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Detecting the re-emergent COVID-19 pandemic after elimination: modelling study of combined primary care and hospital surveillance

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From gorse to ngahere: an emerging allegory for decolonising the New Zealand health system

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Access to intrauterine contraceptives in the Southern District Health Board catchment

Robina Stevens, Antoni Moore, Charlene Rapsey

Long acting reversible intrauterine contraceptive devices like the Mirena are safe, highly effective, reduce the burden of heavy periods and anaemia, and have been fully funded in New Zealand since October 2019. Nevertheless, it remains expensive for people to access these—in the Southern District Health Board (SDHB) catchment it costs an average of $115 for an insertion, and can be as much as $270, in addition to the cost of two or three GP appointments. Access is also logistically difficult, with one in three practices in the SDHB catchment not providing any intra-uterine contraceptives. Of those that do, around 90% unnecessarily require either two or three appointments, despite World Health Organization and RANZCOG (Royal Australian New Zealand College of Obstetricians and Gynaecologists) guidelines saying that with appropriate safety netting, these devices can be safely 'quick started' in just one visit. Most people live within 20km of a potential provider, but distance to providers remains an issue for some thinly populated rural areas.

Thyroid ultrasound and nodule malignancy risk: a "real world" assessment of ultrasound reporting and agreement of ultrasound-based malignancy risk estimates with cytology and histology findings

Cynthia F Benny, Mark J Bolland, Sonal Amin, Adeline Lo

Thyroid nodules are very common and an ultrasound scan of the thyroid is the usual first investigation to help determine whether a nodule might be a thyroid cancer. However, this study found that the reports issued about the scan don't provide sufficient details to allow clinicians to estimate the risk of cancer in a nodule. When radiologists re-assessed the scans and estimated the risk of cancer in a nodule, they often disagreed about both the characteristics of the nodule and the estimates of risk of cancer. Additionally, the agreement of their estimates with the findings from biopsies and surgical specimens was not strong. The findings raise questions about how the results of thyroid ultrasounds should be reported and how the information in those reports should be used in the management of thyroid nodules.

Detecting the re-emergent COVID-19 pandemic after elimination: modelling study of combined primary care and hospital surveillance

Nick Wilson, Markus Schwehm, Ayesha J Verrall, Matthew Parry, Michael G Baker, Martin Eichner

This model-based analysis suggests that a surveillance system with a very high level of routine testing is probably required to detect an emerging or re-emerging outbreak of the COVID-19 pandemic within five weeks of a border control failure in a nation that was assumed to be COVID-19-free.

Cataract surgery in New Zealand: access to surgery, surgical intervention rates and visual acuity

Corina Chilibeck, Jeremy J Mathan, Stephen GJ Ng, James McKelvie

Cataract surgery is one of the most commonly performed surgical procedures in the world, and is associated with improvements in vision, decreased risk of falls and improved quality of life. This study investigated the characteristics of all patients referred for publicly funded cataract surgery in New Zealand over a 4.5-year period and provides a nationwide overview of access to cataract surgery in New Zealand. Eligibility for publicly funded cataract surgery in New Zealand is assessed based on a weighted score, with thresholds varying depending on region. This study found a disparity in access to cataract surgery between regions, and New Zealand Māori and Pasifika ethnic groups had worse visual acuity, and typically severe visual impairment, compared with other ethnic groups at the time of prioritisation.

Trends in the diagnosis of high-grade cervical abnormalities in young women in the post-vaccination era

Avnish D Goyani, Carrie R Innes, Bryony J Simcock, Dianne Harker, Narena M Dudley, Lois Eva, Cecile Bergzoll, Helene MacNab, Peter H Sykes

This paper demonstrates the impact of HPV vaccination on the occurrence of cervical precancerous abnormalities in young women. Pleasingly, there has been a marked reduction in the abnormalities that are most likely to develop into cervical cancer. It is of some concern that this reduction in higher grade abnormalities is less evident for Māori women. Provided women continue to be screened, we can expect a reduction in cervical abnormalities that require treatment and a reduction in cervical cancer for vaccinated cohorts of women. As the National Cervical Screening Program no longer recommends screening for women under 25 years, and abnormalities remain in this population of women, it is imperative that women participate in the screening program from age 25 and that efforts are made to ensure access for Māori women.

Outcomes after ST-elevation myocardial infarction presentation to hospitals with or without a routine primary percutaneous coronary intervention service (ANZACS-QI 46)

Simon Lee, Rory Miller, Mildred Lee, Harvey White, Andrew J Kerr

ST-segment elevation myocardial infarctions are a type of heart attack which requires urgent treatment. In ideal situations, this is done with appropriate medications and timely access to a procedure called primary percutaneous intervention where a blockage in the blood vessels supplying the heart (coronary arteries) can be opened and further damage to the heart is reduced. We examined data for all ST-segment elevation myocardial infarctions in New Zealand patients aged 20–79 years and found that there is no difference in outcomes regardless of the capability of the admitting hospital to provide primary percutaneous intervention in a timely manner. As most hospitals without access to an all-hours primary percutaneous intervention service are situated in the rural regions of New Zealand, this finding demonstrates a step forward in achieving equitable health outcomes between the urban and rural regions.

Exploring Pasifika wellbeing: findings from a large cluster randomised controlled trial of a mobile health intervention programme

Ridvan Firestone, Soo Cheng, Sally Dalhousie, Emily Hughes, Tevita Funaki, Akarere Henry, Mereaumate Vano, Jacqui Grey, Jodie Schumacher, Andrew Jull, Robyn Whittaker, Lisa Te Morenga, Cliona Ni Mhurchu

Our study findings provide new insights on how Pasifika peoples' characteristics and behaviours relate to wellbeing. Our findings point to 'family and community' as being the most important wellbeing factor for Pasifika peoples.

From gorse to ngahere: an emerging allegory for decolonising the New Zealand health system

Heather Came, Isaac Warbrick, Tim McCreanor, Maria Baker

From Gorse to Ngahere is used to deliver a message about the transformative change needed in the health system to make a difference for Māori in New Zealand. Gorse represents the insistent racism and failures in the current health system and the aspiration for a Ngahere that nurtures a holistic health system that is thriving, well, with better control and autonomy by Māori as the sovereign people. To actualise this shift is a call to action to immediately implement the recommendations from Wai 2575 Health Services and Outcomes Kaupapa Inquiry Report.

Nurse prescribing in New Zealand—the difference in levels of prescribing explained

Jane Key, Karen Hoare

This article discusses the three types of nurse prescriber currently registered in New Zealand (nurse practitioners, registered nurse prescribers (RNP) in primary health and specialty teams and registered nurse prescribers (RNPCH) in community health). It also provides an overview of the evolution of each group, as well as a summary of the current legislation, prescribing restrictions and models of supervision required for each type of prescriber.

The case for a bicultural dementia prevalence study in Aotearoa New Zealand

Sarah Cullum, Makarena Dudley, Ngaire Kerse

The prevalence of dementia in Aotearoa New Zealand is projected to triple by 2050 and the cost of caring for dementia is estimated to increase to $2.7 billion by 2030. Research evidence from memory clinics in New Zealand suggests that dementia may be different for Māori compared to NZ Europeans: presenting at an early age but taking a slower course which will have a financial impact on the families who may give up paid work to provide care. We have no accurate information about the epidemiology of dementia in New Zealand, or about differences for Māori, because there has never been a national dementia prevalence study. This viewpoint argues case for a bicultural dementia prevalence study in Aotearoa New Zealand, using culturally unbiased assessment tools that do not over diagnose dementia in Māori, ensuring adequate numbers of Māori are included and engaging whānau and communities in the process. A bicultural dementia prevalence study would provide the information we need to accurately assess current levels of need, evaluate potential inequities in allocation of resources, and to start to develop culturally appropriate services, which will also help to raise public awareness and reduce stigma.

Oh my

Peter Crampton

NZDep is a small area index of relative socioeconomic deprivation based on census data. Ethnicity graphs for NZDep illustrate how socioeconomic advantage and disadvantage are distributed throughout our society. The first time these graphs were produced followed the production of the first version of NZDep based on the 1991 census (Figure 1). They showed that Māori and Pacific people were severely socioeconomically disadvantaged compared to New Zealand European people (approximated in the figure as the prioritised ethnic group 'Non-Maori non-Pacific non-Asian'). Almost 30 years later the 2018 version of the same graphs show that while there is a small reduction in Māori people living in the most socioeconomically deprived neighbourhoods, very little has changed in terms of the overall distribution of socioeconomic advantage and disadvantage.

In interpreting these graphs it is important to keep in mind the use of the prioritised ethnicity classification and the way it handles people with two or more self-identified ethnic affiliations (see footnote to Figure 1), and the changing composition of New Zealand's population (Table 1). Between 1991 and 2018 there were increases in the numbers, and proportions, of Māori, Pacific and Asian people in the population and, while there has been an increase in the number of New Zealand European people, their proportion of the total population has decreased (as measured using the prioritised classification). The shifts in the underlying population structure over this time amplify the consequences of the lack of change in the distribution of privilege and its inverse. In summary, the question has to be asked: has 30 years of 'progress' really amounted to so little change in the underlying structures of opportunity for Māori and Pacific communities? Yes, it would appear so. Hence the title of this editorial.

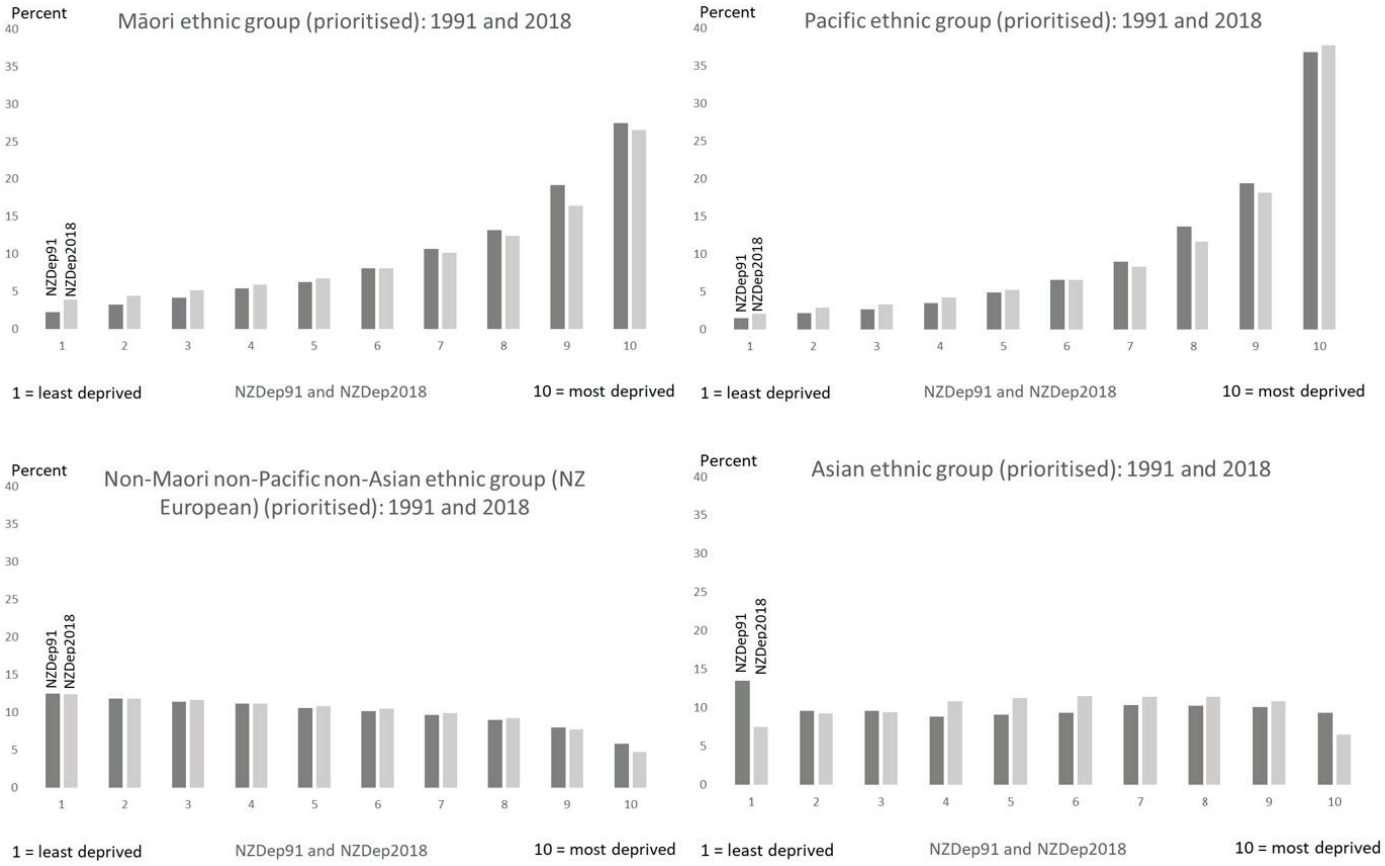
What do we learn from this observation? The boot that is held on the throat of Māori and Pacific people is stubbornly resistant to attempts to shift it. We have not transitioned from a colonial to a postcolonial nation—that transition remains a promise for the future. Our majoritarian political system suffers from something like prostatism and obstruction when it comes to honouring Te Tiriti o Waitangi, leading to dribbling, hesitancy and retention. Successive governments seem unable to lead us out of a state of oppressor/oppressed because of, in part, the dynamics of majoritarian democracy. Yet various components of New Zealand's unwritten constitution provide us with a map of the way ahead:[1] te Tiriti o Waitangi and the New Zealand Bill of Rights Act are two elements of this constitution that provide clear direction.

Disruptors are necessary to alter the fundamental structures of opportunity for Māori and Pacific communities; disruptors that are effective in embedding real change without doing damage to our society. What would these disruptors look like? Historical literacy is a starting point—if Pākehā are to take responsibility for removing the boot from the throat of Māori and Pacific people then Pākehā need to be educated in New Zealand's colonial and pre-colonial history. The moves currently underway to ensure that New Zealand history is taught compulsorily in primary and secondary schools are to be lauded and supported.

One example of a disruptor in the health system could be the increased population of our health system with Māori and Pacific health professionals who have the skills and expertise to drive change from within the system, while, at the same time, providing high-quality and compassionate care to all their patients. Some universities are leading the way in this regard; one role of the entire health sector should be to support Māori and Pacific health professionals and to demand an acceleration and ramping up of the production of these health professionals in the workforce.

Another example of a disruptor in the health system could be the recommen-

Figure 1: NZDep index of socioeconomic deprivation profile for prioritised* ethnic groups: Māori, Pacific, Asian and Non-Maori non-Pacific non-Asian (New Zealand European), 1991 and 2018.



*Prioritised ethnicity: each census respondent is assigned to a mutually exclusive ethnic group by means of a prioritisation system commonly used in New Zealand: Māori, if any of the responses to self-identified ethnicity was Māori; Pacific, if any one response was Pacific but not Māori; Asian, if any one response was Asian but not Māori/Pacific; the remainder non-Māori non-Pacific non-Asian (mostly New Zealanders of European descent, but, strictly speaking, not an ethnic group). The 'Asian' category, as used in the New Zealand health sector, includes respondents from East, South and Southeast Asia but excludes people from the Middle East and Central Asia. This category has acknowledged shortcomings because of the massive ethnic diversity within the category. Ethnic categorisation by 'total ethnicity' is now generally the preferred method, but this categorisation is not available for 1991 data.

Table 1: Population numbers and proportions by prioritised ethnic group (usual resident population), 1991 and 2018.

	1991		**2018**	
	Number	**%**	**Number**	**%**
Māori	434,844	12.9	767,733	16.3
Pacific	152,925	4.5	304,767	6.5
Asian	92,943	2.8	668,898	14.2
NonMāoriNonPacNonAsian (NZ European)	2,665,071	79.0	2,901,099	61.7
Unknown ethnicity	28,116	0.8	57,258	1.2
Total New Zealand population	3,373,899	100.0	4,699,755	100.0

dation from the majority of the Health and Disability Review panel and the Māori Expert Advisory Group to implement an empowered and properly resourced Māori health commissioning agency,[2] a recommendation also mooted by the Waitangi Tribunal.[3] This agency has the potential to bring the requisite expertise, commitment and drive to the task of commissioning services for Māori communities.

The *Black Lives Matter* movement tells us that brushing the legacy of white supremacy under the carpet gets us nowhere and simply foments distrust, social unrest and division. We know that any meaningful pathway to health and wellbeing incorporates agency as a key element; agency in relation to language, culture, worldviews, the environment, health, education and so on. Because of this understanding, the health sector has the capacity to provide leadership that could benefit all of society. The past 30 years have produced very little change in the structures of opportunity for Māori and Pacific people in relation to Pākehā people. If we wish to make the next 30 years count for more than the last 30, then Pākehā New Zealanders have an obligation to create and take opportunities to rid our society of racism and to demand equity in the structures, processes and outcomes for Māori and Pacific New Zealanders.

Competing interests:
Nil.
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REFERENCES:

1. Palmer G, Butler A. Towards democratic renewal: ideas for constitutional change in New Zealand. Wellington: Victoria University Press, 2018.
2. Health and Disability System Review. Health and Disability System Review – Final Report – Pūrongo Whakamutunga. Wellington: Health and Disability System Review, 2020.
3. Baker G, Baxter J, Crampton P. The primary healthcare claims to the Waitangi Tribunal. New Zealand Medical Journal. 2019; 132.

Access to intrauterine contraceptives in the Southern District Health Board catchment

Robina Stevens, Antoni Moore, Charlene Rapsey

ABSTRACT

AIM: Recent changes in funding have reduced the cost of the highly effective levonorgestrel-releasing intrauterine system (LIUS) contraceptives (Mirena and Jaydess). This paper explores equity of access to intrauterine contraceptives for Māori and the general population by locating and surveying all potential providers within the Southern District Health Board catchment area.

METHODS: Using online survey, e-mail or phone, we asked if intrauterine contraceptive insertion was provided, what device was provided, cost and number of appointments required. ArcGIS 10.6.1 software was used to estimate population distribution, and to create service areas showing distance to nearest current providers for Māori and the general population.

RESULTS: All 88 potential providers agreed to participate; two thirds (66.3%) provided some intrauterine contraceptive insertion. Approximately three quarters of the Māori and general population live within 5km of a primary provider. Costs ranged from $0 to $270, in addition to the cost of the required consultations. Number of consultations required varied from one to three.

CONCLUSIONS: Cost and travel time likely remain barriers to accessing intrauterine contraceptives for a significant population within this catchment. Increasing the capacity for all primary providers to offer insertion, funding the insertion process, minimising the number of appointments required and providing mobile services would improve access.

Ensuring access to preferred contraceptive methods is a vital part of any healthcare service. Effective family planning is essential to the wellbeing and autonomy of women, allowing them to choose to have their children when they are physically, psychologically and economically ready.[1,2] The development of highly effective, long-acting reversible contraceptives (LARC) has had a marked impact on family planning. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) recommends LARC as a first-line method for their excellent efficacy, acceptability, cost-effectiveness and reversibility.[3] In New Zealand, increased uptake of the copper intrauterine device (IUD), Mirena intrauterine system and Jadelle implant has been significantly associated with a declining abortion rate, particularly among younger women.[4] Nevertheless, a 2013–2015 survey found LARC were still less commonly used than condoms and the oral contraceptive pill.[5]

The decision by PHARMAC in October 2019 to widen access to levonorgestrel intrauterine systems (LIUS) by funding the Mirena and Jaydess without restriction followed sustained advocacy from many dedicated people.[6] Despite reduced cost to patients of these LIUS, barriers to uptake persist. Barriers include misperceptions about LARC from both patients and clinicians,[7] lack of training of healthcare providers, lack of funding for primary carers for contraceptive procedures, and cost to the patient.[8]

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IUD and LIUS differ from other contraceptives such as the oral contraceptive pill or condoms in two key respects. First, although in New Zealand there is no accredited training scheme for practitioners (unlike in other comparable countries such as the UK), and no system to ensure minimum standards of competency,[8] not all general practitioners (GP) have the facilities or willingness to insert these devices, so people may have to travel further than their closest GP to obtain this service. Second, insertion of these devices often requires two or three separate appointments in close succession: a pre-insertion consultation, the insertion and sometimes a follow-up appointment. This presents a potentially insurmountable logistical and financial challenge to women who lack independent transport, may already be caring for small children, struggle to arrange time off work, or live far from an appropriate provider. This challenge is compounded for people living in socioeconomic deprivation—disproportionately Māori and Pacific populations—for whom cost is already a recognised barrier to LARC access.[9]

The problem in New Zealand is twofold. For one, there is no single authoritative resource that lists and locates providers that can insert IUD or LIUS. This makes it impossible to know how far patients are having to travel to access these effective methods of contraception. Second, as only the device and not the insertion is funded, the costs to patients are unknown.

This descriptive study therefore sought to identify, survey and locate all potential providers of IUD or LIUS within the Southern District Health Board (SDHB) catchment area, and apply spatial analysis using a geographical information system (GIS) to explore to what extent cost and distance to nearest provider may still affect accessibility to intrauterine contraceptive devices.

Methods

Data collection

Using the HealthLink EDI Account Directory for Otago, Southland, Timaru and Oamaru,[10] and on the advice of individuals working within the gynaecological and sexual health services, all the potential providers of IUD/LIUS within the SDHB catchment were identified.

Table 1: Potential providers of IUD/LIUS within the SDHB catchment.

Type of provider	Number identified
General practice	79
Community health clinic	1
Regional outpatient gynaecology clinics	2
Family Planning clinic	2
Youth health clinic	1
Sexual health clinics	3
TOTAL	**88**

Physical locations and e-mail addresses were found online for as many of these practices and clinics as possible. A short survey developed on the Qualtrics software was sent to these e-mail addresses with unique links for each practice. If respondents did not complete the survey within two weeks, a reminder was sent, after which the outstanding respondents were contacted by phone and/or e-mail to ask the survey questions directly. General practices with no e-mail address were asked the survey questions by phone. Information from providers other than general practices was obtained by e-mail correspondence. Data were collected during December 2019 and January 2020.

Survey questions are presented in Table 2.

Ethics approval for data collection was granted by the University of Otago Human Ethics Committee (reference D19/381).

Data preparation

Prior to spatial analysis, population, provider and road network data were collated, collected and prepared. Within the SDHB catchment area, Māori and general electoral population data—hereafter referred to as the Māori and general populations respectively—were obtained from Statistics NZ at the meshblock level,[11] the smallest geographic unit for which data is reported by this organisation. Publicly available data on New Zealand deprivation level (NZDep2018) by meshblock were also obtained.[12]

Meshblocks each enclose a similar number of people, and therefore vary enormously

in size from small city blocks to large tracts of rural land. Using generalised population data at this level can greatly distort calculations of distance to a nearest provider. While populations can reasonably be assumed to be distributed throughout a central city meshblock, in rural areas they are likely clustered in only a small portion of the total meshblock area. This is a common artefact in situations where single values purport to represent an entire area (ecological fallacy).[13] Therefore, to better approximate the actual geographical distribution of population, the general and Māori populations in each meshblock were distributed evenly to each address point within that meshblock in a process known as dasymetric mapping.[14]

The obtained addresses of all potential providers were geolocated by matching them with attributes attached to address point data (source: Land Information New Zealand (LINZ)[15]) in a geocoding process. Where addresses could not be identified, the nearest neighbouring address point was chosen for spatial analytical purposes. Access to facilities by the population was calculated using a roads dataset acquired from LINZ,[16] which was converted using the GIS into a connected network capable of calculating specific distances from the providers.

Spatial analysis

Service areas covering parts of the road network that were within 5km, 20km and 50km of travel along the road network were calculated for each provider able to insert an IUD and/or LIUS. Using the refined estimate of spatial population distribution as described previously, the populations within each of these zones were calculated by summating the population attributed to the address points within that zone. Each point was also given the NZDep2018 score of the meshblock in which it fell,[12] thus the mean deprivation level of address points in each of these service area zones was also able to be calculated.

All spatial data preparation and spatial analysis stages were carried out using ArcGIS 10.6.1 software at the School of Surveying, University of Otago.

Results

Survey results

Of the 86 potential primary IUD/LIUS providers in the SDHB catchment, complete answers were obtained from 77 providers (89.5%). The remaining nine practices indicated that they did provide either IUD or LIUS insertion but were unwilling (one practice) or unable to provide further details (eight practices). Thus 86/86 (100%) of potential primary IUD/LIUS providers in the SDHB catchment provided enough information for the intended spatial analysis to be carried out. This excludes the two regional gynaecology outpatient clinics, which differ from the other providers in that they are not able to be directly accessed by patients but instead require a referral process (ie, they are secondary providers). Results of the survey are summarised in Table 2.

Network analysis

The spatial distribution of current primary providers of IUD/LIUS within the SDHB catchment is wide. This is reinforced by the service areas created, which show that much of the road network is within 50km of such providers. However, significant areas remain more than 50km away, symbolised in red in Figure 1.

In the hypothetical scenario where every primary practice in the SDHB catchment was able to provide IUD/LIUS, the network analysis showed improved coverage, particularly in the areas between Invercargill and Owaka, and to the west of Dunedin. Notably, the area northwest of Kurow remained isolated in this scenario.

Populations within service areas

71.3% of the general population and 79.4% of the Māori population were estimated to live within 5km of a current primary provider. This would increase to 75.2% and 85.9% respectively if every primary practice in the catchment was an IUD/LIUS provider. A further breakdown of these results is shown in Figure 2 and Table 3 below.

NZDep2018 scores for service areas

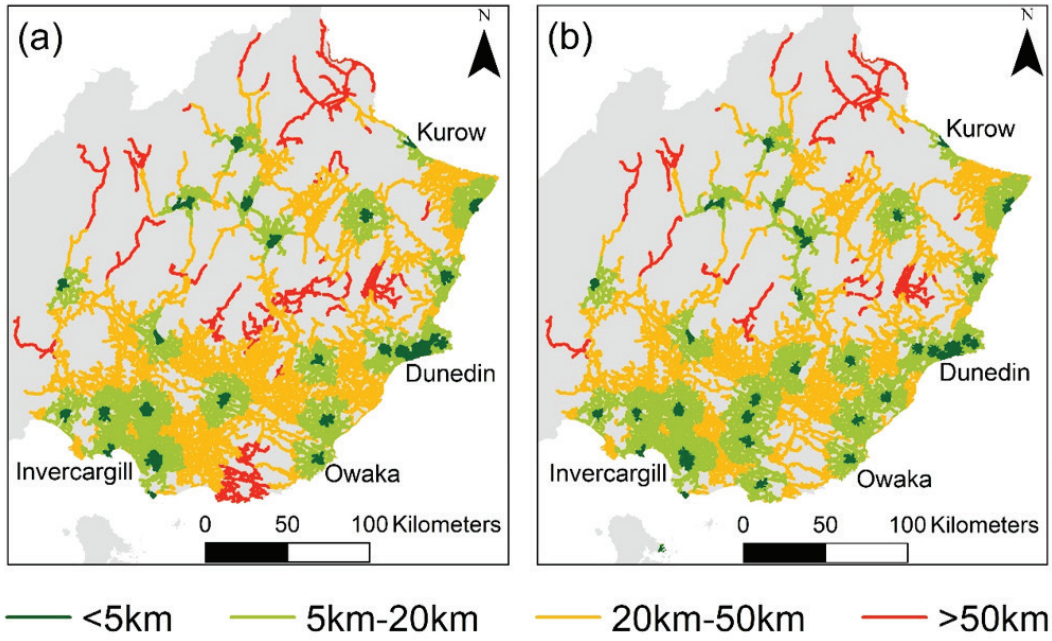
In neither the current nor the hypothetical case was there an observable trend in mean NZDep2018 scores of addresses within each of the service areas from closest to farthest.

Table 2: Survey results.

	% responses (n)
Q1: Does your practice provide intra-uterine contraceptive insertion?	
Yes	66.3 (57)
No	33.7 (29)
Q2: Which intra-uterine contraceptives can be inserted at your practice?*	
Mirena	95.8 (46)
Jaydess	77.1 (37)
Choice Load 375	56.3 (27)
Choice TT380 Short	77.1 (37)
Choice TT380 Standard	85.4 (41)
Multiload (volunteered by one practice)	2.1 (1)
Q3: Who does the insertion?*	
GP	95.8 (46)
Nurse	4.2 (2)
Other	10.4 (5)
Clinical nurse specialist	6.3 (3)
Currently training nurses to do insertions	8.3 (4)
Family planning doctor	4.2 (2)
Gynaecologist	4.2 (2)
Nurse practitioner	6.3 (3)
Specialist medical officer	6.3 (3)
Q4: Which of the following do you require patients to have?*	
Pre-insertion appointment	89.6 (43)
STI check	75.0 (36)
Post-insertion check-up consultation	45.8 (22)
Practices requiring one appointment	10.4 (5)
Practices requiring two appointments	43.8 (21)
Practices requiring three appointments	45.8 (22)
Q5: Does your practice charge an insertion cost in addition to the cost of the consultations required?	
No	20.0 (9)
Yes	80.0 (36)
Mean: $111.89	
Median: $115.00	
Maximum: $270.00	
Q6: Which of the following best describes who can access insertion at your practice / service?	
Only enrolled patients	46.9 (23)
Enrolled and those referred from other general practices	30.6 (15)
Only those from a limited geographical area	0.0 (0)
Anyone	22.4 (11)

*Multiple responses allowed.

Figure 1: Service areas of (a) current primary providers and (b) potential primary providers, if all primary services were able to provide IUD/LIUS. Address and road data from LINZ.[15,16] SDHB outline data from Statistics NZ.[17]



Discussion

Cost and travel time are likely still barriers

The results of the spatial analysis in this study must be considered highly optimistic in that they allocate patients to the nearest possible provider without any regard for what patients can afford or where they are enrolled, which is almost certainly not the case in reality. A further simplification is that no provision is made for the temporal accessibility of services. Many current primary providers have very limited hours, eg, the sexual health clinic in Gore operates one day a month, and many GPs doing insertions work part time.

Figure 2: Percentage of population within service areas of current primary providers.

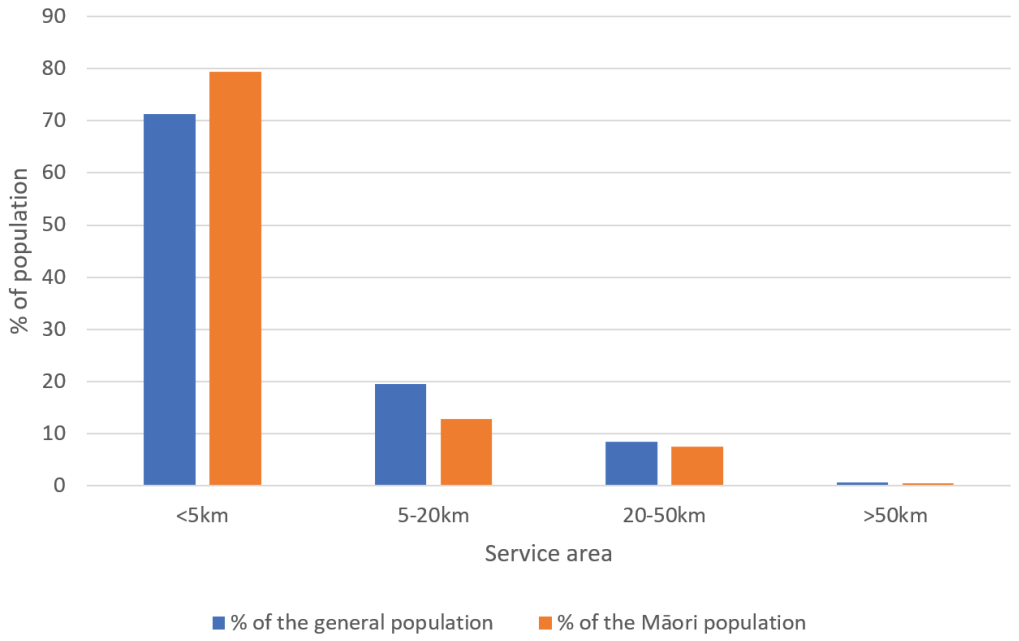


Table 3: Population within service areas of current primary providers (current) and if all primary services were able to provide IUD/LIUS (potential).

	No. general population, n (%)		No. Māori population, n (%)	
Service area	**Current**	**Potential**	**Current**	**Potential**
<5km	218,629 (71.3)	230,588 (75.2)	9,604 (79.4)	10,396 (85.9)
5–20km	60,100 (19.6)	59,487 (19.4)	1,556 (12.9)	1,295 (10.7)
20–50km	26,064 (8.5)	15,332 (5.0)	899 (7.4)	387 (3.2)
>50km	1,840 (0.6)	1,227 (0.4)	43 (0.4)	12 (0.1)

With these simplifications in mind, this study supports the finding in another recent qualitative study that cost is likely still a barrier.[8] Even with the changes in funding for the Mirena and Jaydess, the median insertion cost is $115 with the maximum more than twice this amount at $270, excluding the cost of two or three GP visits. These costs are comparable to, if not in excess of, the estimate of $150 from a recent study.[8] GPs in that study described such costs as forcing them to redirect patients, particularly those in rural areas, to cheaper services, often some distance away.[8]

For some rural populations, the distance to their nearest provider as estimated in this analysis contribute further to the cost. It is encouraging that a large majority of people live within 5km of a current provider (71.3% of the general population, 79.4% of the Māori population). Nonetheless, there remain a significant number of people who need to travel at least 5km to their nearest possible provider, with some needing to travel more than 50km. Using the Inland Revenue Department's 2019 mileage rate of $0.79/km, return journeys to three appointments would cost between $23.70 and $237.00 for patients 5km and 50km away respectively.

The number of providers requiring two (pre-insertion and insertion) and three (additional check-up) appointments were roughly equal. This division is interesting, given that both are rooted in the same current RANZCOG guidelines. On the subject of pre-insertion appointments/tests, the guidelines state that for women at high risk of having a sexually transmitted infection (STI):

"Ideally [STI screening] results should be available prior to IUC insertion. However, in asymptomatic women there is no need to wait for the screening results, nor provide antibiotic prophylaxis, providing the woman can be contacted and treated if a positive result is found."[3]

Regarding post-insertion follow up appointments, they state:

"A follow-up visit at 3–6 weeks may be undertaken to exclude infection, perforation or expulsion. More importantly, the patient should also be advised to present if abnormal bleeding, or symptoms suggestive of infection or pregnancy occur, or if they are unable to locate the string of the device."[3]

Thus, for low risk and asymptomatic high-risk women, there is no need to have STI screening results available prior to

Table 4: Mean NZDep2018 scores for service areas.

	Mean NZDep2018 score of addresses	
Service area	**Current**	**Potential**
<5km	5.6	4.2
5–20km	3.3	3.4
20–50km	5.2	5.0
>50km	4.8	4.6

insertion. Furthermore, it is more important to advise patients of symptoms that might require follow up than require them to attend a compulsory follow-up visit. It therefore appears that there is potential for the number of appointments required by some practices to be reduced, which would further reduce monetary and time costs to patients for whom these may be decision-making factors.

The finding of no simple correlation between distance to nearest provider and NZDep2018 should not be simplistically interpreted as distance not being a barrier for low-income patients. As noted previously, the aggregation of statistical data into meshblocks can introduce an ecological fallacy whereby small pockets of low-income populations are obscured by larger, wealthier areas. It would be more accurate to say that distance and travel time are not the only factors in determining how accessible an IUD or LIUS is for a given person living in a high-deprivation area, but these would certainly be contributing factors.

Increasing the number of providers

Operating under the assumption of people travelling to their nearest provider, the hypothetical scenario in which all general practices were able to insert IUD/LIUS would result in better coverage. The benefits would be relatively greater for the Māori population than the general population when calculating the number of people who would reside within 5km service area of a provider. Anecdotally, it would seem that increasing the number of healthcare professionals able to do insertions would be valuable; many practices spoken to were clear that they were stretched to the limit and were not enrolling new patients, while others who did not insert IUD or LIUS described months-long waiting lists at services to which they referred their patients. With 42 out of 48 providers having only GPs able to do insertions, nurse practitioners and clinical nurse specialists would seem to be a potentially underutilised resource. However, increasing the numbers of providers, and enabling them to perform enough procedures to maintain competence is not without its own challenges.[8] The problems of accessibility of IUD/LIUS for patients and of getting enough procedures for inserters to maintain competence are mutually dependent.

Mobile services for rural populations

SDHB is a challenging catchment to provide services for, given the large and unevenly populated area it encompasses. While populations are concentrated in urban centres, 24.2% of the general population is spread over very low-density meshblocks comprising 99.7% of the area of all inhabited meshblocks (Appendix Table 1). In this catchment, Māori are more urban than the general population, but the proportion of this population in very low-density meshblocks is still sizeable at 11.6% (Appendix Table 1). Thus, the population in low-density areas cannot be ignored in service planning.

It is not the intention of this descriptive study to offer solutions to the challenges of contraceptive provision in the SDHB catchment. Nonetheless, the geographical challenge of a thinly distributed population lends itself to the consideration of a mobile service as a cost-effective solution, and during the course of the study, it was found that Te Waka Wahine Hauora/The Woman's Health Bus has recently been established to provide precisely this. Further geographical analysis could optimise routes for such a service.

Limitations and strengths of the study

This study has several caveats. The electoral populations used are calculated estimates. Statistics NZ suppresses information on meshblocks with fewer than six inhabitants for privacy reasons. The dasymetric mapping used to distribute the populations of meshblocks to address points is a better estimate of reality than simply using meshblocks to estimate population distribution, but it remains a simplification nonetheless. As many rural address points are likely to be uninhabited tracts of land, it is likely that this technique overestimates the population in very remote areas. However, the use of broad service area bands ameliorates this to some extent—this study does not attempt to calculate precise distances to nearest provider for each address point, it merely groups address

points and their corresponding inhabitants into a distance zone. Edge effects may be impacting the analysis along the northern border of the SDHB catchment. As Canterbury providers are not modelled, some of the parts of the network identified as >50km from a current SDHB provider may in fact be within close range of a Canterbury provider.

