled by other indigenous and minority populations around the world).[4] In the context of cancer, these determinants combine to increase Māori exposure to carcinogens, to prevent access to screening and early detection, and to prevent timely access to best-practice curative treatment. These factors occur across cancers, and the extent to which they impact on outcomes will depend on the unique characteristics of each cancer.

A systems-level problem requires system-level solutions. Broad health system actions that impact multiple cancers—such as improving access for Māori to timely diagnosis and appropriate treatment, regardless of income or place of residence—are crucial. Likewise, enhancing Māori access to cancer control decision-making, coupled with the resources required to improve cancer prevention, early detection, treatment and support for Māori-led approaches to providing these services, are also vital. While these broad actions are necessary, dismantling the barriers to equitable cancer outcomes for Māori will require a cancer-specific approach in some instances. In an absolute sense, the cancers identified in Figures 1 and 2 have the most profound impact on overall cancer mortality for Māori. However, systemic actions to reduce Māori cancer deaths should not necessarily be prioritised towards cancers in order of burden. To be effective, such prioritisation must take into account factors such as preventability of the cancer, ability to detect the cancer at an early stage, prognosis of the cancer once it is detected, and the availability of curative treatment options within a given cancer context.

Prevention

More than half of the top 10 most common cancers among Māori (Figure 1) and the top 10 most common causes of cancer death for Māori (Figure 2) have known key aetiological exposures that disproportionately impact Māori compared to non-Māori. These exposures can be broadly grouped as tobacco exposure (lung[5] and pancreatic[6] cancers), infectious diseases (stomach[7] and liver[8] cancers[9]), diet and obesity/diabetes mellitus (breast,[10] uterine,[10] colorectal[10] and pancreatic[6] cancers) and familial genetic predisposition (stomach cancer[11]). Māori are substantially more likely to be exposed to tobacco,[12] to be exposed to infection with *Helicobacter pylori*[7] and the Hepatitis virus,[13] and to be exposed to the structural causes of obesity and diabetes.[14]

Figure 4: Lung cancer age-sex-standardised incidence rate and rate difference (top), mortality rate and rate difference (middle), and differences in survival between Māori and non-Māori (bottom) over the 2007–2016 period. For the survival analysis, shaded areas indicate 95% confidence intervals. (Note: the observed abrupt increase in recorded lung cancer deaths in 2015 is unexplained, and we are investigating this with National Collections.)



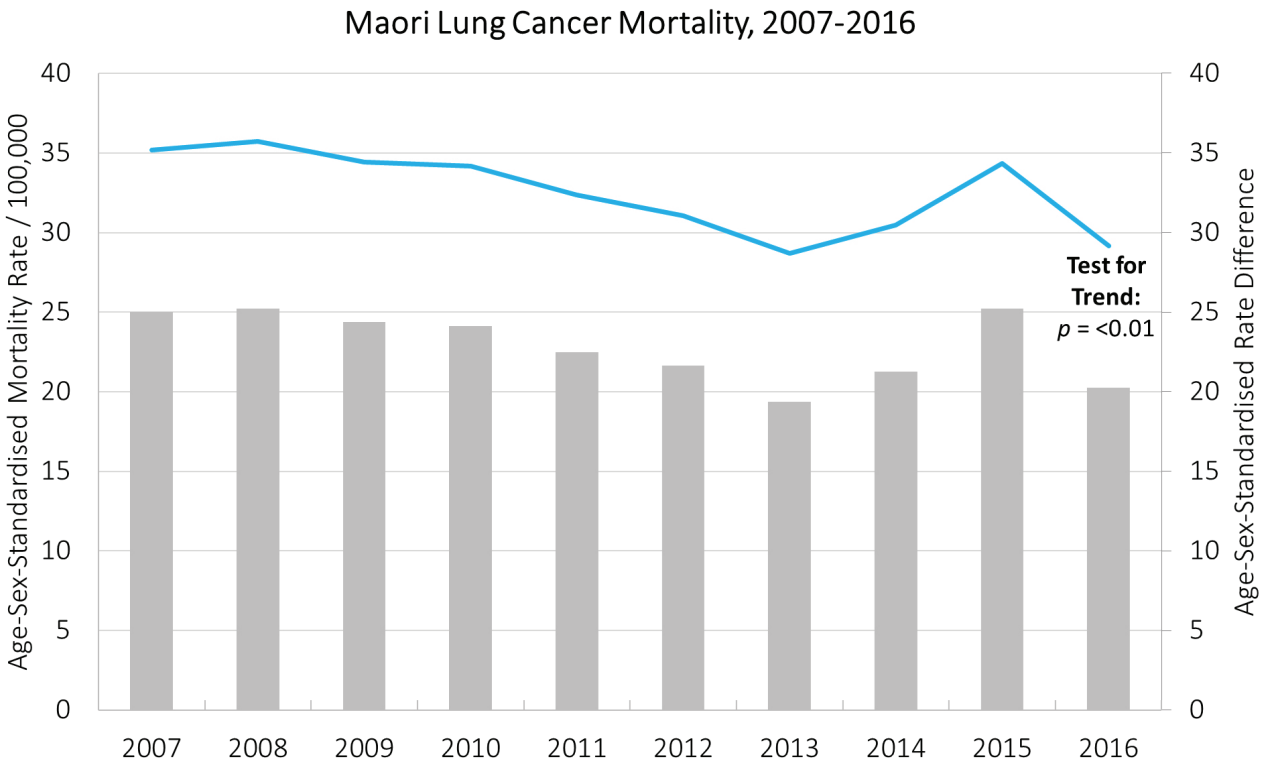
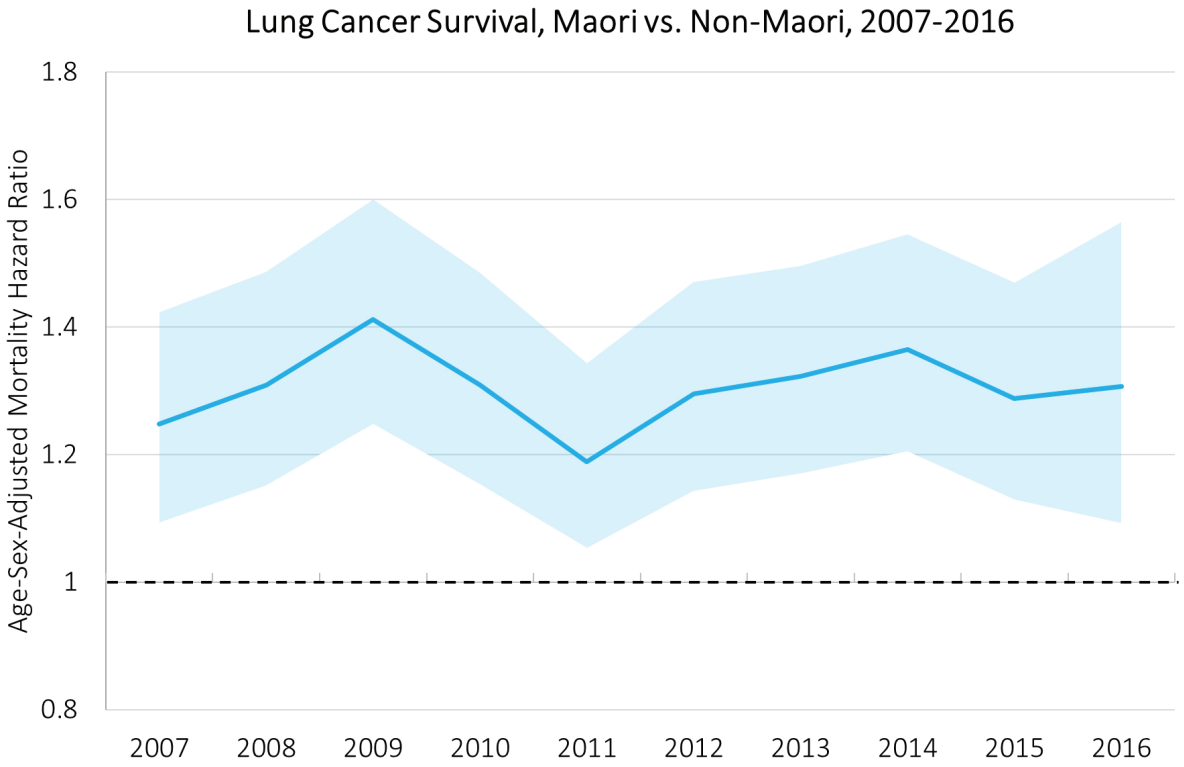


Figure 4: Lung cancer age-sex-standardised incidence rate and rate difference (top), mortality rate and rate difference (middle), and differences in survival between Māori and non-Māori (bottom) over the 2007–2016 period (continued).



The relative contribution of each of these known exposures to the incidence of a given cancer varies depending on the context. The attributable fraction of lung cancer cases caused by tobacco exposure has been estimated to be between 80–90%.[15,16] More than a third of uterine cancers are attributable to obesity.[10] More than two-thirds of all stomach cancers are attributable to *Helicobacter pylori* infection, with this bacterium responsible for 90% of non-cardia stomach cancers—the most common form of stomach cancer diagnosed among Māori.[17] Nearly 40% of all liver cancers worldwide are attributable to the hepatitis B or C virus,[9] and 70% of Māori liver cancer patients will have a history of hepatitis B or C infection, suggesting a much higher attributable fraction for Māori than the international average.[18] In Australia and New Zealand an estimated 17% of pancreatic cancer cases are attributable to high fasting plasma glucose (consistent with pre- or established diabetes mellitus), around the same as that attributable to tobacco exposure.[6]

Thus, a substantial proportion of the most common causes of cancer and cancer death for Māori are attributable to known preventable exposures. We can draw hope and encouragement from the knowledge that the means of preventing a substantial proportion of cancers for Māori are within our grasp—and include steadfast commitment to Smokefree 2025 (with a stronger focus on smoking in Māori), exploration of *Helicobacter pylori* test-and-treat programmes, revived vigour in (and monitoring of) our hepatitis vaccination and surveillance programmes, and a regulatory commitment to ensuring that nutritious and healthy lifestyles are accessible and affordable for all (particularly Māori). This hope must be accompanied with renewed determination and innovation to make these tools work for Māori. National cancer prevention policies should combine a population-based and underserved population approach—known as proportionate universalism[19]—and emphasis on the factors outlined here is consistent with this approach. Perhaps most crucially, an approach that focuses resources on the prevention of cancers that have the strongest impact on Māori is consistent with the principles of the Treaty of Waitangi, which require the Crown to take active measures to restore balance in situations where Māori have been disadvantaged.[20]

Reducing Māori cancer deaths

Five of the top 10 causes of cancer death for Māori (lung, stomach, pancreas, liver, ill-defined) have an expected one-year survival of less than 50%, with the most common cause (lung) having a one-year survival of approximately 35% for both Māori and non-Māori (data not shown). The nature of tumour development within these cancer types means that disease detection often does not occur until tumours have metastasised, at which point the benefit of a curative therapeutic approach is questionable. More than two-thirds of all Māori cancer deaths each year occur among these poor-prognosis cancers (Figure 2).

With this in mind, the primary key to reducing cancer deaths for Māori is by preventing the cancer in the first place (see above). If prevention is unsuccessful, then the next highest priority is early detection, when curative treatment is still possible. Once the cancer is diagnosed, the highest priority becomes ensuring access to timely best-practice treatment for the given tumour type and sub-type. Unfortunately, there is evidence that Māori are underserved in each of these post-diagnosis priorities.[21,22] There are some burgeoning examples of high Māori screening participation in some regions (eg, the Southern DHB bowel screening programme), and these successes should be examined, documented and modeled where appropriate.

Early detection and screening

Early detection of primary lung cancer currently relies on either presentation of a patient with respiratory symptoms, or on chance findings following examination for other conditions (eg, chest x-ray for suspected heart disease). Given the poor prognosis of lung cancer, detection of tumours at the asymptomatic stage may increase the chances of survival via curative treatment. Studies investigating the efficacy of lung cancer screening via computed tomography (CT) scan have been promising.[23–25] Preparatory lung cancer screening work is currently being completed in various locations throughout New Zealand, with the results of this preparatory work of critical importance to Māori health. National screening programmes for breast and colorectal cancers—both in the top three most common causes of cancer death for Māori—are in operation, although the latter programme is in its infancy and as of January 2020 was operational in 10 of the 20 district health boards.[26] With evidence that Māori have poorer access to national screening programmes than non-Māori,[27,28] there is an urgent need for renewed prioritisation and vigour in maximising Māori participation across these programmes.

Many of the most commonly diagnosed cancers among Māori are diagnosed outside of screening programmes. Diagnosis of these cancers principally relies on detection through primary care, although there is evidence that Māori are more likely to be diagnosed following acute admission at an emergency department.[29] Maximising early diagnosis of cancers for Māori requires us to consider the key barriers to early diagnosis that Māori (to a greater extent than non-Māori) face. These include the financial burden of general practitioner visits, transport and travel, as well as other factors including patient comorbidity that may complicate diagnosis.[30] In essence, we need to recognise that our current means of early detection systemically disadvantages Māori, and that this disadvantage contributes to the perpetuation of inequities in cancer death between Māori and non-Māori.

In terms of specific actions that impact on the cancers in Figures 1 and 2, the pathways for achieving important health gain for Māori will vary depending on the cancer. For example, an important pathway in terms of early detection of liver cancer for Māori is hepatitis surveillance. As noted above, hepatitis B and C appear to be the primary causes of liver cancer for Māori; however, Māori are underserved by the current national hepatitis surveillance programme. We previously observed that, in a cohort of Māori liver cancer patients with hepatitis B or C, only around 40% were on surveillance.[31] Crucially, more than three-quarters (77%) of those not on surveillance were diagnosed with stage III or later disease, compared to 33% of those who were on surveillance.[31] These observations, echoes of which can be observed across cancers, strongly suggest that careful scrutiny of the barriers to hepatitis surveillance could yield important health gains for Māori with liver disease.

Access to best-practice and timely treatment

There is compelling evidence that Māori have poorer access to timely best-practice treatment compared to non-Māori.[21] For example, Stevens et al[32] observed that Māori lung cancer patients were 60% less likely to be referred to a medical oncologist than non-Māori and were 70% less likely to receive curative treatment. Because we know that Māori have generally poorer access to best-practice and timely care, cancer care services have an important role to play in reducing the cancer burden for Māori.

In their global report on the drivers of social inequalities in cancer outcomes, the International Agency for Research on Cancer (IARC)[4] summarised the key drivers of equitable access to cancer care as the following three A's:

Availability: The physical availability of high-quality cancer care services, how well resourced and managed these services are, and how well they communicate with patients.

Affordability: The financial accessibility of the cancer treatment journey, from the costs of care to travel/transportation and loss of income.

Acceptability: How cancer care services are perceived by population groups, in terms of factors including effectiveness of care (ie, whether the care will actually work) and cultural competency of the system (ie, how similar/dissimilar the system is to your own culture).[4]

We can identify our own system within these three A's, and how these are likely to be contributing to poorer cancer outcomes for Māori. Relevant factors include where services are located relative to where Māori live, the reality that some best-practice treatments are only available if privately funded, the inflexible nature of pathways of cancer care that do not necessarily reflect the priorities of Māori, and inadequate resourcing of Māori treatment providers and navigators.[33] Each of these factors (and others) are important sources of disparities in access to care and poor cancer outcomes for Māori, and will be occurring to a different extent across cancer types. Underpinning the receipt of best-practice and timely care by Māori is the existence of standards of care (such as those recently published for colorectal cancer[34]) that clearly benchmark what a patient should expect to receive during their cancer care, and an unerring apolitical commitment to ensuring these standards are met for Māori across all district health boards in New Zealand.

Other opportunities for improvements in cancer outcomes for Māori

While we have focused in this manuscript on the cancers that present the greatest overall cancer burden on Māori, this does not detract from the importance of cancers that may be less common but will have equal impact at an individual (and whānau) level. For example, while the number of Māori female deaths from cervical cancer (11/year; Appendix Table 1) is far fewer than lung or breast cancers, the large survival disparity between Māori and non-Māori (adjusted HR: 1.4, 95% CI 1.1–1.8) suggests that there is room within existing systems for substantial improvement that will save the lives of Māori women. The advent self-testing kits for human Papilloma virus (HPV, the cause of all cases of cervical cancer[9]) will likely increase the uptake of cervical screening among those who did not previously access this screening.[35] This is important, and might address the current substantial disparity in access to the national cervical screening programme among screen-age Māori women (64%) compared to European women (81%).[28] Resourcing of cancer control initiatives aimed at reducing the cancer burden for Māori must take into account the burden of a given cancer, as outlined throughout this manuscript—but they must also remain flexible to ensure that we do not miss opportunities for relatively straightforward interventions (such as HPV self-testing) that will invariably save Māori lives.

Conclusions

In this manuscript we have presented the most commonly diagnosed cancers for Māori, the most common causes of cancer death, and contextualised survival disparities between Māori and non-Māori against the actual mortality burden of each given cancer. If our primary objective is to reduce the overall cancer burden for Māori, then

our top priority may be preventing the majority of lung cancers via tobacco eradication while simultaneously detecting lung tumours early (possibly via a targeted lung CT screening programme). Population-based initiatives aimed at the prevention of cancers related to infectious diseases, diet, obesity and diabetes will also result in a substantial reduction in the incidence and mortality cancer burden for Māori. Likewise, overall improvements in early detection and the provision of best-practice timely treatment for Māori patients will close the survival gap between Māori and non-Māori in the short- to medium-term, leading to a further reduction in Maori cancer deaths.

Appendix

Methods

Data sources

We extracted all incident cases of cancer diagnosed between 2007–2016 from the New Zealand Cancer Registry (NZCR), which we then linked to the national Mortality collection to establish those patients that had died over the study period (for the purposes of survival analysis). In addition, to determine the number of cancer deaths for each given cancer we extracted all deaths where cancer was listed as the underlying cause of death from the Mortality Collection.

Variables

Ethnicity was derived from the NZCR for cancer incidence and survival analysis, and from the Mortality collection for mortality analysis. Those with Māori ethnicity were classified as Māori, while those without Māori ethnicity were classified as non-Māori. Cancer type (eg, lung cancer) was determined using ICD codes on the NZCR. In the case of breast cancer, only female breast cancer cases were included in analysis.

Descriptive analysis

In terms of descriptive analysis, we determined the top 10 cancers that were the most commonly diagnosed among Māori over the study period on the NZCR (ie, based on absolute counts). Similarly, we determined the top 10 most common cancers that were listed as an underlying cause of death on the Mortality collection. Further incidence, mortality and survival analysis was then conducted on these cancers.

Age- and sex-standardisation

We used direct age- and sex-standardisation to calculate standardised incidence rates (SIR) and standardised mortality rates (SMR),[36] using the 2001 total Māori population as the standard.[37] This method aligns with that used in the reporting for the WAI2575 report.[38] For incidence, numerator data were the number of cases over the study period as determined from the NZCR. For mortality, numerator data were the numbers of deaths where a given cancer was listed as the underlying cause of death on the Mortality collection. In terms of denominators, we used aggregated estimated population data from Statistics New Zealand across the study period. Age- and sex-stratified denominator data for Māori were derived from mid-year Māori estimated residential population data,[39] while non-Māori denominator data were determined by subtracting the Māori denominator data from the total mid-year estimated residential population data.[40] When calculating temporal trends in incidence and mortality rates, we used the estimated residential populations for each respective year as the relevant denominator (eg, 2007 Māori estimated residential population when calculating 2007 incidence rate).

Where incidence and mortality rates are presented by sex in the Appendix, the denominator and standard population used for these rates is sex-specific. For temporal analyses, we calculated a Cochran-Armitage test for trend.

Survival analysis

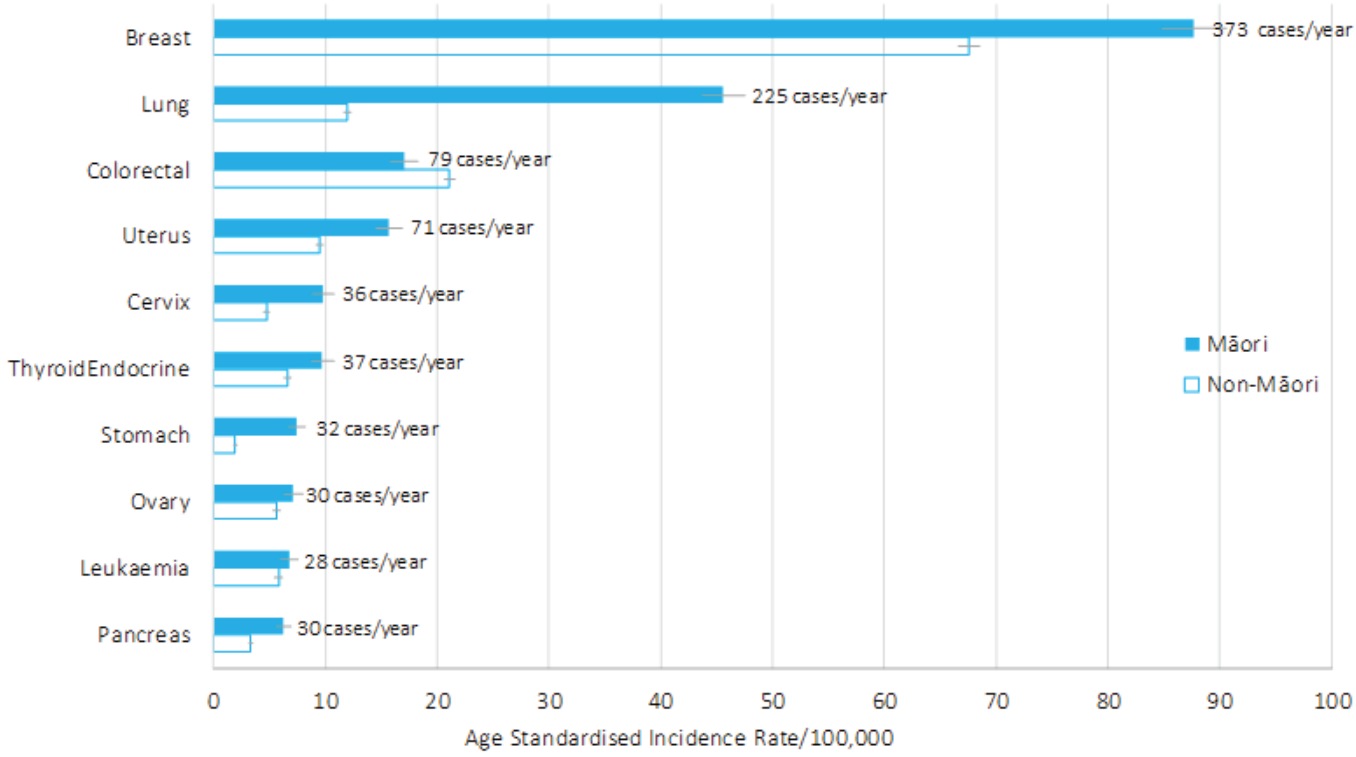
Cox proportional hazards models were used to describe the extent to which Māori were more or less likely to die of their cancer than non-Māori, adjusted for age (continuous variable) and sex where relevant (categorical variable). These results were described using hazard ratios (HR), with non-Māori as the reference group.

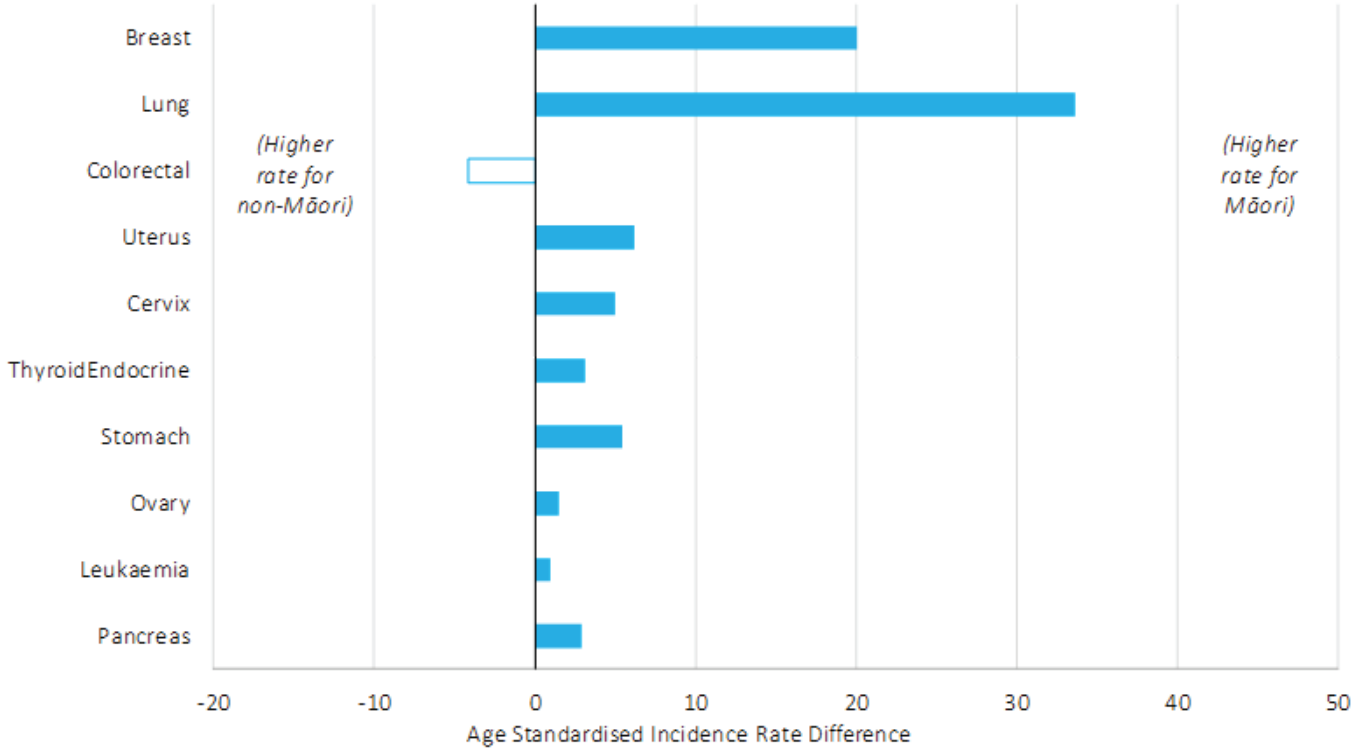
All analyses were conducted in SAS v9.4 (SAS Enterprises Inc.) and Microsoft Excel 2016 (Microsoft Corporation).