As described, complete results were not obtained for all current primary providers. It is possible that there are outliers in terms of cost, but they are unlikely to greatly change the median cost estimate, and the findings from this study support those of others.[8] Also, this analysis assumes people travel to their nearest provider regardless of cost and enrolment, resulting in an over-optimistic estimate of access.

The fact that 100% of potential IUD/LIUS providers in the area provided sufficient information for the spatial analysis (ie, answered whether or not they provided IUD/LIUS insertion) is a particular strength of this study, and thus the spatial distribution of current primary providers can be considered an accurate snapshot at this period of time.

As this study only considers the SDHB catchment, it is unable to draw conclusions about the situation in other areas of the country. There is potential for this approach to be scaled up to a nation-wide level to address this.

Conclusion

While widening funding for the Mirena and Jaydess has been a welcome development, this paper indicates that there are populations in the SDHB catchment for whom distance and cost remain potential barriers to accessing an IUD/LIUS. A combination of increased funding for community insertion of IUD/LIUS, improved training opportunities for both medical and nursing staff, minimising the number of appointments required by providers, and optimised mobile services would improve access for people in this region to some of the most cost-effective and acceptable contraceptives available. With all the physical, social, economic and financial benefits that effective family planning confers to individuals and communities, this is arguably a public health priority.

Appendix

Appendix Table 1: Meshblocks (MB) grouped by general and Māori population density percentiles, with corresponding populations and land areas. Population data from Statistics NZ.[11]

	General population		Māori population	
MB population density band	**% population (n)**	**% inhabited MB area**	**% population (n)**	**% inhabited MB area**
<1st percentile	24.2 (74,211)	99.7	11.6 (1,398)	23.4
1st–10th percentile	49.6 (152,055)	0.23	36.0 (4,353)	57.9
>10th percentile	26.2 (80,367)	0.034	52.5 (6,351)	18.7

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REFERENCES:

1. Starbird E, Norton M, Marcus R. Investing in Family Planning: Key to Achieving the Sustainable Development Goals. Glob Health Sci Pract. 2016; 4:191–210.
2. Naik R and Smith R. 2015. Impacts of family planning on nutrition. Washington, DC: Futures Group, Health Policy Project.
3. RANZCOG. Intrauterine contraception. 2017. Retrieved 28/11/2019 from www.ranzcog.edu.au
4. Whitley C. Improved access to long-acting reversible contraception (LARC) and the declining abortion rate (Thesis, Master of Public Health). University of Otago. 2018. Retrieved 2/12/2019 from http://hdl.handle.net/10523/7935
5. Chesang J, Richardson A, Potter J, Coope P. Prevalence of contraceptive use in New Zealand women. N Z Med J. 2016; 129:58–67.
6. PHARMAC. Decision to widen access to levonorgestrel intrauterine (LIUS) systems (Mirena and Jaydess). 2019. Retrieved 4/2/2020 from http://www.pharmac.govt.nz/news/notification-2019-10-14-lius-mirena-jaydess/
7. Mazza D, Bateson D, Frearson M, et al. Current barriers and potential strategies to increase the use of long-acting reversible contraception (LARC) to reduce the rate of unintended pregnancies in Australia: An expert roundtable discussion. Aust N Z J Obstet Gynaecol. 2017; 57:206–212.
8. McGinn O, Fulcher HJ, Arroll B, McCowan L. Barriers to the prescription of LARCs in general practice in New Zealand – a qualitative research study. N Z Med J. 2019; 132:63–69.
9. Murray C, Roke C. Who can afford a Mirena® for contraception? J Prim Health Care. 2018; 10:201–206.
10. EDI Account Guide – Otago, Southland Timaru & Oamaru. 2019. Retrieved 27/11/2019 from http://nz.healthlink.net/knowledge-base/edi-guides/
11. Statistics NZ. Meshblock electoral populations 2020. 2019. Retrieved 10/1/2020 from http://datafinder.stats.govt.nz/layer/104209-meshblock-electoral-populations-2020/
12. Salmond C, Crampton P, Sutton F, Atkinson J. Socioeconomic Deprivation Indexes: NZDep and NZiDep, Department of Public Health. 2018. Retrieved 11/2/2020 from http://www.otago.ac.nz/wellington/departments/publichealth/research/hirp/otago020194.html#2018
13. Longley P, Goodchild M, Maguire D, Rhind D. Geographic Information Science and Systems (4th ed.). 2015; New York: Wiley.
14. Zandbergen PA. Dasymetric mapping using high resolution address point datasets. Transactions in GIS.15:5–27.
15. Land Information New Zealand. NZ Street Address. 2019. Retrieved 12/12/2019 from http://data.linz.govt.nz/layer/3353
16. Land information New Zealand. NZ Road Centrelines (Topo, 1:50k). 2020. Retrieved 7/2/2020 from http://data.linz.govt.nz/layer/50329-nz-road-centrelines-topo-150k/
17. Statistics NZ. District Health Board 2015. 2015. Retrieved 12/12/2019 from http://datafinder.stats.govt.nz/layer/87883-district-health-board-2015/

Thyroid ultrasound and nodule malignancy risk: a "real world" assessment of ultrasound reporting and agreement of ultrasound-based malignancy risk estimates with cytology and histology findings

Cynthia F Benny, Mark J Bolland, Sonal Amin, Adeline Lo

ABSTRACT

AIMS: Thyroid nodule malignancy risk is increasingly estimated using ultrasound characteristics. We assessed ultrasound reports of nodules and compared ultrasound-based malignancy risk assessments with cytology and histology findings.

METHODS: We identified patients with thyroid ultrasound (55% by private provider, 45% by DHB) and cytology at CMDHB over 18 months. Malignancy risk for each nodule was categorised based on the ultrasound report, then using ultrasound images with the local CMDHB approach and American Thyroid Association guidelines, and then was compared with cytology/histology results.

RESULTS: 36/91 nodules (84 patients) had abnormal (Bethesda 3–6) cytology. Forty-eight patients (54 nodules) underwent thyroid surgery and 13 nodules (12 patients) had thyroid cancers. Most ultrasound reports did not mention nodule malignancy risk characteristics (range 13–98%) or a malignancy risk estimate (66/91). 12/33 nodules with benign (Bethesda 2) cytology and 18/36 nodules with abnormal (Bethesda 3–6) cytology were considered intermediate/high risk of malignancy by ultrasound; none and seven, respectively, had cancer identified subsequently. In 18 nodules considered low risk by ultrasound, four cancers were identified.

CONCLUSIONS: Most ultrasound reports contained insufficient information about nodule malignancy risk to allow an independent assessment. Agreement between cytological/histological findings and malignancy risk estimates using ultrasound was not high.

Thyroid nodules are ubiquitous in modern medical practice, either discovered through palpation or as an incidental finding on radiological examinations. While thyroid nodules themselves are very common,[1] the rate of thyroid cancer in New Zealand is relatively low (356 registered cases in 2016, rate 6.4/100,000 people)[2] but has steadily increased in recent decades. For unknown reasons, rates are three times higher in Pacific women and two times higher in Maori women compared to European women.[3] Guidelines for investigation of thyroid nodules recommend thyroid ultrasound as the first radiological investigation in the presence of normal or low thyroid function tests.[4] While characteristics of a nodule on ultrasound can be used to predict the risk

of malignancy, studies, often from tertiary centres, suggest variable sensitivity and specificity for individual ultrasound characteristics (ranging from <0.5–0.93).[5–7]

At Counties Manukau District Health Board (CMDHB) in Auckland, New Zealand, over half of thyroid ultrasounds are performed in private community practices with the remainder performed in hospital radiology departments. We sought to assess the quality of the thyroid reporting from this diverse range of practices and radiologists, and to compare the estimates of malignancy risk from ultrasound reports with the results of cytological and histological assessments of the nodules. We also compared the results of malignancy risk assessment by sub-specialist radiologists with a radiology registrar and how those assessments compared to the original radiology reports.

Methods

We collated all patients who had a thyroid fine needle aspirate result at CMDHB over an 18-month period spanning 2012–2014, and had a diagnostic ultrasound available. There were no other specific inclusion or exclusion criteria. A total of 91 nodules from 84 patients were included.

Basic demographic details were obtained from the electronic medical record, and all original sonographic imaging was retrieved and viewed on the CMDHB Radiology Department PACS system. A senior radiology registrar and one of two radiology consultants with a Head and Neck sub-specialty interest viewed and interpreted the images blinded to patient details, except for the imprinted age and gender on the images. The sub-specialty radiologists used the current CMDHB thyroid nodule template and their own experience for assessment. The template assessed the maximum size of the nodule and the presence of extra-thyroid extension, micro-calcifications, solid composition, echogenicity, taller than wide dimensions, central vascularity and suspicious lymph nodes. The radiology registrar used the American Thyroid Association (ATA) 2015 guidelines,[4] which were the most up-to-date guidelines at the time. Based on the imaging features, each nodule was categorised as being either low, intermediate or high risk of malignancy. The original radiology reports were also reviewed by the radiology registrar and categorisation of malignancy risk was attempted using only the information and conclusions in the report.

We considered this an audit of current practices regarding thyroid nodule reporting as defined by the New Zealand National Ethics Advisory Committee guidelines,[8] and therefore that it was a low-risk project that did not require ethical approval.

We assessed the inter-rater agreement using Kappa values. Kappa >0.8 was considered almost perfect agreement, 0.61–0.8 to be substantial, 0.41–0.6 moderate, 0.21–0.4 fair and 0–0.2 slight agreement.[9] All analyses were performed using Stata IC 13 or Microsoft Excel 2013. For the comparison of nodule risk assessment and histology, we considered that all patients who did not have a histological sample did not have a thyroid cancer, since there was at least two years of follow-up data for each such patient with no clinical evidence of thyroid cancer.

Results

Demographics and laboratory results

Of the 84 patients with 91 nodules, 76 (90%) were female; the average age was 51 years (range 20–86 years); 40% were New Zealand European, 22% Maori and 16% Polynesian. 55% of the ultrasounds were done in private providers and 45% at CMDHB.

Table 1 shows the Bethesda cytology classification for the 91 nodules. Forty-eight of the 84 patients (54 nodules) had thyroid surgery

Table 1: Bethesda categorisation of the cytology of the 91 nodules.

Category	Number
Bethesda 1—inadequate	20
Bethesda 2—benign	33
Bethesda 3—atypia of uncertain significance	21
Bethesda 4—follicular neoplasm	8
Bethesda 5—suspicious for malignancy	4
Bethesda 6—malignant	3
Unable to be classified	2

Table 2: Thyroid nodule characteristics as described in original ultrasound report.

Characteristic	Feature present	Feature absent	Feature not mentioned
Solid	31 (34%)	39 (43%)	21 (23%)
Hypoechoic	15 (17%)	13 (14%)	63 (69%)
Micro-calcifications	16 (18%)	41 (45%)	34 (37%)
Central vascularity	38 (42%)	26 (29%)	27 (30%)
Irregular margins	8 (9%)	12 (13%)	71 (78%)
Taller than wide	0 (0%)	2 (2%)	89 (98%)
Extra-thyroid extension	0 (0%)	23 (25%)	68 (75%)
Associated cervical lymphadenopathy	3 (3%)	76 (84%)	12 (13%)

for which histology was available. Nineteen nodules from 17 patients had thyroid cancers, of which six cancers in five patients were incidentally found at surgery (ie, the thyroid cancer was not in the pre-operative nodule(s) of interest). One patient with two benign nodules also had parathyroid cancer which was not considered in the analyses. The remaining 33 nodules had benign histological findings.

Assessment of original ultrasound reports

Table 2 shows that the majority of reports did not report on nodule echogenicity (69%), margins (78%), taller-than-wide shape (98%) or extra-thyroid extension (75%), but generally mentioned nodule consistency (77%), vascularity (70%), the presence or absence of micro-calcifications (63%) or cervical lymphadenopathy (87%). Based on the information provided in the ultrasound report, a detailed independent assessment of malignancy risk using all the information recommended in the ATA guidelines was only possible for 1/91 nodules.

Sixty-six of the 91 reports had no definitive indication of the malignancy risk of the relevant nodule. Table 3 shows that in the 25 reports with a definitive risk assessment, 17 (68%) categorised the nodule as intermediate or high risk. Of these 17, 10 (59%) had abnormal cytology (Bethesda 3–6) on FNA. Of the eight nodules categorised as low risk, only one had benign cytology (Bethesda 2) but five had a non-diagnostic FNA (Bethesda 1), and two (25%) had abnormal cytology.

Table 3: Malignancy nodule risk assessment in original report versus cytology results.

	Risk of malignancy in original report			
	Low	Intermediate	High	Not stated
Original report	8	1	16	66
Cytology category				
Bethesda 1	5	0	2	
Bethesda 2	1	0	4	
Bethesda 3	1	1	6	
Bethesda 4	1	0	1	
Bethesda 5	0	0	2	
Bethesda 6	0	0	0	
Unable to be classified	0	0	1	

Table 4: Assessment of thyroid nodule characteristics by sub-specialty radiologist and radiology registrar.

	Feature present		Feature absent		Not able to be assessed		
Nodule characteristic	Sub spec	Reg	Sub spec	Reg	Sub spec	Reg	Inter-rater agreement (kappa)
Micro-calcifications	12	13	73	69	6	9	0.34
Solid	36	46	55	44	0	1	0.57
Hypoechoic	23	17	57	72	11	2	0.23
Taller than wide	3	6	88	83	0	2	-0.04
Central vascularity	20	59	70	32	1	0	0.21
Lymph nodes	3	8	87	82	1	1	0.43
Irregular margins		16		67		8	
Extra-thyroid extension	1	5	89	59	1	27	-0.002

Abbreviations: Sub spec—head and neck subspecialty radiologist, Reg—registrar.

Assessment of nodules by sub-specialty radiologist and radiology registrar

Table 4 shows the characteristics of the 91 nodules assessed. The majority of characteristics could be assessed for each nodule, although irregular margins were only a component of the ATA guidelines not the CMDHB template. There was insufficient information in the original radiology reports to enable a meaningful inter-rater analysis between those reports and the assessment by the sub-specialty radiologist or registrar. Inter-rater agreement between the sub-specialty radiologist and the registrar was fair to moderate.

Of the 91 nodules, 13 were categorised as high risk of malignancy, 25 as intermediate risk, and 53 low risk by the sub-specialty radiologist. The comparable numbers for the radiology registrar were 10 high, 33 intermediate and 48 low risk. The inter-rater Kappa value for malignancy risk assessment between the radiology registrar and sub-specialty radiologist was 0.22, indicating only fair agreement.

Table 5 shows the cytology and histology results compared with the malignancy risk assessments by the sub-specialty radiologist and registrar. Of the 33 nodules in 30 patients with benign (Bethesda 2) cytology, 12 and 15 respectively were categorised by the sub-speciality radiologist and registrar as high/intermediate malignancy risk. At surgery, one patient had two Bethesda 2 nodules and a Bethesda 5 nodule, and a thyroid cancer was diagnosed in the Bethesda 5 nodule. None of the other 17 patients (19 nodules) undergoing surgery had a cancer identified. Of the 36 nodules in 35 patients with abnormal cytology (Bethesda 3–6), 18 and 17 respectively were categorised as intermediate/high risk by the subspecialist and registrar. 26/35 patients (27 nodules) had surgery, and 11 nodules in 10 patients had a cancer identified. Of these 18 and 17 nodules respectively categorised as intermediate/high risk by the sub-specialists or the registrar, seven nodules in 11 patients (39%) and seven nodules in 11 patients (41%), respectively, undergoing surgery had a cancer identified. Of the 18 nodules with abnormal cytology but a low risk ultrasound assessment by the sub-speciality radiologist, 15 patients with 16 nodules had surgery and four had a thyroid cancer identified, and one patient had a parathyroid cancer. Of the 19 nodules with abnormal cytology but a low-risk ultrasound assessment by the registrar, 16 patients with 16 nodules had surgery and four had a thyroid cancer identified, one had parathyroid cancer and one patient had an incidental thyroid cancer.

Table 5: Cytology and histology results versus sub-specialist radiologist and registrar nodule malignancy risk assessment.

	Low risk malignancy by US assessment		Intermediate risk malignancy by US assessment		High risk malignancy by US assessment		Cancer in nodule histology
Cytology category	**Sub spec**	**Reg**	**Sub spec**	**Reg**	**Sub spec**	**Reg**	**(n/N)**
Bethesda 1	14	11	4	6	2	3	1/5
Bethesda 2	21	18	11	13	1	2	0/21
Bethesda 3	13	15	6	3	2	3	4/15
Bethesda 4	3	2	3	6	2	0	1/5
Bethesda 5	1	2	1	1	2	1	3/4
Bethesda 6	1	0	0	3	2	0	3/3
Unable to be classified	0	0	0	1	2	1	1/1
	Low risk by US assessment				**Intermediate or high risk by US assessment**		
Cancer	**Sub spec**	**Reg**			**Sub spec**	**Reg**	
Yes	5	4			8	9	
No	48	44			30	34	

Abbreviations: US—ultrasound, Sub spec—head and neck subspecialty radiologist, Reg—registrar.

Table 5 also shows the comparison of ultrasound-based risk assessment with final histological diagnosis in the 84 patients with 91 nodules, assuming that patients who did not undergo surgery did not have a thyroid cancer. The sensitivity and specificity for the sub-speciality radiologist intermediate/high risk assessment and presence of thyroid cancer in the nodule of interest (ie, not an incidental cancer) was 0.62 (0.32–0.86, 95% CI) and 0.62 (0.50–0.72, 95% CI) respectively, and for the radiology registrar was 0.69 (0.39–0.91, 95% CI) and 0.56 (0.45–0.68, 95% CI) respectively.

Discussion

In this mixture of community-based, private-practice and DHB thyroid ultrasound reports, the majority did not explicitly estimate the malignancy risk of an individual nodule, nor contain sufficient information to allow an independent malignancy risk assessment. However, when the original images were reviewed, the images were almost always sufficient to permit assessment of individual nodule characteristics and an overall malignancy risk. Agreement between the senior sub-speciality radiologists and the registrar for individual nodule characteristics nodules was variable, and for overall malignancy risk was fair. Of nodules with benign (Bethesda 2) cytology, 12/33 (36%) were categorised as intermediate/high risk of malignancy by the sub-speciality radiologist, but no cancer was identified in histology or during clinical follow-up. Of nodules with abnormal cytology (Bethesda 3–6), 18/36 (50%) were categorised as intermediate/high risk of malignancy by the sub-specialty radiologist, and seven of these 18 (39%) were subsequently found to have a thyroid cancer. The sensitivity and specificity of an intermediate/high risk categorisation by the sub-speciality radiologist for thyroid cancer was 62% and 62% respectively.

These findings have a number of clinical implications. From a clinical perspective, the majority of the original ultrasound reports were inadequate to allow an

assessment of the malignancy risk either by reading the report and its conclusions or by an independent assessment of nodule characteristics. Thus, the reports were often inadequate to guide management of thyroid nodules. It is likely that following the thyroid ultrasound, many patients were referred for a specialist review, which may have required a review of the thyroid ultrasound images by the specialist or in a radiology conference to allow an assessment of malignancy risk to be made. In such cases, the second radiology review is a waste of a limited resource, and creates unnecessary delay. Our findings of sub-optimal thyroid ultrasound reports are not unique, with others recently reporting similar results.[10,11] For example, Karkada and colleagues reported that almost half of nodules were not classified for malignancy risk, and 32–91% of reports did not mention key ultrasound characteristics.[10]

A second clinical issue arises when a patient has had a thyroid FNA with abnormal (Bethesda 3–6) cytology. The case may then be discussed at a multidisciplinary meeting with review of both the cytology and radiology findings. However, for the intermediate or high-risk malignancy estimate by a head and neck radiologist, the sensitivity and specificity for malignancy was only 62%, 8/38 (21%) had a cancer diagnosed, and 7/18 (39%) who also had abnormal cytology had a cancer identified. Furthermore, only half of nodules with abnormal cytology were considered intermediate or high risk of malignancy by ultrasound characteristics, but conversely, 22% of those with abnormal cytology who were considered at low risk by ultrasound characteristics had a cancer identified. Taken together, these results suggest that reviewing the thyroid ultrasound after a cytological assessment might not be helpful in increasing the likelihood of a thyroid cancer being diagnosed. This proposition could be tested formally in a clinical study.

Disagreement among radiologists in characterising thyroid nodules is common in clinical practice. However, some previous studies have reported strong agreement between radiologists when assessing individual nodule features.[12,13] The differences between the strong agreement in those studies and the much weaker agreement in the current study might be explained by the study designs: some studies gave specific training prior to the research;[12] and some compare assessments between highly experienced sub-specialists working at single, tertiary-level institutions with high volume throughput.[12,13] In contrast, our study was an audit of current practice and no specific training was provided, with reporting being undertaken by a mixture of radiologists in private and public practice. The senior radiology registrar with specific training and learning for this study might be considered similar in level to a general radiologist working between private and public institutions who may report only a few thyroid ultrasound studies for assessment of nodules each year. The comparisons between the subspecialist and the registrar are therefore clinically relevant.

Since we initiated our study, the American College of Radiologists has released a TI-RADs system for estimating malignancy risk based on ultrasound characteristics of thyroid nodules.[14] It is hoped that this system might overcome some of the problems we identified by creating standardised reports in which each relevant thyroid nodule is assigned a TI-RADS score, with explicit recommendations made based on those scores. However, a likely limiting factor in its widespread use and clinical application is the agreement between radiologists regarding the TI-RADS score. To date, we are not aware of validation studies of TI-RADs as used in a clinical practice similar to CMDHB. It is not clear whether validation studies in highly trained, highly experienced sub-specialists, who are very familiar with the use of TI-RADS and report large volumes of ultrasound reports would be replicated in, or are relevant to, clinical practices in which the majority of thyroid ultrasounds are reported by general radiologists who report only small volumes of thyroid ultrasound. At a practical level, if only a TI-RADs score is reported, without the information upon which the score is based, an independent assessment of malignancy risk will not be possible, which might require review of the ultrasound images, again creating delays and wasting resources. On the other hand, if all the information is reported for multiple nodules, a report can quickly become long and unwieldy.

Limitations

This study is a retrospective review and therefore there is potential for confounding. The assessment of the thyroid nodules was blinded, however we were unable to remove the imprinted information on the ultrasound pictures of age and gender, which may have influenced decisions. Furthermore, reviewing ultrasound static images retrospectively removes the opportunity for real-time evaluation and discussion with the original sonographer. Another issue is that the sub-specialists used their local template for assessing malignancy risk whereas the registrar used the ATA guidelines. This might have accounted for some differences in results, although the underlying nodule characteristics to be assessed do not differ between the systems, except that irregular margins was not included in the local template.

Conclusion

The majority of thyroid ultrasound reports were suboptimal because they did not explicitly estimate the malignancy risk of an individual nodule, or contain sufficient information to allow an independent assessment of malignancy risk. When assessing nodule characteristics, agreement between the sub-speciality radiologists and the registrar was variable for individual characteristics and low for the overall malignancy risk. The level of agreement between cytological and histological findings and the estimated malignancy risk based on ultrasound findings was not high. This raises the question of whether there is any value in reviewing ultrasound findings once cytology is known. This approach, which is commonly followed in our and other institutions, should be assessed formally.

Competing interests:
Nil.

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REFERENCES:

1. Dean DS, Gharib H. Epidemiology of thyroid nodules. Best Pract Res Clin Endocrinol Metab. 2008;22:901-11.
2. Ministry of Health. New cancer registrations. 2016: http://www.health.govt.nz/publication/new-cancer-registrations-2016 (accessed 9/4/20).
3. Meredith I, Sarfati D, Atkinson J, Blakely T. Thyroid cancer in Pacific women in New Zealand. N Z Med J. 2014; 127:52–62.
4. Haugen BR, Alexander EK, Bible KC, et al. 2015 American Thyroid Association Management Guidelines for Adult Patients with Thyroid Nodules and Differentiated Thyroid Cancer: The American Thyroid Association Guidelines Task Force on Thyroid Nodules and Differentiated Thyroid Cancer. Thyroid. 2016; 26:1–133.
5. Bastin S, Bolland MJ, Croxson MS. Role of ultra-

sound in the assessment of nodular thyroid disease. J Med Imaging Radiat Oncol. 2009; 53:177–87.

6. Ahn SS, Kim EK, Kang DR, et al. Biopsy of thyroid nodules: comparison of three sets of guidelines. AJR Am J Roentgenol. 2010; 194:31–7.
7. Brito JP, Gionfriddo MR, Al Nofal A, et al. The accuracy of thyroid nodule ultrasound to predict thyroid cancer: systematic review and meta-analysis. J Clin Endocrinol Metab. 2014; 99:1253–63.
8. National Ethics Advisory Committee. National Ethical Standards for Health and Disability Research and Quality Improvement. Wellington: : Ministry of Health; 2019
9. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics. 1977; 33:159–74.
10. Karkada M, Costa AF, Imran SA, et al. Incomplete Thyroid Ultrasound Reports for Patients With Thyroid Nodules: Implications Regarding Risk Assessment and Management. AJR Am J Roentgenol. 2018; 211:1348–53.
11. Symonds CJ, Seal P, Ghaznavi S, et al. Thyroid nodule ultrasound reports in routine clinical practice provide insufficient information to estimate risk of malignancy. Endocrine. 2018; 61:303–7.
12. Park SH, Kim SJ, Kim EK, et al. Interobserver agreement in assessing the sonographic and elastographic features of malignant thyroid nodules. AJR Am J Roentgenol. 2009; 193:W416–23.
13. Norlen O, Popadich A, Kruijff S, et al. Bethesda III thyroid nodules: the role of ultrasound in clinical decision making. Ann Surg Oncol. 2014; 21:3528–33.
14. Tessler FN, Middleton WD, Grant EG, et al. ACR Thyroid Imaging, Reporting and Data System (TI-RADS): White Paper of the ACR TI-RADS Committee. J Am Coll Radiol. 2017; 14:587–95.

Detecting the re-emergent COVID-19 pandemic after elimination: modelling study of combined primary care and hospital surveillance

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ABSTRACT

AIMS: We aimed to determine the effectiveness of surveillance using testing for SARS-CoV-2 to identify an outbreak arising from a single case of border control failure in a country that has eliminated community transmission of COVID-19: New Zealand.

METHODS: A stochastic version of the SEIR model CovidSIM v1.1 designed specifically for COVID-19 was utilised. It was seeded with New Zealand population data and relevant parameters sourced from the New Zealand and international literature.

RESULTS: For what we regard as the most plausible scenario with an effective reproduction number of 2.0, the results suggest that 95% of outbreaks from a single imported case would be detected in the period up to day 36 after introduction. At the time point of detection, there would be a median number of five infected cases in the community (95% range: 1–29). To achieve this level of detection, an ongoing programme of 5,580 tests per day (1,120 tests per million people per day) for the New Zealand population would be required. The vast majority of this testing (96%) would be of symptomatic cases in primary care settings and the rest in hospitals.

CONCLUSIONS: This model-based analysis suggests that a surveillance system with a very high level of routine testing is probably required to detect an emerging or re-emerging SARS-CoV-2 outbreak within five weeks of a border control failure in a nation that had previously eliminated COVID-19. Nevertheless, there are plausible strategies to enhance testing yield and cost-effectiveness and potential supplementary surveillance systems such as the testing of town/city sewerage systems for the pandemic virus.

One of the challenges with a new pandemic such as COVID-19 is how best to undertake surveillance. Good-quality surveillance is needed to maximise rapid disease control, eg, with case isolation and contact tracing to identify further cases and to quarantine contacts as shown by successful control in China.[1,2] This surveillance and control capacity is particularly critical for nations that decide to eliminate community transmission entirely as New Zealand aimed to[3] and has succeeded with (as per mid-July 2020 and using a definition from other modelling work on elimination for New Zealand).[4] Most Australian States and Territories had also eliminated community transmission of COVID-19 by mid-July 2020, the exceptions being Victoria and New South Wales. Elimination status is also relevant to the following groupings of island jurisdictions, as per WHO data on 15 July 2020:[5]

1. Those jurisdictions which have avoided any COVID-19 cases at the time of writing (eg, via border controls), but which are still at risk if border controls fail. These mainly include island jurisdictions in the Pacific Ocean (eg, American Samoa, Cook Islands, Federated States of Micronesia, Kiribati, Marshall Islands, Nauru, Niue, Palau, Samoa, Solomon Islands, Tokelau, Tonga, Tuvalu and Vanuatu).
2. Those jurisdictions which have only experienced sporadic cases and appear (as per mid-July 2020) to have successfully contained spread. These include some islands in the Pacific (eg, Fiji).
3. Those jurisdictions which have had larger outbreaks of COVID-19, but have instituted tight controls and have declining numbers of new cases or no new cases for many weeks (eg, Taiwan).

A recent Australian study[6] suggested that timely detection and management of community transmission of COVID-19 is feasible. This modelling study concluded that "testing for infection in primary care patients presenting with cough and fever is an efficient, effective and feasible strategy for the detection and elimination of transmission chains". For example, when testing 9,000 people per week (per million population), the authors estimated that no cases of COVID-19 would be missed in some circumstances. This type of surveillance could therefore be relevant to identifying emergent or re-emergent SARS-CoV-2, the pandemic virus causing COVID-19.

Given this background, we aimed to determine the effectiveness of surveillance using testing for the SARS-CoV-2 virus to identify an outbreak arising from a single case of border control failure in a nation that is free of community transmission: New Zealand as per mid-July 2020.

Methods

To run pandemic spread scenarios for New Zealand, we used a stochastic SEIR type model with key compartments for: susceptible [S], exposed [E], infected [I] and recovered/removed [R]. The model is a stochastic version of CovidSIM, which was developed specifically for COVID-19 by two of the authors (http://covidsim.eu; version 1.1). Work has been published from version 1.0 of the deterministic version of the model,[7,8] but in the Appendix we provide updated parameters and differential equations for version 1.1. The stochastic model was built in Pascal and 100,000 simulations were run for each set of parameter values.

The parameters were based on available publications and best estimates used in the published modelling work on COVID-19. Key components were: a single undetected infected case arriving in New Zealand via a border control failure, 80% of infected COVID-19 cases being symptomatic, 39.5% of cases seeking a medical consultation in primary care settings, and 4% of symptomatic cases being hospitalised (see Appendix Table 1 for further details). We assumed that the initial undetected case could be at any stage of infection—to cover both failures of managing quarantine at the border, but also failures around the management of non-quarantined workers such as air crew and ship crew. Scenarios considered different levels of transmission with the effective reproduction number (Re) of SARS-CoV-2 to be 1.5, 2.0, 2.5 and 3.0 (Appendix Table 1). Given some evidence for superspreading phenomena with this pandemic virus,[9–11] we also considered scenarios where just 10% of the cases generated 10 times the number of secondary cases as the other cases.

Other scenarios considered the impact of 75% of symptomatic people seeking a medical consultation (eg, as the result of a potential media campaign); and another considered a possible school outbreak (eg, a border control failure involving a teacher or student returning from overseas). The assumptions for the latter involved: Re = 2.0, only 5% of symptomatic cases seek medical consultation, and only 0.5% being hospitalised.

For the detection of COVID-19 cases, we assume testing of 95% of cases of symptomatic cases of respiratory illness seeking medical attention in primary care and of hospitalised cases of respiratory illness. For parameterising the size of these two groups, we used official statistics and results from the Flutracking surveillance system used in

New Zealand (Appendix Table 1). The sensitivity of the PCR diagnostic test (at 89%) was based on a meta-analysis (Appendix Table 1).

Results

For what we regard as the most plausible scenario with an Re of 2.0 (ie, where people are still practicing some modest level of reduced social contact and/or increased hygiene because of the pandemic in other countries), the results suggest that 50% of outbreaks from a single imported case would be detected in the period up to day 16 and 95% in the period up to day 36 (Table 1, Figure 1). At the time of detection (to day 36), there was an estimated median number of five infected cases in the community (95% range: 1–29). To achieve this level of detection, an ongoing programme of 5,580 tests per day would be required, (1,120 per million people per day) for the whole New Zealand population. The vast majority of this testing (96%) would, however, be in primary care settings and the rest in hospitals.

For all scenarios except for the school scenario, 95% of outbreaks were detected in less than six weeks after introduction. A higher value (71 days) was for the simulated school outbreak where medical consultations were assumed to be much less likely (due to symptoms in young people being typically milder). Increasing the extent by which symptomatic people seek medical consultations to the 75% level (up from that reported by Flutracking at 39.5%), would reduce the time to detection (eg, from 36 to 26 days for the Re = 2.0 scenario at the 95% probability level, Table 1).

When allowing for superspreading events, introductions less frequently lead to outbreaks (Table 1) and these outbreaks have a tendency to be detected earlier (Figure 1).

Discussion

This analysis indicates the challenges for a surveillance system designed to detect the re-emergence of SARS-CoV-2 transmission in a COVID-19-free nation with border controls. A very high level of testing of symptomatic people is typically required in primary care settings and hospitals to detect an outbreak within five weeks after a single border control failure (at least at the 95% level).

Figure 1: Probability of COVID-19 case detection after reintroduction of the infection (the different curves represent the results of 100,000 simulations each, using Re values from 1.5 to 3.0). Dotted lines refer to a scenario where 75% of symptomatic cases seek medical help. Dashed lines refer to scenarios which allow for superspreading events. The dashed-dotted line refers to an outbreak in a school (for further details on parameter settings, see Table 1 and text).

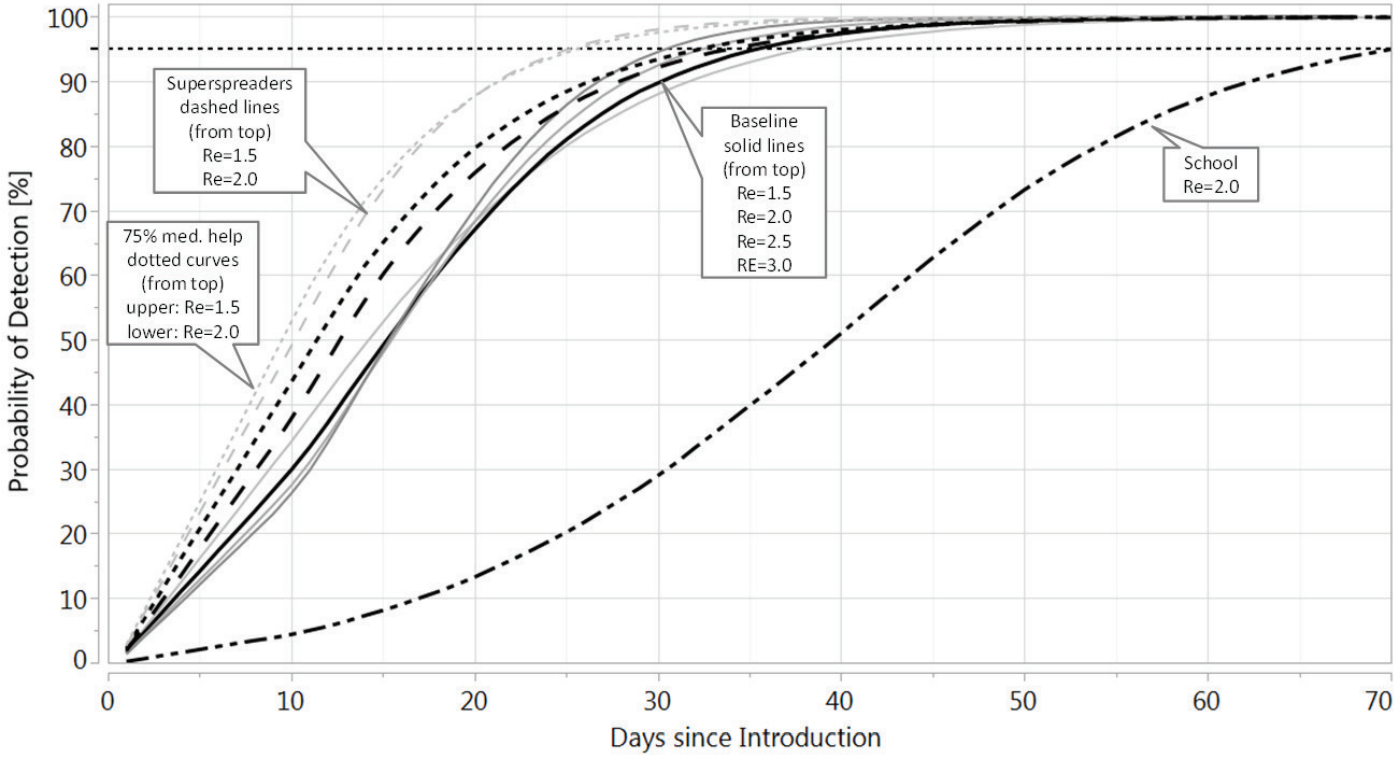

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Table 1: Modelled impacts by the time it takes to obtain at least one positive test result for SARS-CoV-2 infection arising from a border control failure where a single case enters the island nation of New Zealand (all results adjusted for lag times in reporting and obtaining test results, using 100,000 stochastic simulations for each parameter setting).

Scenario with variation in the effective reproduction number (Re)	Introduction leads to no further spread (%)	Day when 50% of outbreaks have been detected (median)	Day when 95% of outbreaks have been detected (median)	Mean day of outbreak detection	Expected no. of total tests*	Median (95% range) number of infections from introduction to detection**
Re = 1.5	45.3	15	38	16.8	93,700	4 (1–21)
Re = 2.0 (most plausible)	36.9	16	36	16.9	94,300	5 (1–29)
Re = 2.5	31.2	16	33	16.6	92,600	7 (1–38)
Re = 3.0	26.9	16	31	16.2	90,400	9 (1–49)
Re = 1.5, but 75% seek medical consultation***	36.1	10	26	11.4	116,000	2 (1–11)
Re = 2.0, but 75% seek medical consultation***	30.8	11	26	11.7	119,000	3 (1–15)
School outbreak#	44.7	40	71	40.4	225,000	44 (2–240)
Re = 1.5, but with superspreaders	56.8	12	33	13.8	77,000	3 (1–26)
Re = 2.0, but with superspreaders	50.3	16	34	15.0	84,000	4 (1–36)

*From the time of the border control failure to the mean day of outbreak detection. This is around 5,600 tests per day for primary care and hospital sectors combined for the first four listed scenarios.
**Includes those in the latent phase, prodromal phase, asymptomatic infections and symptomatic infections (but not recovered or deceased cases).
***These higher levels of consultation seeking result in a proportionate increase in the tests performed.
#The assumed characteristics for this school outbreak involved: Re = 2.0, only 5% of symptomatic cases seeking medical consultation, and only 0.5% being hospitalised. The level of testing was as per the first four listed scenarios.