Appendix Table 1: Numbers of Māori cancer cases (incidence) and the number of cancer deaths (mortality) per year, by cancer type.

Cancer	Cases/year	Deaths/year
Anus	7	2
Bladder	22	11
Bone and cartilage	7	4
Breast	373	77
Cervix	36	11
Colorectal	168	68
Eye, brain and CNZ	33	19
Gallbladder and biliary tract	18	9
Head and neck	50	17
Hodgkin's lymphoma	10	2
Ill-defined, secondary or unspecified	48	39
Kidney	55	19
Leukaemia	71	29
Liver	61	43
Lung	401	311
Melanoma	34	6
Mesothelioma and soft tissue	28	14
Myeloma	35	16
Non-Hodgkin's lymphoma	62	22
Oesophagus	26	19
Other digestive tract	12	11
Other female genital	14	6
Other immune system	3	0
Other male genital	1	0
Other respiratory	6	3
Other urinary	2	1
Ovary	30	15
Pancreas	57	44
Prostate	190	39
Skin (not melanoma)	7	3
Small intestine	14	5
Stomach	73	49
Testis	34	2
Thyroid endocrine	51	7
Uterus	71	16

Appendix Figure 1A: Age standardised incidence rate (SIR) for the top 10 most commonly diagnosed cancers among Māori **females** between 2007–2016 (top), along with the age- and sex-standardised rate difference (RD) between Māori and the non-Māori females (bottom).





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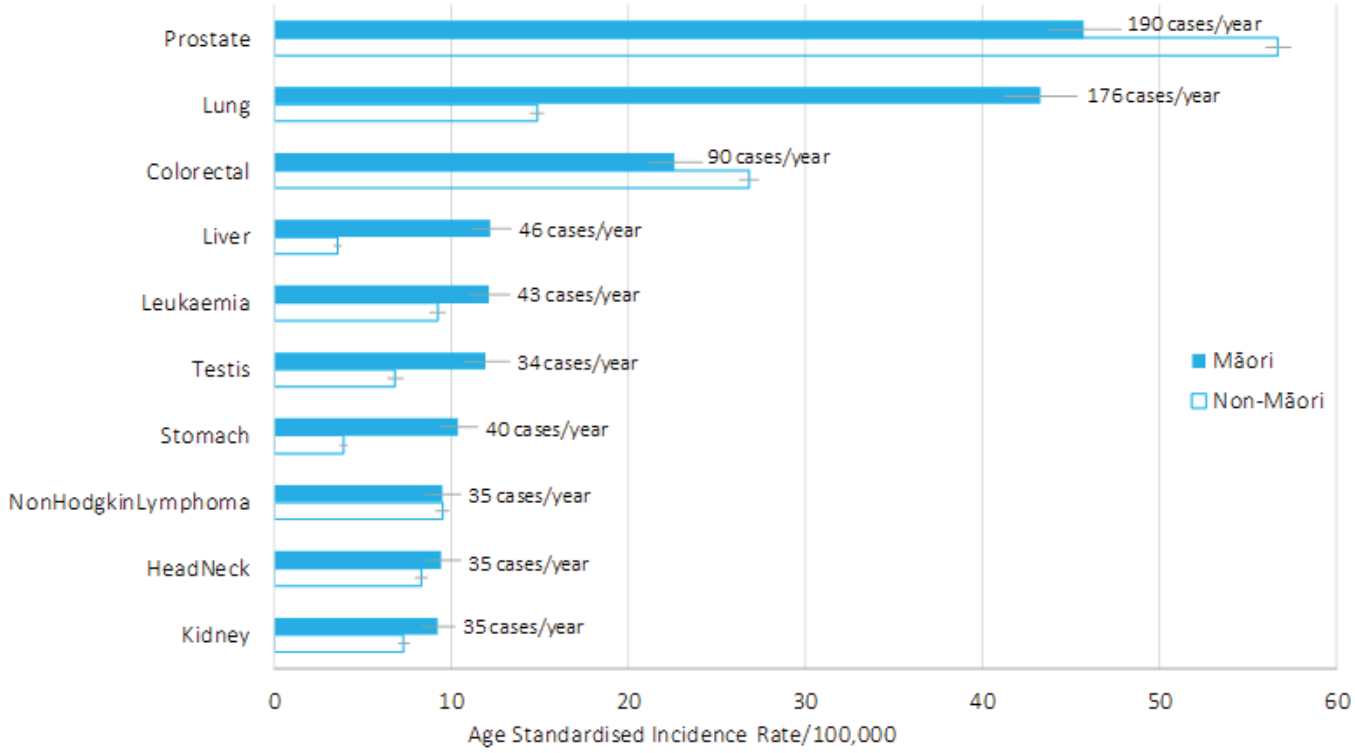
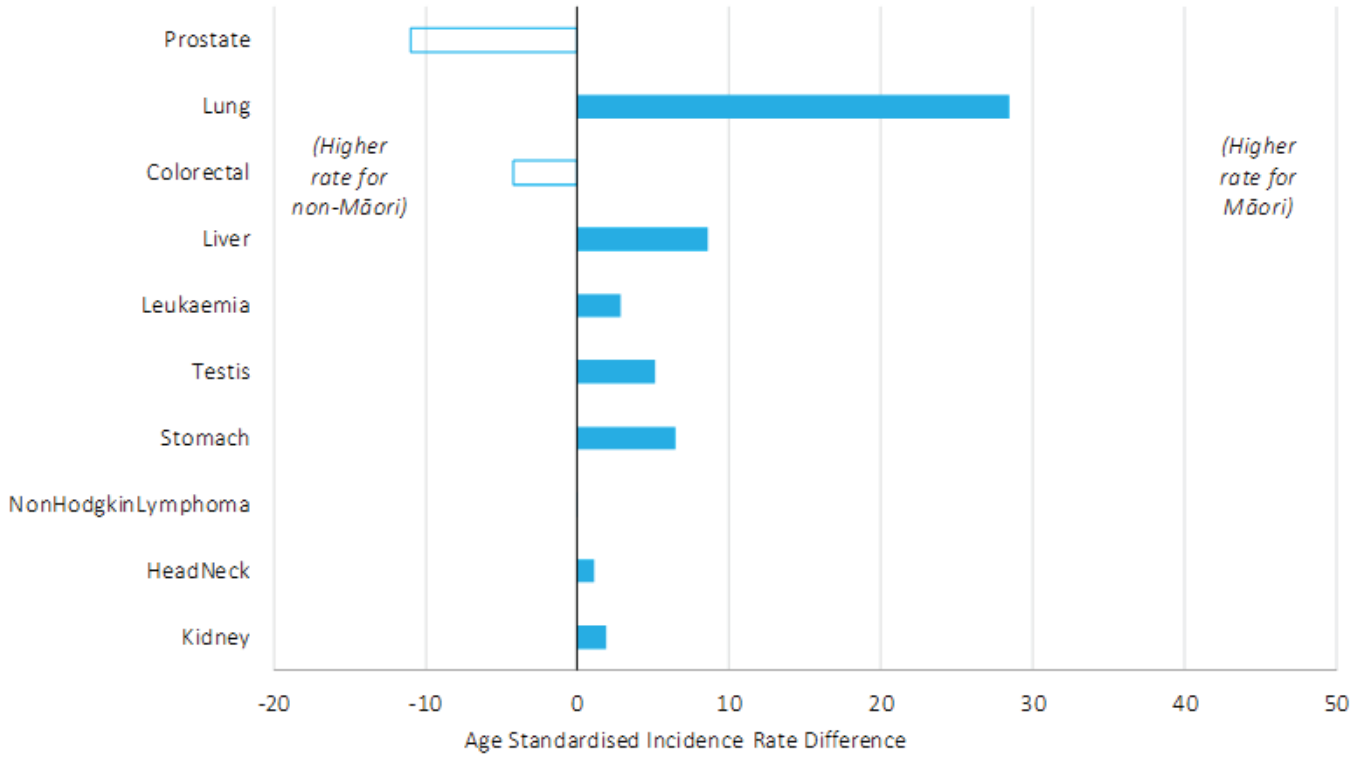
Appendix Figure 1B: Age standardised incidence rate (SIR) for the top 10 most commonly diagnosed cancers among Māori **males** between 2007–2016 (top), along with the age- and sex-standardised rate difference (RD) between Māori and the non-Māori females (bottom).