This relatively ideal testing level, at 5,580 tests per day, is somewhat higher than the levels in New Zealand in early May 2020 (ie, the seven-day rolling average at this time was around 4,200 tests per day,[12] although this included some screening of asymptomatic people). It is even higher than the more recent 2,240 tests per day in early July 2020 (seven-day rolling average from 3 to 9 July). This lower level in July was even in the context of publicised "escapes" from border control facilities and it may drop further in the future with enhancements in quarantine facility security. Possibly there is a need for health authorities to regularly remind health professionals to keep offering testing since there is always some (albeit low) risk of quarantine failures, as some people may still excrete virus beyond the 14-day quarantine period.[13,14] Work could also be done to research any barriers for getting testing (eg, transport issues to primary care, waiting times and perceptions around cost barriers). Research could also explore why Australia has achieved a higher cumulative level of testing (112,000 tests vs 87,500 tests per million by 8 July 2020 [15]), although some of this will be due to the ongoing transmission of disease in states such as Victoria (as per July 2020).

Despite the high level of testing required for this type of surveillance system, there are potential ways that might improve the yield and cost-effectiveness of such testing:

- Prioritising community testing for those with relevant symptoms (as per Ministry of Health criteria updated in June 2020 [16]) in the cities where border control failures are most likely to become evident (ie, those operating international airports and where isolation/quarantine facilities exist: Auckland, Hamilton, Rotorua,

Wellington and Christchurch). Similarly, if cargo ship crews travelling from international ports are permitted shore leave in New Zealand in the future, then testing could be focused on these port cities.

- Pooling samples for PCR testing may preserve reagents and be more efficient[17] and cost-effective,[18] but needs to be balanced against potential loss of sensitivity and associated diagnostic delays.

If it became difficult to maintain high levels of testing even in these priority cities, an additional safeguard might be routinely offering testing to all hospital and emergency department attendees with any respiratory symptom (ie, not just those in the Ministry guidelines[16]). Another safeguard would be enhancements to the contact tracing systems used in New Zealand so that they can effectively address any outbreaks that arise.

Study strengths and limitations

This is the first such modelling analysis for a country that has achieved an elimination goal for COVID-19 with the end of all community transmission. Nevertheless, this work could have been refined further by a focus on a narrower range of acute respiratory diseases (eg, excluding the category of hospital admissions for chronic lower respiratory diseases (ICD10 codes: J40–J47). But since hospital admissions for these often involve an acute aspect, eg, acute bronchitis on top of chronic obstructive respiratory disease, we took the parsimonious approach of considering all respiratory diseases.

Another limitation is that we did not consider the relatively large seasonal fluctuations in the proportion of people consulting primary care for respiratory conditions (ie, with Flutracking data indicating a four-fold variation in cough/fever symptoms between May and October[19]).

This analysis also did not explore other surveillance options such as routine active surveillance of specific groups who might be considered at increased risk (eg, air-crew, ship-crew, port workers and quarantine facility workers). Similarly, not considered was the testing of town and city sewerage systems for the pandemic virus in wastewater, as is being explored in several jurisdictions internationally.[20,21] Indeed, in the New Zealand setting, the Crown Research Institute ESR has reported detecting SARS-CoV-2 in wastewater[22] and is continuing to develop this methodology. Such approaches could improve the speed of early detection in the community and allow for lower routine levels of testing people with respiratory symptoms.

Conclusions

In conclusion, this model-based analysis suggests that a surveillance system with a very high level of routine testing is probably required to detect an emerging or re-emerging SARS-CoV-2 outbreak within five weeks of a border control failure in a nation. But further work is required to improve on this type of analysis and to evaluate other potential surveillance system components, particularly the testing of wastewater in sewerage systems.

Appendix

Mathematical description of the CovidSIM model (version 1.1) and model parameters

The stochastic simulations are based on the following differential equations:

Model dynamics

Number of susceptible individuals

$$\frac{dS}{dt} = -\lambda S$$

Number of individuals in the latent period

$$\frac{dE_1}{dt} = \lambda S - \varepsilon E_1$$

$$\frac{dE_k}{dt} = \varepsilon E_{k-1} - \varepsilon E_k \qquad (1 < k \le n_E)$$

Number of individuals in the prodromal period

$$\frac{dP_1}{dt} = \varepsilon E_{n_E} - \varphi P_1$$

$$\frac{dP_k}{dt} = \varphi P_{k-1} - \varphi P_k \qquad (1 < k \le n_P)$$

Number of individuals in the early infectious period

$$\frac{dI_1}{dt} = \varphi P_{n_P} - \gamma I_1$$

$$\frac{dI_k}{dt} = \gamma I_{k-1} - \gamma I_k \qquad (1 < k \le n_I)$$

Number of individuals in the late infectious period

$$\frac{dL_1}{dt} = \gamma I_{n_I} - \delta L_1$$

$$\frac{dL_k}{dt} = \delta L_{k-1} - \delta L_k \qquad (1 < k \le n_L)$$

Derived variables

Total number in latent period $E_{Sum}(t) = \sum_{k=1}^{n_E} E_k(t)$

Total number in prodromal period $P_{Sum}(t) = \sum_{k=1}^{n_P} P_k(t)$

Total number in early infectious period $I_{Sum}(t) = \sum_{k=1}^{n_I} I_k(t)$

Total number in late infectious period $L_{Sum}(t) = \sum_{k=1}^{n_L} L_k(t)$

Contact rate and force of infection

Contact rate $\beta = \frac{R_e}{c_P D_P + D_I + c_L D_L}$

Force of infection $\lambda(t) = \frac{\beta}{N}(c_P P_{Sum}(t) + I_{Sum}(t) + c_L L_{Sum}(t))$

Stochastic treatment of the differential equations

The kind of epidemiologic events and the duration between two consecutive events are calculated using random numbers. The simulations start with a fully susceptible population in which one individual (index case) is infected. The infection stage of the index case is picked at random, taking into consideration the different lengths of the latent, prodromal, early and late infectious period. In each simulation, the sum of all the rates that change the current state of the system is calculated as

$$\xi = \lambda S + \varepsilon E_{Sum} + \varphi P_{Sum} + \gamma I_{Sum} + \delta L_{Sum}$$

A uniformly distributed random number $r_1 \in [0, \xi]$ is then chosen, and the time $\Delta t = -\ln(r_1)/\xi$ after which the next event occurs is calculated. All transition rates are arranged in an arbitrary order, and cumulative rates are calculated by adding their individual rates. A new uniformly distributed random number $r_2 \in [0, \xi]$ is chosen, and the first transition in the order whose cumulative rate is larger than r_2 is performed. If, for example, the event is an infection, one individual is removed from the group of susceptible individuals and added to the group of latent individuals of stage 1. New rates are calculated after each step, and the procedure is repeated. A more detailed description of the transformation of differential equation models to stochastic models can be found in Gillespie (1976).[23]

Parameters

N	Population size
λ	Force of infection
R_e	Effective reproduction number
β	Effective contact rate
D_E	Average duration of the latent period
n_E	Number of stages for the latent period
ε	Stage transition rate for the latent period $(\varepsilon = n_E / D_E)$
D_P	Average duration of the prodromal period
n_P	Number of stages for the prodromal period
φ	Stage transition rate for the prodromal period $(\varphi = n_P / D_P)$
c_P	Contagiousness in the prodromal period (relative to the contagiousness in the early infectious period)
D_I	Average duration of the early infectious period
n_I	Number of stages for the early infectious period
γ	Stage transition rate for the early infectious period $(\gamma = n_I / D_I)$
D_L	Average duration of the late infectious period
n_L	Number of stages for the late infectious period
δ	Stage transition rate for the late infectious period $(\delta = n_L / D_L)$
c_L	Contagiousness in the late infectious period (relative to the contagiousness in the early infectious period)

Appendix Table 1: Input parameters used for modelling the potential spread of the COVID-19 pandemic with the stochastic version of CovidSIM (v1.1) with New Zealand as a case study.

Parameter	Value/s used	Further details for parameter inputs into the modelling
Population size	4,951,500	We used the estimated New Zealand population as per December 2019 (ie, 4,951,500[24]).
Infections that lead to sickness	80%	We used the same proportion (80%) of symptomatic cases as per a Chinese study,[1] and as per an Australian modelling study.[6] This value is higher than the 57% value found in an Icelandic study[25] but this study did not involve long-term follow-up of the asymptomatic cases (ie, some of the asymptomatic cases might subsequently have developed symptoms). But it is also lower than that found in another Chinese study (at 94% symptomatic).[26]
Sick people who seek medical attention in primary care	39.5% (75% in a scenario analysis)	We used the result from the New Zealand Flutracking surveillance system for people with "fever and cough" in the weekly surveys who report seeking medical attention for these symptoms.[19] This is very similar to international estimates for people with influenza who seeking medical attention at 40%, eg, as used in other modelling.[7] In scenario analyses we raised this to 75% on the assumption that a media campaign could encourage attendance for relatively mild respiratory symptoms.
Sick people need hospitalisation	4%	At the time of writing on 3 May 2020, there were eight people hospitalised in New Zealand with COVID-19 (out of a total of 201 actively infected cases, ie, 4.0%[27]). Of note is that modellers in the UK have used 4.4% (of all infected cases),[28] and for modelling in the US 3%, 5% and 12% have been proposed.[29] The length of hospitalisation was assumed to be 10 days which is similar to other modelling work eg, 10.4 days for the UK.[28]
Effective reproduction number (Re)	2.0 as the most plausible value for New Zealand (1.5, 2.5 and 3.0 used in scenario analyses)	This estimate of 2.0 is in the lower end of the range for the basic reproduction number (R_0) reported on 6 March by the WHO (ie, 2.0–2.5[30]). This is because we assumed some level of ongoing physical distancing and enhanced hygiene practices in New Zealand relative to the pre-pandemic world. Of note is that an earlier review of 12 studies,[31] suggested estimates that ranged from 1.4–6.49, with a mean of 3.28, a median of 2.79 and interquartile range of 1.16. UK modelling work has used an estimate of 2.4 (range: 2.0–2.6).[28] Australian modelling studies have used R_0 values in the 2.2–2.7 range.[32] For the Re = 1.5 and 2.0 values we also considered scenarios with superspreading (as explained in the main text).
Relative contagiousness in the prodromal period	50%	There is uncertainty around this value but we used the same estimate as in recent UK modelling.[28] This has biological plausibility as while there is similarity in viral loads between asymptomatic and symptomatic COVID-19 patients,[33] it would be expected that those who are fully symptomatic (with a cough, etc) would be more likely to transmit infection. Of note is an estimate from the Diamond Princess cruise ship outbreak, that 17.9% of COVID-19 infections were from asymptomatic individuals (95% credible interval 15.5–20.2%).[34] But it is unclear how generalisable this finding is given the crowded cruise ship conditions and the typically elderly nature of the passengers.
Latency period	4 days	We used an average duration of four days as per Read et al,[35] with a standard deviation (SD) of 25% (one day) (calculated using 16 stages; Erlang distribution). This is similar to the estimate in a Chinese study which reported a median latent period of 3.69 days.[36]
Prodromal period	1 day	There is as-yet insufficient data on this for COVID-19, so we used an assumed value for influenza (SD = 25%; 0.25 days, Erlang distribution).
Symptomatic period	10 days (split into 2 periods of 5 days each)	The WHO-China Joint Mission report stated that "the median time from onset to clinical recovery for mild cases is approximately two weeks and is 3–6 weeks for patients with severe or critical disease".[2] But given that mild cases may have been missed in this particular assessment, we used a slightly shorter total time period of 10 days (SD = 25%; 2.5 days, Erlang distribution).

Appendix Table 1: Input parameters used for modelling the potential spread of the COVID-19 pandemic with the stochastic version of CovidSIM (v1.1) with New Zealand as a case study (continued).

Parameter	Value/s used	Further details for parameter inputs into the modelling
Contagiousness during the two symptomatic periods	100% and 50%	In the first five days of symptoms, cases were considered to be fully contagious. In the second five-day period, this was assumed to be at 50%. The latter figure is highly uncertain, but is broadly consistent with one study on changing viral load.[37]
Provision of testing and test sensitivity assumed		
Background hospital admissions for respiratory conditions in New Zealand	234 admissions per day	Using 85,439 respiratory disease admissions to New Zealand public hospitals in the year 2016/2017 (for all Chapter X ICD10 codes: J00 to J99).[38]
Background medical consultations in primary care for respiratory conditions in New Zealand	5,640 consultations per day	Data from the New Zealand arm of the Flutracking surveillance system was used. This indicates that approximately 3% of respondents in the period from April to October report "fever and cough" in the weekly surveys.[19] Of these 39.5% report seeking medical attention for their symptoms. However, we assumed a lower annual rate of 2% to account for the period outside of the influenza season (eg, Flutracking reporting is closer to 1% for weekly "fever and cough" at the start of May when the surveillance system begins for the year). In the New Zealand population of five million this would suggest 14,300 new cases developing "cough and fever" per day of whom 5,640 would be expected to seek medical attention.
Coverage in patients with respiratory symptoms who seek medical attention in primary care	95% coverage	These coverage values were further adjusted for the test sensitivity of 89% (see below). With 95% coverage and 89% test sensitivity, 84.55% of cases would be detected.
Coverage in hospitalised patients with respiratory symptoms	95% coverage	As above.
PCR test sensitivity	89%	A meta-analysis has reported this as 89% (95%CI: 81 to 94%).[39] This sensitivity is not ideal as while infection can be in the lungs, the sampling is from the nasopharynx, which may contain lower levels of virus at some stages of infection. Specificity is close to 100% for the PCR test.
Lag times (for health sector interaction and testing delays)		
Delay from symptom onset until a test has been performed and the result has become available	5 days plus 1 day for the testing delay	There is a delay between symptom onset and the performance of the test for SARS-CoV-2. For the first part of the delay we considered a study in Beijing, China, which reported the interval time from between illness onset and seeing a doctor was 4.5 days.[40] Another Chinese study of 710 patients with pneumonia[41] reported that those dying had a median duration from onset of symptoms to radiological confirmation of pneumonia of 5 (IQR: 3–7) days. For the testing delay we noted that the aim in New Zealand is to obtain the result of the tests in under 24-hours regardless of the primary care or hospital setting. But this may not always be the case for rural and small-town settings. In our simulations, test results were available on average 5.94 days after symptom onset (SD: 1.36 days).

Competing interests:
Dr Verrall reports this paper was written in Dr Verrall's capacity as Senior Lecturer at the University of Otago, not in her capacity as a candidate for Parliament. The views in this paper are not necessarily the views of the New Zealand Labour Party.

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REFERENCES:

1. Bi Q, Wu Y, Mei S, Ye C, Zou X, Zhang Z, Liu X, Wei L, Truelove SA, Zhang T, Gao W, Cheng C, Tang X, Wu X, Wu Y, Sun B, Huang S, Sun Y, Zhang J, Ma T, Lessler J, Feng T. Epidemiology and transmission of COVID-19 in 391 cases and 1286 of their close contacts in Shenzhen, China: a retrospective cohort study. Lancet Infect Dis 2020;(E-publication 27 April) S1473-3099(20)30287-5. doi: 10.1016/S1473-3099(20)30287-5.
2. WHO-China Joint Mission. Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19). 2020;(16-24 February). http://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf
3. Baker M, Kvalsvig A, Verrall A, Telfar-Barnard L, Wilson N. New Zealand's elimination strategy for the COVID-19 pandemic and what is required to make it work. N Z Med J 2020; 133(1512):10–14.
4. Wilson N, Parry M, Verrall A, Baker M, Schwehm M, Eichner M. When can elimination of SARS-CoV-2 infection be assumed? Simulation modelling in a case study island nation. medRxiv 2020;(20 May). http://medrxiv.org/cgi/content/short/2020.05.16.20104240v1
5. World Health Organization. Coronavirus disease (COVID-19) Situation Report – 177. 2020; (15 July). http://www.who.int/emergencies/diseases/novel-coronavirus-2019/situation-reports
6. Lokuge K, Banks E, Davies S, Roberts L, Street T, O'Donovan D, Caleo G, Glass K. Exit strategies: optimising feasible surveillance for detection, elimination and ongoing prevention of COVID-19 community transmission. medRxiv 2020;(23 April). doi: http://doi.org/10.1101/2020.04.19.20071217
7. Wilson N, Telfar Barnard L, Kvalsig A, Verrall A, Baker M, Schwehm M. Modelling the potential health impact of the COVID-19 pandemic on a hypothetical European country. medRxiv 2020; (23 March). http://medrxiv.org/cgi/content/short/2020.03.20.20039776v1
8. Wilson N, Telfar Barnard L, Kvalsvig A, Baker M. Potential health impacts from the COVID-19 pandemic for New Zealand if eradication fails: Report to the NZ Ministry of Health. Wellington: University of Otago Wellington. 2020; http://www.health.govt.nz/system/files/documents/publications/report_for_moh_-_covid-19_pandemic_nz_final.pdf
9. Endo A, Abbott S, Kucharski AJ, Funk S, Centre for the

Mathematical Modelling of Infectious Diseases COVID-19 Working Group. Estimating the overdispersion in COVID-19 transmission using outbreak sizes outside China. Wellcome Open Res 2020, 5:67 (http://doi.org/10.12688/wellcomeopenres.15842.1).

10. Miller D, Martin M, Harel N, Kustin T, Tirosh O, Meir M, Sorek N, Gefen-Halevi S, Amit S, Vorontsov O, Wolf D, Peretz A, Shemer-Avni Y, Roif-Kaminsky D, Kopelman N, Huppert A, Koelle K, Stern A. Full genome viral sequences inform patterns of SARS-CoV-2 spread into and within Israel. medRxiv 2020; (22 May). http://doi.org/10.1101/2020.05.21.20104521
11. Hamner L, Dubbel P, Capron I, Ross A, Jordan A, Lee J, Lynn J, Ball A, Narwal S, Russell S, Patrick D, Leibrand H. High SARS-CoV-2 attack rate following exposure at a choir practice - Skagit County, Washington, March 2020. MMWR 2020; 69:606–10.
12. Ministry of Health. Lab testing and capacity. New Zealand Ministry of Health. [Updated 3 May 2020]. http://www.health.govt.nz/our-work/diseases-and-conditions/covid-19-novel-coronavirus/covid-19-current-situation/covid-19-current-cases#lab
13. Lauer SA, Grantz KH, Bi Q, Jones FK, Zheng Q, Meredith HR, Azman AS, Reich NG, Lessler J. The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: Estimation and application. Ann Intern Med 2020; (E-publication 10 March).
14. Wilson N, Baker M, Eichner M. Estimating the impact of control measures to prevent outbreaks of COVID-19 associated with air travel into a COVID-19-free country: A simulation modelling study. medRxiv 2020; (17 June). http://www.medrxiv.org/content/10.1101/2020.06.10.20127977v3
15. Ritchie H, Ortiz-Ospina E, Hasel J, Beltekian D, Mathieu E, Hasell J, Macdonald B, Giattino C, Roser M. Coronavirus Pandemic (COVID-19) – the data (Assessed 11 July 2020). http://ourworldindata.org/coronavirus-data?country=~AUS 2020.
16. Ministry of Health. Case definition of COVID-19 infection (updated 24 June 2020). http://www.health.govt.nz/our-work/diseases-and-conditions/covid-19-novel-coronavirus/covid-19-resources-health-professionals/case-definition-covid-19-infection
17. Mallapaty S. The mathematical strategy that could transform coronavirus testing. Nature 2020.
18. Hogan CA, Sahoo MK, Pinsky BA. Sample pooling as a strategy to detect community transmission of SARS-CoV-2. JAMA 2020; (E-publication 7 April).
19. Flutracking. NZ participant annual report 2018. http://info.flutracking.net/wp-content/uploads/2020/02/NZ-participant-annual-report-2018.pdf
20. Mallapaty S. How sewage could reveal true scale of coronavirus outbreak. Nature 2020;(3 April). http://www.nature.com/articles/d41586-020-00973-x
21. Lesté-Lasserre C. Coronavirus found in Paris sewage points to early warning system. Science 2020; (21 April) http://www.sciencemag.org/news/2020/04/coronavirus-found-paris-sewage-points-early-warning-system
22. ESR. Update on ESR's wastewater testing for the COVID-19 virus. (Media Release 13 May 2020). http://www.esr.cri.nz/home/about-esr/media-releases/update-on-esrs-wastewater-testing-for-the-covid-19-virus/
23. Gillespie D. A general method for numerically simulating the stochastic time evolution of coupled chemical reactions. J Comput Phys 1976; 22:403–34.
24. Statistics New Zealand. Population (December 2019). Statistics New Zealand. http://www.stats.govt.nz/topics/population http://www.stats.govt.nz/topics/population
25. Gudbjartsson DF, Helgason A, Jonsson H, Magnusson OT, Melsted P, Norddahl GL, Saemundsdottir J, Sigurdsson A, Sulem P, Agustsdottir AB, Eiriksdottir B, Fridriksdottir R, Gardarsdottir EE, Georgsson G, Gretarsdottir OS, Gudmundsson KR, Gunnarsdottir TR, Gylfason A, Holm H, Jensson BO, Jonasdottir A, Jonsson F, Josefsdottir KS, Kristjansson T, Magnusdottir DN, le Roux L, Sigmundsdottir G, Sveinbjornsson G, Sveinsdottir KE, Sveinsdottir M, Thorarensen EA, Thorbjornsson B, Love A, Masson G, Jonsdottir I, Moller AD, Gudnason T, Kristinsson KG, Thorsteinsdottir U, Stefansson K. Spread of SARS-CoV-2 in the Icelandic Population. N Engl J Med 2020.
26. Luo L, Dan L, Xin-long L, Xian-bo W, Qin-long J, Jia-zhen Z, al. e. Modes of contact and risk of transmission in COVID-19 among close contacts. medRxiv 2020;(24 March). http://www.medrxiv.org/content/10.1101/2020.03.24.20042606v1

27. Ministry of Health. COVID-19 - current cases. [Data as of 7 April, 2020]. http://www.health.govt.nz/our-work/diseases-and-conditions/covid-19-novel-coronavirus/covid-19-current-situation/covid-19-current-cases

28. Ferguson N, Laydon D, Nedjati-Gilani G, Imai N, Ainslie K, Baguelin M, Bhatia S, Boonyasiri A, Cucunubá Z, Cuomo-Dannenburg G, Dighe A, Dorigatti I, Fu H, Gaythorpe K, Green W, Hamlet A, Hinsley W, Okell L, van Elsland S, Thompson H, Verity R, Volz E, Wang H, Wang Y, Walker P, Winskill P, Whittaker C, Donnelly C, Riley S, Ghani A. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. Imperial College 2020; (16 March):1–20.

29. Fink S. Worst-case estimates for U.S. coronavirus deaths. New York Times 2020;(Updated 14 March). http://www.nytimes.com/2020/03/13/us/coronavirus-deaths-estimate.html

30. World Health Organization. Coronavirus disease 2019 (COVID-19) Situation Report – 46. 2020;(6 March). http://www.who.int/docs/default-source/coronaviruse/situation-reports/20200306-sitrep-46-covid-19.pdf?sfvrsn=96b04adf_4

31. Liu Y, Gayle AA, Wilder-Smith A, Rocklov J. The reproductive number of COVID-19 is higher compared to SARS coronavirus. J Travel Med 2020.

32. Group of Eight Australia. COVID-19 Roadmap to Recovery: A Report for the Nation. The Group of Eight Ltd. 2020. http://go8.edu.au/research/roadmap-to-recovery

33. Zou L, Ruan F, Huang M, Liang L, Huang H, Hong Z, Yu J, Kang M, Song Y, Xia J, Guo Q, Song T, He J, Yen HL, Peiris M, Wu J. SARS-CoV-2 Viral Load in Upper Respiratory Specimens of Infected Patients. N Engl J Med 2020.

34. Mizumoto K, Kagaya K, Zarebski A, Chowell G. Estimating the asymptomatic proportion of coronavirus disease 2019 (COVID-19) cases on board the Diamond Princess cruise ship, Yokohama, Japan, 2020. Euro Surveill 2020;25:pii=2000180. http://doi.org/10.2807/1560-7917

35. Read J, Bridgen J, Cummings D, Ho A, Jewell C. Novel coronavirus 2019-nCoV: early estimation of epidemiological parameters and epidemic predictions. MedRxiv 2020. doi: http://doi.org/10.1101/2020.01.23.20018549

36. Li R, Pei S, Chen B, Song Y, Zhang T, Yang W, Shaman J. Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV2). Science 2020.

37. Wolfel R, Corman VM, Guggemos W, Seilmaier M, Zange S, Muller MA, Niemeyer D, Jones TC, Vollmar P, Rothe C, Hoelscher M, Bleicker T, Brunink S, Schneider J, Ehmann R, Zwirglmaier K, Drosten C, Wendtner C. Virological assessment of hospitalized patients with COVID-2019. Nature 2020.

38. Ministry of Health. Publicly funded hospital discharges – 1 July 2016 to 30 June 2017. http://www.health.govt.nz/publication/publicly-funded-hospital-discharges-1-july-2016-30-june-2017

39. Kim H, Hong H, Yoon SH. Diagnostic Performance of CT and Reverse Transcriptase-Polymerase Chain Reaction for Coronavirus Disease 2019: A Meta-Analysis. Radiology 2020:201343.

40. Tian S, Hu N, Lou J, Chen K, Kang X, Xiang Z, Chen H, Wang D, Liu N, Liu D, Chen G, Zhang Y, Li D, Li J, Lian H, Niu S, Zhang L, Zhang J. Characteristics of COVID-19 infection in Beijing. J Infect 2020; 80:401–06.

41. Yang X, Yu Y, Xu J, Shu H, Xia J, Liu H, Wu Y, Zhang L, Yu Z, Fang M, Yu T, Wang Y, Pan S, Zou X, Yuan S, Shang Y. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. Lancet Respir Med 2020;Published Online (21 February). http://doi.org/10.1016/S2213-2600(20)30079-5

Cataract surgery in New Zealand: access to surgery, surgical intervention rates and visual acuity

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ABSTRACT

AIMS: To analyse the surgical intervention rate (SIR), best spectacle-corrected visual acuity (BSCVA) and disparities in access to public-funded cataract surgery in New Zealand. The New Zealand Ministry of Health uses the National Prioritisation Web Service (NPWS) to prioritise all patients for public-funded cataract surgery. BSCVA at prioritisation, ethnic, demographic and geographic disparities have not previously been assessed.

METHODS: A retrospective cohort study. Between November 2014 and March 2019, 61,095 prioritisation events for 44,403 unique patients were identified. Cataract prioritisation events extracted from the NPWS were merged with date of birth and ethnicity extracted from the National Health Index database. All data were de-identified prior to statistical analysis.

RESULTS: Mean age at prioritisation was 74.4 years, with female preponderance (56%). Overall ethnicity was 'European' in 69.8% and 'New Zealand Māori' in 9.6%. Mean Snellen BSCVA was 6/30-2 (prioritised eye), and 6/12-1 (binocular). Māori and Pasifika presented on average 10 years earlier than other ethnic groups with significantly worse BSCVA. Surgery was approved in 74.4% of prioritisation events with mean Snellen BSCVA of 6/38-2. Only 34.9% of New Zealand patients had Snellen BSCVA of 6/12 or better in the prioritised eye, compared to 58.4% in the European Union. Cataract SIR varied by region.

CONCLUSIONS: New Zealand's cataract SIR is lower than most Organisation for Economic Co-operation and Development countries and patients have significantly worse BSCVA at prioritisation. Access to cataract surgery in New Zealand varies according to region. Māori and Pasifika present younger with worse BSCVA, suggesting potential barriers in accessing timely referral and prioritisation.

Cataracts are the leading cause of vision impairment and blindness in the world.[1] In developed countries, cataract surgery is one of the most commonly performed elective surgical procedures.[2] Cataract surgery is associated with improvements in visual acuity, decreased risk of falls and improved quality of life.[3–5] These benefits, coupled with an ageing population at high risk of cataract-related visual impairment, have increased the demand for cataract surgery worldwide.

With improvements in surgical capacity, recovery time, decreased complication rates and improved visual outcomes, the surgical intervention rate (SIR) for cataract surgery in most of the Organisation for Economic Co-operation and Development (OECD) countries has increased dramatically over the past two decades.[2] Government spending on cataract surgery typically produces a large return on investment,[6] and the cost per quality-adjusted life year gained is one of the highest of any operation or medical intervention.[5,6] Despite the significant benefits of cataract surgery, healthcare resources are finite and prioritisation for surgery is an important strategy to ensure that those with the greatest need are prioritised highest for surgery.

Public-funded cataract surgery accounts for approximately half of all cataract surgery currently completed in New Zealand. Eligibility for public-funded cataract surgery in New Zealand is assessed based on a weighted combination of visual acuity, cataract morphology and patient-reported quality of life. These variables are combined to produce a Clinical Prioritisation and Assessment Criteria (CPAC) score that ranges from 0 to 100 points. District health boards (DHBs) each set their own regional CPAC threshold based on demand and available funding for cataract surgery. To be prioritised for publicly funded cataract surgery a patient needs to score above the CPAC threshold in their region. CPAC thresholds may change over time in response to a number of factors and are not typically published; however, a 2019 report documented that the nationwide CPAC thresholds ranged from 40 to 61 in 2018.[7] The New Zealand Ministry of Health provides access to the National Prioritisation Web Service (NPWS) to calculate the CPAC score, assess eligibility, and to identify those patients who will benefit most from surgery. The prioritisation system allows for clinical overrides to prioritise patients who have cataract that makes it difficult to monitor AMD, glaucoma or diabetic eye disease. For these patients at risk of permanent visual loss, prioritisation is assured and visual acuity and quality of life data is optional.

The lack of a single nationwide CPAC threshold for cataract surgery in New Zealand may introduce disparities in access to surgery. The demographic and ethnic composition of the New Zealand population varies from region to region. Certain demographic subgroups, including New Zealand Māori and Pasifika ethnicity, may experience barriers in accessing healthcare, and commonly endure worse health outcomes.[8,9] Until now, the impact of regional prioritisation thresholds on ethnic and demographic disparity in accessing public-funded cataract surgery has not been assessed.

This study aims to investigate the characteristics of all patients referred for public-funded cataract surgery in New Zealand and to provide a nationwide overview of access to cataract surgery in New Zealand.

Methods

This study conformed to the tenets of the Declaration of Helsinki and the National Ethics Advisory Committee guidelines.[10] Criteria for exemption from formal review by the New Zealand Health and Disability Ethics Committee was met.[11] This is a retrospective cohort study analysing all patients referred for cataract surgery in the New Zealand public healthcare system between November 2014 to March 2019.

National prioritisation data for cataract surgery were extracted from the New Zealand Ministry of Health NPWS database. Clinical variables included best spectacle corrected visual acuity (BSCVA) in the eye prioritised for surgery alone and with both eyes open, cataract morphology, clinician-estimated visual potential, patient-reported impact on life and DHB of domicile. Using the National Health Identifier (NHI) as a primary key, date of birth, gender and patient-reported ethnicity data were merged to all prioritisation events for analysis. Age at prioritisation was calculated as the difference in years between the date of birth and the date of prioritisation. All data were de-identified prior to analysis.

New Zealand national census data were used to normalise regional prioritisation events by gender, age, ethnicity, location and duration of data collection to enable comparison between regions.[12] OECD cataract surgery data were used to compare New Zealand prioritisation for cataract surgery rates with cataract surgery rates in other OECD countries.[2]

All patients prioritised for publicly funded cataract surgery in New Zealand using the Ministry of Health Web Service between November 2014 and March 2019 were included in this study. Where a clinician, on the same day, submitted more than one prioritisation event for the same patient with differing clinical variables (a same-day re-prioritisation event), the number of events submitted and patient ethnicity were analysed. Same-day re-prioritisation events were excluded from further analysis. Visual acuity was converted to LogMAR units for statistical analysis, which was completed using R statistical software (R Foundation for Statistical Computing, Vienna, Austria).[13]

Results

A total of 61,095 prioritisation events involving 44,403 unique patients, spanning 52 months from November 2014 to March 2019, were identified for analysis. Two thousand four hundred and twenty-eight prioritisation events were identified as duplicates or had incomplete data submitted (in the case of overrides), and were removed from analysis. Two patients did not have ethnicity coded and were excluded from analysis that included ethnicity-related endpoints.

The mean age at prioritisation was 74.5 years for females and 73.7 years for males, with a female preponderance (56.0%). Of all prioritisation events, the mean Snellen BSCVA was 6/30-2 for the prioritised eye, and 6/12-1 binocular. For females and males, BSCVA in the prioritised eye was 6/30-1 and 6/38+2 respectively, and binocular BSCVA was 6/12-2 and 6/12-1 respectively. BSCVA in both the prioritised eye and binocular was similar across the regions. Surgery was approved in 72.6% of all prioritisation events. After removing duplicates, clinical overrides and same-day re-prioritisation events, surgery was approved in 74.4% of prioritisation events with mean LogMAR BSCVA 0.84 (6/38-2 Snellen equivalent, prioritised eye). Of all prioritisation events approved for surgery, 34.9% had LogMAR BSCVA 0.3 (6/12 Snellen equivalent, prioritised eye) or better.

Self-reported ethnic origins were 'European' in 69.8% and 'New Zealand Māori' in 9.6% of all prioritisation events, with other ethnic minorities comprising the remainder. Ethnicity, mean age and visual acuity of all prioritisation events that were made once on a given day are presented in Table 1. Where patients had same-day re-prioritisation events, the initial visual acuity submitted was used for analysis. The ethnicity of all patients prioritised is not significantly different to the proportions seen in the wider population of New Zealand (Chi squared = 20, degrees of freedom = 16, P-value = 0.22). The visual acuity differed between ethnic groups (Kruskal Wallace p-value <0.001) with the worst mean LogMAR visual acuity noted for New Zealand Māori and Pasifika ethnic groups.

New Zealand Māori and Pasifika were also noted to have younger mean age at prioritisation than other ethnic groups. Age at prioritisation stratified by ethnicity is shown in Figure 1. Analysis of variance confirmed a statistically significant difference between different ethnic groups (P-value <0.001).

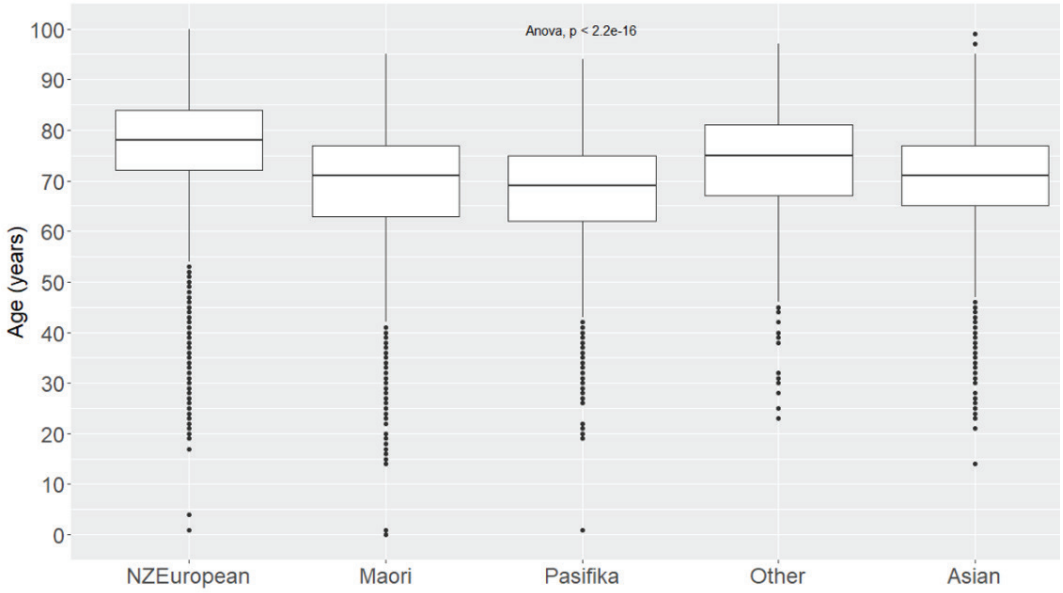
There were a total of 1,954 patients who were prioritised by the same clinician more than once on the same day, with different values submitted in each prioritisation event (Table 2). The ethnicity of patients with same-day re-prioritisation events is not significantly different to the proportions seen in the wider population (Chi squared = 20, degrees of freedom=16, P-value = 0.22)

Table 1: Ethnicity, mean age and visual acuity of prioritisation events compared with the New Zealand population.

Ethnicity	Number of patients† (percentage)	Percentage of NZ population	Mean age (years)	Mean BSCVA prioritised eye (Snellen)	Binocular mean BSCVA (Snellen)
European	39,467 (69.6%)	65.7%	77.0	6/30+1	6/12-1
Asian	5,735 (10.1%)	10.4%	70.3	6/38+2	6/12-2
NZ Māori	5,460 (9.6%)	13.3%	69.5	6/60+2	6/12
Pasifika	4,410 (7.8%)	6.6%	68.0	6/45	6/12-1
Other	1,641 (2.9%)	4.0%	73.2	6/30-2	6/12-1
Total	**56,713**		**74.4**	**6/30-2**	**6/12-1**

†There were a total of 44,403 unique patients. Some patients included in this summary were prioritised more than once on a different day, eg, for second eye surgery.
BSCVA = best spectacle corrected visual acuity.

Figure 1: Age at prioritisation stratified by ethnicity. New Zealand Māori and Pasifika were noted to have younger mean age at prioritisation than other ethnic groups. Analysis of variance demonstrates a statistically significant difference between the mean age at prioritisation between different ethnic groups (P-value <0.001).