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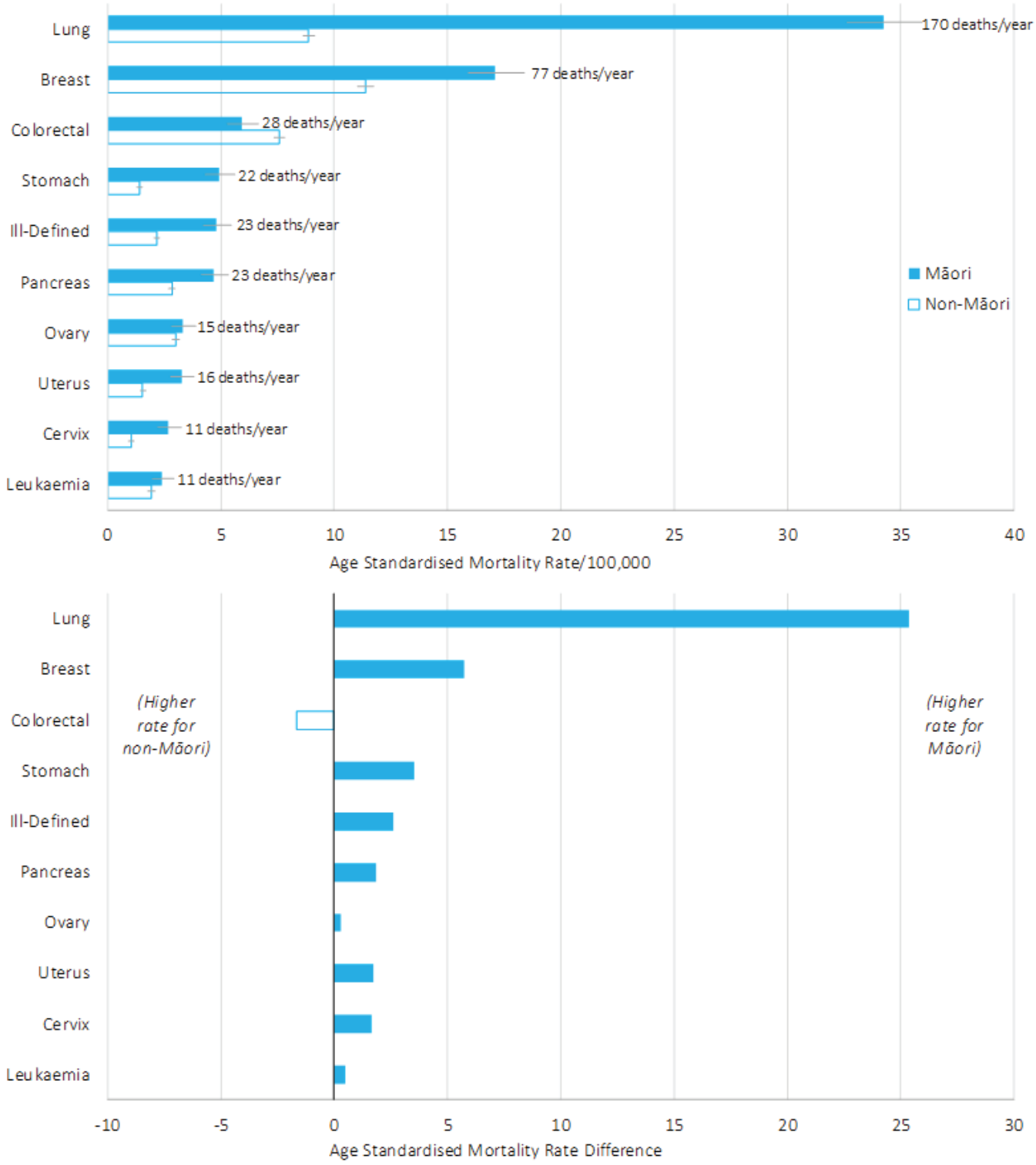
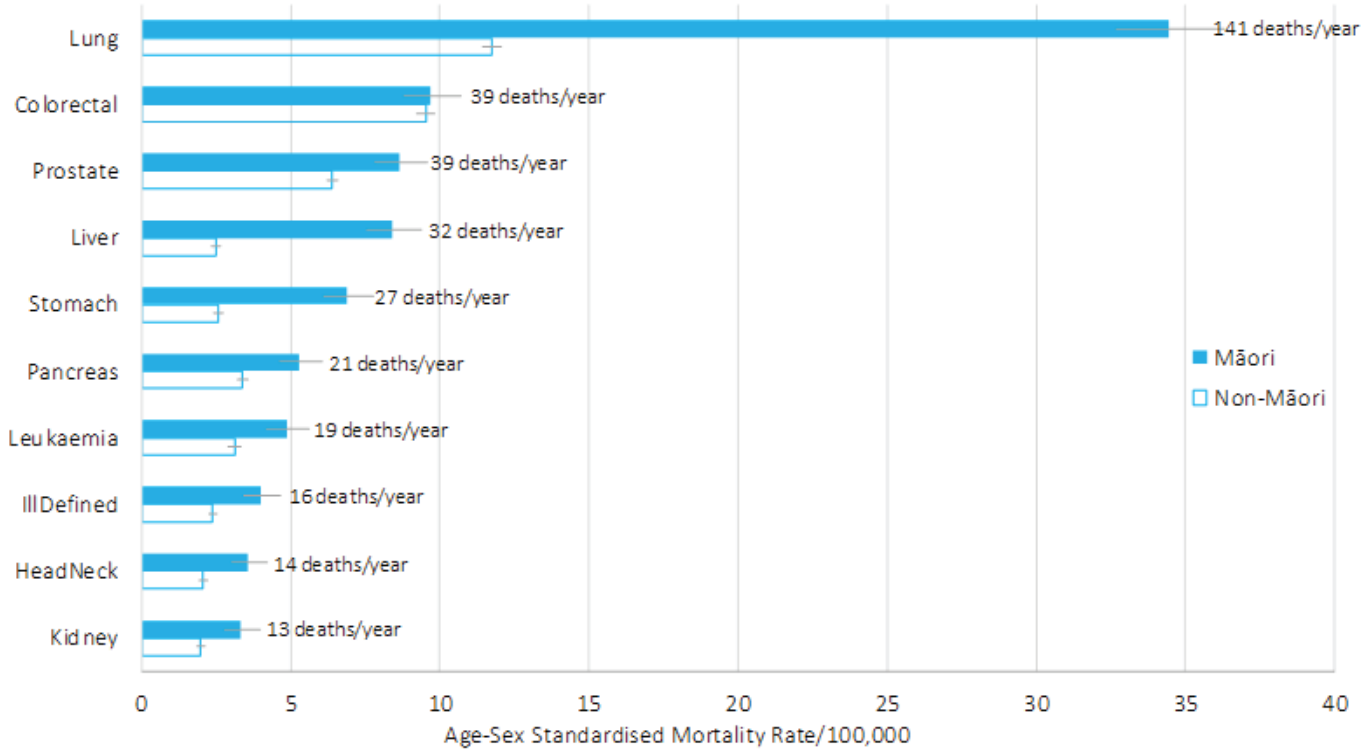
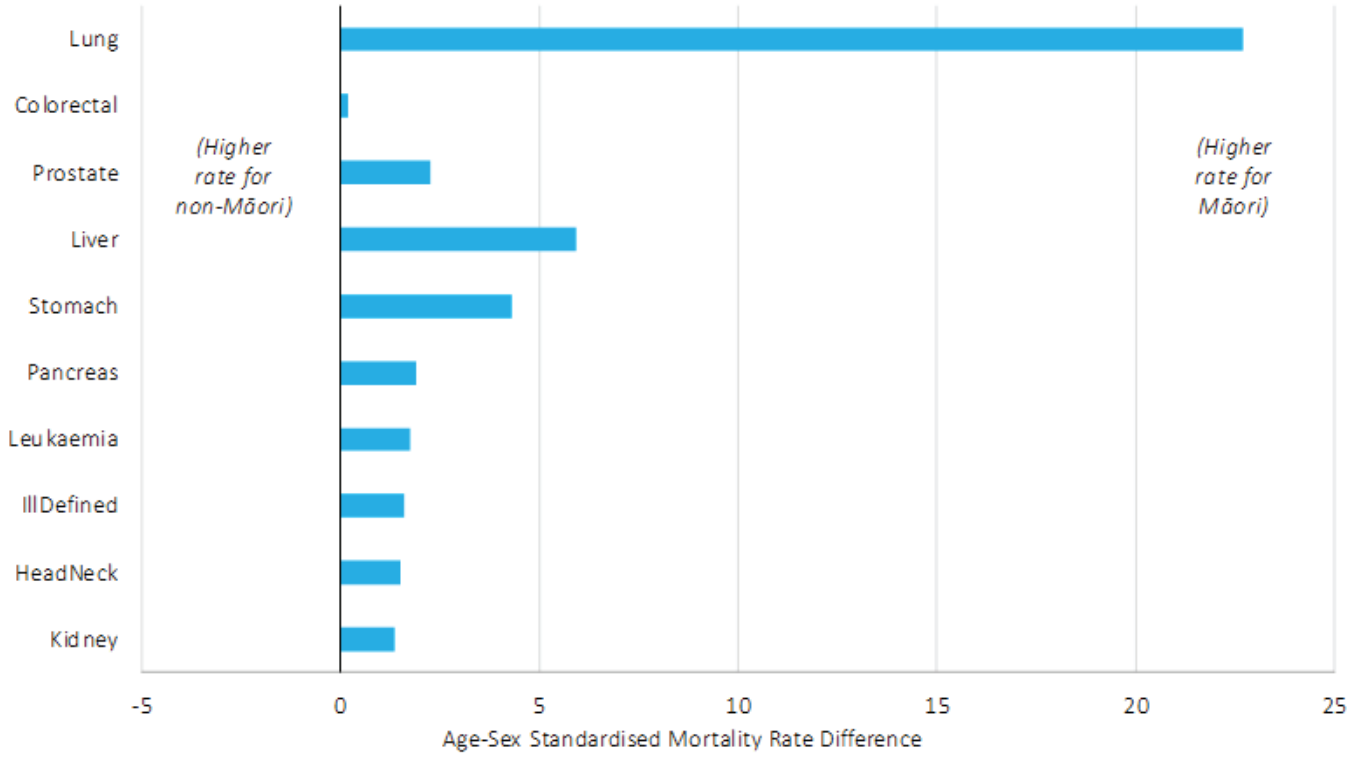
Appendix Figure 2A: Age- and sex-standardised mortality rate (SMR) and absolute numbers of cases for the top 10 most common causes of cancer death for Māori **females** between 2007–2016 (top), along with the age- and sex-standardised rate difference (RD) between Māori and the non-Māori population (bottom).



Appendix Figure 2B: Age- and sex-standardised mortality rate (SMR) and absolute numbers of cases for the top 10 most common causes of cancer death for Māori **males** between 2007–2016 (top), along with the age- and sex-standardised rate difference (RD) between Māori and the non-Māori population (bottom).





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Appendix Table 2: Adjusted hazard ratios (HRs) and 95% confidence intervals, comparing the adjusted likelihood of cancer-specific death between Māori and non-Māori over the follow-up period. Total HRs are for combined sexes and are adjusted for age and sex, while sex-specific HRs are adjusted for age. HRs for cancers where fewer than 10 deaths occurred among Māori over the follow-up period are not shown.

		Māori		
		Total	**Females**	**Males**
Cancer	**Non-Māori**	*Adj. HR (95% CI)*	*Adj. HR (95% CI)*	*Adj. HR (95% CI)*
Anus	Ref	1.6 (1–2.6)	2.1 (1.1–4.2)	-
Bladder	Ref	1.4 (1.1–1.7)	1.5 (1.1–2.1)	1.3 (1–1.7)
Bone and cartilage	Ref	2.4 (1.6–3.7)	3.6 (1.9–6.8)	1.7 (0.9–3.1)
Breast	Ref	1.4 (1.2–1.5)	1.4 (1.2–1.5)	-
Cervix	Ref	1.4 (1.1–1.8)	1.4 (1.1–1.8)	-
Colorectal	Ref	1.5 (1.4–1.6)	1.4 (1.2–1.6)	1.7 (1.5–1.8)
Eye, brain and CNZ	Ref	1.4 (1.2–1.7)	1.3 (1–1.7)	1.5 (1.2–1.8)
Gallbladder and biliary tract	Ref	1 (0.8–1.2)	1 (0.8–1.4)	0.9 (0.6–1.3)
Head and neck	Ref	2.3 (1.6–3.2)	3 (1.5–5.9)	2.1 (1.4–3)
Hodgkin's lymphoma	Ref	2 (1–3.9)	-	-
Ill-defined, secondary or unspecified	Ref	1.3 (1.2–1.5)	1.3 (1.1–1.5)	1.3 (1.1–1.6)
Kidney	Ref	1.6 (1.4–1.9)	1.4 (1.1–1.9)	1.7 (1.4–2.1)
Leukaemia	Ref	1.6 (1.4–1.9)	1.7 (1.4–2.1)	1.6 (1.3–1.9)
Liver	Ref	1.3 (1.2–1.5)	1.3 (1–1.6)	1.3 (1.2–1.5)
Lung	Ref	1.3 (1.2–1.4)	1.3 (1.2–1.4)	1.3 (1.2–1.4)
Melanoma	Ref	2.6 (2–3.3)	3.1 (2.1–4.5)	2.2 (1.6–3.3)
Mesothelioma and soft tissue	Ref	1.2 (1–1.4)	1.2 (0.9–1.6)	1.1 (0.9–1.4)
Myeloma	Ref	1.6 (1.3–1.9)	1.5 (1.1–2)	1.7 (1.3–2.2)
Non-Hodgkin's lymphoma	Ref	2 (1.7–2.3)	2.1 (1.7–2.6)	1.9 (1.5–2.3)
Oesophagus	Ref	1.4 (1.2–1.6)	1.5 (1.2–2)	1.3 (1.1–1.6)
Other digestive tract	Ref	1.3 (1–1.6)	1.6 (1.2–2.2)	1 (0.7–1.5)
Other female genital	Ref	1.6 (1.1–2.1)	1.6 (1.1–2.1)	-
Other immune system	Ref	-	-	-
Other male genital	Ref	-	-	-
Other respiratory	Ref	0.9 (0.5–1.4)	0.7 (0.3–1.8)	0.9 (0.5–1.6)
Other urinary	Ref	1.9 (1–3.7)	-	-
Ovary	Ref	1.6 (1.4–1.9)	1.6 (1.4–1.9)	-
Pancreas	Ref	1.1 (1–1.2)	1.1 (1–1.3)	1.1 (1–1.3)
Prostate	Ref	2.1 (1.8–2.4)	-	2.1 (1.8–2.4)
Skin (not melanoma)	Ref	1.4 (0.7–2.8)	0.7 (0.2–2.9)	1.9 (0.8–4.4)
Small intestine	Ref	1 (0.7–1.4)	0.8 (0.4–1.3)	1.2 (0.8–2)
Stomach	Ref	1.2 (1.1–1.4)	1.1 (0.9–1.3)	1.3 (1.2–1.5)
Testis	Ref	1.8 (0.9–3.5)	-	1.8 (0.9–3.5)
Thyroid endocrine	Ref	1.6 (1.2–2.2)	1.7 (1.1–2.6)	1.6 (1–2.6)
Uterus	Ref	1.6 (1.3–1.9)	1.6 (1.3–1.9)	-

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Diagnosis of metastatic lung cancer from a colonic polyp: case report of a rare histological diagnosis

Hannah Scowcroft, Richard Flint

Lung cancer is the leading cause of cancer-related death in New Zealand, followed by colorectal cancer.[1,2] Common sites of metastases for lung cancer include the brain, bone, liver, adrenal glands, contralateral lung and distant lymph nodes.[3] Here, we present a rare case of non-small cell lung cancer metastasis found incidentally in a colonic polyp.