Prioritisation events were compared by region after controlling for age, gender, ethnicity and catchment population size (Figure 2). This data includes prioritisation events both above and below eligibility threshold. Prioritisation events that were approved for public-funded cataract surgery were compared by region after controlling for age, gender, ethnicity and catchment population size (Figure 3).

The percentage of referrals declined by each DHB varied significantly (Figure 4). Three DHBs declined over 40% of referrals received. Based on the number of referrals made to each DHB, Lakes DHBs had the lowest approval rate of 51.4% and West Coast DHB had the highest approval rate of 93%.

Although the mean BSCVA of all patients that were prioritised for surgery were comparable across the regions, the

Table 2: All prioritisation events grouped by the number of same-day prioritisation events.

Number of same-day prioritisation events	Number of patients†	Total number of prioritisation events
1	56,713	56,713
2	1,617	3,234
3	248	744
4	61	244
5	18	90
6	3	18
7	4	28
8	3	24

†A number of the 44,403 unique patients will have also been scored on a different day, eg, for second eye surgery; these patients will therefore be represented more than once in this table.

Figure 2: Total number of prioritisation events, including those approved and declined for public-funded cataract surgery by district health board (DHB). Results are adjusted for age, gender, ethnicity and DHB catchment population size, with blue dots representing unadjusted rates. The nationwide mean overall number of prioritisation events (459/100,000/year) is indicated by the horizontal line.

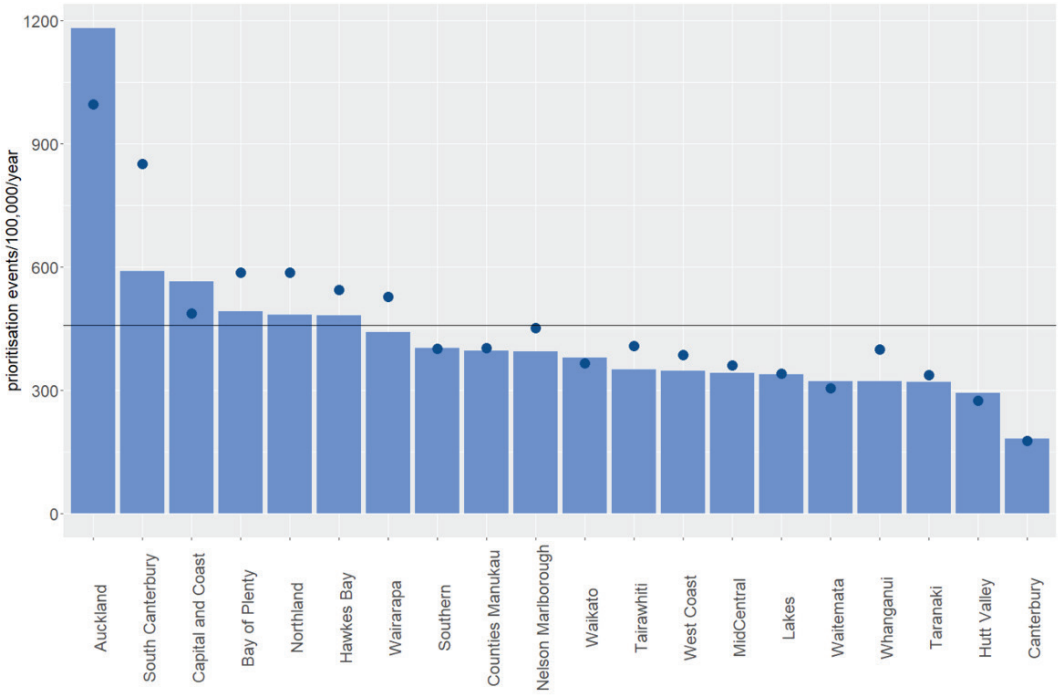


Figure 3: Number of approved prioritisation events per year by each district health board (DHB). Results are adjusted for age, gender, ethnicity and DHB catchment population size, with green dots representing unadjusted rates. The nationwide mean overall number of approved prioritisation events (340/100,000/year) for public-funded surgery is indicated by the horizontal line.

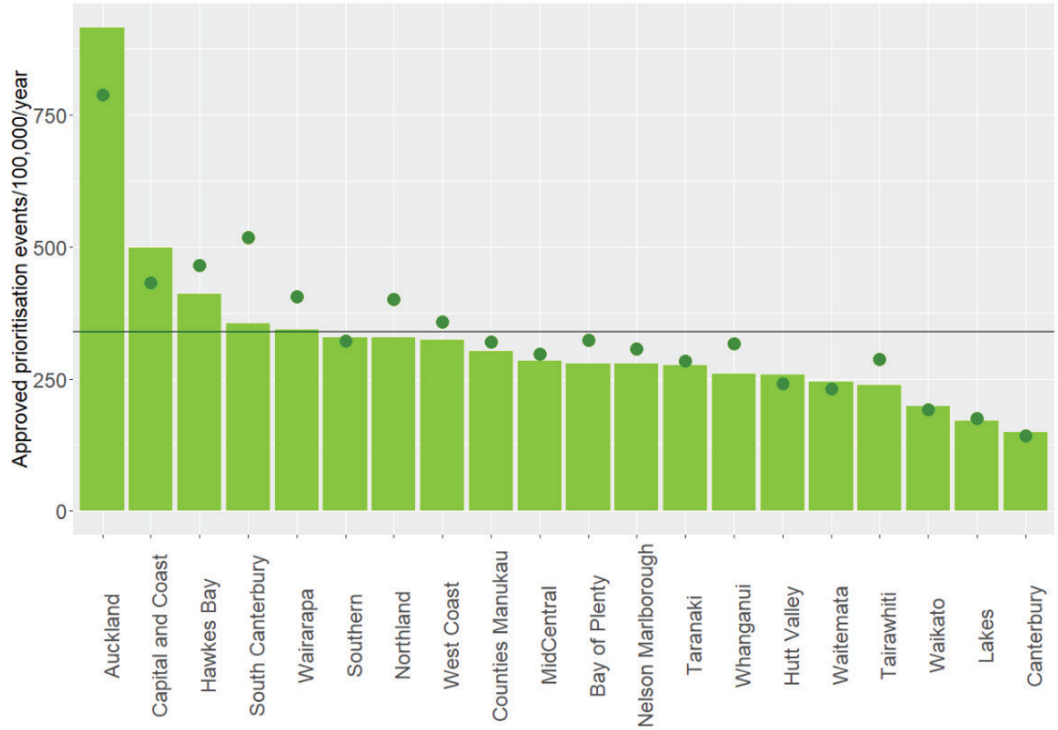


Figure 4: The percentage of prioritisation events declined by district health boards throughout New Zealand. The national percentage of declined prioritisation events (25.6%) is indicated by the horizontal line.

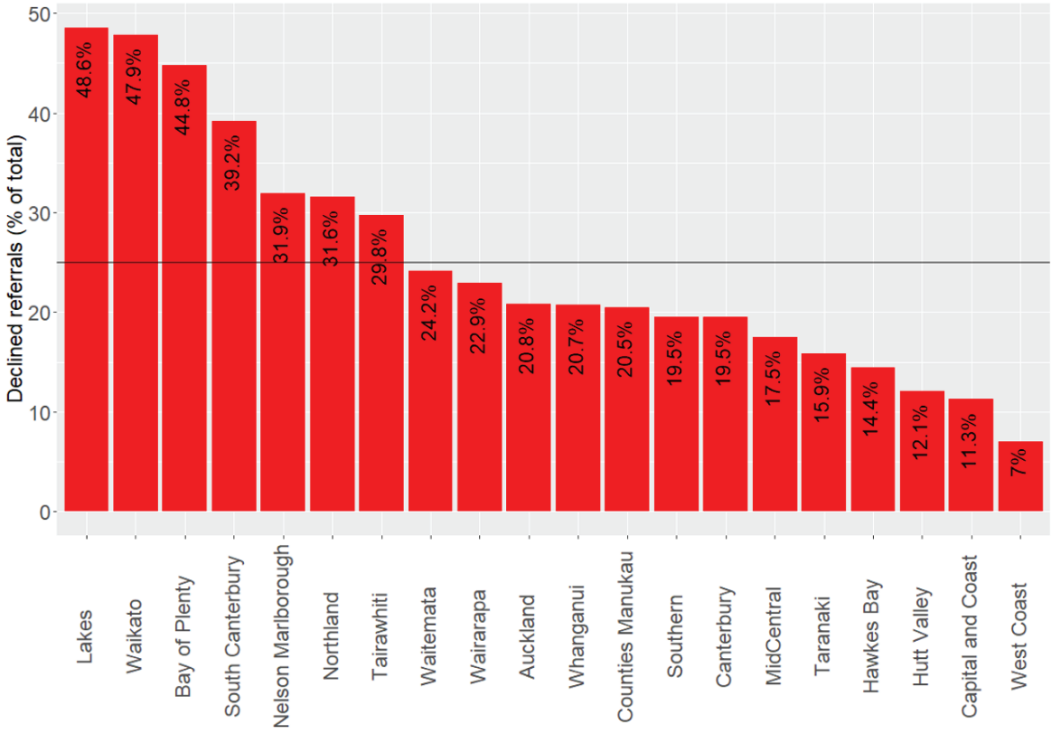
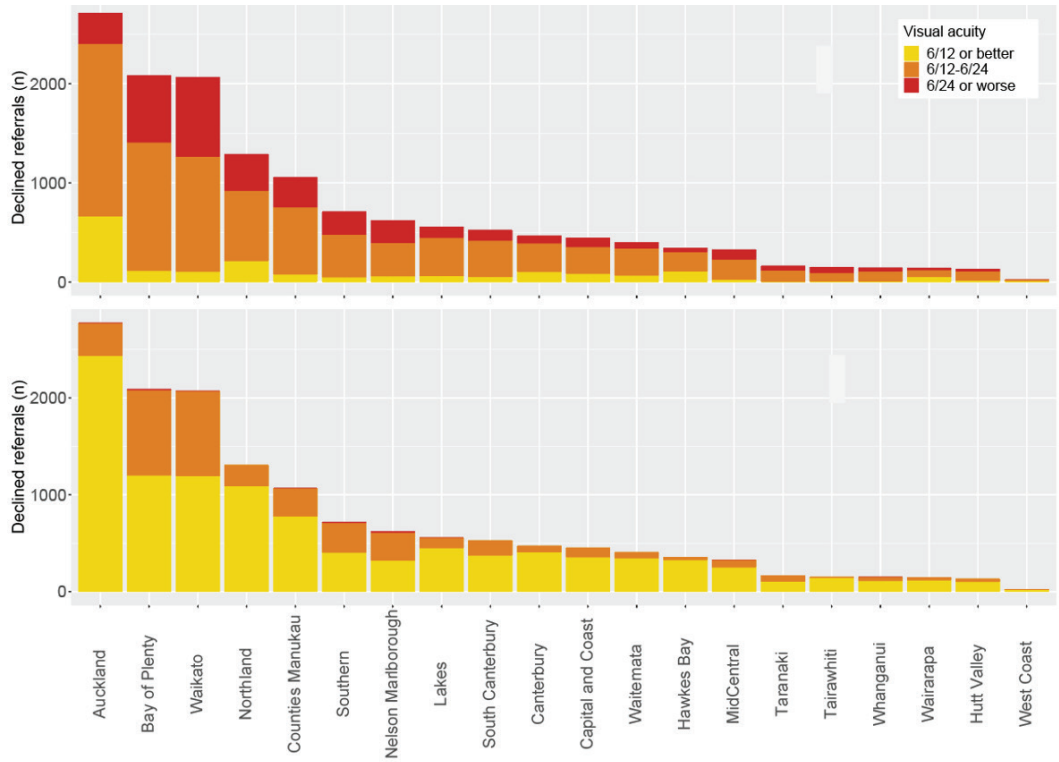


Figure 5: Number of declined referrals by district health boards. Bars are colour coded according to the proportion of patients in each visual acuity group in the prioritised eye (upper plot), or both eyes (lower plot). Red bars indicate patients with visual acuity worse than 6/24, orange bars indicate patients with visual acuity worse than 6/12, yellow bars indicate visual acuity better than 6/12.



binocular BSCVA of patients declined for cataract surgery varied significantly (range 6/6 to 6/36 Snellen, summarised in Figure 5). In total, 27.7% of all declined prioritisation events had binocular BSCVA 6/12 Snellen or worse, and 8.6% had BSCVA 6/15 Snellen or worse. The majority of all declined patients with a binocular BSCVA of 6/12 Snellen or worse were from the Waikato and Bay of Plenty DHB regions; however, per population, the highest rates were seen in Southern and Nelson-Marlborough DHBs. Overall, 0.3% of declined patients had binocular BSCVA worse than 6/24 Snellen from seven different DHBs.

The patient-reported quality of life score used for prioritisation was calculated from the Patient Impact on Life Questionnaire, which is assigned by the Ministry of Health to all elective surgical prioritisation schemes. This questionnaire is explained in detail elsewhere.[14]

Discussion

The current study assessed access to public-funded cataract surgery in New Zealand. Regional differences in mean patient characteristics and disparity in access to surgery between DHBs were identified. These findings highlight inequity in access to elective cataract surgery, a finding that is consistent with reports evaluating access to surgery in other specialties in New Zealand.[15,16]

The age and gender of patients prioritised for cataract surgery in New Zealand were similar to rates seen in other OECD countries.[17–19] The visual acuity of patients prioritised for surgery in New Zealand was significantly worse than vision at the time of surgery in other OECD countries.[19,20] The nationwide mean BSCVA of 6/30 (prioritised eye) at the time of prioritisation indicates that a significant level of visual impairment is required to access public-funded surgery in New Zealand. New Zealand Māori and Pasifika ethnic groups have worse visual acuity, and typically severe visual impairment, compared with other ethnic groups at the time of prioritisation. These ethnic disparities are consistent with reports from other specialities of the New Zealand health system and highlight the urgent need to improve access and the provision of culturally appropriate services for these ethnic groups.[21,22]

Despite the significantly worse visual acuity at prioritisation noted for New Zealand Māori and Pasifika, the proportion of prioritisation events for these ethnic groups was not significantly different to their proportion in the wider population of New Zealand. New Zealand Māori and Pasifika patients who were prioritised for cataract surgery, however, were on average 8–9 years younger than other ethnic groups and 6–7 years younger than the national mean. These results demonstrate that New Zealand Māori and Pasifika patients develop more advanced cataract associated with a greater degree of visual impairment at a younger age. New Zealand Māori and Pasifika patients face barriers to accessing timely referral for cataract surgery. As a result of severe visual impairment, these patients will, in many cases, have significantly decreased quality of life, increased risk of falls and decreased independence while waiting for treatment.[3,23,24]

The ethnic distribution of same-day re-prioritisation events did not differ significantly in comparison to the wider New Zealand population. Data for same-day re-prioritisation events did not include a reason for this activity. It is possible clinicians re-prioritised due to data-entry errors or in some cases clinicians may have intentionally re-scored patients who did not initially meet the threshold for surgery.

The overall mean Snellen visual acuity for prioritisation events accepted for cataract surgery in New Zealand is 6/38-2, significantly worse than the visual acuity reported in most OECD countries at the time of surgery. The 2018 Euroquo report that has data for over 2.8 million cataract surgeries completed in the European Union, reports 58.4% of European cataract surgery patients have visual acuity of 6/12 or better in the operated eye at the time of surgery.[20] Canadian guidelines recommend cataract surgery when the visual acuity decreases to 6/15 in the operative eye, or 6/12 with symptoms of glare, and/or anisometropia.[25] In contrast, only 34.9% of New Zealand patients in the current study had visual acuity of 6/12 or better in the operative eye at the time of prioritisation.

The current study noted that 25.6% of prioritisation events for public-funded cataract surgery were declined with significant regional variation noted. The lack of a single national CPAC threshold for public-funded cataract surgery, which ranged between 40–61 in 2018,[7] creates significant geographic disparity in access. Over one quarter of patients who were declined for surgery did not meet the visual acuity requirement for driving a private vehicle in New Zealand.[26] Although it is not possible to exclude the presence of visual comorbidities from data analysed in the current study, a small but significant number of patients who were declined for public-funded surgery had such advanced visual impairment they would be eligible for registration with Blind Low Vision New Zealand (formerly the Blind Foundation). The difference between declined rates based on the prioritised eye BSCVA and binocular BSCVA suggests that most prioritisation events that were declined were prioritisation events for second eye surgeries.

Although the mean BSCVA of all prioritisation events for public-funded cataract surgery in New Zealand were comparable across the regions, the SIR varied significantly from 95 to 737/100,000 population/year, with the Auckland region SIR more than double the national mean. Other OECD countries with regional variations in SIR[27,28] have proposed that this could be due to regional variation in the indication for surgery, or related to ocular health provider proximity; these studies however, reported SIR in isolation without reporting regional variations in BSCVA so further analysis is not possible. In the current study, the reason for geographic disparity in access to cataract surgery is the lack of a single national CPAC threshold.

The mean overall SIR for cataract surgery in New Zealand is lower than most OECD countries. Canada and Australia have cataract SIR's in excess of 1,000/100,000 population/year, over double the current rate in New Zealand even after adjusting for private surgery volumes.[2] The cataract SIR in the UK public-funded National Health Service has been over double the rate of public-funded surgery in New Zealand since 2014. Comparing with other OECD countries that have a similar ratio of gross domestic product to health spending to that of New Zealand, in 2018 New Zealand ranked 23 out of 27 for cataract SIR. The total New Zealand government spend on healthcare (7.5% of GDP) was ranked 14 out of 27 OECD countries.[2,29] New Zealand has relatively high rates of certain cancers,[30,31] obesity[32] and obesity-related chronic illnesses[32] that may limit funding elsewhere.

There are several limitations to the current study. The data includes only patients referred for public-funded cataract surgery and does not include data for surgery completed in the private healthcare sector. Visual acuity data were rounded to the nearest line at the time of prioritisation and may contain rounding errors in some cases. It is possible that a small number of patients who received public-funded cataract surgery may not have been prioritised prior to surgery and will not have been recorded in the current data set. Some patients who were prioritised for surgery may not have undergone surgery if for some reason they declined treatment.

Cataract surgery is relatively inexpensive with significant economic and quality of life benefits.[4–6] Visual impairment contributes to poor quality of life, falls, depression and loss of independence.[3,4,23] This is the first study reporting nationwide prioritisation for public-funded cataract surgery in New Zealand spanning a period of four years. New Zealand Māori and Pasifika patients prioritised for public-funded cataract surgery are typically younger and have significantly worse vision than other ethnic groups. Regional variation in CPAC thresholds creates significant geographic disparity for patients in New Zealand who have cataracts to access public-funded surgery. Given the well-established return on investment and dramatic improvement in quality of life associated with cataract surgery, increasing the cataract SIR to match other OECD rates, introducing a single national CPAC threshold, and improving access for New Zealand Māori and Pasifika would provide significant benefits for the New Zealand population.

Competing interests:
Dr McKelvie is an ophthalmologist. He performs cataract surgery on a regular basis at several DHBs and in private practice at Hamilton Eye Clinic. He is a co-founder for CatTrax, a web application designed to digitise cataract pathway data.

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www.nzma.org.nz/journal-articles/cataract-surgery-in-new-zealand-access-to-surgery-surgical-intervention-rates-and-visual-acuity

REFERENCES:

1. Pascolini D, Mariotti SP. Global estimates of visual impairment: 2010. Br J Ophthalmol 2012; 96:614–18.
2. OECD. Health Care Utilisation : Surgical procedures. OECD Stat. Accessed Nov 2019 Available from: http://stats.oecd.org/index.aspx?queryid=30167
3. Lord SR, Dayhew J. Visual risk factors for falls in older people. J Am Geriatr Soc 2001; 49:508–15.
4. Heemraz BS, Lee CN, Hysi PG, Jones CA, Hammond CJ, Mahroo OA. Changes in quality of life shortly after routine cataract surgery. Can J Ophthalmol 2016; 51:282–87.
5. Busbee BG, Brown MM, Brown GC, Sharma S. Incremental cost-effectiveness of initial cataract surgery. Ophthalmology 2002; 109:606–12; discussion 612–13.
6. Brown GC, Brown MM, Menezes A, Busbee BG, Lieske HB, Lieske PA. Cataract Surgery Cost Utility Revisited in 2012: A New Economic Paradigm. Ophthalmology. 2013; 120:2367–76.
7. Arrowsmith M. A response to your request for official information. Document number h201901181. Ministry of Health Manatū Hauora, 2019.
8. Robson B, Harris R. Hauora: Màori Standards of Health IV. A study of the years 2000–2005. Wellington: Te Ropu Rangahau Hauora a Eru Pomare 2007. Accessed Available from: http://www.moh.govt.nz/NoteBook/nbbooks.nsf/0/C3C40E20B25D301ECC2573B500014445/$file/hauora-iv.pdf
9. Kahukura T. Māori Health Chart Book 2015. Wellington: Ministry of Health, 2015 Accessed Available from: http://www.moh.govt.nz/NoteBook/nbbooks.nsf/0/28ACB9EA9A3C9D3FCC257EE5006B14F8/$file/tatau-kahukura-maori-health-chart-book-3rd-edition-oct15.pdf
10. New Zealand Ministry of Health. National Ethics Advisory Committee. Ethical Guidelines for Observational Studies: Observational research, audits and related activities. National Ethics Advisory Committee, 2012.
11. New Zealand Ministry of Health. Standard Operating Procedures for Health and Disability Ethics Committees. In: Ministry of Health, ed. Standard Operating Procedures for Health and Disability Ethics Committees. New Zealand Ministry of Health: Wellington, NZ, 2014.
12. NZ.Stat. Accessed Nov 2019 Available from: http://nzdotstat.stats.govt.nz/wbos/Index.aspx?DataSetCode=TABLECODE7501.
13. R: The R Project for Statistical Computing. Accessed Nov 2019 Available from: https://www.r-project.org/
14. Li SS, Misra S, Wallace H, Hunt L, McKelvie J. Patient-reported quality of life for cataract surgery: prospective validation of the 'Impact on Life' and Catquest-9SF questionnaires in New Zealand. N Z Med J 2019; 132:34–45.
15. Derrett S, Bevin TH, Herbison P, Paul C. Access to elective surgery in New Zealand: considering equity and the private and public mix. Int J Health Plann Manage 2009; 24:147–60.
16. Murphy R, Ghafel M, Beban G, Booth M, Bartholomew

K, Sandiford P. Variation in public-funded bariatric surgery intervention rate by New Zealand region. Intern Med J 2019; 49:391–95.

17. Behndig A, Montan P, Stenevi U, Kugelberg M, Lundström M. One million cataract surgeries: Swedish National Cataract Register 1992–2009. J Cataract Refract Surg 2011; 37:1539–45.
18. Schein OD, Cassard SD, Tielsch JM, Gower EW. Cataract surgery among Medicare beneficiaries. Ophthalmic Epidemiol 2012; 19:257–64.
19. Noertjojo K, Mildon D, Rollins D, Law F, Blicker J, Courtright P, et al. Cataract surgical outcome at the Vancouver Eye Care Centre: can it be predicted using current data? Can J Ophthalmol 2004; 39:38–47.
20. Lundström M, Brocato L, Dickman M, Henry Y, Manning S, Rosen P, Stenevi U, Tassignon MJ. ESCRS EUREQUO Annual Report 2018. European Society of Cataract and Refractive Surgeons, 2018 Accessed Available from: http://www.eurequo.org/wp-content/uploads/2020/02/EUREQUO_Annual_Report2018_final.pdf
21. Davis P, Lay-Yee R, Dyall L, Briant R, Sporle A, Brunt D et al. Quality of hospital care for Māori patients in New Zealand: retrospective cross-sectional assessment. Lancet 2006; 367:1920–25.
22. Gillies TD, Tomlin AM, Dovey SM, Tilyard MW. Ethnic disparities in asthma treatment and outcomes in children aged under 15 years in New Zealand: Analysis of national databases. Prim Care Respir J 2013; 22:312–18.
23. Javitt JC, Zhou Z, Willke RJ. Association between vision loss and higher medical care costs in Medicare beneficiaries costs are greater for those with progressive vision loss. Ophthalmology 2007; 114:238–45.
24. Ivers RQ, Cumming RG, Mitchell P, Simpson JM, Peduto AJ. Visual risk factors for hip fracture in older people. J Am Geriatr Soc 2003; 51:356–63.
25. Ministry of Health. Cataract - Treatment of Adults - Province of British Columbia. Accessed Nov 2019 Available from: http://www2.gov.bc.ca/gov/content/health/practitioner-professional-resources/bc-guidelines/cataract#recommendation2
26. 6 | NZ Transport Agency. Accessed Nov 2019 Available from: http://nzta.govt.nz/resources/medical-aspects/6.html
27. Lundström M, Stenevi U, Thorburn W. The Swedish National Cataract Register: A 9-year review. Acta Ophthalmol Scand 2002; 80:248–57.
28. Bernth-Petersen P, Bach E. Epidemiologic aspects of cataract surgery. II: Regional variation in frequencies. Acta Ophthalmol 1983; 61:397–405.
29. OECD. Health resources - Health spending. OECD Data. Accessed Nov 2019 Available from: http://data.oecd.org/healthres/health-spending.htm
30. Karimkhani C, Green AC, Nijsten T, Weinstock MA, Dellavalle RP, Naghavi M, et al. The global burden of melanoma: results from the Global Burden of Disease Study 2015. Br J Dermatol 2017; 177:134–40.
31. World Health Organization. Table by Cancers. International Agency for Research on Cancer. Accessed May 2020 Available from: http://ci5.iarc.fr/CI5plus/Pages/table3_sel.aspx
32. Lal A, Moodie M, Ashton T, Siahpush M, Swinburn B. Health care and lost productivity costs of overweight and obesity in New Zealand. Aust N Z J Public Health 2012; 36:550–56.

Trends in the diagnosis of high-grade cervical abnormalities in young women in the post-vaccination era

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ABSTRACT

BACKGROUND: Most cervical cancers are associated with human papillomavirus (HPV) types 16 and 18. In 2008, New Zealand commenced a quadrivalent HPV (virus-like particles of types 6, 11, 16 and 18) vaccination programme.

AIM: Document trends in number of colposcopy referrals and number and grade of cervical abnormalities diagnosed in women (20–24 years) referred to three large colposcopy clinics over time.

METHOD: Retrospective analysis of colposcopy clinic data.

RESULTS: The dataset included 5,012 episodes from 4,682 women. In Auckland (2013–2017), there was a 38% decrease in colposcopy referrals and 55% decrease in cervical intraepithelial neoplasia grade 2 (CIN2) or worse diagnoses. In Waikato (2011–2017), there was an 8% decrease in referrals and 22% reduction in CIN2 or worse diagnoses. In Canterbury (2011–2017), there was a 24% decrease in referrals and 49% reduction in CIN2 or worse diagnoses. Across all centres, the decrease in cervical intraepithelial neoplasia grade 3 (CIN3) or worse diagnoses was marked and more consistent than in CIN2 diagnoses. However, while the proportion of biopsies reported as CIN3 or worse decreased in non-Māori (24% in 2013 vs 16% in 2017, nptrend z=-4.24, p>|z| <.001), there was no change in Māori women (31% in 2013 vs 29% in 2017, nptrend z=-0.12, p>|z| =.90).

CONCLUSIONS: We observed a decreased number of CIN diagnoses in young women over time, with a particularly large drop in the number of CIN3/AIS/CGIN diagnoses. However, compared to non-Māori, Māori women having biopsies are more likely to have CIN3 or worse and there was a smaller reduction in the total number of Māori women diagnosed with CIN2 or worse.

In 2008, the quadrivalent HPV vaccine (containing HPV virus-like particles of types 6, 11, 16 and 18) was introduced to the New Zealand National Immunisation Schedule. HPV types 16 and 18 are associated with around 70% of all cervical cancers and about 50–60% of high-grade cervical precancerous abnormalities (CIN2/3).[1,2] HPV types 6 and 11 are associated with 90% of anogenital warts.[3]

When introduced, fully subsidised HPV vaccination was first offered to New Zealand young women born in 1990 and 1991. A catch-up programme was then offered to girls and young women aged 9–20 years.[4] From 1 January 2017 onwards, HPV immunisation became funded for everyone aged 9–26 years, including boys and young men.[4] HPV vaccination coverage in New Zealand has increased from 39% (for all three HPV

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doses) for the cohort born in 1990 to 67% (for all three HPV doses) for the cohort born in 2003.[5]

International research has demonstrated a decrease in vaccine-type HPV infections and the number of high-grade cervical cell abnormalities in young women following HPV vaccine introduction compared to pre-vaccine introduction[6–11] and in HPV-vaccinated compared to unvaccinated women.[12–16]

The New Zealand National Cervical Screening Programme (NCSP) was established in 1990 and until very recently recommended regular three-yearly cervical screening for women aged 20–69 years.[17] The incidence of cervical cancer in New Zealand has decreased from 10.5 per 100,000 women in 1996 to 5.5 per 100,000 women in 2014 for all ethnicities.[18] For Māori women, rates have decreased from 25 to 10.8 per 100,000 women over the same time period.[18] Women born in 1990 turned 20 and became eligible for cervical screening in 2010.

Many countries are currently reconsidering cytology-based cervical screening programs.[19] Australia, among other countries, has already transitioned to five-yearly primary HPV screening for women aged 25–74 years.[19] New Zealand increased the commencement age for cervical screening from 20 to 25 years in November 2019 and plans to commence HPV primary screening in 2021.[20] The justification for increasing the commencement age comes from local[21] and international[22,23] screening data and research showing that screening women aged 20–24 does not reduce the incidence of cervical cancer in these women.

Women in New Zealand aged under 25 have high incidences of cervical abnormalities but information regarding them will greatly diminish when they cease to be screened. Furthermore, there is little published information regarding the trends of change in cervical abnormalities in women under 25 in New Zealand, since the vaccination programme started.

This study's aims were to document, in a vaccine-eligible population of women aged 20–24 years, any change over time in (a) colposcopy referrals and (b) the number of histologically confirmed high-grade CIN (CIN2 and CIN3). A secondary aim was to investigate any differences over time regarding histologically confirmed high-grade CIN in Māori women compared to non-Māori.

Methods

Ethical approval for this retrospective audit was received from the University of Otago Human Research Ethics Committee (approved 24 August 2017, reference number HD17/033). Data were collected from the databases of three major colposcopy clinics serving three district health boards (DHBs) in New Zealand: National Women's Health, Auckland City Hospital, Auckland (Auckland), Waikato Hospital, Hamilton (Waikato) and Christchurch Women's Hospital, Christchurch (Canterbury). At each centre, data was exported from Gynaecology Plus colposcopy databases (Version 7, Solutions Plus, Auckland, New Zealand, http://www.solutionsplus.co.nz/index.php/gynaecology-plus/). Exported data included the woman's date of birth, ethnicity, referral indication, referral cervical cytology sample grade, number of visits, histological biopsy results (if applicable) and type of treatment (if applicable). The age attributed to each woman was the age she was when she attended her first visit. Each woman was attributed a single ethnicity using the NCSP priority order: Māori, Pacific, Asian, European/Other, ie, a woman identifying as New Zealand European and Māori, is counted as Māori.[24]

Data was episode-based, with an episode including data from all visits, starting from being referred, to colposcopy, through to discharge. Following discharge, a woman could have another abnormal cervical cytology sample and be re-referred, which would then mark the start of second episode. It was possible to export the self-reported vaccination status for a subset of women from the Canterbury cohort; however, the option to extract vaccination status was not available from Auckland and Waikato at the time of data extraction.

All episodes were included in the colposcopy referrals analysis, but only episodes from women referred following an abnormal cervical cytology sample were used to examine the trend of histologically confirmed high-grade CIN (CIN2 and CIN3)

changes over time. Women with clinical symptoms or an abnormal appearing cervix, vulva or genital tract may also be referred to colposcopy in the absence of abnormal cytology. However, the vast majority of women referred without abnormal cytology will not have CIN or any other significant cervical abnormalities. Referral patterns for women without abnormal cytology may vary and it was considered that inclusion of these women may lead to a misleading bias.

It is possible that occasionally no histological samples are taken following referral for an abnormal cervical cytology sample or the histological sample is deemed to be unsatisfactory. These episodes cannot contribute to the trends of histologically diagnosed CIN, but the number of episodes in which this occurs will be provided for completeness.

The time-period for which data was extracted was in a non-calendar year format from 1 November 2010 to 31 October 2017 for Waikato and Canterbury. For Auckland data was extracted from 1 November 2012 to 31 October 2017. This was done to maintain consistency in the data parameters obtained as previous to this Auckland was using a different system. All women who had been referred and had their first attended visit within these respective time frames for each colposcopy clinic, and were between 20 and 24 years of age at their first attended visit, were included in the data. Using non-calendar years allowed maximal utilisation of available data and the most recent data up till late 2017. For brevity, years are specified based on the 'year ending' when referring to each year, eg, the 12 months between 1 November 2012 and 31 October 2013, is referred to as 2013.

Cervical glandular intraepithelial neoplasia (CGIN) and adenocarcinoma in situ (AIS) were grouped with CIN3 for analysis. For completeness, low-grade CIN changes (CIN1) and microinvasive or invasive carcinoma were also included in analyses. The main endpoint of this study was the highest (worst) histologically confirmed diagnosis per episode contributed by each woman. It was possible for a woman to contribute more than one episode (ie, the patient may be re-referred following discharge), but she could only contribute one outcome per episode. The number of low-grade CIN (CIN1), and high-grade CIN (CIN2 and CIN3/AIS/CGIN) diagnoses per year served as indicators of trends.

The number of episodes with each grade of cervical cell abnormality was determined for each centre (and for combined centres) each year and change over time in CIN diagnoses was investigated using a non-parametric test for trend across ordered groups as implemented in STATA (nptrend StataCorp. 2013. Stata Statistical Software: Release 13. College Station, TX: StataCorp LP). Significance level was set at alpha =.05

Results

The dataset included 5,012 episodes from 4,682 women. Of this dataset 4,460 episodes from 4,209 women were used to examine the trend of histologically confirmed high-grade CIN (CIN2 and CIN3) changes over time as these episodes came from women referred following an abnormal cervical cytology sample. The remaining 552 episodes (from 473 women) were excluded from the high-grade CIN trend analysis due to referral for other reasons including clinical reasons (eg, pelvic pain, abnormal bleeding, abnormal appearing cervix, suspicious symptoms or vaginal and vulval inflammation, metaplasia and atrophy). Of the episodes excluded from the high-grade CIN trend analysis, there was no biopsy taken in 280 (51%), there was a low-grade or normal biopsy result in 227 (41%), CIN2 was diagnosed in 25 (5%), and CIN3 or AIS was diagnosed in 20 (4%). There were no cervical carcinoma diagnoses in the excluded episodes.

Auckland

One thousand one hundred and sixty-five episodes (from 1,126 women) were recorded at National Women's Health, Auckland City Hospital, Auckland (2013–2017). There was a 38% decrease in total referrals over time (292 episodes in 2013 vs 182 episodes in 2017).

One thousand and sixty-four episodes (from 1,036 women) were referrals following an abnormal cervical cytology sample. There was one cervical carcinoma diagnosis. See Appendix for the histology results for all women.

There was a 45%, 55%, and 54% reduction in the number of CIN1, CIN2 and CIN3/AIS/CGIN diagnoses over time, respectively (see

Figure 1). However, there was a transient increase in CIN1 diagnoses in 2016. Overall (2013–2017), there was a 50% reduction in CIN1 or worse diagnoses over time and a 55% reduction in total CIN2 or worse diagnoses over time.

Waikato

One thousand four hundred and eighteen episodes (from 1,295 women) were recorded at Waikato Hospital (2011–2017). There was an 8% decrease in total referrals over time (221 episodes in 2011 vs 204 episodes in 2017).

One thousand two hundred and ninety episodes (from 1,192 women) were referrals following an abnormal cervical cytology sample. There was one cervical carcinoma diagnosis. See Appendix for the histology results for all women.

The number of CIN3/AIS/CGIN diagnoses increased from 2011 to 2014, following which there was a 50% decrease in diagnoses between 2014 and 2017 (see Figure 2). The number of CIN1 and CIN2 diagnoses remained relatively consistent, other than a transient decrease in CIN 1 diagnoses in 2015. Overall (2011–2017), there was a 16% reduction in total CIN1 or worse diagnoses and a 22% reduction in total CIN2 or worse diagnoses.

Canterbury

Two thousand four hundred and twenty-nine episodes (from 2,261 women) were recorded at Christchurch Women's Hospital (2011–2017). There was a 24% decrease in total referrals over time (367 episodes in 2011 vs 278 episodes in 2017).

Two thousand one hundred and six episodes (from 1,981 women) were referrals following an abnormal cervical cytology sample. There were five cervical carcinoma diagnoses. See Appendix for the histology results for all women.

Overall CIN diagnoses increased from 2011 to 2012 and then began to decrease, with a 74% decrease in CIN3/AIS/CGIN diagnoses and 36% decrease in CIN1 diagnoses between 2012 and 2017 (see Figure 3). The number of CIN2 diagnoses were relatively stable over time with a transient increase in diagnoses in 2016. Overall (2011–2017), there was a 38% reduction in total CIN1 or worse diagnoses and a 50% reduction in total CIN2 or worse diagnoses.

Impact of HPV vaccination in Canterbury

HPV vaccination status was known for 823 episodes from 752 women (367 unvaccinated and 385 vaccinated) referred

Figure 1: Number of CIN diagnoses by grade (2013–2017) at National Women's Health, Auckland City Hospital, (Auckland) in women aged 20–24 years referred following an abnormal cervical cytology sample.

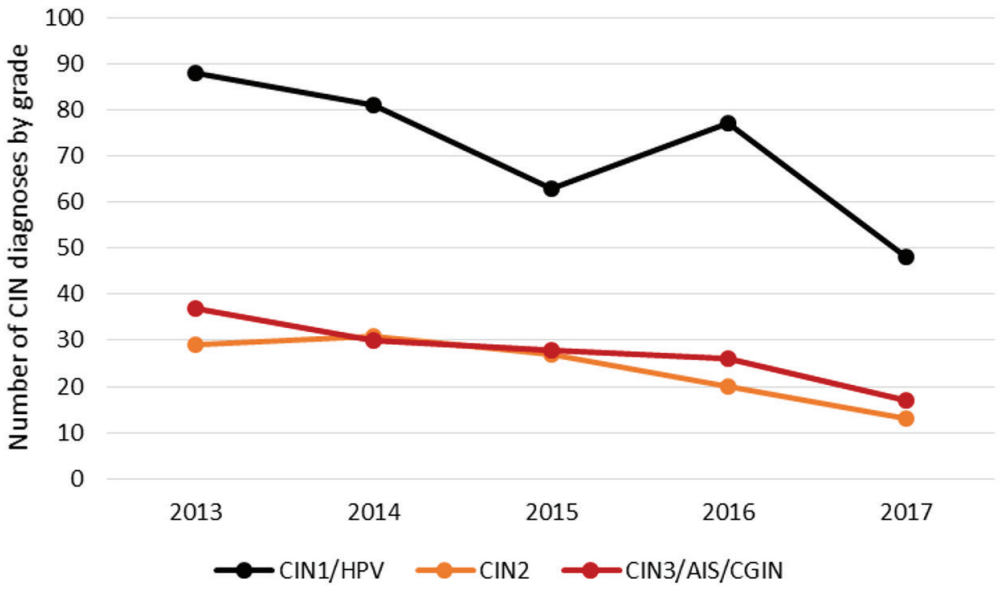
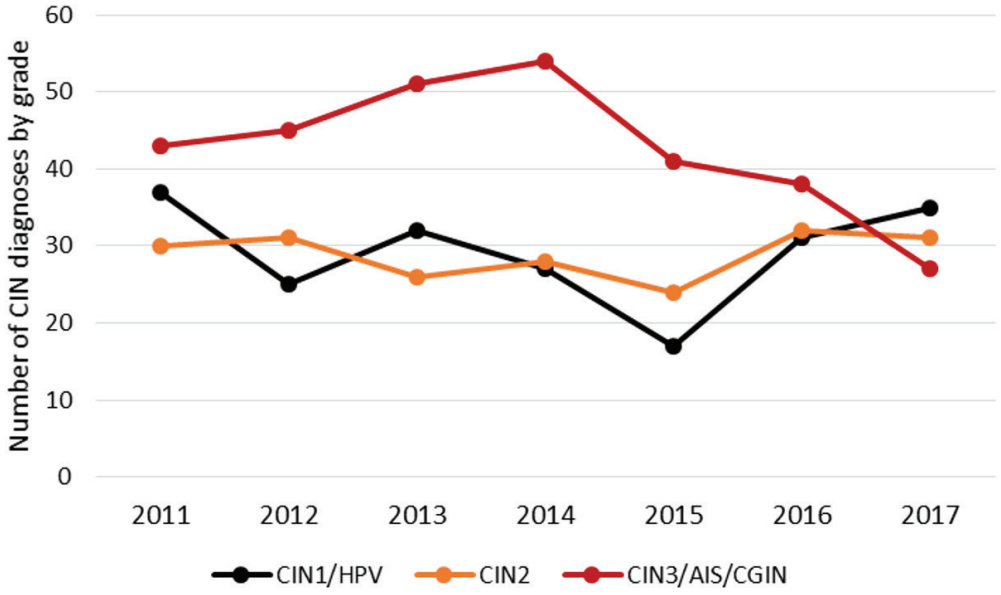


Figure 2: Number of CIN diagnoses by grade (2011–2017) at Waikato Hospital in women aged 20–24 years referred following an abnormal cervical cytology sample.



following an abnormal cervical cytology sample (See Table 1).

Of the 823 episodes where vaccination status was known, 789 had a satisfactory histological biopsy, of which 382 (48%) episodes were for unvaccinated women and 407 (52%) were for vaccinated women. CIN3 or worse was diagnosed in a significantly higher proportion of episodes for unvaccinated women than vaccinated women (31% vs 19%, X^2=15.18 p<.001). There was no evidence of a difference in the proportion of CIN2 episodes for unvaccinated and vaccinated women (29% vs 24%, X^2=2.64 p=.104).

Figure 3: Number of CIN diagnoses by grade (2011–2017) in women aged 20–24 years at Christchurch Women's Hospital (Canterbury).

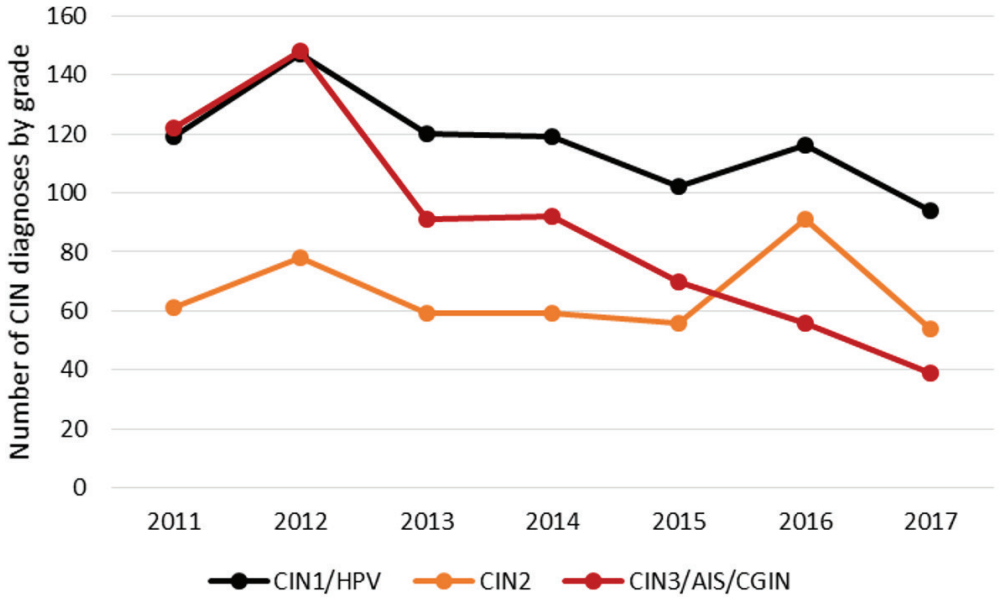


Table 1: Number of episodes and contributing women by vaccination status.

Vaccination status	Number of women
Vaccination status known	752
• Unvaccinated	367
• Vaccinated	385
• Vaccinated—complete	241
• Vaccinated—incomplete	25
• Vaccinated—number of doses not reported	119

There was weak evidence that CIN1 or better was diagnosed in a higher proportion of episodes for vaccinated women than unvaccinated women (52% vs 45%, X^2=3.68 p=.055).

Impact of ethnicity

Ethnicity was not recorded for 302 episodes from 282 women across the three centres (2013–2017). These episodes were excluded from ethnicity analyses.

Four hundred and fifty-six episodes from 409 Māori women were recorded at the three centres (2013–2017). There was a 27% decrease in total referrals for Māori women over time (108 in 2013 vs 79 in 2017). Four hundred and eighteen episodes from 379 Māori women were referrals following an abnormal cervical cytology sample. Of these episodes, 359 had a satisfactory histological biopsy. See Appendix for the histology results for Māori vs non-Māori women.

Three non-Māori women and no Māori women were diagnosed with cervical carcinoma following an abnormal cytology sample (2013–2017). Between 2013 and 2017, Māori women had a 58% reduction in CIN1, no reduction in CIN2 and a 33% reduction in CIN3 or worse. This pattern appears different in non-Māori women who had a more marked reduction in CIN3 or worse (48% decrease) but less reduction in CIN1 (13% decrease). The number of high grade CIN diagnoses by grade (2013–2017) in women aged 20–24 years by ethnicity is shown in Figure 4.

If we consider CIN3 or worse as a proportion of all satisfactory biopsies, overall Māori women had a higher proportion of CIN3 or worse than non-Māori (29% vs 23%, X^2=5.69, p=.02).

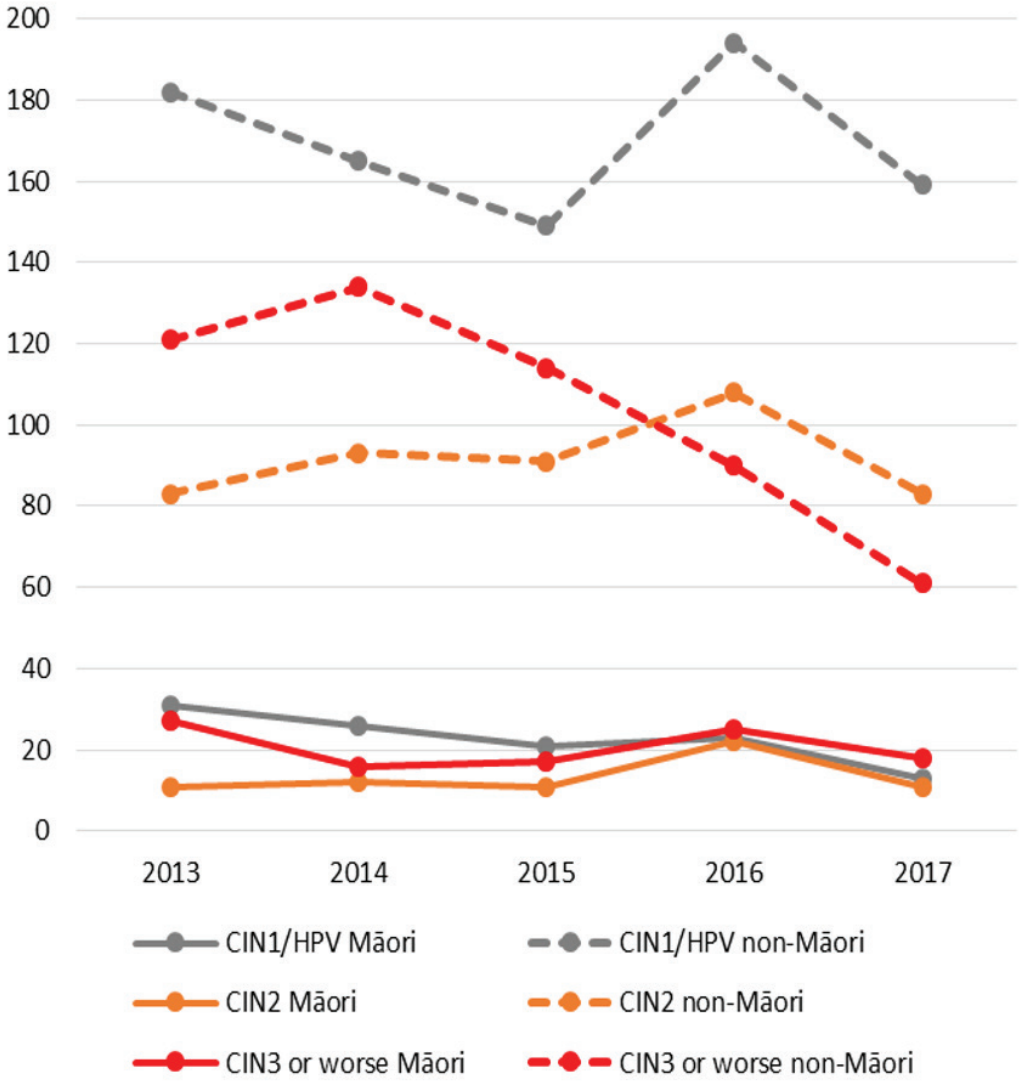
Furthermore, for Māori women there was no change over time in CIN3 or worse diagnoses as a proportion of all satisfactory biopsies (31% in 2013 vs 29% in 2017, nptrend z=-0.12, p>|z| =0.90). In contrast, in non-Māori women there was a decrease over time in CIN3 or worse diagnoses as a proportion of all satisfactory biopsies (24% in 2013 vs 16% in 2017, nptrend z=-4.24, p>|z| <0.001).

Impact of smoking

Smoking status was recorded for 61% of women (2,323 episodes from 2,110 women) across the three centres (2013–2017). Where smoking status was recorded, 30% (563/1,892) of referrals following an abnormal cervical cytology sample self-reported smoking. Overall, smoking rates decreased over time (35% in 2013 vs 25% in 2017, z=-3.93, p>|z| <.001). Women who smoked were more likely to be diagnosed with CIN3 or worse than women who did not smoke (32% vs 21%, X^2=23.3 p<.001).

A higher proportion of Māori women self-reported smoking than non-Māori women (55% vs 26%, X^2=85.5 p<.001). Smoking rates decreased over time in non-Māori women (28% in 2013 vs 23% in 2017, z= -2.79, p>|z|=.005). However, although smoking rates also appeared to decrease over time in Māori women (62% in 2013 vs 47% in 2017), trend analysis did not reach significance (z=-1.65, p>|z| =.099). See Appendix for a figure showing the proportion of women who self-reported smoking (2013–2017) by ethnicity.

Figure 4: Number of CIN diagnoses by grade (2013–2017) in women aged 20–24 years by ethnicity.*



*Excludes n=258 women where ethnicity was unknown.

Discussion

In this study, we describe the changes over time in the number and type of cervical abnormalities diagnosed in women (aged 20–24 years) in three large public hospital colposcopy clinics following the introduction of a national HPV vaccination programme. We observed in all three clinics a reduction in women diagnosed with high-grade abnormalities (CIN2 or worse). The reduction was particularly marked and consistent for CIN3 or worse. In comparison, changes in CIN1 and CIN2 varied between clinics and, overall, the reduction was less marked.

The changes observed in our study are consistent with data from NCSP reports. Nationally, histologically confirmed high-grade CIN for 20–24-year-old women decreased between 2011 and 2017.[25] This decrease is expected in the context of the introduction of HPV vaccination. Using data from the vaccination registry and the NCSP, we have previously demonstrated that vaccinated women had a 31% reduced cumulative incidence of high-grade abnormalities but only a 15% reduction of low-grade abnormalities when compared to non-vaccinated women.[26]

In this study, as laboratories reporting to these colposcopy clinics routinely differentiate between a histological diagnosis of CIN2 and CIN3 in young women we were able to report these occurrences separately. It is notable in this study that the reduction of CIN3 is more marked and consistently observed in all three clinics, whereas the pattern of CIN2 diagnoses over time was

more similar to CIN1. This would suggest that a greater proportion of CIN3 diagnosis are due to HPV 16 or 18 than CIN2.

While we do not have HPV typing data for this study, previous studies examining HPV genotypes found in high-grade cervical disease have reported a prevalence of types 16 and/or 18 of 40–53% in CIN2 and 58–75% for CIN3 lesions.[2,27] Other oncogenic types (excluding types 16 and 18) have been shown to be more prevalent in CIN2 compared to CIN3.[27] Furthermore, studies indicate that CIN3 lesions associated with HPV 16 occur at a significantly younger age compared to lesions associated with other high-risk HPV infections.[28,29] Thus, vaccination may be more effective at preventing CIN3 disease than CIN2 disease, especially in young women.

As expected in the sub-population of women where vaccination status was known, the proportion of women with CIN3 or worse was lower in vaccinated women than in unvaccinated women, indicating a protective effect of vaccination. It is likely, however, that the decreases in CIN3 or worse observed are not just due to reductions in vaccinated women. It is also likely that a reduction of prevalent HPV 16 and 18 in the community has resulted in a reduction of HPV 16/18 in non-vaccinated women via the herd effect. We have previously described a reduction of HPV 16 in young women with CIN2 regardless of vaccination status.[30] Other populations have demonstrated reductions in vaccination HPV types in non-vaccinated women belonging to vaccination eligible cohorts.[31–33]

An important observation of the study was that the proportion of satisfactory biopsies that were CIN3 or worse was greater in Māori women and this proportion decreased over time in non–Māori women but did not change in Māori women. We hypothesise that this inequity is due to reduced vaccination rates for Māori women in this cohort. New Zealand Ministry of Health data on vaccination coverage is published for birth cohort years 1990 to 2003 and women are grouped as either Māori, Pacific, Asian or Other. The 'Other' category is comprised predominantly of New Zealand European women.[4] Compared to 'Other' women, HPV vaccination coverage for Māori was lower in earlier birth cohorts (ie, those born in 1990 [23% vs 47%] to 1993 [47% vs 54%]). Other research examining the pre-vaccination HPV type prevalence between Māori and non-Māori women with high-grade cytological abnormalities, found no significant difference between Māori and non-Māori regarding the prevalence of HPV 16 and/or 18.[34] In addition, data matching between the NCSP register and the vaccination registry revealed that the cumulative incidence of high-grade CIN was dependent on vaccination status but did not vary between Māori and European women.[26] Reassuringly, Māori have had either similar or slightly higher vaccination coverage compared to 'Other' women for birth cohorts from 1994 onwards so hopefully this inequity will not persist.

A confounding risk factor is smoking. Although our data is incomplete, smokers had a higher proportion of CIN3 or worse than non-smokers and Māori women were more likely to report being smokers.

The study limitations include its retrospective nature, some missing data, and the involvement of only some colposcopy units, which although large and cover demographically different populations, may not provide findings that accurately reflect trends in the entire New Zealand population.

The three clinics showed large variation particularly regarding trends in the numbers of CIN1. We have no clear explanation of these trends. We acknowledge the quadrivalent HPV vaccine introduced in New Zealand, includes vaccination against types 6 and 11 which may cause low-grade but not high-grade abnormalities, and we have little information regarding the regional epidemiology of these virus types. It is of note that the denominator of the referral populations cannot be accurately described and the proportion of women with screen-detected abnormalities from each DHB that are referred to that clinic stratified by age is undocumented. It is therefore difficult to compare the trends seen in three different clinics.

Referral patterns may vary between DHB and also over time. However, approximately 10% of colposcopies nationwide are performed in private practice and it has stayed at this proportion over the years, hence it is unlikely to have influenced trends over time in our study.[25]

Public colposcopy clinics in each DHB have remained the same for the duration of our study period.[25] In addition, there have been no systematic changes that we are aware of that are likely to have changed referral patterns over this time. However, there is inter-observer variation in the reporting of cervical cytology and histology, which may influence histology findings; this is somewhat reflected in the differing positive predictive value of abnormal smears reported in different laboratories as seen in the NCSP monitoring reports.[25]

An important confounding variable is that screening rates for 20–25-year-olds have decreased between 2013 and 2017, nationally. Using the NCSP's interactive screening coverage app,[35] we are able to observe that from 2011 to 2017 there has been a decrease of 9.8% in three-yearly screening coverage, nationally. This decrease has not been uniform as screening rates remained relatively stable from 2010 to 2015 (53,000–54,000 satisfactory cervical cytology samples per annum) but subsequently dropped to 51,000 per annum in 2016 and dropped further to 48,000 per annum in 2017, nationally.[25] Per DHB there has been a 5.5%, 6.7% and 9.9% decrease in screening coverage for Auckland, Waikato and Canterbury, respectively.[35] These reductions in screening rates undoubtedly explain a proportion of the reductions we observed but are unlikely to explain the different reductions depending on CIN grade. Unfortunately, we are unable to account for ethnicity-related changes in screening rates in young women.

The population of women aged 20–24 years has been steadily increasing in each of the DHBs during our study period,[35] hence we can exclude the scenario of a decreasing population as a contributing reason for a decrease in the number of CIN lesions.

HPV vaccination status was not known for a substantial proportion of women in our study across all years but especially for women referred prior to 2013. In addition, for women referred prior to 2013, a very low proportion were known to be vaccinated. Both reporting of vaccination status and the proportion of women who were vaccinated increased from 2013 onwards. However, missing vaccination status data, especially in earlier years, does limit the power of our analyses.

This study adds to the information describing the impact of the National HPV vaccination programme. Overall, the drop in cervical abnormalities in this age group have been modest. This is likely a result of relatively low HPV vaccination coverage in New Zealand, the limited time since the introduction of the vaccination programme, and type-specific coverage of the vaccine. A meta-analysis demonstrated the dose-response link between vaccination coverage and the reduction in cervical disease.[6] This is supported by studies showing that populations with a higher vaccination coverage such as Australia and Scotland show a reduction in cervical disease spanning all three CIN types.[6,14,16] In contrast the impressive reduction of CIN3 or worse abnormalities is an indication of the effectiveness of the vaccination programme in reducing the occurrence of these precancerous abnormalities.

These data demonstrate that following the introduction of the HPV vaccination programme in 2008 there has been a subsequent marked decrease in young women with CIN3 or worse. As women under 25 years with CIN2 are no longer routinely treated, this translates to a major reduction in the requirement for destructive cervical treatments in this age group. In time, a decrease in CIN3 is also likely to result in a reduction in the incidence of cervical cancer provided cervical screening rates can be maintained. From 2017, HPV vaccination became fully funded for males and females, aged 9—26.[4] Also available from 2017 onwards is a second-generation nonavalent HPV vaccine (Gardasil-9; Merck), which includes the addition of the next five most prevalent oncogenic HPV types. Together, the seven high-risk types covered by this nonavalent vaccine are associated with 90% of cervical cancers, thus providing more benefit than the quadrivalent vaccine. This should herald a greater reduction in the incidence of cervical disease.[36]

It is of concern that, in this study, CIN3 or worse was observed in a higher proportion of Māori women compared to non-Māori women. In addition, the reduction in CIN3 or worse does not appear to be shared

equally with Māori women. As the NCSP no longer recommends screening for women under age 25, this inequity can no longer be explored. It would appear essential that access to screening is prioritised for young Māori women and that, for screen eligible women, pre-invasive and invasive disease rates are carefully monitored.

In conclusion, over time we observed a decreased number of CIN diagnoses with a particularly large drop in the number of CIN3/AIS/CGIN. However, compared to non-Māori women, Māori women having cervical biopsies are more likely to have CIN3 or worse and there was a smaller reduction in the total number of Māori women diagnosed with high-grade disease. We hypothesise that the overall decreases are largely due to the prevention of infections with HPV 16 and 18 as a result of HPV vaccination. Further measures need to be taken to reduce inequities for New Zealand women.

Appendix

Appendix Table 1: Histology results at National Women's Health, Auckland City Hospital, (Auckland) per year for young women referred following an abnormal cervical cytology sample.

	2013	2014	2015	2016	2017	Total	% change 2013–2017
No biopsy or unsatisfactory biopsy	69	47	36	50	40	242	-42%
Normal or benign*	51	47	27	43	38	206	-25%
CIN1/HPV	88	81	63	77	48	357	-45%
CIN2	29	31	27	20	13	120	-55%
CIN3/AIS/CGIN	37	30	28	26	17	138	-54%
>Stage 1a1 carcinoma	1	0	0	0	0	1	-100%
Total episodes	275	236	181	216	156	1,064	-43%

*Includes normal, cervicitis, inflammation only, squamous metaplasia.
Abbreviations: CIN1/HPV – cervical intraepithelial neoplasia grade 1 or human papillomavirus effect, CIN2 – cervical intraepithelial neoplasia grade 2, CIN3 – cervical intraepithelial neoplasia grade 3, AIS – adenocarcinoma in situ, CGIN – cervical glandular intraepithelial neoplasia.

Appendix Table 2: Histology results at Waikato Hospital per year for young women referred following an abnormal cervical cytology sample.

	2011	2012	2013	2014	2015	2016	2017	Total	% change 2011–2017
No biopsy or unsatisfactory biopsy	47	27	36	45	17	32	35	239	-26%
Normal or benign*	46	49	62	41	40	46	61	345	+33%
CIN1/HPV	37	25	32	27	17	31	35	204	-5%
CIN2	30	31	26	28	24	32	31	202	+3%
CIN3/AIS/CGIN	43	45	51	54	41	38	27	299	-37%
Stage 1a1 carcinoma	1	0	0	0	0	0	0	1	-100%
Total	204	177	207	195	139	179	189	1,290	-7%

*Includes normal, cervicitis, inflammation only, squamous metaplasia.
Abbreviations: CIN1/HPV – cervical intraepithelial neoplasia grade 1 or human papillomavirus effect, CIN2 – cervical intraepithelial neoplasia grade 2, CIN3 – cervical intraepithelial neoplasia grade 3, AIS – adenocarcinoma in situ, CGIN – cervical glandular intraepithelial neoplasia.

Appendix Table 3: Histology results at Christchurch Women's Hospital per year for young women referred following an abnormal cervical cytology sample.

	2011	2012	2013	2014	2015	2016	2017	Total	% change 2011–2017
No biopsy or unsatisfactory biopsy	19	20	12	13	6	10	16	96	-16%
Normal or benign*	15	16	14	12	18	22	15	112	0%
CIN1/HPV	119	147	120	119	102	116	94	817	-21%
CIN2	61	78	59	59	56	91	54	458	-11%
CIN3/AIS/CGIN	122	148	91	92	70	56	39	618	-68%
Invasive adenocarcinoma	1	0	0	0	0	0	0	0	-100%
Stage 1a1 carcinoma	0	1	0	0	0	0	0	1	0%
>Stage 1a1 carcinoma	1	0	2	0	0	0	0	3	-100%
Total episodes	338	410	298	295	252	295	218	2,106	-36%

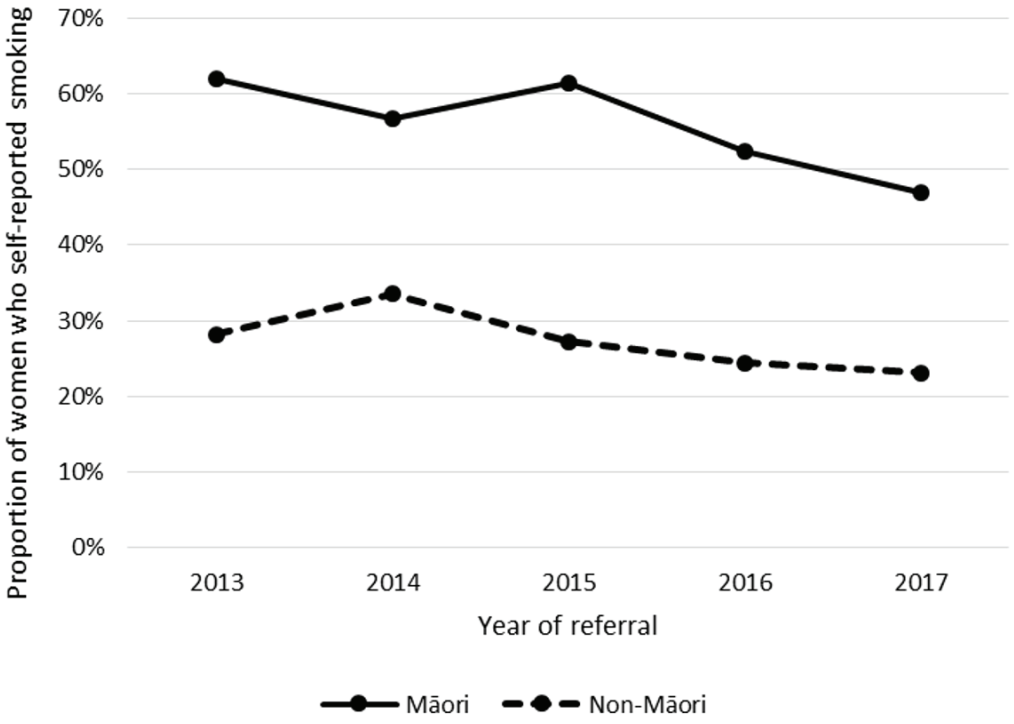
*Includes normal, cervicitis, inflammation only, squamous metaplasia.
Abbreviations: CIN1/HPV – cervical intraepithelial neoplasia grade 1 or human papillomavirus effect, CIN2 – cervical intraepithelial neoplasia grade 2, CIN3 – cervical intraepithelial neoplasia grade 3, AIS – adenocarcinoma in situ, CGIN – cervical glandular intraepithelial neoplasia.

Appendix Table 4: Histology results for young Māori versus non-Māori women* referred following an abnormal cervical cytology sample.

		2013	2014	2015	2016	2017	Total	% change 2013–2017
No biopsy or unsatisfactory biopsy	Māori	15	11	11	15	7	59	-53%
	Non-Māori	99	90	48	74	76	387	-23%
Normal or benign^	Māori	17	11	10	17	20	75	+18%
	Non-Māori	108	82	72	89	90	441	-17%
CIN1/HPV	Māori	31	26	21	23	13	114	-58%
	Non-Māori	182	165	149	194	159	849	-13%
CIN2	Māori	11	12	11	22	11	67	0%
	Non-Māori	83	93	91	108	83	458	0%
CIN3/AIS/CGIN	Māori	27	16	17	25	18	103	-33%
	Non-Māori	118	134	114	90	61	517	-48%
>1a invasive carcinoma	Māori	0	0	0	0	0	0	0%
	Non-Māori	3	0	0	0	0	3	-100%
Total	Māori	101	76	70	102	69	418	-32%
	Non-Māori	593	564	474	555	469	2,655	-21%

*Excludes women where ethnicity was unknown (n=258) ^ Includes normal, cervicitis, inflammation only, squamous metaplasia.
Abbreviations: CIN1/HPV – cervical intraepithelial neoplasia grade 1 or human papillomavirus effect, CIN2 – cervical intraepithelial neoplasia grade 2, CIN3 – cervical intraepithelial neoplasia grade 3, AIS – adenocarcinoma in situ, CGIN – cervical glandular intraepithelial neoplasia.

Appendix Figure 1: Proportion of women who self-reported smoking in those aged 20–24 years referred following an abnormal cervical cytology sample (2013–2017) by ethnicity.*



*Excludes n=258 women where ethnicity was unknown and n=1,191 where smoking status was unknown.

Competing interests:
Dr Sykes reports grants from Cancer Society of New Zealand during the conduct of the study.

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REFERENCES:

1. Smith JS, Lindsay L, Hoots B, et al. Human papillomavirus type distribution in invasive cervical cancer and high-grade cervical lesions: a meta-analysis update. Int J Cancer. 2007; 121:621–32.
2. Simonella LM, Lewis H, Smith M, Neal H, Bromhead C, Canfell K. Type-specific oncogenic human papillomavirus infection in high grade cervical disease in New Zealand. BMC Infectious Diseases. 2013; 13:114.
3. Garland SM, Steben M, Sings HL, et al. Natural history of genital warts: analysis of the placebo arm of 2 randomized phase III trials of a quadrivalent human papillomavirus (types 6, 11, 16, and 18) vaccine. J Infect Dis. 2009; 199:805–14.
4. Ministry of Health. HPV immunisation programme. 2018: Ministry of Health; Access date: 12/12/2018, Available from:^http://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/hpv-immunisation-programme
5. Final Dose HPV Immmunisation Coverage All DHBs: girls born between 1990 and 2002. 2016. New Zealand: Ministry of Health; Access date: 14 June, 2018. Available from:^http://www.health.govt.nz/our-work/preventative-health-wellness/immunisation/hpv-immunisation-programme
6. Drolet M, Bénard É, Boily M-C, et al. Population-level impact and herd effects following human papillomavirus vaccination programmes: a systematic review and meta-analysis. Lancet Infect Dis. 2015; 15:565–80.
7. Cameron RL, Kavanagh K, Pan J, et al. Human Papillomavirus Prevalence and Herd Immunity after Introduction of Vaccination Program, Scotland, 2009–2013. Emerg Infect Dis. 2016; 22:56.
8. Hariri S, Johnson ML, Bennett NM, et al. Population-based trends in high-grade cervical lesions in the early human papillomavirus vaccine era in the United States. Cancer. 2015; 121:2775–81.
9. Brotherton JM, Saville AM, May CL, Chappell G, Gertig DM. Human papillomavirus vaccination is changing the epidemiology of high-grade cervical lesions in Australia. Cancer Causes Control. 2015; 26:953–4.
10. Berenson AB, Hirth JM, Chang M. Change in Human Papillomavirus Prevalence Among U.S. Women Aged 18–59 Years, 2009–2014. Obstet Gynecol. 2017; 130:693.
11. Mesher D, Panwar K, Thomas SL, Beddows S, Soldan K. Continuing reductions in HPV 16/18 in a population with high coverage of bivalent HPV vaccination in England: an ongoing cross-sectional study. BMJ Open. 2016; 6:e009915.
12. Baldur-Felskov B, Dehlendorff C, Munk C, Kjaer SK. Early impact of human papillomavirus vaccination on cervical neoplasia--nationwide follow-up of young Danish women. J Natl Cancer Inst. 2014; 106:djt460.
13. Crowe E, Pandeya N, Brotherton JML, et al. Effectiveness of quadrivalent human papillomavirus vaccine for the prevention of cervical abnormalities: case-control study nested within a population based screening programme in Australia. BMJ. 2014; 348.
14. Pollock KG, Kavanagh K, Potts A, et al. Reduction of low- and high-grade cervical abnormalities associated with high uptake of the HPV bivalent vaccine in Scotland. Br J Cancer. 2014; 111:1824–30.
15. Cameron RL, Kavanagh K, Cameron Watt D, et al. The impact of bivalent HPV vaccine on cervical intraepithelial neoplasia by deprivation in Scotland: reducing the gap. J Epidemiol Community Health. 2017; 71:954–60.
16. Brisson M, Bénard É, Drolet M, et al. Population-level impact, herd immunity, and elimination after human papillomavirus vaccination: a systematic review and meta-analysis of predictions from transmission-dynamic models. Lancet Public Health. 2016; 1:e8–e17.
17. National Screening Unit. Guidelines for Cervical Screening in New Zealand - Incorporating the Management of Women with Abnormal Cervical Smears. In: Lewis H, Fentiman G, Bethwaite P, eds. Wellington: Ministry of Health, 2008.
18. Smith M, Leanne R, Canfell K. National Cervical Screening Programme Annual Report 2014. 2017. ^http://www.nsu.govt.nz/system/files/page/nsu_screening_report_2014.pdf
19. Simms KT, Smith MA, Lew JB, Kitchener HC, Castle PE, Canfell K. Will cervical screening remain cost-effective in women offered the next generation nonavalent HPV vaccine?

Results for four developed countries. Int J Cancer. 2016; 139:2771–80.

20. National Screening Unit. HPV primary screening. 2017 Ministry of Health; Access date: 2 Sep 2018, 2018. Available from:^http://www.nsu.govt.nz/health-professionals/national-cervical-screening-programme/hpv-primary-screening

21. Smith MA, Edwards S, Canfell K. Impact of the National Cervical Screening Programme in New Zealand by age: analysis of cervical cancer trends 1985–2013 in all women and in Māori women. Cancer Causes Control. 2017; 28:1393–404.

22. Sasieni P, Castanon A, Cuzick J. Effectiveness of cervical screening with age: population based case-control study of prospectively recorded data. BMJ. 2009; 339.

23. Smith M, Canfell K. Impact of the Australian National Cervical Screening Program in women of different ages. The Medical Journal of Australia 2016; 205:359–64.

24. Ethnicity Data Protocols. 2017. Wellington, New Zealand: Ministry of Health. ^http://www.health.govt.nz/system/files/documents/publications/hiso-10001-2017-ethnicity-data-protocols-v2.pdf

25. Smith M, Walker R, Edwards S, Rumlee L, Yap S, Canfell K. Monitoring Reports 33–48. 1 January 2010–31 December 2017. 2018. National Cervical Screening Programme.

26. Innes C, Williman J, Simcock B, et al. Impact of human papillomavirus vaccination on rates of abnormal cervical cytology and histology in young New Zealand women. N Z Med J. 2020; 133:72–84.

27. Guan P, Howell-Jones R, Li N, et al. Human papillomavirus types in 115,789 HPV-positive women: a meta-analysis from cervical infection to cancer. Int J Cancer. 2012; 131.

28. Baandrup L, Munk C, Andersen KK, Junge J, Iftner T, Kjaer SK. HPV16 is associated with younger age in women with cervical intraepithelial neoplasia grade 2 and 3. Gynecol Oncol. 2012; 124.

29. Brotherton JM, Tabrizi SN, Garland SM. Does HPV type 16 or 18 prevalence in cervical intraepithelial neoplasia grade 3 lesions vary by age? An important issue for postvaccination surveillance. Future Microbiol. 2012; 7.

30. Innes CR, Sykes PH, Harker D, et al. Changes in human papillomavirus genotypes associated with cervical intraepithelial neoplasia grade 2 lesions in a cohort of young women (2013–2016). Papillomavirus Research. 2018; 6:77–82.

31. Berenson AB, Hirth JM, Chang M. Change in human papillomavirus prevalence among U.S. women aged 18–59 Years, 2009–2014. Obstet Gynecol. 2017; 130:693–701.

32. Cameron RL, Kavanagh K, Pan J, et al. Human papillomavirus prevalence and herd immunity after introduction of vaccination program, Scotland, 2009–2013. Emerg Infect Dis. 2016; 22:56–64.

33. Tabrizi SN, Brotherton JM, Kaldor JM, et al. Assessment of herd immunity and cross-protection after a human papillomavirus vaccination programme in Australia: a repeat cross-sectional study. Lancet Infect Dis. 2014; 14:958–66.

34. Kang YJ, Lewis H, Smith MA, et al. Pre-vaccination type-specific HPV prevalence in confirmed cervical high grade lesions in the Maori and non-Maori populations in New Zealand. BMC Infect Dis. 2015; 15:365.

35. National Cervical Screening Programme Coverage Report. 2020. Wellington, New Zealand: National Screening Unit; Access date: June, 2020. Available from:^http://minhealthnz.shinyapps.io/nsu-ncsp-coverage

36. Serrano B, Alemany L, Tous S, et al. Potential impact of a nine-valent vaccine in human papillomavirus related cervical disease. Infect Agent Cancer. 2012; 7:38–38.

Outcomes after ST-elevation myocardial infarction presentation to hospitals with or without a routine primary percutaneous coronary intervention service (ANZACS-QI 46)

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ABSTRACT

AIM: Primary percutaneous coronary intervention (PCI) is the optimal reperfusion strategy to manage ST-elevation myocardial infarction (STEMI). Where timely primary PCI cannot be achieved, an initial pharmacological reperfusion strategy is recommended with subsequent transfer to a PCI-capable hospital. The study aim was to assess STEMI outcomes according to the interventional capability of the New Zealand hospital to which patients initially present.