Case report

An 80-year-old female was referred to the General Surgery Department for investigation of rectal bleeding. Two years prior, she had had a high anterior resection for pT2N0 sigmoid adenocarcinoma. She had recently commenced Dabigatran for newly diagnosed atrial fibrillation. She was also an ex-smoker, with a 40-pack-year history.

Diagnostic colonoscopy revealed an intact healthy appearing end-to-end colo-colonic anastomosis, with three 5mm polyps, as well as one 10mm polyp (Figure 1) at the hepatic flexure.

Histological examination after excision of the largest polyp revealed poorly differentiated non-small cell carcinoma (Figure 2).

The immunohistochemistry profile was not in keeping with a primary colorectal tumour, instead favouring a metastasis. The cells were positive for CK7, CK20 (patchy and weak), GATA3 (weak) and broad-spectrum cytokeratin, while negative for CDX2, CEA, SOX10,

Figure 1: Endoscopic view of hepatic flexure polyp.

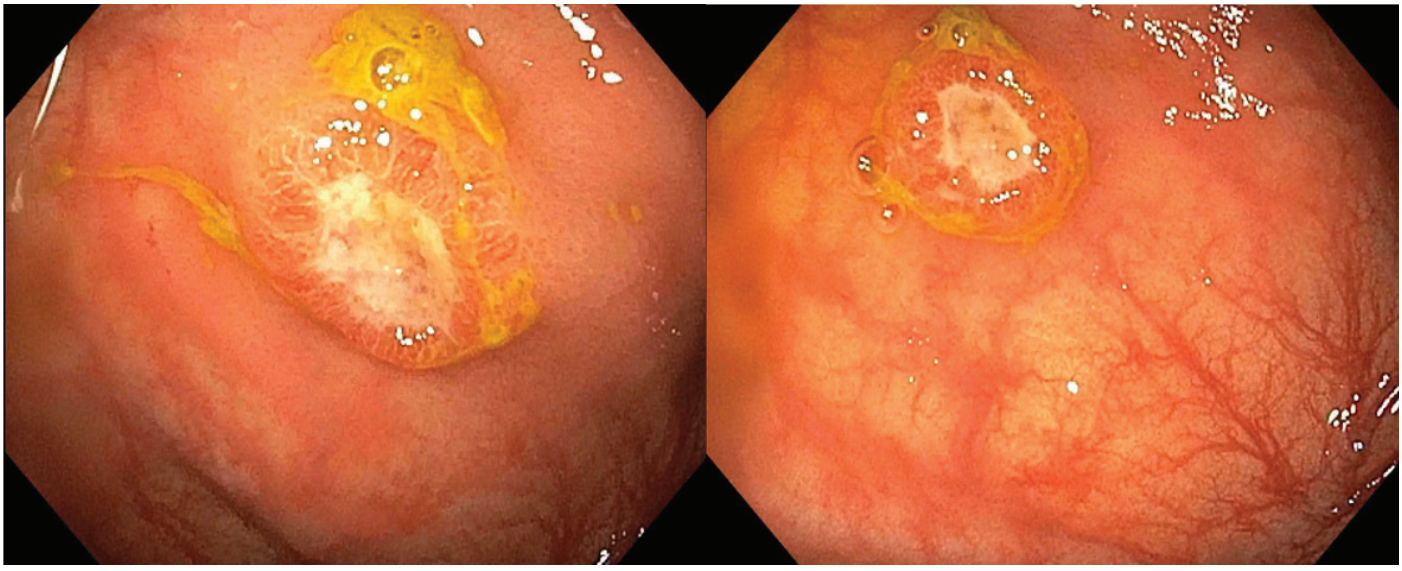
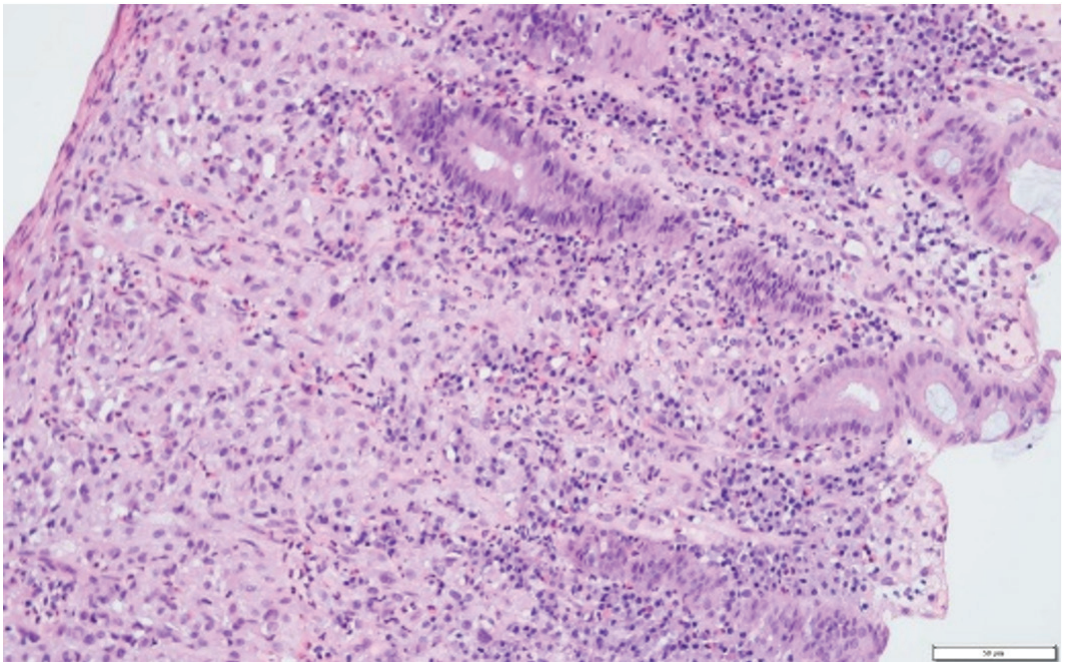


Figure 2: H&E stain of tumour infiltration (left side of specimen).



LCA/CD45, calretinin, BerEP4, oestrogen receptor, GCFP15, progesterone receptor, p40, TTF1 and PAX8 (Figures 3 and 4).

The patient underwent cross-sectional chest imaging which showed a 13mm left upper lobe nodule (Figure 5).

This was radiologically suspicious for a primary adenocarcinoma pulmonary malignancy, and the patient was subsequently referred to the Medical Oncology service.

Discussion

Metastatic disease to the colon from an extra-colonic primary malignancy is very rare. This is reflected by the limited number of reported cases. In a large multi-centre European study that examined 10,365 colorectal malignant tumour patients,[4] only 35 (0.34%) were found to have metastasis to the colon from an extra-colonic primary tumour. The most common primary site was breast with 17 cases. Most cases are asymptomatic; however, can present drastically as severe anaemia or bowel perforation.[5–7]

Primary lung adenocarcinoma metastasis to the colon is exceedingly rare. Pulmonary metastases to the small intestine are more common than to the stomach or colon.[8] Most cases of primary lung cancer that metastasised to the gastrointestinal tract were squamous cell carcinoma and large cell carcinoma, not adenocarcinoma.[9] The exact incidence of adenocarcinoma metastases to the colon is unknown. Most publications found in the literature were isolated case reports with a known pulmonary malignancy background, which further highlights the rarity of this clinical finding of a lung cancer initially manifesting as GI-tract involvement.[10,11]

Adenocarcinoma of the lung presenting as metastatic colonic polyp has not been reported in New Zealand before.

Figure 3 and 4: Immunohistochemical stain showing strong CK7 and patchy very weak CK20 staining.

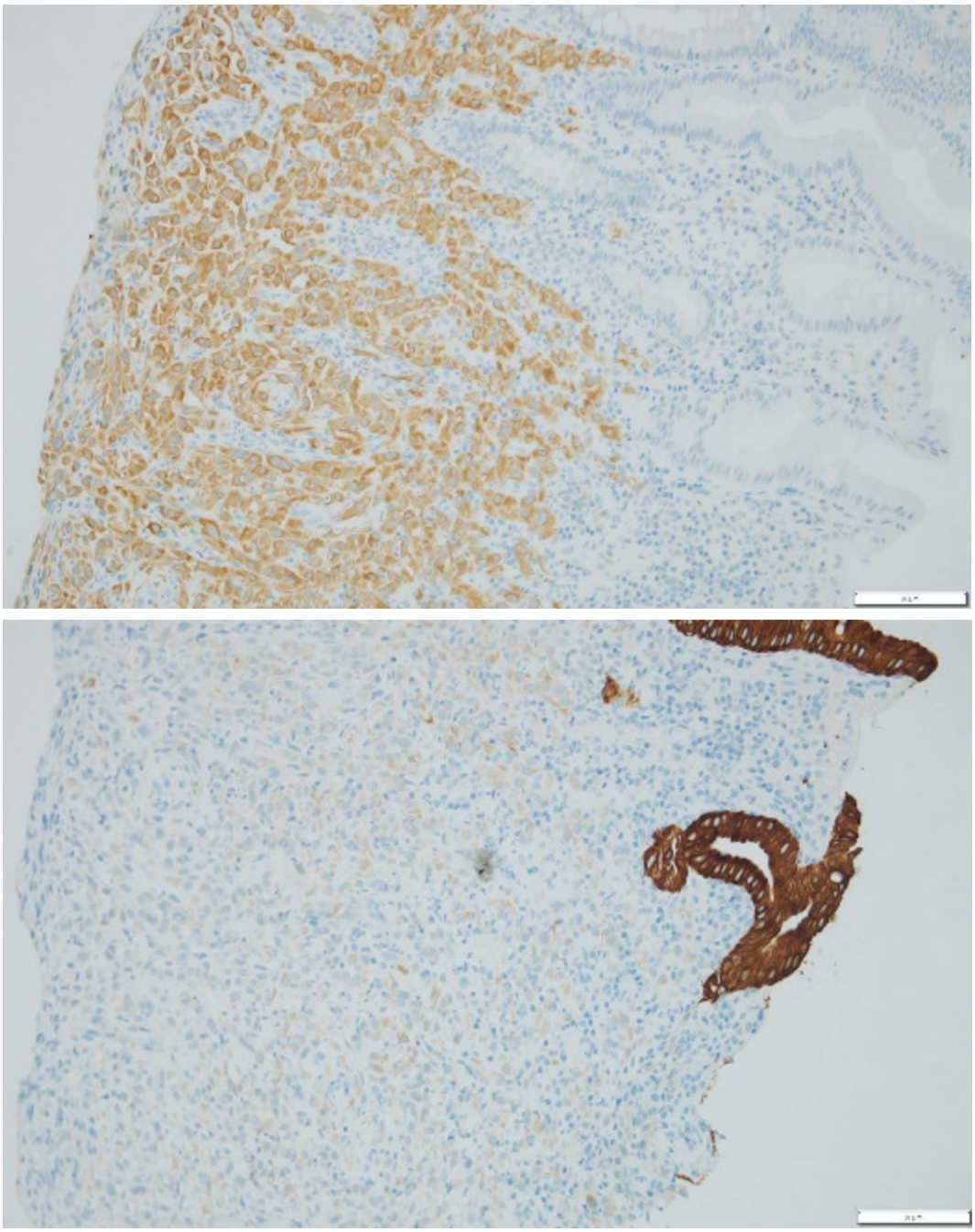
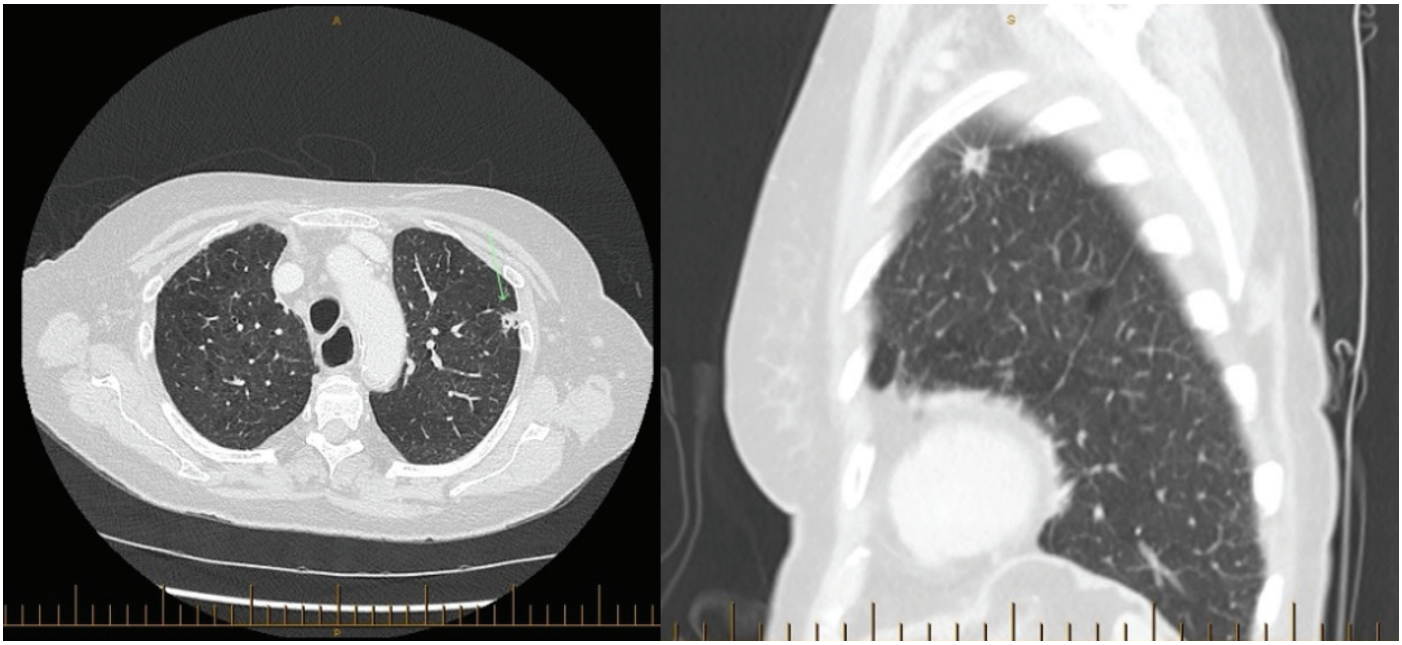


Figure 5: Transverse and sagittal CT images of the left upper lobe nodule.



Competing interests:
Nil.

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The mule who took us for a ride

Islam El-Abbassy, Benjamin Perakath

ABSTRACT

Foreign body ingestion is not uncommon in patients with mental disorders, alcohol intoxication and for purposes of drug trafficking. Small objects pass spontaneously; however, larger ones may get stuck in the oesophagus, stomach or at narrow areas of the bowel. 'Body packers' is a term used to describe persons who swallow or insert drug-filled packets into a body cavity. They are also called 'swallowers', 'internal carriers', 'couriers' or 'mules'. We report a 37-year-old previous drug abuser who presented with dysphagia. Upper GI endoscopy showed an oblong foreign body covered in plastic in the lower oesophagus. This could not be extracted and hence was pushed into the stomach. Three weeks later, he presented with bowel obstruction that was shown on abdominal radiograph and confirmed by CT indicating multiple dilated small bowel loops with a transition point in the terminal ileum where the ingested package was identified. The package was then removed through a longitudinal enterotomy. Ingested foreign bodies causing dysphagia should ideally be extracted endoscopically. If not possible, then a watch-and-wait policy may be justified. While most ingested objects pass spontaneously, unusual and larger ones may require surgical extraction. The contents, nature and reason for ingesting this strange object remain a mystery. With history of drug abuse and the consistent denial of knowingly swallowing that object, we can only conclude that the patient was trying to transport an illicit drug in the packet.

Foreign body ingestion is not uncommon in patients with mental disorders, alcohol intoxication and for purposes of drug trafficking. Small objects pass spontaneously; however, larger ones may get stuck in the oesophagus, stomach or at narrow areas of the bowel.[1]

'Body packers' is a term used to describe persons who swallow or insert drug-filled packets into a body cavity in an attempt to smuggle them. They are also called 'swallowers', 'internal carriers', 'couriers' or 'mules'.[2]

Case report

We present a 37-year-old male who presented with dysphagia. Upper GI endoscopy showed a foreign body in a plastic bag in the lower oesophagus, which could not be retrieved (Figure 1). Therefore, it was pushed into the stomach.

Three weeks later, he presented with symptoms and signs of bowel obstruction. Abdominal radiograph showed dilated loops of small bowel and a possible foreign body. Computerised tomography (CT) confirmed bowel obstruction with a transition point at the terminal ileum where the package was identified (Figure 2).

Laparotomy revealed the package in the terminal ileum with proximal dilated and distal collapsed bowel. It was removed via a longitudinal enterotomy (Figure 3). On opening the plastic cover, it turned out to be a wad of folded and tightly rolled up paper with illegible handwriting.

While the patient had a past history of substance abuse and had several gastroscopies for swallowing illicit substances, he claimed to have been reformed, and adamantly denied swallowing that object being surprised at its nature and contents.

Discussion

Endoscopic management of oesophageal foreign body include en bloc removal, piecemeal approach or advancement into the stomach.[3] Conservative outpatient management is indicated in cases where the object has entered the stomach.[4]

Figure 1: Upper GI endoscopy showing a plastic bag in the oesophagus.

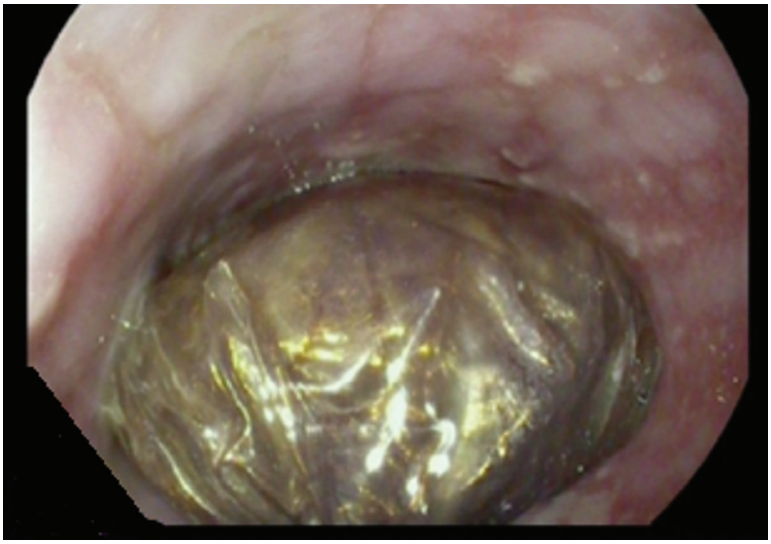
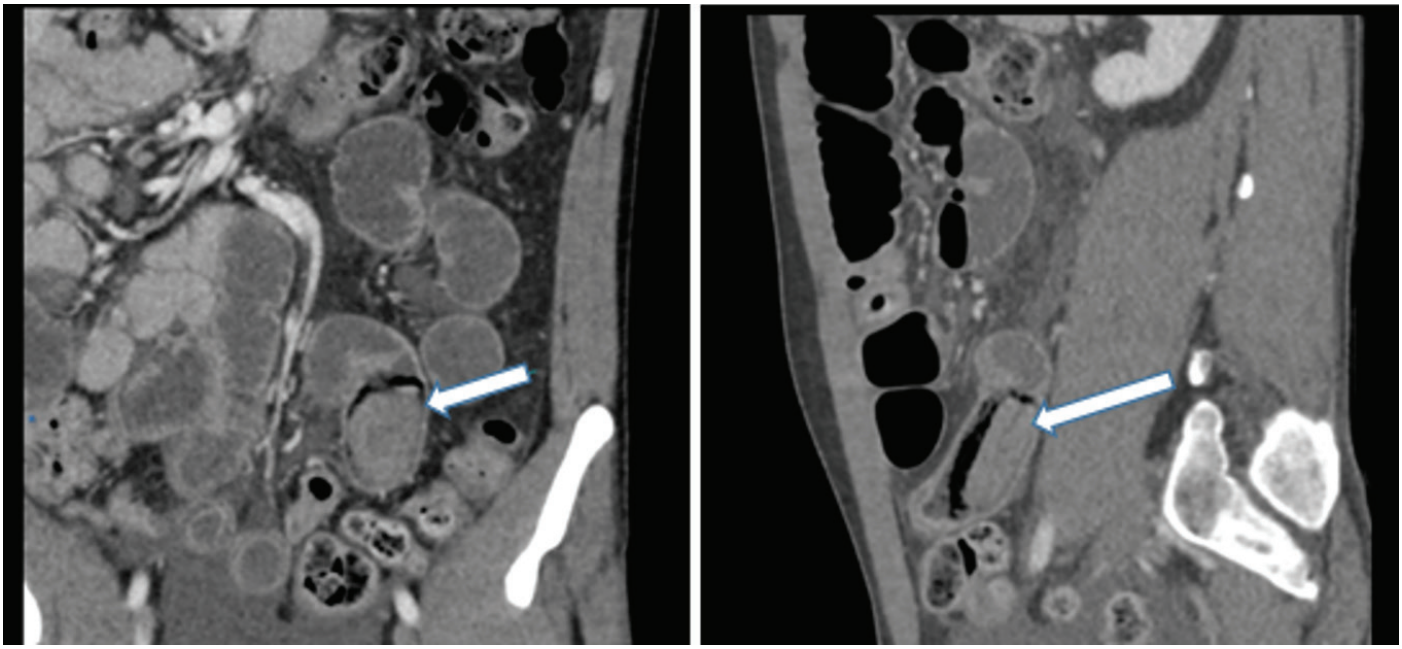


Figure 2: Coronal and sagittal CT views showing a rim of gas around the package.

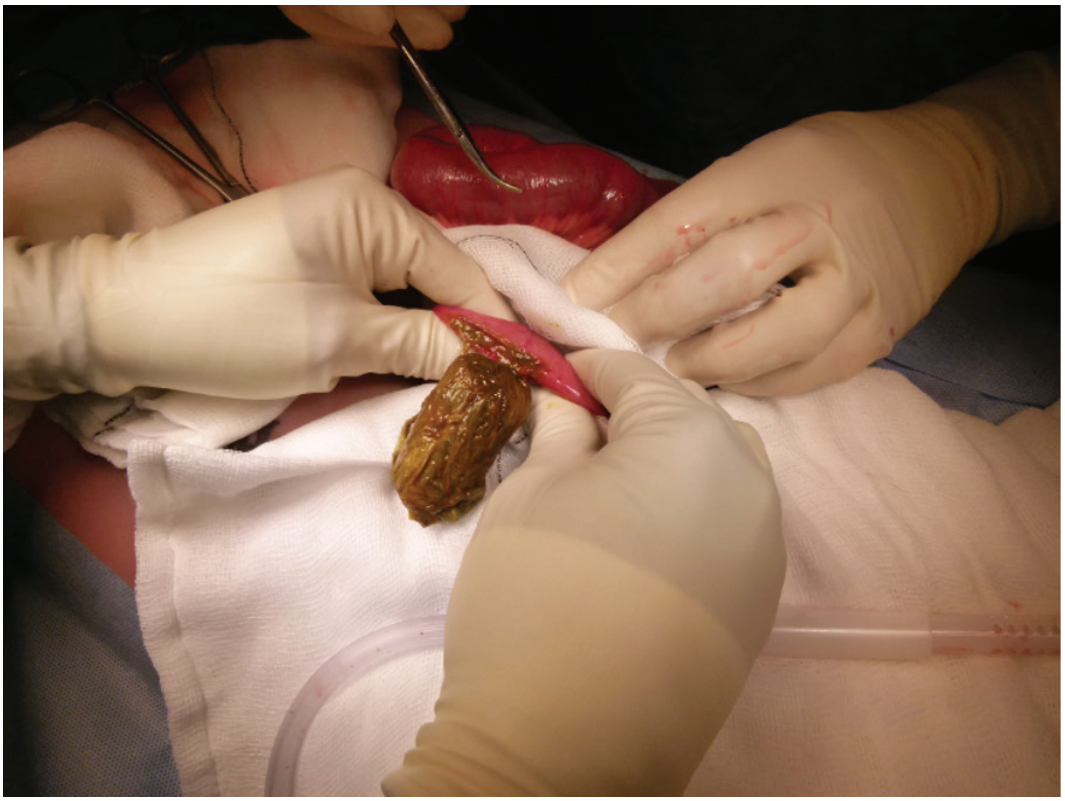


Most ingested objects pass spontaneously within four days to four weeks. Surgical intervention should be considered if the object passes the stomach and remains in the same location for more than a week.[5]

We could not retrieve the package during the upper GI endoscopy. Given that it was plastic and soft (measuring around 5x3cm), it was pushed into the stomach, hoping it would pass spontaneously. There is still controversy regarding the push technique because it carries a risk of perforation when performed without examining the distal oesophagus first.[6] Some studies have reported that this technique has a success rate of over 90% and is the primary method for managing food bolus ingestion with minimal complications.[7,8]

Plain abdominal radiography has been reported to be diagnostic for swallowed packets (with sensitivity of 90%) as it may show 'rosette-like' or 'double-condom' signs; however, contrast CT is more sensitive.[9] In our case, plain radiography showed signs of obstruction, with a suspicious foreign body.