METHODS: Nine thousand four hundred and eighty-eight New Zealand patients, aged 20–79 years, admitted with STEMI to a public hospital were identified. Patients were categorised into three groups—metropolitan hospitals with all-hours access to primary PCI (routine primary PCI cohort), metropolitan hospitals without routine access to PCI, and rural hospitals. The primary outcome was all-cause mortality. Secondary outcomes were major adverse cardiac events (MACE) and major bleeding.

RESULTS: Invasive coronary angiography was more frequent in the routine primary PCI cohort compared to metropolitan hospitals without routine access to PCI and rural hospitals (90.6 vs 83.0 vs 85.0% respectively; p<0.001) and occurred more commonly on the day of admission (78.9 vs 28.7 vs 25.7% respectively; p<0.001). There were no differences in multivariable adjusted all-cause mortality, MACE or major bleeding between patients admitted to any of the hospital groupings.

CONCLUSION: Outcomes after STEMI in New Zealand are similar regardless of the interventional capability of the hospital where they first present.

When a patient presents with an ST-elevation myocardial infarction (STEMI), acute reperfusion therapy by either acute percutaneous coronary intervention (primary PCI) or fibrinolysis improves outcomes.[1,2] When it can be performed in a timely fashion primary PCI is the preferred approach;[3] however, in all other cases current guidelines from the European Society of Cardiology and the American Heart Association advocate that fibrinolysis followed by early PCI is the recommended strategy for STEMI patients with symptom onset less than 12 hours who cannot be transferred to undergo PCI within 120 minutes.[3,4] This recommendation is reflected in local guidelines from the New Zealand Branch of the Cardiac Society of Australia and New Zealand (CSANZ)[5] and the National Out-of-Hospital STEMI Pathway co-developed by the National Cardiac Network and New Zealand ambulance services.[6] The

CSANZ guidelines further note that all STEMI patients with successful reperfusion via fibrinolysis should be transferred to a PCI-capable hospital for rescue PCI if appropriate, or invasive coronary angiography ±PCI within 3–24 hours after fibrinolytic therapy.

There are 47 public hospitals that receive STEMI patients in New Zealand, but only nine of these have round-the-clock access to interventional cardiac catheterisation laboratories allowing participation in a routine primary PCI service.[7] When acute reperfusion is considered, each of the 38 remaining hospitals and their ambulance service, without local access to primary PCI, must decide whether to pursue a reperfusion strategy of primary PCI (via transfer to a PCI capable hospital without fibrinolysis), or an approach of fibrinolysis by pre-hospital providers or the in-hospital team, followed by a transfer to a PCI-capable hospital for further definitive PCI.[8] This is often termed the 'pharmaco-invasive' strategy.[9,10] These 38 remaining hospitals can be grouped according to their level of service provision into metropolitan hospitals without routine PCI, and rural hospitals. While two of the metropolitan hospitals, Tauranga and Nelson, without routine PCI have a "mixed" service with limited primary PCI availability on certain times and days of the week only, the others have no on-site interventional service. The hospitals without local PCI capability predominantly serve regional or rural communities.[11] Rural hospitals specifically differ from the metropolitan hospitals with or without routine access to PCI, in that they are predominately staffed by generalist doctors and nurses without any local specialist support[12–14] and have limited access to both basic and advanced diagnostic tests as well as resources such as acute cardiac care unit facilities.[15] The potential for both variation in clinical practice and varying thresholds for angiography referral, together with the greater delays to invasive coronary angiography in patients requiring transfer for angiography might adversely affect outcomes following STEMI in these patients.

The aim of this observational cohort study was to assess the outcomes for patients with STEMI according to whether they first present to a metropolitan hospital with a routine all-hours primary PCI service, a metropolitan hospital without routine primary PCI, or to a rural hospital.

Methods

Patient cohorts and data collection

The All New Zealand Acute Coronary Syndrome Quality Improvement (ANZACS-QI) programme is a clinician-led initiative which aims to advocate for appropriate management of acute coronary syndrome (ACS) patients and to close the gap between evidence-based treatment and daily clinical practice.[16] Data sources for the ANZACS-QI programme include the ANZACS-QI registry which collects an in-depth dataset on the limited subset of ACS patients who have a coronary angiogram (approximately 60% of all ACS patients nationwide), and the National administrative datasets which collects limited data for all hospitalisations for ACS and its sub-types recorded using the International Statistical Classification of Diseases and Related Health Problem (ICD10) coding as well as coronary procedure codes. A principle strength of the National datasets are that they collect standardised demographic and clinical data for all New Zealand residents who are admitted to public hospitals.[16] To report the burden of non-cardiac comorbidity, we modified the Charlson comorbidity index[17] by excluding congestive heart failure. Data from the ANZACS-QI registry has been used to validate the accuracy of the National administrative dataset ACS sub-types (STEMI, NSTEMI, UA) and procedure codes.[18]From the National datasets, we identified all confirmed STEMI cases in New Zealand resident patients between 20–79 years of age who presented to a New Zealand public hospital between November 2011–November 2016. Both primary and secondary STEMI diagnostic codes were used. For this analysis we used only the first admission with a STEMI during the time period. This search method was validated against the ANZACS-QI registry to ensure accuracy.[18] Post-STEMI mortality, hospitalisation and medication dispensing data were individually linked from mortality and pharmaceutical collections.[19] Secondary prevention medications dispensed within three months of hospital

discharge are reported. Appendix Table 1 shows the types of data collected in these respective databases.

Patients who present with STEMI can be rapidly transferred between hospitals to undergo appropriate management. We thus applied a previously validated process to "bundle" ACS hospitalisations to ensure a temporally continuous admission under a single index admission episode of care.[20] Patients were divided depending on the local STEMI services provided by the hospital that first received the patient.

Patients 80 years and older were excluded. They are a heterogenous group that contribute to a minority of cases (6.1–6.5%), often have comorbidity requiring more individualised treatment decisions, and have disproportionately high rates of mortality (19.8–75%).[21–25]

Hospital cohorts

Hospitals were divided into three different cohorts with differing STEMI management policies. They were—metropolitan hospitals with routine all-hours access to primary PCI service for STEMI ("routine primary PCI" cohort), metropolitan hospitals that do not provide a routine primary PCI service ("metropolitan without routine PCI" cohort) and mostly provide a pharmaco-invasive strategy (two of these hospitals, Tauranga and Nelson, provide primary PCI at certain times and days of the week) and rural hospitals that also predominately pursue a pharmaco-invasive strategy. These hospital groupings are supported by ANZACS-QI registry data over a similar time period that confirms the dominant management strategies for each cohort (Appendix Table 2). A list of New Zealand hospitals with and without access to routine all-hours primary PCI services are listed in Appendix Table 3.

STEMI management pathways

Patients were managed according to prevailing guidelines.[5,6] Patients who presented with STEMI who were clinically eligible for acute reperfusion therapy received either primary PCI or fibrinolysis. Those with a delayed presentation of greater than 12 hours of symptoms, or in whom acute reperfusion was considered clinically inappropriate due to comorbidities received medical therapy only. Those undergoing primary PCI received a loading dose of anti-platelet medications and proceeded to the cardiac catheterisation laboratory for invasive coronary angiography and PCI. Patients undergoing the pharmaco-invasive approach typically received a loading dose of aspirin or another anti-platelet agent and received pharmacological fibrinolysis with bolus intravenous tenecteplase. Patients were then routinely transferred to another hospital with PCI capabilities in order to receive an early invasive coronary angiography and PCI if appropriate. Patients who underwent fibrinolysis which resulted in less than 50% resolution of the elevated ST segment at 60 minutes after fibrinolysis, or recurrence of ST elevation, or ongoing ischemic symptoms, or continuing haemodynamic instability were defined as failed fibrinolysis and were urgently transferred for rescue PCI.

Clinical end points and definitions

Outcomes were available from hospital admission to the end of 2017, to ensure each patient had a minimum possible follow-up of one year. The primary outcome measured was all-cause mortality. The two secondary outcomes were 1) a composite of all major adverse cardiac events (MACE), comprising of a composite of all-cause mortality, myocardial re-infarction, ischaemic or haemorrhagic stroke and new heart failure, and 2) the rate of fatal and non-fatal major bleeding.

Re-infarction, ischaemic stroke, hemorrhagic stroke and heart failure were defined by their respective ICD-10 definitions. Fatal and non-fatal major bleeding were defined by the ICD-10 code for "fatal bleeding", a primary ICD-10 code for bleeding or a secondary ICD-10 code for bleeding requiring transfusion.

Statistical analysis

Categorical variables were summarised as frequency and percentage. Pearson's chi-square test was used to compare different types of hospitals. Continuous variables were presented as mean and standard deviation (SD) and/or median with interquartile range (IQR), and the comparisons between types of hospitals were done using nonparametric Mann-Whitney *U* test as the continuous data was not normally distributed.

Cox proportional hazard regression models were constructed to estimate the hazard ratios and 95% confidence interval for the

outcomes after ensuring that the assumption of proportional hazards was met.

All P-values reported were two tailed and a P-value <0.05 was considered significant. Data were analysed using SAS statistical package, version 9.4 (SAS Institute, Cary, NIC). Outcomes were displayed using Kaplan-Meier survival curves using R Studio.

Ethics approval

ANZACS-QI is part of the wider Vascular Informatics Using Epidemiology and the Web (VIEW) study. The VIEW study was approved by the Northern Region Ethics Committee in 2003 (AKY/03/12/314), with subsequent amendments to include the ANZACS-QI registries, and with annual approvals by the National Multi-region Ethics Committee since 2007 (MEC07/19/EXP).

Results

We identified 13,265 records from patients that presented with a STEMI to a New Zealand hospital between 1 November 2011 to 30 November 2016. Of these, 201 records were non-New Zealand residents, 39 had missing socioeconomic data and 2,955 were outside the stated age bracket of 20–79 years old. Five hundred and eighty-two records were for patients identified to have presented with a repeated STEMI episode during the study period. Thus, a total of 9,488 eligible patients participated in the study.

Six thousand one hundred and seventy-nine participants first presented to a metropolitan hospital providing a routine all-hours primary PCI service (routine primary PCI cohort), 2,801 participants first presented to a metropolitan hospital that does not provide a routine primary PCI service (metropolitan without routine PCI cohort) and 508 participants first presented to a rural hospital that does not provide a routine primary PCI service (rural hospital cohort).

Baseline characteristics (Table 1)

Patients presenting to the metropolitan without routine PCI and rural hospital cohort were older, more likely to be female and of Māori ethnicity than patients presenting to a routine primary PCI hospital.

The hospitals without access to a routine primary PCI service received a larger proportion of patients who lived in socioeconomically deprived areas. 32.0% of patients in the metropolitan without routine PCI cohort and 28.4% patients in the rural hospital cohort were from the most deprived quintile (NZ Dep13, 9–10) compared to 22.8% of patients in the routine primary PCI cohort. The reciprocal was also true where a larger proportion of patients from the most affluent quintile (NZDep13, 1–2) were initially seen in hospitals of the routine primary PCI cohort.

Prior cardiovascular disease, myocardial infarction, congestive heart failure, PCI and the modified non-cardiac Charlson comorbidity score[17] were comparable among the three hospital cohorts. The prevalence of prior CABG was slightly higher within the metropolitan without routine PCI cohort (p=0.045).

Coronary procedures and timing (Table 2)

Eight thousand three hundred and fifty-two (88%) of the 9,488 study participants proceeded for invasive coronary angiography. More patients in the routine primary PCI hospital cohort had angiography during their admission (90.6%) compared to the metropolitan hospital without routine PCI cohort (83.0%), and rural hospital cohort (85.0%) (p<0.001). Of those that proceeded for invasive coronary angiography during the index admission, 91.6% of patients in the routine primary PCI cohort received PCI or CABG surgery during their index admission compared to 80.0% of patients in the metropolitan hospital without routine PCI cohort, and 79.6% in the rural hospital cohort (p<0.001).

Of those undergoing angiography, more patients presenting to a routine primary PCI hospital (78.9%) received invasive coronary angiography within the first 24 hours of presentation than those to a metropolitan hospital without routine PCI (28.7%), or a rural hospital (25.7%) (p<0.001). Only 13.2% of patients presenting to a routine primary PCI hospital had angiography 24–72 hours after presentation compared to 42.0% of those presenting to a metropolitan without routine PCI hospital, and 46.3% of those to a rural hospital. 7.9% of patients in the routine PCI hospital cohort had invasive coronary angiography beyond 72 hours after presentation compared to 29.3% and 28.0% of the metropolitan without routine PCI, and rural hospital cohorts.

Table 1: Baseline patient characteristics.

	Total (N=9,488) (% of total)	**Routine primary PCI** (n=6,179) (% of group)	**Metropolitan without routine PCI** (n=2,801) (% of group)	**Rural hospital** (n=508) (% of group)	**P-value**
Age group, years					<.001
<40	270 (2.9)	203 (3.3)	59 (2.1)	8 (1.6)	
40–<50	1,130 (11.9)	786 (12.7)	291 (10.4)	53 (10.4)	
50–<60	2,492 (26.3)	1,679 (27.2)	694 (24.8)	119 (23.4)	
60–<70	2,976 (31.4)	1,929 (31.2)	889 (31.7)	158 (31.1)	
70–<80	2,620 (27.6)	1,582 (25.6)	868 (31.0)	170 (33.5)	
Age, years					<.001
Mean (SD)	61.5 (10.9)	60.9 (11.0)	62.6 (10.7)	63.2 (10.6)	
Median (IQR)	63 (54–70)	62 (53–70)	64 (55–71)	64 (55.5–72)	
Sex					<.001
Male	6,898 (72.7)	4,611 (74.6)	1,930 (68.9)	357 (70.3)	
Female	2,590 (27.3)	1,568 (25.4)	871 (31.1)	151 (29.7)	
Ethnicity					<.001
Māori	1,198 (12.6)	655 (10.6)	480 (17.1)	63 (12.4)	
Pacific	467 (4.9)	425 (6.9)	38 (1.4)	4 (0.8)	
Indian	416 (4.4)	392 (6.3)	20 (0.7)	4 (0.8)	
Asian	237 (2.5)	216 (3.5)	18 (0.6)	3 (0.6)	
European	7,061 (74.4)	4,400 (71.2)	2,235 (79.8)	426 (83.9)	
Other/unknown	109 (1.1)	91 (1.5)	10 (0.4)	8 (1.6)	
NZ Dep13*					<.001
1–2	1,477 (15.6)	1,185 (19.2)	253 (9.0)	39 (7.7)	
3–4	1,579 (16.6)	1,058 (17.1)	446 (15.9)	75 (14.8)	
5–6	1,796 (18.9)	1,176 (19.0)	513 (18.3)	107 (21.1)	
7–8	2,188 (23.1)	1,352 (21.9)	693 (24.7)	143 (28.2)	
9–10	2,448 (25.8)	1,408 (22.8)	896 (32.0)	144 (28.4)	
Coexisting conditions					
Prior CVD	1,992 (21.0)	1,262 (20.4)	625 (22.3)	105 (20.7)	0.124
Prior MI	1,076 (11.3)	683 (11.1)	336 (12.0)	57 (11.2)	0.426
Prior CHF or use of loop diuretics in the previous six months	687 (7.2)	434 (7.0)	212 (7.6)	41 (8.1)	0.496
Modified non-cardiac Charlson score					0.385
0	7,895 (83.2)	5,153 (83.4)	2,328 (83.1)	414 (81.5)	
1–2	1,217 (12.8)	774 (12.5)	374 (13.4)	69 (13.6)	
3+	376 (4.0)	252 (4.1)	99 (3.5)	25 (4.9)	
Previous cardiac procedure					
Prior CABG	35 (0.4)	17 (0.3)	17 (0.6)	1 (0.2)	0.045
Prior PCI	263 (2.8)	161 (2.6)	93 (3.3)	9 (1.8)	0.060

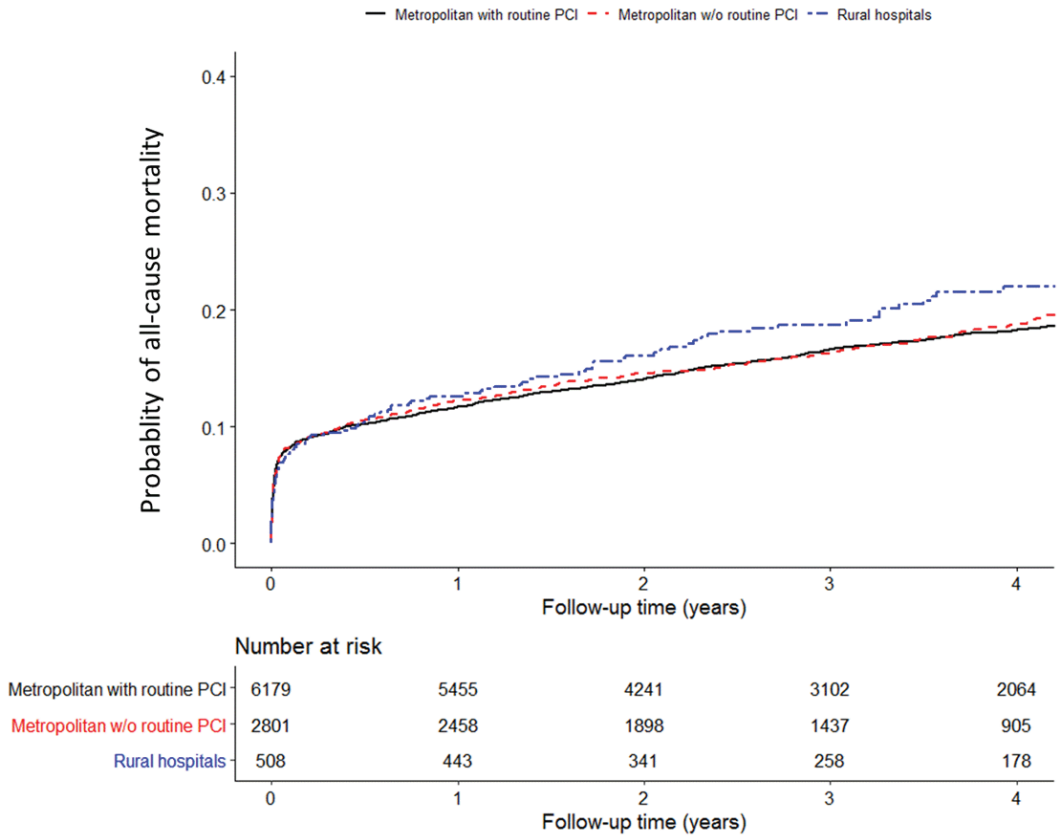
SD = standard deviation; IQR = inter-quartile range; NZ Dep 13 = New Zealand Index of Deprivation 2013 (* = higher score = more deprived); CVD = cardiovascular disease; MI = myocardial infarction; CHF = congestive heart failure; CABG = coronary artery bypass surgery; PCI = percutaneous coronary intervention.

Table 2: Angiography, percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG) time intervals and rates of medical therapy.

	Total (N=9,488) (% of total)	**Routine primary PCI** (n=6,179) (% of group)	**Metropolitan without routine PCI** (n=2,801) (% of group)	**Rural hospital** (n=508) (% of group)	**P-value**
Angiogram during index admission	8,352 (88.0)	5,595 (90.6)	2,325 (83.0)	432 (85.0)	<.001
PCI during index admission	6,780 (71.5)	4,807 (77.8)	1,667 (59.5)	306 (60.2)	<.001
CABG during index admission	551 (5.8)	320 (5.2)	193 (6.9)	38 (7.5)	0.002
Secondary prevention medication post discharge	n=8,775	n=5,708	n=2,595	n=472	
Aspirin	8,252 (94.0)	5,403 (94.7)	2,404 (92.6)	445 (94.3)	0.002
$P2Y_{12}$ inhibitor	7,250 (82.6)	4,772 (83.6)	2,110 (81.3)	368 (78.0)	0.001
DAPT	7,029 (80.1)	4,632 (81.1)	2,036 (78.5)	361 (76.5)	0.002
Statin	8,231 (93.8)	5,363 (94.0)	2,432 (93.7)	436 (92.4)	0.383
ACEi/ARB	7,086 (80.8)	4,680 (82.0)	2,047 (78.9)	359 (76.1)	<0.001
Beta-blocker	7,805 (89.0)	5,059 (88.6)	2,325 (89.6)	421 (89.2)	0.423
Spironolactone	596 (6.8)	382 (6.7)	175 (6.7)	39 (8.3)	0.425
Hemorrhagic stroke during index admission	37 (0.39)	14 (0.23)	21 (0.75)	2 (0.39)	0.001
Subgroup analysis of those who underwent coronary angiogram	**Total** (N=8,352) (% of total)	(n=5,595) (% of group)	(n=2,325) (% of group)	(n=432) (% of group)	
Admission to angiogram (days)					<.001
0	5,190 (62.1)	4,413 (78.9)	666 (28.7)	111 (25.7)	
1–2	1,913 (22.9)	737 (13.2)	976 (42.0)	200 (46.3)	
3+	1,249 (15.0)	445 (7.9)	683 (29.3)	121 (28.0)	
Subgroup analysis of those who underwent inpatient PCI	**Total** (N=6,780) (% of total)	(n=4,807) (% of group)	(n=1,667) (% of group)	(n=306) (% of group)	
Admission to PCI (days)					<.001
0	4,604 (67.9)	3,983 (82.9)	528 (31.7)	93 (30.4)	
1–2	1,336 (19.7)	532 (11.1)	667 (40.0)	137 (44.8)	
3+	840 (12.4)	292 (6.1)	472 (28.3)	76 (24.8)	
Subgroup analysis of those who underwent inpatient CABG	**Total** (N=551) (% of total)	(n=320) (% of group)	(n=193) (% of group)	(n=38) (% of group)	
Admission to CABG (days)					<.001
0	23 (4.2)	21 (6.6)	2 (1.0)	0 (0)	
1–2	56 (10.2)	44 (13.8)	9 (4.7)	3 (7.9)	
3+	472 (85.7)	255 (79.7)	182 (94.3)	35 (92.1)	

PCI = percutaneous coronary intervention; CABG = coronary artery bypass surgery; $P2Y_{12}$ inhibitor = $P2Y_{12}$ receptor blocker (clopidogrel, ticagrelor, prasugrel); DAPT= dual antiplatelet therapy; ACEi = angiotensin-converting-enzyme inhibitors, ARB = angiotensin II receptor blockers.

Figure 1: All-cause mortality.



Secondary prevention medications dispensed post-discharge (Table 2)

The dispensing of secondary prevention medications was high across groups but with a slightly lower use of anti-platelet agents and ACEI/ARBs in the non-routine primary PCI groups.

Outcomes (Figures 1,2 and Table 3)

The average follow-up duration for all-cause mortality, was 3.03 years. The all-cause mortality, MACE and major bleeding outcomes are shown using Kaplan Maier survival plots in Figures 1 and 2. The Kaplan-Maier mortality at one and three years was 11.7% and 16.6% for patients presenting to the metropolitan with routine PCI cohort, 12.2% and 16.2% for metropolitan hospitals without routine PCI and 12.6% and 18.7% for rural hospitals.

The Kaplan-Maier MACE at one and three years was 22.0% and 30.1% for patients presenting to the metropolitan with routine PCI cohort, 24.4% and 31.9% for metropolitan hospitals without routine PCI and 21.5% and 31.0% for rural hospitals.

The Kaplan-Maier major bleeding at one and three years was 6.9% and 8.6% for patients presenting to the metropolitan with routine PCI cohort, 7.5% and 9.1% for metropolitan hospitals without routine PCI and 5.5% and 7.1% for rural hospitals.

After adjusting for age, sex, ethnicity, deprivation score, modified Charlson score and prior CVD, there were no differences in outcomes for patients admitted to each of the three hospital groups (Table 3).

Discussion

This nationwide, real-world study describes the interventional management and outcomes for all hospitalised STEMI patients in New Zealand according to the interventional capability of the hospitals to which they were first admitted. Patients presenting first to a routine primary PCI capable hospital, as opposed to a metropolitan without routine PCI, or a rural hospital, received slightly higher overall rates of coronary angiography and revascularisation and received these more quickly. Of those treated with PCI who presented

Figure 2: A) All-cause mortality/non-fatal MI/HF/stroke (MACE); B) fatal/non-fatal bleeding.

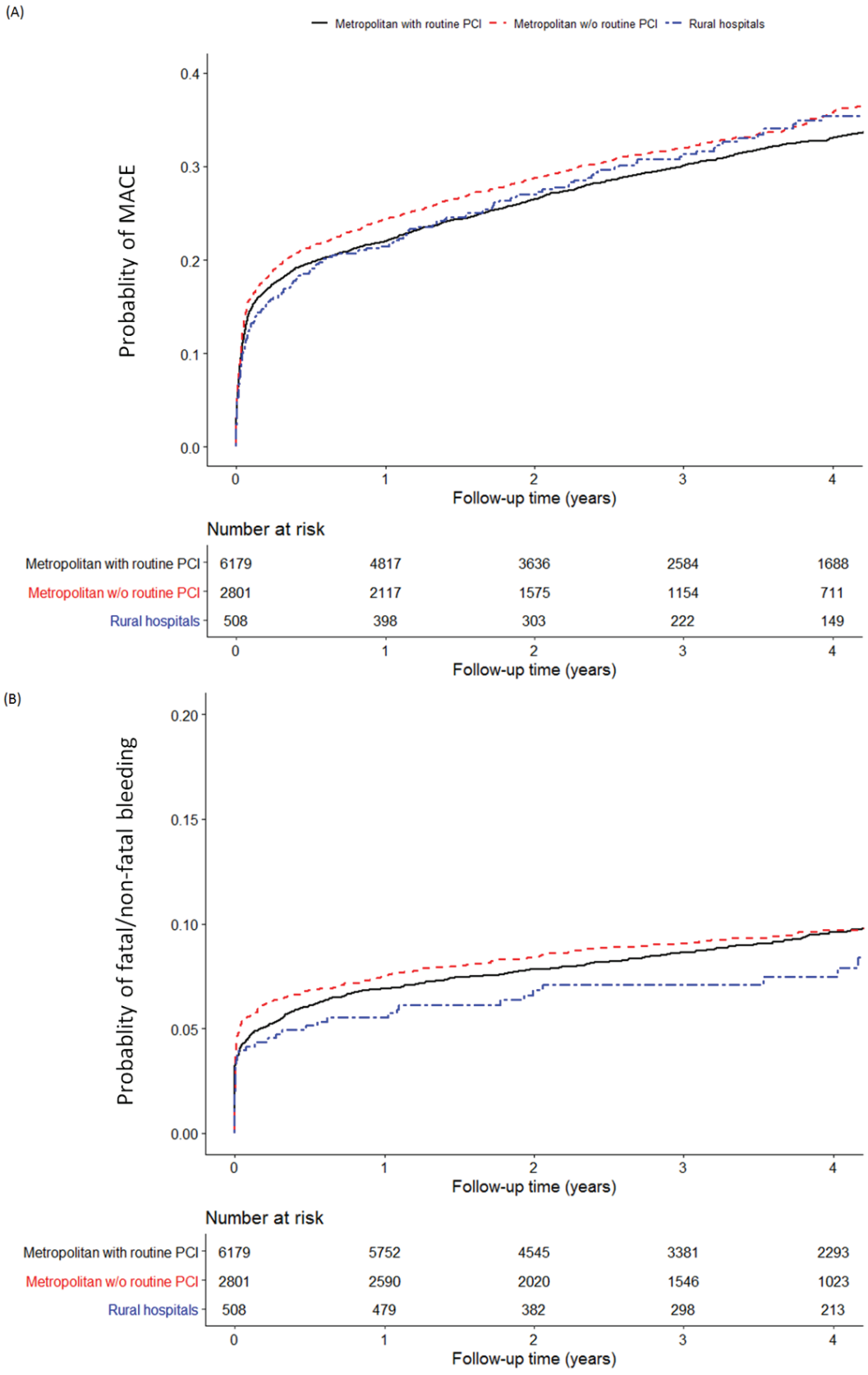


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Table 3: Outcomes.

	All-cause mortality		
	# events/N	**HR (95% CI)**	**P-value**
Unadjusted			
Routine primary PCI	1,084/6,179	Reference	-
Metropolitan without routine PCI	514/2,801	1.05 (0.95–1.17)	0.366
Rural hospital	103/508	1.16 (0.95–1.42)	0.157
Adjusted			
Routine primary PCI		Reference	-
Metropolitan without routine PCI		0.96 (0.86–1.06)	0.403
Rural hospital		0.99 (0.81–1.21)	0.903
	All-cause mortality/non-fatal MI/HF/stroke		
	# events/N	**HR (95% CI)**	**P-value**
Unadjusted			
Routine primary PCI	1,942/6,179	Reference	-
Metropolitan without routine PCI	951/2,801	1.10 (1.02–1.19)	0.015
Rural hospital	169/508	1.05 (0.89–1.22)	0.583
Adjusted			
Routine primary PCI		Reference	-
Metropolitan without routine PCI		1.01 (0.93–1.10)	0.786
Rural hospital		0.92 (0.79–1.08)	0.308
	Fatal/non-fatal bleeding		
	# events/N	**HR (95% CI)**	**P-value**
Unadjusted			
Routine primary PCI	561/6,179	Reference	-
Metropolitan without routine PCI	266/2,801	1.05 (0.91–1.21)	0.517
Rural hospital	38/508	0.81 (0.58–1.13)	0.208
Adjusted			
Routine primary PCI		Reference	-
Metropolitan without routine PCI		1.01 (0.86–1.17)	0.943
Rural hospital		0.77 (0.55–1.07)	0.123

Adjusted by age (continuous), sex, ethnicity, NZDep13, Modified Charlson non-cardiac comorbidities, prior CVD.
events = number of events; CI = confidence interval; PCI = percutaneous coronary intervention; MI = myocardial infarction; HF = congestive heart failure.

first to a routine primary PCI capable hospital, nearly four in five received PCI on the day of admission, compared to a quarter of those presenting to other hospitals. Despite the differences in management, all clinical outcomes over a mean of three years follow-up did not differ between patients presenting to each of the three hospital groupings.

Efficacy of reperfusion strategies

This finding is congruent with previous literature examining different STEMI management strategies. Patients who are reperfusion candidates presenting with STEMI to New Zealand hospitals providing all-hours routine primary PCI services proceed for invasive coronary angiography with an aim for performing primary PCI.

Reperfusion candidates presenting with STEMI to hospitals without local PCI facilities are transferred to receive primary PCI where possible. However, in most cases a transfer cannot be completed within an appropriate time period (120 minutes) and patients then are administered fibrinolytic therapy as the immediate reperfusion strategy prior to transfer to a hospital with PCI facilities for invasive coronary angiography ± PCI. This strategy is commonly called the pharmaco-invasive approach.[9,10]

Over the preceding decades, it has been established that primary PCI delivers superior outcomes compared to pharmacological fibrinolysis monotherapy.[26] However, despite the delay in accessing PCI inherent within the pharmaco-invasive approach, there is similar efficacy to primary PCI. In a registry study from the Mayo Clinic STEMI network comparing the rates of all-cause mortality between patients undergoing the pharmaco-invasive approach versus primary PCI, showed that the rates of early and late mortality were comparable between the two strategies.[27] In a study from the University of Ottawa Heart institute regional STEMI system which employs a policy of primary PCI for patients presenting within a 90km radius of the PCI centre and a pharmaco-invasive strategy for those outside this limit[28] displayed the rates of mortality, stroke or reinfarction were no different between the two strategies. The landmark Strategic Reperfusion Early After Myocardial Infarction (STREAM) trial[29] assigned STEMI patients to undergo primary PCI versus fibrinolysis with transfer to a PCI capable hospital for a coronary angiography within 6–24 hours. The rate of the 30-day primary endpoint, a composite of death, shock, CHF and reinfarction, was similar among both groups. Other studies have noted similar findings.[30–33] Lastly, a meta-analysis consisting of studies up to 2017 [34] have concluded there is no difference in short-term and long-term mortality between the two reperfusion strategies as long as symptom onset to device time in primary PCI did not exceed 200 minutes.

Timing of angiography

A prominent finding in our results is the delay in proceeding for angiography for patients presenting to rural and metropolitan hospitals with no routine PCI available. 28.7% of metropolitan hospitals without PCI, and 25.7% of the rural hospital cohort were able to access invasive angiography within 24 hours. This contrasts with 78.9% of patients who presented to hospitals providing routine primary PCI proceeding to invasive angiography within 24 hours.

The New Zealand Cardiac Clinical Network and the Ministry of Health recommend a "three-day door-to-catheter target" for all acute coronary syndrome (including unstable angina, NSTEMI and STEMI) admissions.[35] The Cardiac Society of Australia and New Zealand recommends all STEMI patients with successful reperfusion via fibrinolysis should be transferred to a PCI capable hospital for cardiac catherisation within 24 hours after fibrinolytic therapy.[5] While our study was unable to determine when the transfer to a PCI centre took place, it showed that only one-quarter of patients who present to a rural or metropolitan hospital with no routine primary PCI had angiography within this 24-hour window. This finding is similar to a nationwide audit in 2012 that showed 22% of patients presenting with STEMI to non-interventional hospitals received routine angiography within 24 hours.[20] Nearly one-third of patients who present to a rural or metropolitan hospital with no routine primary PCI were awaiting cardiac catheterisation more than 72 hours post-STEMI.

Delays in in receiving angiography primarily reflect shortfalls in processes to ensure early transfer of patients to an interventional hospital together with appropriate prioritisation on arrival. This has previously been noted in a New Zealand study which reported that patients are more likely to wait longer for cardiac catherisation in districts without interventional facilities after ACS.[35] New Zealand has a small and geographically dispersed population with smaller regional and rural centres. These hospitals do not have the concentration of healthcare resources and specialist care as seen in hospitals with a routine primary PCI service. Instead, metropolitan hospitals without routine primary PCI and rural hospitals are more likely to be served by general physicians in metropolitan hospitals or generalist rural hospital doctors in rural hospitals[13] leading to a range of inter-physician differences in the threshold for

referrals, delays in transfer and proceeding for cardiac catherisation. Reduced rates of investigations (eg, ETT, CT scans) for geographically isolated areas has been demonstrated previously in New Zealand.[36,37] The optimal management of STEMI involves prompt inter-disciplinary and inter-regional co-operation and co-ordination among frontline ambulance staff, helicopter crews, STEMI co-ordinators, rural nurses, general practitioners, general and emergency physicians, cardiac catherisation laboratory staff and interventional cardiologists. Any misaligned communication or expectations within this chain of care results in prolonged times from first medical contact to crossing of the coronary occlusion or stenosis with a wire. It has been recognised that it is essential to implement standardised pathways for management to reduce uncertainty and inequality nationwide. This has resulted in the New Zealand out-of-hospital STEMI pathway in 2016.[6]

Differences in rates of angiography and revascularisation

There were lower rates of invasive coronary angiography in patients presenting to hospitals without routine primary PCI services. Patients in these cohorts were slightly less likely to receive invasive coronary angiography during their admission, with 83–85% proceeding for cardiac catheterisation compared to 90.6% of patients presenting to hospitals providing a routine all-hours primary PCI service. This may be contributed by the different baseline characteristics of the three cohorts, in particular the older age of the non-routine PCI hospital cohorts. Among patients who did have a diagnostic angiogram, the gap for revascularisation was even greater; 91.6% of these patients in the routine primary PCI group received revascularisation by either PCI or CABG during their index admission compared to 80.0% of patients in the metropolitan hospitals without routine PCI cohort and 79.6% in the rural hospital groups. This is likely in part due to a higher rate of non-obstructive coronary artery disease in those already treated with fibrinolytic therapy, which may have lysed the thrombus who therefore do not need revascularisation.[38]

Rural-urban differences

Although we have primarily created hospital cohorts based on STEMI management capabilities, these groups also represent a rural-urban divide. Hospitals providing routine primary PCI are located within the largest metropolitan centres, whereas hospitals of the non-routine PCI cohorts are in regional urban areas and rural communities. We previously reported that ≥80% of patients with STEMI who lived in predominantly rural district health boards (DHBs) received pharmaco-invasive therapy.[39] Patients who presented to metropolitan hospitals without routine PCI and rural hospitals were more likely to be older, female, from a more deprived socioeconomic quintile and of Māori or European ethnicity. After adjustment, there were no significant differences in outcomes between patients initially presenting to a rural compared with a routine primary PCI hospital. This is congruent with previous Australian[40] and Chinese studies[41] which found that there was no difference in mortality post STEMI between metropolitan and rural regions. This study shows that great progress has been made in New Zealand over the last two decades with a closing of the gap, especially for mortality, in outcomes and access to intervention between hospitals with PCI and those without.[42]It is a concern that patients presenting to non-primary PCI hospitals tended to have greater levels of socioeconomic deprivation. Delays in angiography associated with location of care and socioeconomic status have been demonstrated previously in a study in the US.[43] Most other studies have examined the broader topic of acute coronary syndrome, with varied findings. In Canada, patients who presented with ACS from non-metropolitan areas were less likely to receive cardiac catheterisation within one day and those from the lowest income area within non-metropolitan areas were less likely to have a coronary angiogram within seven days compared to their more affluent counterparts living in metropolitan areas.[44] Also, women from poor-income neighbourhoods were associated with a poorer odds of having coronary angiography and a higher mortality within 30 days.[45] In contrast, a

study in Australia found that socioeconomic status was not related to differences in having coronary angiography after ACS.[46]

Safety outcomes

There was no statistically significant difference in major bleeding between the three cohorts. There was a small, but significant, increase in haemorrhagic stroke within the metropolitan without routine PCI cohort (Table 2). The literature is varied when examining the safety aspects of the pharmaco-invasive strategy. Two studies[28,29] demonstrated increased rates of haemorrhagic stroke with the pharmaco-invasive method. This increased safety risk was found to be confined to patients greater than 75 years of age in a sub-analysis of one study.[29] This has led to the recommendation to use lower dose bolus fibrinolytic therapy in patients greater than 75 years of age.