Figure 3: Package removed through enterotomy.



In body packers, surgical intervention is required in less than 1% of cases. Perforation is an absolute indication, whereas unsuccessful endoscopic retrieval is a relative one.[10] In our case, the patient underwent laparotomy and the package retrieved as soon as obstruction was confirmed radiologically.

Conclusion

Ingested foreign bodies causing dysphagia should ideally be extracted endoscopically. If not possible, then a watch-and-wait policy may be justified. While most ingested objects pass spontaneously, unusual and larger ones may require surgical extraction.

The contents, nature and reason for ingesting this strange object remain a mystery. With history of drug abuse and the consistent denial of knowingly swallowing that object, we can only conclude that the patient was trying to transport an illicit drug in the packet.

Competing interests:
Nil.

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Doctor narratives on burnout and allergic reactions to talking about feelings: what are the unspoken rules when talking to doctors?

Kathryn Russell

The views expressed in this piece are the opinion of the author and do not reflect the views of any institution.

Problem 1: Bunch of blimmin' high-achieving, over-intelligent perfectionists (some of my favourite people are doctors)

Kevin, are you okay? How you going with all this?—Yes I'm fine, a little busy; I'm worried about Bob though.

Bob, I just want to check in with how you are doing.—Yes I'm fine, it's what I trained for, but I'm worried about Kevin.

'I'm fine' definitions

1. I think I'm coping but people keep asking (Cryptic clue: Germany, river in Egypt (6)).
2. I'm not coping but don't want to talk about it because I fear I might crumble into a heap on the floor—so don't be kind to me and piss off with your mamby pamby feelings crap.
3. I *will* be fine as soon as I get my very large glass of wine.
4. I'm on autopilot and "I'm fine" is a conditioned response which did not pass higher cortical gatekeeping.
5. I'm coping quite well, I have insight and reflect on my feelings often and I'm well supported and not experiencing any concerning signs of built up stress. I attempt self-compassion, and occasionally mindfulness. I may or may not have a scented candle (Don't judge).

Problem 2: Unspoken doctor rules of what is and isn't allowed to be said, as unscientifically collected through 15 years of informal doctor observation (ethical approval not obtained/not peer reviewed)

Okay to discuss: General references to burnout, workload, hours, pressure and being busy.

Okay to discuss: Teamwork, what we learn from this, communication, improving team function, team and inter-team relationships, concern for others and systems.

Okay to discuss: General vague references to "support" preferably of 'other' not 'self'.

Less discussed: Daily burden of the consequences of decisions, habituation to risk and stress, self-protection strategies in response to emotional distress and trauma—depersonalisation and dissociation.

Less discussed: Carrying the day home, riding an emotional roller coaster of success and sadness and stoic responses in the face of unbearable pressure.

Not discussed: Reality of burnout, depression, anxiety, suicidal thoughts, relationship breakdown and alcohol or drug use.

Problem 3: The truth of mental health for doctors vs the 'I'm fine' narrative

Suicide, depression and anxiety rates in doctors are higher than age-matched non doctors.[1,2]

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- Ten percent of doctors have suicidal thoughts in previous year vs general adult population ~4%.[1]
- Fifty percent will experience burnout in career.[3]
- Alcohol misuse five times higher than general population.[4,5]
- Low rates of doctors get their own regular healthcare with a GP—it is compulsory in the UK.[6]
- Depression rates may be higher than general population, but only 16% of doctors with depression seek any treatment.[7]

Problem 4: Silence…why?

- Embarrassment
- Fear of impacts on registration—rights to confidential treatment?
- Inter-doctor stigma—a doctor who has sought help is inferior? Weak? Tainted? Inept?
- Pessimistic view of the value of mental health services—*Talking therapy is a bit stupid, the therapist won't be as smart as I am, otherwise they would have become a real doctor*.
- Fear of psychotropic meds.

Problem 5: More barriers and excuses

- I can manage by myself.
- No time.
- Fear of being reported.
- Burnout more "acceptable" than depression.
- Doctors treat doctors differently—engage in medical talk, discuss papers (this defence mechanism is called intellectualisation)—this limits the doctor-patient norms and keeps the relationship doctor-to-doctor, which may interfere with effective treatment.

What to do?

Drop the defences dude. Doctors need to take a break from self-diagnosis (and self-medicating) and just be the patient for a little bit.[8]

Mindfulness, self-care, time off, exercise and healthy work environment—some of these are seen as too touchy feely, and there is a general tone of cynicism expressed. To combat this you need a bit of humour and peers who are wellbeing enthusiasts due to their own experiences.

Doctors need to start to talk about vulnerability and responses to trauma to reduce the stigma and model good behaviour. It is not okay to just talk about what can be learnt from stressful situations.

Changes are needed at group, social and institutional levels to transcend the barriers. Schwartz rounds are trending.[8]

Get a GP, for $%^*'# sake, you could get your cholesterol checked as a cover story. Maybe find your own therapist—if you don't like them then get another one, get recommendations for someone good or try an online option if you are persistently allergic. If you have a therapist-in-waiting you can get in more easily when you are ready. The first time is the always the most difficult.

You're worth it.

(I saw that eye roll.)

Competing interests:
Nil.

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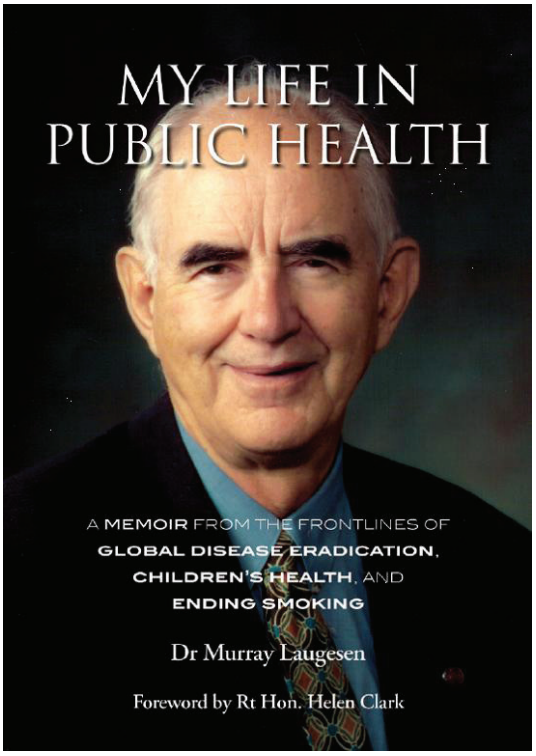
My Life in Public Health

Frank Frizelle



Dr Murray Laugesen. Published by Health NZ, 2019. ISBN 9780473470913. Contains 344 pages. Price NZ$45.00. Foreword by Rt Hon. Helen Clarke.

In the time of Covid, some of the superheroes of the health sector are the public health physicians. This book outlines the active and varied personal and professional life of one New Zealand public health physician. The 341-page paperback book, with coloured photographs and easy-to-read text, spans Murray Laugesen's life, ending with 2018 election of the Ardern-led government. The book is broken into 12 chapters, and as one might expect from an academic, is well referenced and has an appendix, a section on abbreviations and a glossary.

The book starts as usual at the beginning of his life, with an outline of his family history in New Zealand, his early education, and his time and reflections on medical school and his subsequent training in surgery. At several points he deviates away from the narrative to comment on smoking-related activities and how this has changed from a harmless common pastime for all, (including doctors) to today's perspective of a harmful and damaging addiction. The following chapters explore his time in India as a missionary surgeon in India, and the effect of the Indo-Pakistani war and the Bangladesh war of independence, and leads from this to his developing understanding of the importance of public health issues, leading to his first real public health position with his appointment to the coordinating agency for health planning in India. In this position he was involved in the management of children's health and infectious diseases such as small pox, polio, leprosy and tuberculosis.

Returning to New Zealand in in 1978, he was appointed to the Department of Health as principal medical officer (child health) and became involved in many aspects of policy development of child health, and subsequently tobacco control, where his

efforts had a lasting influence on this aspect of health policy in New Zealand. Murray was involved with the Public Health Commission and subsequently retired just before his 60th birthday in 1995.

After retiring from the public sector Murray established his own firm, Health NZ (who published this book), contracting himself out on health policy, research and planning, of which was tobacco related. The book continues to comment on such contemporary issues as his thoughts on religion, the Canterbury earthquakes, his own experience of ill health and the present Labour-led government.

This book describes the evolution of a public health physician and beautifully describes the extent and impact of public health across many sectors. The book would be of interest to most doctors from young doctors trying to work out which career direction to go, to the older who enjoy reflecting on how things have changed.

Competing interests:
Nil.
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Streptococcal Infections and Infectious Mammitis of the Cow

By W. E. STEVENS, M.R.C.S. (Eng.), L.R.C.P. (Lond.), New Brighton

A great many cases of these infections have come under my care during the last few years, and some of them I have been able to trace directly and certainly to infected milk. So conclusive has the evidence been that I decided to quote notes of my cases to you, gentlemen, and the conclusions that I have come to regarding them.

I shall bring forward evidence to prove that this infectious mammitis of cow is due chiefly to a streptococcal infection, although other germs are also frequently present. l may say in passing that it is probably only those of us who have practised in country districts who have had the opportunity of proving these factors in producing these and mixed infections.

On 19th October, 1919, I was called in to attend the three children of Mr. R—, of Geraldine, they having previously been treated by Dr. Hyslop of that town, who recognised that they were suffering from some form of germ infection, and as they were too far from him to treat them as he would like, they were removed to Christchurch and then on to New Brighton. When I first saw them their condition was as follows:—

Tom, aged 3 years.—Temp. 102 to 103 every night; glands on both sides of neck very swollen, also tonsils, and one ear discharging profusely. Dirty spotty tongue.

Janet, aged about 5 years.—Sores resembling impetigo all over feet, legs, and part way up thighs; hands and arms covered with a rash which looked like a cross between scarlet fever and measles (which parts subsequently peeled). Spotty tongue.

Elizabeth, between 7 and 8, was running about, but looked pale and feverish, evidently not well, and had a dirty spotty tongue, and on 28th October she developed well-marked scarlet fever and was sent to Bottle Lake Hospital for that disease. Here she developed two mastoid abscesses, which were operated on by Dr. McGibbon, and afterwards ran a very high temperature (probably due to some septic inflammation of some of the deep veins), and was treated by anti-streptococcus and other serums, and ultimately recovered.

The previous history of the cases was as follows:—

Up to the beginning -of August, 1919, these children had never had a day's illness, and were all fine, healthy specimens of childhood.

On 8th August a new cow calved on the station, a good cow, and apparently quite sound. This cow was milked by two members of a neighbouring family, both of whom were suffering from sores on hands at the time the trouble occurred (probably impetigo).

On 25th August all the children's temperatures were over 100deg. Fah.

On 30th August Tom very feverish and treated for tonsillitis.

On 4th or 5th September Janet started sores, and all the children had been unwell all the week.

On 15th September Janet treated for impetigo. Tom had very swollen glands of neck.

On 7th October Tom's neck still very swollen and ear discharged slightly. He was again taken to Dr. Hyslop, who said they had some form of germ infection.

On 9th October Dr. Hyslop thought Tom was sickening for something else. Brought children in to Christchurch.

On 10th October consulted Dr. Irving of Christchurch for Tom and Janet. Tom's ear discharged enormously.

On 14th October came to New Brighton.

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On 19th October I was called in, and thought I recognised streptococcal infection, probably from the milk, as I could ascertain no other source, and I procured a specimen and had it examined by Dr. Pearson, and obtained the following report. The sample of milk was taken from cow by Mr. R. himself and all outside contamination eliminated:—"Microscopic examination: Profuse pus cells of polymorph type. Gram positive cocci resembling streptococcus in very large numbers. Cultures showed a profuse growth of streptococcus. The reaction of this organism has not yet been worked out."

From this report it was evident that the milk was the cause, but whether the cow had become infected from the man or the man from the cow it is hard to say; but probably, as the children were all perfectly healthy until this cow's milk was used, the man's hands became infected from the cow; or it may have been a coincidence that the man had sores on his hands.