Limitations

We compared practice and outcomes according to which three types of hospital a patient initially presented to. Hospitals were categorised according to their levels of availability of primary PCI services and access to specialist services. Although each hospital has defined reperfusion policies, STEMI management is not necessarily consistent for each hospital within these three groups. ANZACS-QI data shows that 10.5–14% of patients who present to hospitals that do not provide a routine primary PCI service do indeed proceed for primary PCI (Appendix Table 2). The most prominent example of this are a subset of hospitals such as Tauranga or Nelson Hospital who offer primary PCI within limited hours or within the limits of staffing availability. In some regions a small number of patients are flown directly to a PCI capable centre for primary PCI and so may bypass their local hospital. In addition to the effect of acute reperfusion therapy, outcomes are also likely to be dependent on the other components of the STEMI pathway management including pharmacological management, pre-hospital vs in-hospital fibrinolysis and the availability of appropriate and timely transfer to tertiary centres. These may also vary between hospitals within our three service groups. Furthermore, the national datasets used in this study do not reliably record whether patients were treated with acute reperfusion as primary PCI and fibrinolysis are not coded. Primary PCI cases and those receiving fibrinolysis and subsequent angiography can be identified using the separate ANZACS-QI registry[16,38] but there is no data source which reliably identifies patients treated with fibrinolysis who do not proceed to an angiogram. A consequence of this is that for hospitals without routine primary PCI availability we cannot reliably report the proportion of STEMI patients who do not receive acute reperfusion therapy. We were also unable to capture any patient who died without reaching a hospital or who died after STEMI discharge outside of New Zealand.

Conclusion

Our study has demonstrated that patients who present with STEMI to hospitals without a routine primary PCI service are less likely to receive coronary angiography, wait longer for angiography and are less likely to receive coronary revascularisation. This is likely due to the geographical isolation of these hospitals from PCI facilities that results in the differences in STEMI management, however there may be other factors that influence the timing of angiography and primary PCI. Despite differences in management, we did not find any differences in mortality, MACE or major bleeding rates following STEMI between the three different cohorts of hospitals. This is a tribute to the current systems of STEMI care including timely pharmacological reperfusion, appropriate bypass of selected non-interventional hospitals with transfer of patients to interventional centres and use of secondary prevention medication.

This study adds to the growing body of international evidence that the pharmaco-invasive approach is a viable method in STEMI patients who present to hospitals without PCI capabilities. In the New Zealand context, this may mean future resources could be efficiently used in further optimising existing STEMI networks.

Appendix

Appendix Table 1: Outline of data collected for the ANZACS-QI national Routine Information cohort.[16]

Name of dataset and data contained	Variables
National Minimum Dataset[47]	**Admission-related data:** date of admission, date of discharge, ICD-coded discharge diagnoses, ICD-coded procedural diagnoses (including angiography, PCI, CABG), DHB of domicile. **Demographic data:** age at admission, sex, ethnicity, deprivation quintile, domicile, rurality of residence. **Previous hospitalisations:** previous ACS and ischaemic heart disease admissions; Charlson comorbidities (MI, peripheral vascular disease heart failure chronic obstructive pulmonary disease, connective tissue disease, ulcers, dementia, cerebrovascular disease, hemiplegia, diabetes, liver disease, renal disease, neoplasms, AIDS); total Charlson comorbidity score.
Pharmaceutical Collection[48]	Government-subsidised medications dispensing claims from community pharmacies.
Mortality Collection[49]	Date of death and ICD-coded underlying and contributing causes of death.

ICD = International Statistical Classification of Diseases and Related Health Problem, PCI = percutaneous coronary intervention, CABG = coronary artery bypass grafting, DHB = District Health Board, ACS = acute coronary syndrome, MI = myocardial infarction, AIDS = acquired immune deficiency syndrome.

Appendix Table 2: Proportion of STEMI reperfusion strategies between hospital groupings. Data collected from the ANZACS-QI registry between 1 September 2013–30 November 2016.

	Routine primary PCI (n=3,520) (% of group)	**Metropolitan without routine PCI** (n=1,785) (% of group)	**Rural hospital** (n=430) (% of group)
Primary PCI	2,704 (77.6%)	185 (10.5%)	60 (14.0%)
Fibrinolysis	94 (2.70%)	1,122 (63.6%)	253 (59.1%)
No reperfusion	688 (19.7%)	456 (25.9%)	115 (26.9%)

PCI = percutaneous coronary intervention.

Appendix Table 3: List of New Zealand public hospitals that receive STEMI patients, with and without access to cardiac catherisation services.

Hospitals participating in a routine primary PCI service	Metropolitan hospitals with no routine primary PCI service	Rural hospitals
Auckland City Hospital	Blenheim/Wairau Hospital	Ashburton Hospital
Christchurch Hospital	Gisborne Hospital	Bay of Islands Hospital
Dunedin Hospital	Hawke's Bay Hospital	Chatham Islands Hospital
Hutt Hospital	Masterton/Wairarapa Hospital	Clutha Health First
Middlemore Hospital	Nelson Hospital	Dargaville Hospital
North Shore Hospital	Palmerston North Hospital	Dunstan Hospital
Waikato Hospital	Rotorua Hospital	Golden Bay Hospital
Waitakere Hospital	Southland Hospital	Gore Hospital
Wellington Hospital	Taranaki Base Hospital	Grey Base Hospital
	Tauranga Hospital	Hawera Hospital
	Timaru Hospital	Kaikoura Hospital
	Whakatane Hospital	Kaitaia Hospital
	Whanganui Hospital	Lakes District Hospital
	Whangarei Hospital	Maniototo Hospital
		Oamaru Hospital
		Rawene/Hokianga Hospital
		Taumarunui Hospital
		Taupo Hospital
		Te Kuiti Hospital
		Te Puia Springs Hospital
		Thames Hospital
		Tokoroa Hospital
		Wairoa Hospital
		Westport/Buller Hospital

PCI = percutaneous coronary intervention.

Competing interests:
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REFERENCES:

1. Vogel B, Claessen BE, Arnold SV, et al. ST-segment elevation myocardial infarction. Nat Rev Dis Primers. 2019; 5:39.
2. Reed GW, Rossi JE, Cannon CP. Acute myocardial infarction. Lancet. 2017; 389:197–210.
3. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2018; 39:119–177.
4. O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2013; 127:e362–425.
5. ST-Elevation Myocardial Infarction Guidelines Group; New Zealand Branch of Cardiac Society of Australia and New Zealand. ST-elevation myocardial infarction: New Zealand Management Guidelines, 2013. N Z Med J. 2013; 126:127–64.
6. New Zealand out-of-hospital STEMI pathway. New Zealand National Cardiac Network, 2018.
7. Kerr A, Williams MJ, White H, et al. 30-day mortality after percutaneous coronary intervention in New Zealand public hospitals (ANZACS-QI 18). N Z Med J. 2017; 130:54–63.
8. White HD. Systems of care: need for hub-and-spoke systems for both primary and systematic percutaneous coronary intervention after fibrinolysis. Circulation. 2008; 118:219–22.
9. Di Mario C, Dudek D, Piscione F, et al. Immediate angioplasty versus standard therapy with rescue angioplasty after thrombolysis in the Combined Abciximab REteplase Stent Study in Acute Myocardial Infarction (CARESS-in-AMI): an open, prospective, randomised, multicentre trial. Lancet. 2008; 371:559–68.
10. Bøhmer E, Hoffmann P, Abdelnoor M, et al. Efficacy and safety of immediate angioplasty versus ischemia-guided management after thrombolysis in acute myocardial infarction in areas with very long transfer distances results of the NORDISTEMI (NORwegian study on DIstrict treatment of ST-elevation myocardial infarction). J Am Coll Cardiol. 2010; 55:102–10.
11. Kerr A. Acute Reperfusion for STEMI Patients in New Zealand Hospitals July 2015–June 2017 - an ANZACS-QI Registry Report. [In Press].
12. Nixon G. Rural generalism: the New Zealand way. Address for the Eric Elder Medal. RNZCGP Conference July 2017. J Prim Health Care. 2018; 10:102–105.
13. Lawrenson R, Reid J, Nixon G, Laurenson A. The New Zealand Rural Hospital Doctors Workforce Survey 2015. N Z Med J. 2016; 129:9–16.
14. Blattner K, Stokes T, Nixon G. A scope of practice that works 'out here': exploring the effects of a changing medical regulatory environment on a rural New Zealand health service. Rural Remote Health. 2019; 19:5442.
15. Miller R, Stokes T, Nixon G. Point-of-care troponin use in New Zealand rural hospitals: a national survey. N Z Med J. 2019; 132:25–37.
16. Kerr A, Williams MJ, White H, et al. The All New Zealand Acute Coronary Syndrome Quality Improvement Programme: Implementation, Methodology and Cohorts (ANZACS-QI 9). N Z Med J. 2016; 129:23–36.
17. Quan H, Li B, Couris CM, et al. Updating and validating the Charlson comorbidity index and score for risk adjustment in hospital discharge abstracts using data from 6 countries. Am J Epidemiol. 2011; 173:676–82.
18. Kerr AJ, Lee M, Jiang Y, et al. High level of capture of coronary intervention and associated acute coronary syndromes in the all New Zealand acute coronary syndrome quality improvement cardiac registry and excellent agreement with national administrative datasets (ANZACS-QI 25). N Z Med J. 2019; 132:19–29.
19. Kerr A, Exeter D, Hanham G, et al. Effect of age, gender, ethnicity, socioeconomic status and region on dispensing of CVD secondary prevention medication in New Zealand: the Atlas of Health Care Variation CVD cohort (VIEW-1). N Z Med J. 2014; 127:39–69.
20. Ellis C, Gamble G, Devlin G, et al. The management of acute coronary syndrome patients across New Zealand in 2012: results of a third comprehensive nationwide audit and observations of current interventional care. N Z Med J. 2013; 126:36–68.
21. Turk J, Fourny M, Yayehd K, et al. Age-Related Differences in Reperfusion Therapy

and Outcomes for ST-Segment Elevation Myocardial Infarction. J Am Geriatr Soc. 2018; 66:1325–1331.

22. Yudi MB, Jones N, Fernando D, et al. Management of Patients Aged ≥85 Years With ST-Elevation Myocardial Infarction. Am J Cardiol. 2016; 118:44–8.

23. Khera S, Kolte D, Palaniswamy C, et al. ST-elevation myocardial infarction in the elderly--temporal trends in incidence, utilization of percutaneous coronary intervention and outcomes in the United States. Int J Cardiol. 2013; 168:3683–90.

24. Forman DE, Chen AY, Wiviott SD, et al. Comparison of outcomes in patients aged <75, 75 to 84, and ≥85 years with ST-elevation myocardial infarction (from the ACTION Registry-GWTG). Am J Cardiol. 2010; 106:1382–8.

25. Yudi MB, Hamilton G, Farouque O, et al. Trends and Impact of Door-to-Balloon Time on Clinical Outcomes in Patients Aged <75, 75 to 84, and ≥85 Years With ST-Elevation Myocardial Infarction. Am J Cardiol. 2017; 120:1245–1253.

26. Keeley EC, Boura JA, Grines CL. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet. 2003; 361:13–20.

27. Siontis KC, Barsness GW, Lennon RJ, et al. Pharmacoinvasive and Primary Percutaneous Coronary Intervention Strategies in ST-Elevation Myocardial Infarction (from the Mayo Clinic STEMI Network). Am J Cardiol. 2016; 117:1904–10.

28. Rashid MK, Guron N, Bernick J, et al. Safety and Efficacy of a Pharmacoinvasive Strategy in ST-Segment Elevation Myocardial Infarction: A Patient Population Study Comparing a Pharmacoinvasive Strategy With a Primary Percutaneous Coronary Intervention Strategy Within a Regional System. JACC Cardiovasc Interv. 2016; 9:2014–2020.

29. Armstrong PW, Gershlick AH, Goldstein P, et al. Fibrinolysis or primary PCI in ST-segment elevation myocardial infarction. N Engl J Med. 2013; 368:1379–87.

30. Larson DM, Duval S, Sharkey SW, et al. Safety and efficacy of a pharmaco-invasive reperfusion strategy in rural ST-elevation myocardial infarction patients with expected delays due to long-distance transfers. Eur Heart J. 2012; 33:1232–40.

31. Sim DS, Jeong MH, Ahn Y, et al. Pharmacoinvasive Strategy Versus Primary Percutaneous Coronary Intervention in Patients With ST-Segment-Elevation Myocardial Infarction: A Propensity Score-Matched Analysis. Circ Cardiovasc Interv. 2016; 9.

32. Pu J, Ding S, Ge H, et al. Efficacy and Safety of a Pharmaco-Invasive Strategy With Half-Dose Alteplase Versus Primary Angioplasty in ST-Segment-Elevation Myocardial Infarction: EARLY-MYO Trial (Early Routine Catheterization After Alteplase Fibrinolysis Versus Primary PCI in Acute ST-Segment-Elevation Myocardial Infarction). Circulation. 2017; 136:1462–1473.

33. Danchin N, Puymirat E, Steg PG, et al. Five-year survival in patients with ST-segment-elevation myocardial infarction according to modalities of reperfusion therapy: the French Registry on Acute ST-Elevation and Non-ST-Elevation Myocardial Infarction (FAST-MI) 2005 Cohort. Circulation. 2014; 129:1629–36.

34. Siddiqi TJ, Usman MS, Khan MS, et al. Meta-Analysis Comparing Primary Percutaneous Coronary Intervention Versus Pharmacoinvasive Therapy in Transfer Patients with ST-Elevation Myocardial Infarction. Am J Cardiol. 2018; 122:542–547.

35. Williams MJ, Harding SA, Devlin G, et al. National variation in coronary angiography rates and timing after an acute coronary syndrome in New Zealand (ANZACS-QI 6). N Z Med J. 2016; 129:66–78.

36. Nixon G, Samaranayaka A, de Graaf B, et al. The impact of a rural scanner in overcoming urban versus rural disparities in the utilisation of computed tomography. Aust J Rural Health. 2015; 23:150–4.

37. Blattner K, Nixon G, Horgan C, et al. Evaluation of a rural primary-referred cardiac exercise tolerance test service. N Z Med J. 2014; 127:63–70.

38. Kerr A, Lee M, Grey C, et al. Acute reperfusion for ST-elevation myocardial infarction in New Zealand (2015-2017): patient and system delay (ANZACS-QI 29). N Z Med J. 2019; 132:41–59.

39. Kerr A. Fibrinolysis for STEMI in New Zealand Public Hospitals July 2015-June 2017 - an ANZACS-QI Registry Report. [In Press].

40. Huynh LT, Rankin JM, Tideman P, et al. Reperfusion therapy in the acute management of ST-segment-elevation myocardial infarction in Australia: findings from

the ACACIA registry. Med J Aust. 2010; 193(9):496–501.

41. Li X, Murugiah K, Li J, et al. Urban-Rural Comparisons in Hospital Admission, Treatments, and Outcomes for ST-Segment-Elevation Myocardial Infarction in China From 2001 to 2011: A Retrospective Analysis From the China PEACE Study (Patient-Centered Evaluative Assessment of Cardiac Events). Circ Cardiovasc Qual Outcomes. 2017; 10.

42. Tang EW, Wong CK, Herbison P. Community hospital versus tertiary hospital comparison in the treatment and outcome of patients with acute coronary syndrome: a New Zealand experience. N Z Med J. 2006; 119:U2078.

43. Yong CM, Abnousi F, Asch SM, Heidenreich PA. Socioeconomic inequalities in quality of care and outcomes among patients with acute coronary syndrome in the modern era of drug eluting stents. J Am Heart Assoc. 2014; 3:e001029.

44. Fabreau GE, Leung AA, Southern DA, et al. Area Median Income and Metropolitan Versus Nonmetropolitan Location of Care for Acute Coronary Syndromes: A Complex Interaction of Social Determinants. J Am Heart Assoc. 2016; 5.

45. Fabreau GE, Leung AA, Southern DA, et al. Sex, socioeconomic status, access to cardiac catheterization, and outcomes for acute coronary syndromes in the context of universal healthcare coverage. Circ Cardiovasc Qual Outcomes. 2014; 7:540–9.

46. Hyun K, Redfern J, Woodward M, et al. Socioeconomic Equity in the Receipt of In-Hospital Care and Outcomes in Australian Acute Coronary Syndrome Patients: The CONCORDANCE Registry. Heart Lung Circ. 2018; 27:1398–1405.

47. National Minimum Dataset (Hospital Events) Data Dictionary. Wellington: Ministry of Health; 2019.

48. Pharmaceutical Claims Data Mart (PHARMS) Data Dictionary. Wellington: Ministry of Health; 2019.

49. Mortality Collection Data Dictionary. Wellington: Ministry of Health; 2019.

Exploring Pasifika wellbeing: findings from a large cluster randomised controlled trial of a mobile health intervention programme

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ABSTRACT

AIM: The primary objective of this study was to determine the effect of a mobile health (mHealth) intervention on the wellbeing of Pasifika peoples, and to explore factors associated with Pasifika wellbeing.

METHODS: The OL@-OR@ mHealth programme was a co-designed smartphone app. Culturally relevant data was collected to examine holistic health and wellbeing status, at baseline, and at 12 weeks (end of the trial). The concept of wellbeing was examined as part of a two-arm, cluster randomised trial, using only the Pasifika data: 389 (of 726) Pasifika adults were randomised to receive the mHealth intervention, while 405 (of 725) Pasifika adults were randomised to receive a control version of the intervention. Culturally relevant data was collected to examine holistic health and wellbeing status, at baseline, and at 12 weeks (end of the trial). The intervention effects and the association of demographic and behavioural relationships with wellbeing, was examined using logistic regression analyses.

RESULTS: Relative to baseline, there were significant differences between the intervention and control groups for the 'family/community' wellbeing, at the end of the 12-week trial. There were no significant differences observed for all other wellbeing domains for both groups. Based on our multivariate regression analyses, education and acculturation (assimilation and marginalisation) were identified as positively strong factors associated to Pasifika 'family and community' wellbeing.

CONCLUSION: Our study provides new insights on how Pasifika peoples' characteristics and behaviours align to wellbeing. Our findings point to 'family and community' as being the most important wellbeing factor for Pasifika peoples.

In New Zealand, obesity, prediabetes and T2DM are serious non-communicable (NCD) diseases that have an impact on overall health and wellbeing.[1] New Zealand has the third highest obesity (ie, BMI >30kg/m^2) rates (31%) (following Mexico and the US).[2] These diseases pose a major challenge for healthcare in New Zealand, place a substantial social-economic burden on the health system,[3–6] and are the leading drivers for health inequalities, particularly among New Zealand Pacific peoples.[1] Pacific peoples in New Zealand make up 7% of the total population,[7] and they have the highest rate (67%) of obesity, compared to Māori (indigenous people of New Zealand) (47%), and the non-Māori non-Pacific population (32%).[8] Little is known about how New Zealanders manage the challenges imposed by these conditions on wellbeing. Reducing the incidence and disease impact remains a key issue in disease prevention, and there is a major knowledge gap in how to tailor prevention programmes for sustainable healthier lifestyle change.[9]

With the scale of the rising NCD problem being more evident among Pacific peoples, recent work has highlighted that a focus on health and wellbeing required in-depth knowledge of: lifestyle factors (eg, poor diet); systemic issues (eg, lack of knowledge); and further understanding of the role of cultural and family responsibilities on Pacific people's overall health. Through better knowledge and understanding of these issues, effective prevention programmes that place health and well-being as a holistic focus[10] are considered to be better aligned with Pacific peoples' cultural and value systems. Researchers have also called for interventions to be ethnic-specific and culturally safe,[11] and for programmes that are inclusive of health and wellbeing from a Pacific viewpoint.[10] There have been many efforts to develop and implement culturally appropriate intervention programmes;[12,13] however, these programmes were not planned, developed, piloted or evaluated with the Pacific communities playing an equal partnership role at the helm of the project.

Mobile health (mHealth) programmes, that is, the use of mobile and wireless tools,[14] have been shown to aid the improvement in reducing NCD risk factors and develop healthy behavioural changes.[15,16] The OL@-OR@ project was a culturally tailored mHealth programme,[17] co-designed between New Zealand health researchers and Māori and Pasifika (defined as a collective group of people representing different Pacific Island Nations[18]) communities.[19] We will refer to Pacific peoples as Pasifika peoples from here onwards. The project employed co-design principles and methods to develop a pragmatic mHealth intervention tool with communities to support better health and wellbeing, through improved nutrition, healthy behaviours and to build better knowledge and awareness of community-level activities, resources and social cohesion. The co-design principles aligned well with indigenous health frameworks, and therefore it was considered to be a good fit, and likely to be well accepted,[9] by Pasifika communities.

This paper presents analyses of secondary outcomes of the cRCT and aims to determine the effects of the co-designed mHealth intervention on the wellbeing of Pasifika communities, and to identify the demographic and behavioural factors associated with enhanced wellbeing.

Methods

The OL@-OR@ mHealth programme focused on managing or reducing the key risk factors for NCDs (eg, diet, physical activity, smoking, alcohol). The co-design approach enabled Pasifika communities to include a cultural measure of health that was holistic, Pasifika values-based, and included family and cultural identity as the foundation of health and wellbeing. The Pacific model of health (Fonofale)[20] includes four dimensions of health, namely: spiritual, physical, mental and other, and was used to inform the wellbeing measurements (Appendix 1) used in the OL@-OR@ mHealth programme.

The OL@-OR@ mHealth programme was implemented in a 12-week, community-based two-arm, cluster randomised control trial (cRCT) design, administered from between January–December 2018. Eligibility to participate in the trial included self-identification as being Māori or Pasifika, aged ≥18 years, regular mobile device access (eg, smartphone, laptop), regular internet access, and an email account. The main findings of the cRCT intervention have been published, and the trial protocol adheres to the SPIRIT guidelines, which has been published elsewhere and included as Appendix 1.[21] However, briefly, the participants were recruited predominantly via face-to-face from 64 community clusters (32 Māori, 32 Pasifika), and these were defined as a distinct New Zealand community context with an average of 20 participants per cluster. For Pasifika clusters, these included groups or communities (eg, churches, sports clubs), as identified by the Pasifika community coordinators (employed by the Pasifika community research partners). All clusters were randomly allocated (1:1 ratio) to either the intervention (mHealth tool) or the control (a control version of the mHealth tool that only selected collected data) group using a computer-generated randomisation list, and block randomisation was used to stratify Pasifika clusters by locality (Auckland/urban or Waikato/rural). The risk of contamination between cluster arms was minimised by

recruitment across large geographic areas and multiple diverse community.

Ethical approval for the trial was received from the Northern B Health and Disability Ethics Committee of New Zealand (OL@-OR@) in 2017. All clusters provided written informed consent, and individual participants provided informed consent via an online questionnaire completed at registration.

Pasifika participants in the initial phase of this study, provided an end-user perspective, contributing to the design of the intervention tool, design of outcome measures, analysis of qualitative data, and recruitment pathways for the cRCT. As a secondary outcome measure of the overall cRCT, we included the focus on holistic health and wellbeing status from a Pasifika perspective,[22] and this was compared between trial arms.

Study outcomes and analyses

The original sample size calculation was based on the primary outcome for the overall cRCT; self-reported adherence to health-related behaviour guidelines,[17] and included complete data from 69 clusters (based on 80% power at a 5% level of significance (two-sided) to detect between group absolute difference of 15% in the primary outcome at 12 weeks post-randomisation). At baseline there were 69 clusters and 1,451 participants (657 Māori and 794 Pasifika), and 84% completed the 12-week follow-up questionnaire (n=1,224).

For the current paper, the Pasifika wellbeing data was extracted as a focus for this investigation, and therefore, all Pasifika participants were included in the analyses, irrespective of whether the participants in each cluster received or used the intervention. In addition, clusters that withdrew from the study or did not register any participants at baseline were excluded. Thus, the overall sample included in this paper was 794 Pasifika (controls n=405 and intervention group n=389). Continuous and categorical variables were presented as numbers observed, means and 95% confidence limits. Analyses were performed using SAS 9.4 (SAS Institute Inc., Cary NJ, US). All statistical tests of significance were based on paired t-tests, t-tests, using the conventional $p<0.05$.

Defining wellbeing

We defined five areas of 'wellbeing' based on the individual variables that were aligned to the four pillars of the Fonofale model of health, and according to *a priori* knowledge and understanding of Pasifika health and wellbeing, as developed from our previous work.[10] This included; **spiritual wellbeing**: defined as, 'spiritual beliefs supporting health' measured on a 5-point Likert scale (*1=not very likely to 5=very likely*); **physical wellbeing** defined as, 'being physically ambulant (without pain)', all answers were measured on a 5-point Likert scale (*1=not very able to 5=very able*); **mental wellbeing** defined as 'how likely are setting family goals' and 'having a positive outlook about life in general'. Each question were measured on a 5-point Likert scale (*1=not very able/not very positive to 5=very able/very positive*); and '**family and community life'** was determined by 'how families rated their capacity to support healthier choices' and 'environments that support healthy choices'. All answers were measured on a 5-point Likert scale (1=*not very strong at all/not very well at all to 5=very strong/very well*). We also aggregated other variables that had indicated significance (data not shown in this paper), to formulate a single '**combined wellbeing'** variable. This included spiritual, physical and mental domains (including: spiritual beliefs, eating the right-sized portions at social events, mental goals and positive outlook on life). They were considered to be important aspects of wellbeing to our study participants, but not a sufficiently meaningful variable on its own, hence the aggregated approach. Each of these wellbeing variables were measured on a 5-point Likert scale (*1=not very able/not very confident at all/not very positive at all to 5=very able/very confident/very positiv*e). From here onwards, we will refer to these as the 'domains of wellbeing'.

Complementing the findings of the overall cRCT[21] this paper presents Pasifika data examining the relationship between the 'factors of wellbeing' (dependent variables) with demographics/behaviours (independent variables): **Socio-demographic data:** age, gender, ethnicity, highest education level; **Anthropometry:** self-reported weight (in kilograms) and height

(in centimetres); **Co-morbidities:** self-reported health condition(s) defined as being told by a doctor that they have high blood pressure, high cholesterol, diabetes and/or heart disease; and **Acculturation: Pasifika and Kiwi-New Zealand Heritage and Lifestyle:** Attitudes and beliefs about Pacific and Kiwi/New Zealand heritage and lifestyle measured using an eight-item cultural affiliation questionnaire.[23] The acculturation tool used in this study was developed by researchers of the Kohala Health Research Project,[24] and is a validated tool for adult Pasifika peoples examining similar health outcomes (metabolic health problems).[24] In accordance with the Kohala Health Research Project guidance, we analysed the responses by grouping the summed responses into the following categories: *integrated* (high affiliation with Pacific heritage and mainstream culture); *tradition* (high affiliation with Pacific heritage only); *assimilated* (high affiliation with mainstream culture only); and *marginalised* (low affiliation with both Pacific heritage and mainstream culture).

Results

Demographics

Table 1 shows the characteristics of all Pasifika study participants. The study communities were mostly located in urban centres (69.5%). The highest education qualifications obtained from the participants was at secondary school (45.0%) and tertiary (32.8%) levels, and the majority of participants were female (65.5%).

A wide range of Pacific Island nations were represented in the study, with the majority being Samoan (28.3%), Cook Island Māori (25.3%), and Tongan (19.7%). We grouped the remainder under 'Other Pacific Islands' because the numbers were too small to include independent island nations on their own.

Age was categorised into approximate quartiles: 18–24 (17.5%); 25–34 years (26.3%); 35–44 years (26.3%) and 45+ (29.9%).

A large proportion (67.6%) of the study participants assessed their acculturation mode as being '*marginalised*', indicating they had a low degree of affiliation with *both* their Pacific heritage and the mainstream culture.

Overwhelmingly, obesity BMI (30+) was highly prevalent among the entire Pasifika study sample (69.9%), and this is analogous for both the intervention (69.6%) and control (70.3%) groups. The participants presented with co-morbidities based on known diagnosis: high blood pressure being the most commonly reported. We also included a grouped variable ('any') to include all known morbidities. There were no significant differences indicated between the co-morbidity groups.

Table 2 examines the group means (standard deviations) for intervention and control participants at baseline and at 12 weeks. At baseline, there was no significant difference between these two groups. However, at 12 weeks, there was a significant difference between intervention and control groups, for the 'family and community' wellbeing (t-test p-value=0.007).

Relative to baseline, based on the mean differences (95%CI), both groups showed no change for 'spiritual', 'physical' and 'mental' and 'combined' factors of wellbeing. However, there was a significant difference between intervention and control groups for the 'family and community', at the end of the 12-week trial (t-test p-value=0.006).

Table 3 summarises the univariate analyses, examining the relationships between each 'factor of wellbeing' and the demographic and behavioural variables, at baseline.

For **spiritual wellbeing:** the strongest associations were age (oldest group) (p=0.0001); Other Ethnicity (p=0.0001); being *assimilated* (p=0.0001) and *marginalised* (p=0.0001).

As to the **physical wellbeing** factor, the strongest associations were: the older age groups (35–44 (p=0.0005); and 45 years (p=0.0008); those reporting extreme obesity (BMI 40+) (p=0.0001), and missing obesity data (p=0.015); and participants who identified as being *assimilated* (p=0.035); *traditional* (p=0.0005); and *marginalised* (p=0.0001), and having a co-morbidity (p=0.004).

Table 1: Distribution of Pasifika participant characteristics, at baseline.

	All		Intervention		Control	
	794 (n)	%	389 (n)	%	405 (n)	%
Gender						
Male	232	34.5	117	34.2	115	34.9
Female	440	65.5	225	65.8	215	65.2
Missing	1					
Ethnicity						
Tokelauan	16	2.0	9	2.3	7	1.7
Fijian	7	0.9	4	1.0	3	0.7
Niuean	59	7.4	21	5.4	38	9.4
Tongan	156	19.7	114	29.3	42	10.4
Cook Island Māori	201	25.3	94	24.2	107	26.4
Samoan	225	28.3	95	24.4	130	32.1
Other Pacific Island	8	1.0	5	1.3	3	0.7
Māori	48	6.1	12	3.1	36	8.9
NZ/Other European	46	5.8	13	3.3	33	8.2
Other	28	3.5	22	5.7	6	1.5
Highest education						
Secondary school	335	45.0	167	46.4	168	43.6
Trade certificates	52	7.0	27	7.5	25	6.5
Tertiary (any level)	244	32.8	103	28.6	141	36.6
None	114	15.3	63	17.5	51	13.3
Missing	49					
Age group (quartiles)						
18–24 years	139	17.5	67	17.2	72	17.8
25–34 years	209	26.3	81	20.8	128	31.6
35–44 years	209	26.3	104	26.7	105	25.9
45+	237	29.9	137	35.2	100	24.7
Region						
Urban	552	69.5	279	71.7	273	67.4
Rural	242	30.5	110	28.3	132	32.6
BMI class						
Underweight (<18.50)	2	0.3	1	0.3	1	0.3
Healthy weight (18.50-24.99)	73	11.3	41	12.7	32	9.8
Overweight (25.00-29.99)	120	18.5	56	17.4	64	19.6
Obese (30+)	453	69.9	224	69.6	229	70.3
BMI missing	146					

Table 1: Distribution of Pasifika participant characteristics, at baseline (continued).

Co-morbidities						
High blood pressure	108	13.6	57	14.7	51	12.6
High cholesterol	65	8.2	31	8.0	34	8.4
Diabetes	76	9.6	38	9.8	38	9.4
Heart disease	19	2.4	11	2.8	8	2.0
Acculturation						
Integrated	118	14.9	49	12.6	69	17.1
Traditional	66	8.3	25	6.4	41	10.2
Assimilated	73	9.2	39	10.0	34	8.4
Marginalised	535	67.6	276	71.0	259	64.3
Missing	2					

For the **mental wellbeing** factor, the relationships were evident among those: in the oldest age group (45+years), p=0.011; participants who were from the Other Pacific Island nations (p=0.009) and Others (p=0.007); having only secondary school qualifications (p=0.010); and participants whose scores identified them as being *assimilated* (p=0.0001)*, traditional* (p=0.009) and *'marginalised'* (p=0.0001).

Under the **family/community** wellbeing factor, the following positive associations with demographic factors were highlighted: from those among the oldest age group (p=0.0001); being from 'Other Pacific Island' nations (p=0.002); participants with the lowest education qualifications: 'none' and 'secondary', p=0.021 and p=0.006, respectively; those who aligned with being *'assimilated'* (p=0.004); *'traditional'*

Table 2: Effect of wellbeing factors based on the mHealth programme, at 12 weeks from baseline.

Factors of wellbeing	**Intervention baseline (n=389)**		**Control baseline (n=405)**		**Intervention 12 weeks (n=347)**		**Control 12 weeks (n=369)**		**Intervention vs control 12 weeks**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Intervention mean* (95%CI)**	**Control mean* (95%CI)**
Spiritual	3.8	1.2	3.7	1.3	3.8	1.2	3.7	1.3	0.00	0.00
Physical	4.2	0.9	4.1	1.0	4.2	0.9	4.1	1.0	0.10 (-0.04–0.24)	-0.02 (-0.13–0.8)
Mental[a]	7.9	1.7	7.8	1.7	8.1	1.6	7.9	1.5	0.22 (0.04–0.40)	0.04 (-0.14–0.21)
Family/ community[a]	7.1	1.8	7.1	1.8	7.6	1.6	7.3	1.6	**0.54 (0.34–0.74)**	**0.17 (-0.03–0.36)**
Combined wellbeing[b]	15.2	3.1	15.0	2.9	15.6	2.9	15.4	2.8	0.43 (0.11–0.75)	0.38 (0.07–0.69)

[a,b]=aggregate variables (see earlier for composition); SD=standard deviation; *=Difference between means, 95%CI= 95% confidence intervals.

Table 3: Wellbeing model: univariate analyses at baseline, by participant characteristics.

	Factors of Pacific wellbeing					
	(n)	Spiritual wellbeing	Physical wellbeing	Mental wellbeing	[a]Family and community	[b]Combined wellbeing
Characteristics		**mean (95CI)**	**mean (95CI)**	**mean (95CI)**	**mean (95CI)**	**mean (95CI)**
Gender						
Male	253	3.79 (3.63–3.94)[R]	4.16 (4.04–4.28)[R]	7.88 (7.67–8.09)[R]	7.01 (6.79–7.23)[R]	15.15 (14.78–15.52)[R]
Female	540	3.77 (3.66–3.87)	4.16 (4.08–4.24)	7.88 (7.74–8.03)	7.19 (7.04–7.34)	15.13 (14.87–15.38)
Missing	1					
Age group (quartiles)						
18–24 years	139	3.53 (3.32–3.73)[R]	4.46 (4.30–4.62)[R]	7.78 (7.50–8.06)[R]	6.82 (6.53–7.11)[R]	14.93 (14.45–15.42)[R]
25–34 years	209	3.61 (3.44–3.77)	4.20 (4.07–4.33)	7.68 (7.45–7.91)	6.76 (6.52–6.99)	14.47 (14.08–14.87)
35–44 years	209	3.71 (3.55–3.88)	**3.98 (3.85–4.11)****	7.76 (7.53–7.99)	7.19 (6.95–7.43)	14.99 (14.59–15.38)
45+ years	237	**4.12 (3.96–4.27)****	**4.11 (3.99–4.24)****	**8.23 (8.02–8.45)***	7.60 (7.38–7.82)**	**15.96 (15.58–16.33)****
Ethnicity						
Samoan	225	3.91 (3.75–4.07)[R]	4.16 (4.04–4.29)[R]	7.95 (7.73–8.17)[R]	7.07 (6.84–7.30)[R]	15.21 (14.83–15.60)[R]
Tokelauan	16	3.81 (3.22–4.40)	4.13 (3.65–4.60)	7.75 (6.93–8.57)	6.31 (5.45–7.18)	15.44 (14.00–16.88)
Fijian	7	4.00 (3.11–4.89)	4.00 (3.28–4.72)	7.57 (6.33–8.82)	7.43 (6.12–8.74)	15.71 (13.53–17.89)
Niuean	59	3.71 (3.40–4.02)	4.31 (4.06–4.55)	7.75 (7.32–8.17)	6.97 (6.52–7.42)	14.59 (13.84–15.34)
Tongan	156	4.15 (3.96–4.34)	4.15 (3.99–4.30)	8.08 (7.82–8.35)	7.22 (6.94–7.50)	15.76 (15.29–16.22)
Cook Island Māori	201	3.70 (3.53–3.86)	4.24 (4.10–4.37)	8.04 (7.81–8.28)	7.39 (7.14–7.63)	15.32 (14.91–15.73)
Other Pacific Island	8	3.25 (2.41–4.09)	3.63 (2.95–4.30)	**6.38 (5.21–7.54)***	**5.13 (3.90–6.35)***	13.00 (10.96–15.04)*
Other	122	**3.20 (2.99–3.42)****	4.03 (3.86–4.21)	**7.44 (7.14–7.74)***	7.02 (6.71–7.34)	14.20 (13.67–14.72)*
Highest education qualification						
Tertiary (any level)	244	3.81 (3.66–3.97)[R]	4.22 (4.10–4.34)[R]	8.10 (7.89–8.32)[R]	7.40 (7.18–7.63)[R]	15.34 (14.96–15.71)[R]
None	114	3.77 (3.54–4.00)	4.16 (3.98–4.34)	7.75 (7.44–8.06)	**6.94 (6.61–7.26)***	15.11 (14.57–15.66)
Secondary	335	3.71 (3.58–3.84)	4.16 (4.05–4.26)	**7.74 (7.56–7.92)***	**6.99 (6.80–7.18)***	14.93 (14.61–15.25)
Trade	52	3.83 (3.49–4.16)	4.00 (3.74–4.26)	7.92 (7.46–8.38)	7.04 (6.56–7.52)	15.21 (14.40–16.02)
Missing	49	3.96 (3.61–4.31)	4.12 (3.85–4.40)	8.06 (7.59–8.53)	7.33 (6.83–7.82)	15.45 (14.61–16.28)
Obese class						
Not obese	195	3.64 (3.46–3.81)[R]	4.34 (4.20–4.47)[R]	7.78 (7.54–8.02)[R]	7.21 (6.96–7.46)[R]	15.20 (14.78–15.62)[R]
Obese class 1 (BMI 30-34.99)	144	3.83 (3.62–4.03)	4.28 (4.13–4.44)	7.90 (7.62–8.17)	7.06 (6.76–7.35)	15.15 (14.66–15.63)
Obese class 2 (BMI 35-39.99)	131	3.78 (3.57–3.99)	4.24 (4.08–4.41)	7.99 (7.70–8.28)	6.87 (6.56–7.18)	15.07 (14.56–15.58)
Obese class 3 (BMI 40+)	178	3.80 (3.62–3.99)	**3.88 (3.74–4.02)****	7.97 (7.72–8.22)	7.24 (6.98–7.50)	14.97 (14.53–15.40)
Missing	146	3.86 (3.66–4.06)	**4.08 (3.93–4.24)***	7.80 (7.53–8.08)	7.21 (6.92–7.50)	15.29 (14.81–15.78)

Table 3: Wellbeing model: univariate analyses at baseline, by participant characteristics (continued).