I was led to this train of thought by two other cases of streptococcal poisoning from milk some years ago, when resident at Kurow.

A Stock Inspector asked me to make up a Winchester quart of 4 per cent. boracic acid lotion, which, on enquiry, was required to treat the udder of a cow suffering from "infectious mammitis," which is due to a streptococcal infection, and sometimes attacks one quarter of the udder only, sometimes, of course, several quarters.

As this was a very valuable Ayrshire cow which gave over a bucketful of milk at each milking, the Inspector and his agents persevered with her until they thought they had her cured, and afterwards purchased her. At the next calving he milked her and used the milk, but whether he was in the habit of drinking it warm from cow or not I don't remember; but I was called to treat him soon afterwards for pelvic cellulitis, with retention of urine. He was in great agony and had to have catheter used night and morning for a good long time. On examination per rectum, all the pelvic organs were set, as in a hard block; an abscess subsequently formed beside bowel and pointed, and was opened in ischiorectal fossa, the pus from which contained streptococci. I also examined stools a considerable time after he was able to go about, and still found streptococci; he was about six months ill altogether. I cleared up the bowel infection at last with methylene blue.

One of his agents who also used the milk became infected soon afterwards with a troublesome colitis, which impelled him to lie up for a considerable time.

I persuaded them to have cow dried off and fattened; as I felt certain she was the cause of trouble.

There are so many cases of streptococcal poisoning, from throat downwards. that there is evidently some prevalent and general cause for the trouble, and what more likely than that one of the main causes is infected milk? This infectious mammitis (known amongst dairymen as "bad quarter") is one of the commonest troubles amongst dairy herds, and unfortunately the owners do not recognise the seriousness of the trouble sufficiently to throw the milk from the cow away, and the danger to the public in putting it in the cans. In the suburbs of Christchurch we have large numbers of cases of streptococcal infections of throat, some of them no doubt due to open drains leading into holes in gardens; and this condition of affairs is very common in some of the rapidly-growing suburbs of Christchurch and other cities, where no scheme of proper drainage is in existence to carry off the wash-up water from the dishes. I have just had three cases of streptococcal poisoning in one house where the following condition of affairs existed: Untrapped sink in kitchen leading into an open wooden drain in garden, which drain is moved about in different directions in garden to allow contents to soak in.

I took a swab from this drain and obtained a bacteriological examination of same from Christchurch Hospital. Here it is: "On cultivation the specimen showed a mixed growth of bacillus subtilis coli and streptococcus."

One of the children had high temperature, spotty tongue, enlarged tonsils and glands and endocarditis, with loud murmurs. Now all are quite cleared up with the treatment I mention later on. The two other members of family had the sore throat and spotty tongue. So that I know that this state of affairs also is responsible for a great many sore throats of streptococcal type. What more favourable condition could these aerobes have for their growth than an open drain?

But there are many other cases in which no cause can be traced and are most likely due to milk which has been infected.

Two cases of Henock's purpura in which I had stools examined were both due to streptococcal infections of the intestines; one of them also had streptococci in urine; both recovered. One of them had many doses of anti-streptococcal serum given. either by the rectum or mouth, and both took a salol mixture for some time. I could not find any definite cause for Henock's purpura from a bacteriological point of view laid down in any text-books, but certainly both of my cases were due to that form of infection.

Then there are those rare cases of streptococcal peritonitis which one drops across occasionally, one of which I have in my mind at present, in which abdomen was opened and drained and all infection from appendix excluded, as it was perfectly healthy.

How often when a mother's milk supply fails and one puts the infants on humanised milk do we meet with disappointment! And why? Again and again I have done so, and in a few days' time what do we have? Green motions, diarrhoea, and vomiting, clearly pointing to some infection in the milk. So often has this happened in my practice in New Brighton that I always insist on the milk being brought up to 155deg. Fah. and sterilised, both for infants and household use; and this I would do even if we could exclude tubercle baccili.

Again, one of the most pathognomonic symptoms of this kind of germ infection (and the most persistent) is the "spotty" tongue. It differs somewhat from the scarlet fever strawberry tongue in being "muddy" (as well as spotty), instead of red. Wherever one sees it, one can look for the streptococcus and his works. Take a lot of cases of endocarditis in children (very common in these parts), where there is absolutely no history of rheumatism or any of the ordinary infectious diseases, and look at tongue, and in a great majority you will find the "spotty" condition, and you can go for that germ with success; and here, again, other causes being excluded, we are driven to suspect the milk.

Another very common form of ailment in children is an enteritis or gastro-enteritis, where child has been off colour for some time, and where no cause such as ptomaine poisoning can be proved; here, again, the spotty tongue points to streptococcus poisoning, and milk, being the only uncooked food, is often the offender.

Other cases of acute gastro-enteritis, where child has been perfectly healthy a day or two previously and is suddenly stricken down and in a state of collapse, point to a large dose of some poison which has entered by the alimentary canal, and often there is nothing in the way of food to which the illness can be traced; here, also, the milk supply should be suspected.

A few years ago l was called to see a child whose case puzzled me very much at the time. She was vomiting and purging as if suffering from irritant poison, but I could not discover any possible cause in the food or anything growing about the place; moreover, the temperature was 105deg. Fah. Shortly afterwards the child developed broncho-pneumonia, ran a very high temperature, and finally died. Streptococci were found in sputum and polyvalent anti-streptococcal serum was given, and case improved for a time, but relapsed and died.

Shortly afterwards a second child in same family was taken with broncho-pneumonia, and although I tried the serum again this one also died. With the light of my present knowledge I should say that the reason those children did not sustain the improvement was because I insisted on feeding them on milk; and that milk was in all probability the cause of the trouble. The milk came from the family cow and would have been easily traced had I thought of it. The people themselves were very clean and kept their house very clean also.

Another case that came under my care some years ago: A little boy about eight years old was in convulsions; his face and body were dropsical and he was covered all over legs and part of body with impetigo-looking sores; his urine contained blood and was almost solid on being boiled. Here was another case in which the child had evidently had a large dose of some food or fluid containing streptococci, and if I had a similar case now I should suspect the milk.

The great prevalence of tonsillitis is due to either streptococcal (the most common), Vincent's angina, diphtheritic or tuberculous infection, and of these the first and last are most likely to lead to chronic enlargement,

and what more likely cause of infection can we have than milk, when we remember how children are fed with this article of diet?

One of the most interesting and peculiar developments of these three cases I have mentioned was the third child, Elizabeth, developing scarlet fever. Dr. Pearson, in his report, said that "the reaction of this organism has not yet been worked out." I was most anxious to know whether he could isolate a streptococcus identical with the scarlet fever type, but owing to his leaving so soon for the Old Country he had no time to finish his researches.

As far as I could ascertain, this child had not been subjected to any scarlet fever infection. All three had kept together and the house they went into in New Brighton had never had any scarlet fever in it; and again, if she developed it from outside infection, why did not the other children also have it? If it could have been proved that infectious mammitis in the cow could start and spread an epidemic of scarlet fever, it would explain many of our recent outbreaks; and I would say that there is a very strong supposition in this case that the elder child did so develop it. For, if the same source of infection could affect the two younger children so differently, why should the third child not develop some different form of streptococcal infection such as scarlet fever? I do not know how far this form of reasoning will influence pathologists, but to my mind the evidence seems sufficiently grave to warrant further research.

Some text-books describe a form of scarlet fever "sine eruptione," and how often do we see one member of a family of children affected with what was formerly known as "scarlatinal sore throat," and then other members of same family develop a really fine scarlatinal rash! Does not this point to some common infection?

It has long been recognised that the infection of scarlet fever (as well as typhoid and diphtheria) can be carried in milk; but I do not think it has ever been suspected to be due to milk from a cow suffering from infectious mammitis; and yet this disease of cow is most prevalent right through the country, and I have already shown what havoc it wrought in Mr. R.'s family, and that the resulting developments were different in the case of each child; and to my mind it needs no great stretch of imagination to believe that this streptococcal infection of udder is one probable cause of scarlet fever.

A word as to treatment of these children: My favourite prescription for these chest, throat, and heart infections is a mixture of spt. amm. co., spts. chlorof., tr. cinchona co., sod. sal., syrup aurantii.

The two younger children improved so fast on this mixture (which has a double shot at the germ) that they were practically well in ten days. The girl's sores were carefully washed with an antiseptic solution and dressed with Lassar's paste with 2 per cent. acid salicylic, and healed very swiftly.

I prefer Lassar's paste with the 2 per cent. acid salicylic where large surfaces are affected with these sores, and ungt. hyd. ammon. chlor. where only few sores are to be dealt with.

The mixture quoted also works well in cases of streptococcal endocarditis.

In these cases of enteritis of streptococcal origin I found nothing equal to salol as a disinfectant; if there is vomiting as well as diarrhoea, I give hydrg. c creta with a small proportion of opium for a start and then castor oil, and follow up with salol and bismuth and pulv. ipec. co. or tr. opii if necessary. And such is my treatment of infantile diarrhoea with green stools—usually omitting the opium, except where compelled to give it for pain and exceptionally frequent stools.

As to prevention, it seems to me that the medical profession should urge much more rigid and thorough examination of dairy herds, cow-sheds, dairies, and the actual milk supply itself. At present it is done in a very half-hearted, inefficient, and untutored way, and is a continual source of danger to the public.

More inspectors and more inspection is required. All herds of dairy cows should be tested for T.B. once or twice yearly.

Frequent inspections as to conditions of udders; also of sheds, dairies, and the milkers, say, every three months.

Not only should milk be tested for additions of water and preservatives, but, what is more to the point, samples should be taken unawares and systematically examined by bacteriologists, or it may in the course of time be done by the inspectors

themselves after a proper course of instruction by bacteriologists. At present the inspection of herds is very inadequate, both as to the frequency of the examinations and the omission of these bacteriological examinations that are so much more necessary than the mere finding out how much water has been added to the milk. Even if they do add a little water, if it is sterilised no great harm is done; but when they dispense germs to the public they should be taught how to exclude them, and my experience amongst a large number of dairymen is that the majority would do their best to supply germ-free milk if they were properly taught. Where a tuberculous cow or bull is found in a dairy herd, the Government pay half the value of the animal to the owner and destroy it. This consideration, in a modified way, could be extended to those owners who possess cows affected with infectious mammitis, and some compensation allowed for drying them off.

To show how prevalent infectious mammitis of cow has become, a dairyman in South Canterbury tells me that one of his neighbours who started the milking season with 60 cows has 25 of them out of commission, suffering from this disease. I obtained two samples of milk from two cows known to be suffering from this lesion; both were supplied by this same friendly dairyman. Here is the report from the Bacteriological Depot of Christchurch Hospital (both were very bad cases):—

No. 1.—Naked eye: Milk of a brown colour. Microscopic: Profuse polymorph pus cells; profuse gram positive cocci in chains; gram negative baccili. Cultures: A mixed growth of streptococcus, staphylococcus aureus, and baccili coli.

No. 2.—Microscopic: Polymorph pus cells; gram positive cocci in chains. Cultures: A mixed growth of streptococcus and staphylococcus albus.

It will be noticed that whilst the streptococcus is constantly present in infectious mammitis, other germs are also frequently present, and would no doubt account for some of those puzzling cases of mixed infection which we occasionally are called upon to treat.

SUMMARY.

That infectious mammitis in cows is so very common that it is becoming a source of danger to the community.

That it is due chiefly to a streptococcal infection which is constantly present.

That other germs may also be present in this disease and thus give rise to mixed infections in children and others taking the milk.

That milk from a cow suffering from it may cause streptococcal infection in any of the organs of body or skin, or give rise to some special form of disease such as purpura, Henock's purpura, or even probably account for some outbreaks of scarlet fever.

That milk in the unsterilised state is a highly dangerous food and is no doubt responsible for a great many cases of illness amongst children.

When a cow's udder has become infected the first thing noticed by dairyman is that a little curdled milk first comes.

If dairyman milks an infected cow and then milks other cows, the disease soon spreads through the herd.

ADDENDA.

Since writing the above I have come across one case of infectious mammitis of cow with a very hard nipple and quarter, and when the sample of milk was submitted to bacteriologist only the staphylococcus aureus was found and no streptococci; but this seems to be an exception, as I have always previously found streptococci. But is it any wonder that people get an attack of boils at times?

URL:
www.nzma.org.nz/journal-articles/streptococcal-infections-and-infectious-mammitis-of-the-cow

Transparency in the year of COVID-19 means tracking and publishing performance in the whole health system: progress on the public reporting of acute coronary syndrome data in New Zealand

Andrew Kerr, Carl Shuker, Gerry Devlin

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In the last edition of the *Journal* on 21 August, this manuscript was incorrectly published as an editorial rather than as a viewpoint.

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