Acculturation						
Integrated	118	3.12 (2.91–3.33)[R]	3.75 (3.58–3.93)[R]	6.60 (6.32–6.89)[R]	5.95 (5.64–6.25)[R]	12.77 (12.27–13.27)[R]
Assimilated	73	**3.88 (3.61–4.15) ****	**4.05 (3.83–4.27)***	**7.85 (7.49–8.21)****	**6.67 (6.28–7.06)***	**15.11 (14.48–15.74)****
Traditional	66	3.09 (2.81–3.38)	**4.27 (4.04–4.50)****	**7.24 (6.86–7.62)***	**6.83 (6.43–7.24)****	**13.92 (13.26–14.59)***
Marginalised	535	**3.98 (3.88–4.08) ****	**4.25 (4.17–4.33)****	**8.25 (8.11–8.38)****	**5.95 (5.64–6.25)****	**15.81 (15.57–16.04)****
Missing	2					
Region						
Urban	552	3.83 (3.72–3.93)[R]	4.14 (4.06–4.22)[R]	7.86 (7.72–8.01)[R]	7.04 (6.89–7.18)[R]	15.07 (14.82–15.32)[R]
Rural	242	3.65 (3.50–3.81)	4.21 (4.08–4.33)	7.93 (7.71–8.14)	**7.35 (7.13–7.58)***	15.28 (14.91–15.66)
Comorbidities (Any)						
No	608	3.79 (3.69–3.89)[R]	4.22 (4.14–4.29)[R]	7.92 (7.79–8.06)[R]	7.12 (6.98–7.26)[R]	15.21 (14.97–15.45)[R]
Yes	186	3.72 (3.54–3.90)	**3.98 (3.84–4.12)***	7.76 (7.51–8.00)	7.17 (6.92–7.43)	14.88 (14.45–15.31)

[a]=Aggregate of family and community goals, active participation in community life, and rating of family's ability to make healthy choices; [b]=Aggregate of spiritual, dietary portions, mental wellbeing goals for family and positive view on life; R=referent group; *=p<0.05; **=p<0.001.

Table 4: Wellbeing model: multivariate regression (mean differences), from 12 weeks to baseline.

Characteristics	**(n)**	**Spiritual wellbeing**	**Physical wellbeing**	**Mental wellbeing**	**[a]Family and community**	**[b]Combined wellbeing score**
Gender						
Male	226	0.19 (-0.09–0.46)[R]	0.07 (-0.17–0.30)[R]	0.32 (-0.06–0.70)[R]	0.72 (0.30–1.14)[R]	0.56 (-0.11–1.24)[R]
Female	489	0.22 (-0.05–0.49)	0.07 (-0.16–0.29)	0.43 (0.05–0.80)	0.81 (0.40–1.22)	0.83 (0.17–1.49)
Missing	1					
Age group (quartiles)						
18–24 years	118	0.39 (0.05–0.73)[R]	-0.08 (-0.36–0.21)[R]	0.34 (-0.13–0.80)[R]	0.76 (0.25–1.28)[R]	0.90 (0.07–1.72)[R]
25–34 years	190	**0.08 (-0.20–0.36)***	0.16 (-0.08–0.40)	0.29 (-0.10–0.69)	0.85 (0.42–1.28)	0.58 (-0.11–1.27)
35–44 years	190	0.23 (-0.07–0.53)	**0.19 (-0.07–0.44)***	0.57 (0.15–0.98)	0.83 (0.37–1.28)	0.77 (0.04–1.50)
45+ years	218	0.11 (-0.18–0.39)	0.00 (-0.24–0.24)	0.30 (-0.09–0.69)	0.62 (0.19–1.05)	0.54 (-0.16–1.23)
Ethnicity						
Samoan	203	0.24 (-0.04–0.51)[R]	-0.08 (-0.36–0.21)[R]	0.16 (-0.21–0.54)[R]	0.65 (0.24–1.06)[R]	0.84 (0.18–1.50)[R]
Tokelauan	14	0.77 (0.11–1.42)	0.16 (-0.08–0.40)	0.37 (-0.53–1.28)	0.89 (-0.11–1.89)	1.07 (-0.53–2.68)
Fijian	7	0.08 (-0.82–0.97)	0.19 (-0.07–0.44)	0.80 (-0.43–2.03)	1.04 (-0.32–2.40)	1.11 (-1.07–3.29)
Niuean	54	0.11 (-0.27–0.49)	0.00 (-0.24–0.24)	0.40 (-0.13–0.92)	0.62 (0.04–1.20)	0.94 (0.01–1.87)
Tongan	141	0.20 (-0.10–0.50)	-0.08 (-0.36–0.21)	0.43 (0.02–0.84)	0.84 (0.39–1.30)	0.49 (-0.24–1.22)
Cook Island Māori	185	0.20 (-0.07–0.47)	0.16 (-0.08–0.40)	0.07 (-0.31–0.44)	0.40 (-0.01–0.81)	0.68 (0.02–1.34)
Other Pacific Islands	6	0.04 (-0.95–1.02)	0.19 (-0.07–0.44)	0.38 (-0.97–1.74)	1.27 (-0.22–2.76)	-0.09 (-2.48–2.31)
Other	106	-0.01 (-0.31–0.28)	0.00 (-0.24–0.24)	0.38 (-0.02–0.79)	0.40 (-0.05–0.85)	0.52 (-0.21–1.24)

Table 4: Wellbeing model: multivariate regression (mean differences), from 12 weeks to baseline (continued).

Highest education qualification						
Tertiary (any level)	223	0.16 (-0.11–0.44)[R]	0.13 (-0.10–0.37)[R]	0.22 (-0.16–0.61)[R]	0.53 (0.11–0.95)[R]	0.48 (-0.19–1.16)[R]
None	107	0.18 (-0.14–0.50)	0.12 (-0.15–0.39)	0.58 (0.13–1.02)	**1.06 (0.57–1.55)***	1.05 (0.26–1.83)
Secondary	293	0.11 (-0.15–0.38)	0.08 (-0.15–0.30)	0.39 (0.02–0.76)	0.75 (0.34–1.15)	0.71 (0.07–1.36)
Trade	46	0.24 (-0.18–0.65)	0.03 (-0.32–0.38)	0.37 (-0.20–0.94)	1.01 (0.38–1.64)	0.79 (-0.22–1.80)
Missing	47	0.31 (-0.11–0.74)	-0.02 (-0.38–0.33)	0.31 (-0.27–0.90)	0.47 (-0.17–1.11)	0.45 (-0.59–1.48)
Acculturation						
Integrated	104	0.52 (0.20–0.83)[R]	0.28 (0.01–0.55)[R]	0.81 (0.37–1.25)[R]	1.24 (0.76–1.72)[R]	1.80 (1.03–2.58)[R]
Assimilated	70	**0.01 (-0.36–0.37)***	0.01 (-0.29–0.32)	**-0.01 (-0.51–0.49)***	**0.47 (-0.08–1.02)***	**-0.10 (-0.99–0.79)****
Traditional	59	0.17 (-0.24–0.57)	0.06 (-0.28–0.40)	0.43 (-0.13–0.98)	0.83 (0.22–1.45)	0.88 (-0.11–1.86)
Marginalised	481	**0.12 (-0.13–0.37)***	**-0.09 (-0.29–0.12)***	**0.27 (-0.07–0.61)***	**0.51 (0.14–0.89)****	**0.20 (-0.40–0.81)****
Missing	2					
Region						
Urban	503	0.16 (-0.10–0.41)[R]	0.01 (-0.20–0.22)[R]	0.29 (-0.05–0.63)[R]	0.66 (0.29–1.04)[R]	0.44 (-0.64–1.51)[R]
Rural	213	0.25 (-0.11–0.60)	0.13 (-0.13–0.38)	0.46 (0.04–0.88)	0.86 (0.40–1.33)	0.51 (-0.63–1.65)
Comorbidities (any)						
No	553	0.19 (-0.06–0.44)[R]	0.01 (-0.20–0.22)[R]	0.29 (-0.05–0.63)[R]	0.66 (0.29–1.04)[R]	0.44 (-0.64–1.51)[R]
Yes	163	0.21 (-0.09–0.52)	0.13 (-0.13–0.38)	0.46 (0.04–0.88)	0.86 (0.40–1.33)	0.51 (-0.63–1.65)

[R]=Referent group; [a]=Aggregate of family and community goals, active participation in community life, and rating of family's ability to make healthy choices; [b]=Aggregate of spiritual, diet, mental wellbeing goals for family and positive view on life; *=p<0.05; **=p<0.001.

(p=0.0007); and *'marginalised'* (p=0.0001), and participants from the 'rural' cluster localities (p=0.021).

The **combined wellbeing** factor showed significant positive relationships with participants: in the oldest age group (45+yrs) (p=0.0011); being from Other Pacific Island' nations (p=0.037), and 'Other' (p=0.002) ethnic groups; and those who rated their acculturation status as being *assimilated* (p=0.0001); *traditional* (p=0.007), and *marginalised* (p=0.0001), all significantly reported alignment with this wellbeing factor.

Informed by our univariate analyses (Table 3), Table 4 includes the *potential* independent variables in our multivariate analyses of all participants that provided data at both baseline and at 12 weeks. We excluded BMI and obesity class variables from this analyses as (from earlier models) their significant levels consistently diminished and it was no longer meaningful to retain them in the model. The independent variables were examined by way of mean differences (95CI) from 12 weeks to baseline, for each factor of wellbeing. Notably, only the significant relationships are highlighted in the table.

For the **spiritual wellbeing factor**, after adjusting for all co-variates: being of young age (25–34 years) p=0.031; and acculturation (*assimilation and marginalised)* p=0.008 and 0.003, respectively, sustained significant improved relationships with this wellbeing factor.

Under the **physical wellbeing factor**, after adjusting for sex, age, ethnicity, education, cluster region and having any comorbidity, only the participants who were aged 35–44 years (p=0.030) retained a positive association with physical wellbeing. Conversely, those who rated as being *'marginalised'* (p=0.001) had a very small negative mean difference that was significant, albeit indicating no improvement (-0.09) compared to the *'integrated'* group, by the end of the trial.

As for the **mental wellbeing**, after adjusting for all variables, only acculturation sustained a significant relationship with this wellbeing factor. Participants that aligned with being *'assimilated'* had shown a very small negative association, indicating no improvement (-0.01, 95CI: -0.51–0.49, p=0.002) at the end of the trial. Those participants that affiliated with being *'marginalised'* had sustained a positive significant relationship and they reported a mild improvement with mental wellbeing (0.27, 95CI: -0.07–0.61, p=0.004), compared to the *'integrated'* group.

For the **family/community** wellbeing, after controlling for all co-variates, the participants with 'no education' qualifications (1.06, 95CI: 0.57–1.55, p=0.019) showed a large significant (positive) improvement, compared to those participants with any 'tertiary level' qualifications (0.53, 95CI: 0.11–0.95). Regarding all acculturation modes, there were significant positive improvements for the *'marginalised'* group (p=0.0004), followed by the *'assimilated'* group (p=0.008), compared to the *'integrated'* group.

Finally, the **combined wellbeing** factor, after adjusting for all variables, the significant relationships were evident among those participants that corresponded to being *'assimilated'* (-0.10, 95CI: -0.99–0.79, p=0.0001) – showing no improvement for this wellbeing; and being *'marginalised'* (0.20, 95CI: -0.40–0.81, p=0.0001), when compared to the *'integrated'* group.

Discussion

In our large mHealth cRCT programme, we defined the 'domains of wellbeing' as being: spiritual, physical, mental, family/community and a combined wellbeing domain, which was an aggregate of various wellbeing measurement scores (Appendix 2). These wellbeing factors were arbitrarily defined by how well the Pasifika participants rated their wellbeing status according to a range of individual characteristics (Table 1).

Principal findings

There are three major findings from our analyses. Firstly, Table 2 showed significant differences between the intervention and control groups for 'family/community' wellbeing factor, by the end of the 12-week trial. This is not surprising, given that Pasifika peoples traditionally and have continue to live and participate in social cohesion. There were no differences between intervention and control groups for the remaining wellbeing factors, and this was analogous with the findings from the overall study,[21] that also demonstrated that the mHealth programme did not significantly improve adherence to health-related behaviours for all participants. This finding may be explained by the short duration of the trial (12 weeks), and it is possible that a longer duration may have provided more meaningful information.[21]

The remaining major findings were based on our multivariate analyses (Table 4). The second major finding was 'acculturation' as being a major determinant of wellbeing for our Pasifika participants. In particular, the acculturated modes, of being *'assimilated'* (*high affiliation with mainstream culture only*) and *'marginalised'* (*low affiliation with both Pacific heritage and mainstream culture*) were independently negatively associated with all wellbeing factors. Specifically, those participants who classified themselves as being *'assimilated'* showed either little or no association with 'spiritual', 'physical', 'mental' and 'combined' wellbeing factors. A possible explanation could relate to issues of adapting to the changing dynamics of traditional and cultural practices and values. On the other hand, significant positive associations were evident for those who classified themselves as being *'marginalised'* for all wellbeing factors (but not physical—very small negative association), and this could be an indicator of cultural resilience. Previous research has shown that some groups facing chronic stresses created by poverty, racism and discrimination due to a lack of security in identity and traditional values,[25] and therefore the scores in our study may reflect a lack of bicultural and societal identity.

Of note, the young and working age participants (25–34 years and 35–44 years) showed significant associations with the 'spiritual' and 'physical' wellbeing factors, which characterises their level of participation in community and church activities.

The third major finding of our study showed clear positive relationships between: 'no education', and acculturation modes: 'assimilation', and being

'marginalised', with the 'family/community' wellbeing factor. The high scores highlighted significant positive improvements by the end of the 12-week trial. A possible explanation for having a strong and diverse relationship of acculturation to this wellbeing factor could be related to how Pasifika peoples in our study connect to the Pasifika way of life (ie, cultural values and protocol). This may indicate the growing disconnect between and within Pasifika communities.[26] For example, symptoms of living in diasporic communities may be manifested in the way Pasifika peoples view and define their cultural identity as being 'born' or 'raised',[27] and the degree of 'how well' they affiliate with the mainstream and, or their Pasifika heritage.[22] In relation to education, participants with 'no education' had improved because of the programme, and this was evident in our qualitative data (not published), where participants reportedly learnt a lot about healthy lifestyles, because the mHealth tool was relevant to Pasifika culture and values.

Implications of study

Acculturation has recently been redefined from a linear process in which one ethnic/cultural group adopt the beliefs and behaviours of another group,[28] to a multi-dimensional process where people engage in different ways.[29,30] The finding of associations among *'marginalised'* participants and 'family/community' wellbeing may be indicative of other complex psychosocial factors, such as attitudes, beliefs, emotions and learned behaviours, that have not been catered for in the current study.

Additionally, as the family/community context is a primary environment in which its members grow up and develop their identity, it is possible that the participants in this study experienced different distress and intra-familial stressors as a result of acculturation.[25] Therefore, acculturation responses are likely to be different, or conflicting based on personal experiences of acculturation and family/community cohesion.[31–33] Thus, the acculturation modes used in this study may only be representative of the participants' perspectives in relation to how we have defined 'family/community' wellbeing. Alternatively, the acculturation tool may not be adequately sufficient to measure the degree and variation of cultural heritage and affiliation. Observing how family and community members function as a nucleus or an extended network system of shared interests, values and experiences maybe a better alternative to understand wellbeing. Unfortunately, our study was not able to gauge participants' in-depth understanding of acculturation and family/community cohesion.

Study limitations and future work

A major limitation is the potential for selection bias of study participants that may have led to the high proportion of participants indicating their acculturation status as being predominantly 'marginalised', and lower education background. Also, there is the potential for participation bias based on the limited use of the mobile/electronic platform of the intervention tool and due to the duration of the mHealth trial (12 weeks), that may have been too short to be able to measure the wellbeing factors at a comprehensive level. Finally, to better understand wellbeing from a Pasifika perspective, further research will be needed to include other domains outside of established health models, including the role of family and community.

Conclusion

Our study utilised Pasifika-only participant data from a large cRCT[21] study, to examine the relationship between demographic and behavioural factors and Pasifika wellbeing. From the cRCT findings (Table 2), the programme appears to have supported positive changes, particularly for the intervention participants in 'family and community' wellbeing, compared with the controls. Additionally, it was clear from our multivariate analyses that at an individual level, the study participants who identified as being 'marginalised' had significantly positive associations with family/community wellbeing. Although the study findings do not fully explain the reasons behind the acculturation, education and age characteristics associations, it does point to the importance of 'family/community' as being the most important wellbeing factor for Pasifika peoples. Future work could focus on more in-depth understanding of the psychosocial factors and an up-to-date knowledge of intra-familial and inter-generational perception of acculturation, and its effect on overall wellbeing.

Appendix

Appendix Table 1: Wellbeing questions: Pasifika version.

Spiritual	1. How do your spiritual beliefs support you to have a healthy life? Likert scale not very well at all—> very well Comment: *Free text*
Physical (also covered with primary outcomes)	2. How able are you to move about without pain or discomfort? Likert scale not very able at all—> very able Comment: *Free text*
	3. How confident are you in eating the right-sized portions at community events? Likert scale not very confident at all—> very confident Comment: *Free text*
Mental	4. How able do you feel to set goals for yourself? Likert scale not very able at all—> very able Comment: *Free text*
	5. How likely are you to set goals for yourself or your family? Likert scale not very likely at all—> very likely Comment: *Free text*
	6. How positive are you about life in general? Likert scale not very positive at all—> very positive Comment: *Free text*
	7. How much do you like participating in community activities? Likert scale not very much at all—>very much Comment: *Free text*
Family	8. How strong would you rate your family's ability to make healthy choices? Likert scale not very strong at all—> very strong Comment: *Free text*
Other	9. How well does the environment support you to make healthy choices? (environment includes physical, social, economic and political environment(s) and a range of settings such as schools, churches, food stores, sports clubs, etc) Likert scale not very well at all—> very well Comment: *Free text*
	10. How well do you know how to access healthy services in your local community, eg, local markets, low-cost exercise classes, etc? Likert scale not very well at all—> very well Comment: *Free text*

Pacific and Kiwi/New Zealand heritage and lifestyle

Next are questions about your attitude and beliefs about Pacific and Kiwi/New Zealand heritage and lifestyle. Please provide the answer that best describes you after each question					
Pacific/Kiwi-New Zealand Heritage and Lifestyle					
1 = Very Knowledgeable, 2 = Somewhat Knowledgeable, 3 = Neutral or No response, 4 = Somewhat not knowledgeable, 5 = Not at all Knowledgeable					
Questions	1	2	3	4	5
1. How knowledgeable are you of traditional **Pacific** culture and lifestyle?					
2. How knowledgeable are you of traditional **Kiwi/ New Zealand** culture and lifestyle?					
1 = Very involved, 2 = Somewhat involved, 3 = Neutral or No response, 4 = Somewhat not knowledgeable, 5 = Not at all involved					
Questions	1	2	3	4	5
3. How involved are you in **Pacific** culture and lifestyle?					
4. How involved are you in **Kiwi/New Zealand** culture and lifestyle?					
1 = Very Positive, 2 = Somewhat Positive, 3 = Neutral or No response, 4 = Somewhat negative, 5 = Very Negative					
Questions	1	2	3	4	5
5. How do you feel towards the **Pacific** culture and lifestyle?					
6. How do you feel towards the **Kiwi/New Zealand** culture and lifestyle?					
1 = Very Important 2 = Somewhat Important, 3 = Neutral or No response, 4 = Very little importance, 5 = Not important at all					
Questions	1	2	3	4	5
7. How important is it for you to maintain a **Pacific** lifestyle and identity?					
8. How important is it for you to maintain a **Kiwi/New Zealand** lifestyle and identity?					

Appendix Table 2:

Section/item	Item No	Description	Addressed on page number
Administrative information			
Title	1	Descriptive title identifying the study design, population, interventions and, if applicable, trial acronym	1
Trial registration	2a	Trial identifier and registry name. If not yet registered, name of intended registry	2
	2b	All items from the World Health Organization Trial Registration Data Set	
Protocol version	3	Date and version identifier	14
Funding	4	Sources and types of financial, material, and other support	15
Roles and responsibilities	5a	Names, affiliations and roles of protocol contributors	1
	5b	Name and contact information for the trial sponsor	1
	5c	Role of study sponsor and funders, if any, in study design; collection, management, analysis, and interpretation of data; writing of the report; and the decision to submit the report for publication, including whether they will have ultimate authority over any of these activities	15
	5d	Composition, roles and responsibilities of the coordinating centre, steering committee, endpoint adjudication committee, data management team and other individuals or groups overseeing the trial, if applicable (see Item 21a for data monitoring committee)	15
Introduction			
Background and rationale	6a	Description of research question and justification for undertaking the trial, including summary of relevant studies (published and unpublished) examining benefits and harms for each intervention	3–4
	6b	Explanation for choice of comparators	7
Objectives	7	Specific objectives or hypotheses	3–4
Trial design	8	Description of trial design including type of trial (eg, parallel group, crossover, factorial, single group), allocation ratio, and framework (eg, superiority, equivalence, non-inferiority, exploratory)	4

Methods: participants, interventions, and outcomes			
Study setting	9	Description of study settings (eg, community clinic, academic hospital) and list of countries where data will be collected. Reference to where list of study sites can be obtained	4–5
Eligibility criteria	10	Inclusion and exclusion criteria for participants. If applicable, eligibility criteria for study centres and individuals who will perform the interventions (eg, surgeons, psychotherapists)	4–5
Interventions	11a	Interventions for each group with sufficient detail to allow replication, including how and when they will be administered	6–7
	11b	Criteria for discontinuing or modifying allocated interventions for a given trial participant (eg, drug dose change in response to harms, participant request, or improving/worsening disease)	N/A
	11c	Strategies to improve adherence to intervention protocols, and any procedures for monitoring adherence (eg, drug tablet return, laboratory tests)	7
	11d	Relevant concomitant care and interventions that are permitted or prohibited during the trial	N/A
Outcomes	12	Primary, secondary and other outcomes, including the specific measurement variable (eg, systolic blood pressure), analysis metric (eg, change from baseline, final value, time to event), method of aggregation (eg, median, proportion), and time point for each outcome. Explanation of the clinical relevance of chosen efficacy and harm outcomes is strongly recommended	7–10
Participant timeline	13	Time schedule of enrolment, interventions (including any run-ins and washouts), assessments, and visits for participants. A schematic diagram is highly recommended (see Figure)	11
Sample size	14	Estimated number of participants needed to achieve study objectives and how it was determined, including clinical and statistical assumptions supporting any sample size calculations	10
Recruitment	15	Strategies for achieving adequate participant enrolment to reach target sample size	5

Methods: assignment of interventions (for controlled trials)			
Allocation:			
Sequence generation	16a	Method of generating the allocation sequence (eg, computer-generated random numbers), and list of any factors for stratification. To reduce predictability of a random sequence, details of any planned restriction (eg, blocking) should be provided in a separate document that is unavailable to those who enrol participants or assign interventions	6
Allocation concealment mechanism	16b	Mechanism of implementing the allocation sequence (eg, central telephone; sequentially numbered, opaque, sealed envelopes), describing any steps to conceal the sequence until interventions are assigned	6
Implementation	16c	Who will generate the allocation sequence, who will enrol participants, and who will assign participants to interventions	6
Blinding (masking)	17a	Who will be blinded after assignment to interventions (eg, trial participants, care providers, outcome assessors, data analysts), and how	6
	17b	If blinded, circumstances under which unblinding is permissible, and procedure for revealing a participant's allocated intervention during the trial	N/A
Methods: Data collection, management and analysis			
Data collection methods	18a	Plans for assessment and collection of outcome, baseline and other trial data, including any related processes to promote data quality (eg, duplicate measurements, training of assessors) and a description of study instruments (eg, questionnaires, laboratory tests) along with their reliability and validity, if known. Reference to where data collection forms can be found, if not in the protocol	7–10
	18b	Plans to promote participant retention and complete follow-up, including list of any outcome data to be collected for participants who discontinue or deviate from intervention protocols	7–10
Data management	19	Plans for data entry, coding, security, and storage, including any related processes to promote data quality (eg, double data entry; range checks for data values). Reference to where details of data management procedures can be found, if not in the protocol	12
Statistical methods	20a	Statistical methods for analysing primary and secondary outcomes. Reference to where other details of the statistical analysis plan can be found, if not in the protocol	11–12
	20b	Methods for any additional analyses (eg, subgroup and adjusted analyses)	
	20c	Definition of analysis population relating to protocol non-adherence (eg, as randomised analysis), and any statistical methods to handle missing data (eg, multiple imputation)	11–12

Methods: Monitoring			
Data monitoring	21a	Composition of data monitoring committee (DMC); summary of its role and reporting structure; statement of whether it is independent from the sponsor and competing interests; and reference to where further details about its charter can be found, if not in the protocol. Alternatively, an explanation of why a DMC is not needed	13
	21b	Description of any interim analyses and stopping guidelines, including who will have access to these interim results and make the final decision to terminate the trial	N/A
Harms	22	Plans for collecting, assessing, reporting, and managing solicited and spontaneously reported adverse events and other unintended effects of trial interventions or trial conduct	N/A
Auditing	23	Frequency and procedures for auditing trial conduct, if any, and whether the process will be independent from investigators and the sponsor	13
Ethics and dissemination			
Research ethics approval	24	Plans for seeking research ethics committee/ institutional review board (REC/IRB) approval	13
Protocol amendments	25	Plans for communicating important protocol modifications (eg, changes to eligibility criteria, outcomes, analyses) to relevant parties (eg, investigators, REC/IRBs, trial participants, trial registries, journals, regulators)	13
Consent or assent	26a	Who will obtain informed consent or assent from potential trial participants or authorised surrogates, and how (see Item 32)	13
	26b	Additional consent provisions for collection and use of participant data and biological specimens in ancillary studies, if applicable	14–15
Confidentiality	27	How personal information about potential and enrolled participants will be collected, shared, and maintained in order to protect confidentiality before, during and after the trial	12

Declaration of interests	28	Financial and other competing interests for principal investigators for the overall trial and each study site	15
Access to data	29	Statement of who will have access to the final trial dataset, and disclosure of contractual agreements that limit such access for investigators	13
Ancillary and post- trial care	30	Provisions, if any, for ancillary and post-trial care, and for compensation to those who suffer harm from trial participation	13
Dissemination policy	31a	Plans for investigators and sponsor to communicate trial results to participants, healthcare professionals, the public, and other relevant groups (eg, via publication, reporting in results databases, or other data sharing arrangements), including any publication restrictions	13–14
	31b	Authorship eligibility guidelines and any intended use of professional writers	14
	31c	Plans, if any, for granting public access to the full protocol, participant-level dataset, and statistical code	N/A
Appendices			
Informed consent materials	32	Model consent form and other related documentation given to participants and authorised surrogates	Appendix 2
Biological specimens	33	Plans for collection, laboratory evaluation and storage of biological specimens for genetic or molecular analysis in the current trial and for future use in ancillary studies, if applicable	N/A

Competing interests:
Nil.

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REFERENCES:

1. Ministry of Health. Long term conditions Wellington: Ministry of Health; 2016 [http://www.health.govt.nz/our-work/diseases-and-conditions/long-term-conditions].
2. OECD Health Data. Obesity and the Economics of Prevention: Fit not Fat. 2011.
3. Ministry of Health. Health Loss in New Zealand: A report from the New Zealand Burden of Diseases, Injuries and Risk Factors Study, 2006–2016. Wellington: Ministry of Health; 2013.
4. Ministry of Health. National Diabetes Work Programme 2014/15. Wellington: Ministry of Health; 2014.
5. Ministry of Health. Annual Update of Key Results 2014/15: New Zealand Health Survey Wellington: Ministry of Health; 2015 [http://www.health.govt.nz/publication/annual-update-key-results-2014-15-new-zealand-health-survey].
6. National Health Committee. Meeting the needs of people with chronic conditions: Hapai te whanau mo ake ake tonu. Wellington: Ministry of Health; 2007.
7. Statistics New Zealand. 2013 Census QuickStats about culture and identity. Wellington: Statistics New Zealand; 2014.
8. Ministry of Health. Annual Update of Key Results 2016/17: New Zealand Health Survey. Wellington: Ministry of Health; 2017.
9. Eyles H, Jull A, Dobson R, Firestone R, Whittaker R, Te Morenga L, et al. Co-design of mHealth Delivered Interventions: A Systematic Review to Assess Key Methods and Processes. Current Nutrition Reports. 2016:1–8.

10. Firestone R FT, Dalhousie S, Henry A, Vano M, Grey J, Jull A, Whittaker R, Te Morenga L, Ni Mhurchu C,. Identifying and overcoming barriers to healthier lives. Pacific Health Dialog. 2018; 21(2):54–66.
11. Wilson D. New perspectives on health disparities and obesity interventions in youth. J Pediatric Psychology. 2009; 34(3):231–44.
12. Counties Manukau District Health Board. LotuMoui Health Programme. Auckland: Counties Manukau District Health Board; 2010.
13. Scragg R, et al. Obesity in Pacific Island Communities. University of Auckland: Health Research Council of New Zealand; 2002.
14. World Health Organisation. mHealth: New horizons for health through mobile technologies. Geneva: World Health Organisation; 2011.
15. Ghorai K AS, Khatun F, et al. mHealth for Smoking Cessation Programs: A Systematic Review. J Pers Med. 2014; 4(3):412–23.
16. Whittaker R MH, Bullen C, et al. Mobile phone-based interventions for smoking cessation. Cochrane Database Syst Rev. 2016; 4(CD006611).
17. Verbiest M SB, Firestone R, Funaki T, Goodwin D, Grey J, Henry A, Hughes E, Humpfrey G, Jiang Y, Jull A, Pekepo C, Schumacher J, Te Morenga L, Tunks M, Vano M, Whittaker R, Ni Mhurchu C. A Code-signed, Culturally-Tailored mHealth Tool to Support Healthy Lifestyles in Māori and Pasifika Communities in New Zealand: Protocol for a Cluster Randomized Controlled Trial. JMIR Research Protocols 2018; 7(8):e10789.
18. Statistics New Zealand. QuickStats about Pacific peoples: 2006 Census. Wellington: Statistics New Zealand; 2010.
19. Verbiest M CC, Dalhousie S, Firestone R, Funaki T, Goodwin D, Grey J, Henry A, Humpfrey G, Jull A, Vano M, Pekepo C, Te Morenga L, Whittaker R, Ni Mhurchu C,. Using co-design to develop a culturally-tailored, behavior change mHealth intervention for indigenous and other priority communities: a case study in New Zealand. Transl Behav Med. 2018; 8:1–17.
20. Pulotu-Endemann K. Fonofale Model of Health. Auckland: Health Promotion; 2001.
21. Ni Mhurchu C, Te Morenga L, Tupai-Firestone R, Grey J, Jiang Y, Jull A, et al. A co-designed mHealth programme to support healthy lifestyles in Māori and Pasifika peoples in New Zealand (OL@-OR@): a cluster-randomised controlled trial. The Lancet Digital Health. 2019; 1(6):e298–e307.
22. Firestone R TH, Manukia M, Kaholokula K, Foliaki S, Kingi TK, Kruger R, Breier B, O'Connell A, Borman B, Ellison-Loschmann L. Understanding Pasifika youth and the obesogenic environment, Auckland & Wellington, New Zealand. NZ Med J. 2016; 129(1434).
23. Kaholokula JK, Nacapoy AH, Grandinetti A, Chang HK. Association between acculturation modes and type 2 diabetes among Native Hawaiians. Diabetes Care. 2008; 31(4):698–700.
24. Kaholokula JK, Nacapoy AH, Grandinetti A, Chang HK. Association between acculturation modes and type 2 diabetes among Native Hawaiians. Diabetes Care. 2008; 31(4):698–700.
25. Rothe E, Tzuang D, Pumariega AJ. Acculturation, Development, and Adaptation Child Adolesc Psychiatric Clin N Am 2010; 19:681–96.
26. Bedford R. Pacific Islanders in New Zealand. Espace Populations Sociétés. 1994:187–200.
27. Tiatia J. Pasifika Cultural Competencies: A literature review. Wellington: Ministry of Health; 2008.
28. Birman D. Acculturation and human diversity in a multicultural society. In: Trickett E, Watts R, Birman D, editors. Human diversity: Perspectives on people in context San Fancisco: JossevBass; 1994:261–84.
29. Berry JW. Conceptual approaches to acculturation. In: Chun KM, Organista PM, Mar´ın G, editors. Acculturation: Advances in theory, measurement, and applied research. Washington DC: American Psychological Association; 2003.
30. Chun KM, Organista PB, Mar´ın G. Acculturation: Advances in theory, measurement, and applied research. Washington DC: American Psychological Association; 2003.
31. Cespedes YM, Huey SJ Jr. Depression in Latino adolescents: a cultural discrepancy perspective. Cultur Divers Ethnic Minor Psychol 2008; 14(2):168–72.
32. Rogler LH. International migrations: a framework for directing research. Am Psychol 1994; 49:701–8.
33. Saldana DH. Acculturative stress and minority status. Journal of Behavioral Health Sciences 1994; 16:117–25.

From gorse to ngahere: an emerging allegory for decolonising the New Zealand health system

Heather Came, Isaac Warbrick, Tim McCreanor, Maria Baker

ABSTRACT

Prior to colonisation, Māori had a well-developed holistic health system based on maintaining balance between people, place and spirit. The colonial imposition of British economic, religious, educational, legal, health and governance, through warfare, immigration, legislation and social coercion had a devastating effect on Māori health outcomes. With the release of the WAI 2575 Waitangi Tribunal report exposing the failings of our health system in relation to Māori health, the need to decolonise our health system becomes more pressing. A key difficulty in this work is the poverty of transformative language, concepts and frameworks in our workforce. This paper is the product of an anti-racism think tank that occurred in April 2019. While working through a system change analysis on our colonial health system, Māori and Tauiwi activists and scholars created an allegory—*from gorse to ngahere*. The allegory depicts the ongoing impact of the colonial health system as represented by gorse, and the possibilities of a decolonised health system represented by ngahere—a self-sustaining and flourishing native forest. Racism has a geographic specificity. The allegory we developed is a mechanism for conceptualising decolonisation for the context of Aotearoa. It serves to reinforce the different roles and responsibilities of the descendants of the colonisers and the colonised in the pursuit of decolonisation.

The colonial health system in Aotearoa is failing Māori. This is evident through the findings of the landmark WAI 2575 report of the Waitangi Tribunal that found institutional racism and systemic breaches of Te Tiriti o Waitangi across the infrastructure of the health system, including in health legislation, policy, contracting, governance and investment practices of the Crown.[1] Ultimately the failings manifest as enduring ethnic health inequities.[2] This complex problem is not unique to Aotearoa—rather, it is a global challenge[3,4] facing colonial health systems and nation states committed to equity and social justice. Evidence shows political processes of colonisation and forced assimilation have devastated Indigenous health.[5,6] For example, dispossession and forceful detachment from ancestral lands, shifted access to healthy food and water, while diminishing cultural identity that was tied to the whenua where whānau had lived for many generations. The challenge before us now is how to reconfigure colonial power relations and to decolonise health systems.

There are loose parallels with the challenges facing Indigenous forest ecosystems—the ngahere—in the face of colonial economic development. How can we achieve the sustainable, equitable diversity vital to the future and well-being of the ngahere (and the population!) without destroying the economic fabric of society? Our allegory 'gorse to ngahere' is designed (recognising its limitations) to stimulate thinking about how to approach changing the colonial impacts of the health system on Māori. We hope that by looking to te taiao (the natural environment) for metaphor, models, and understanding relating to human wellbeing, as Māori and Indigenous epistemologies continue to do, that processes within the natural world

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will provide guidance for ways forward. In this particular narrative the introduced species gorse became an invasive weed that colonised vast areas of forest which Pākehā settlers cut and burned to make farmland. This is gradually replaced, with a regenerating Indigenous tree canopy. Such gradualism is inherent to the transformative processes envisaged by the landmark Matike Mai[7] report on constitutional transformation. This report developed through an extensive engagement process with Māori led by Moana Jackson and Margaret Mutu modelling a Te Tiriti o Waitangi and tikanga-based framework for decolonising the constitutional arrangements of the nation. Came, Baker and McCreanor[8] have articulated the possible implications of Matike Mai for the health sector.

Decolonisation

Decolonisation is both an individual and collective process of revealing and analysing the historic and contemporary impact of colonisation, and institutional racism, combined with political commitment towards the recognition of Indigenous sovereignty. Tuhiwai Smith[9] describes it is as a "…long-term process involving the bureaucratic, cultural, linguistic and psychological divesting of colonial power".

McGuire-Adams and Giles[10] argued decolonisation requires the development of critical consciousness about the cause of oppression, the distortion of history and the degrees to which one has colluded with and internalised deficit colonial ideology. One response to decolonisation is to focus on strengths and return to one's ancestors' teachings, values, ethics and knowledge, Such as Heke's[11] 'Atua-Matua Māori Health Framework', which realigns health and wellness with the characteristics of, and relationship to and between Atua Māori (Māori environmental deities). McGuire-Adams maintains decolonisation requires a refusal to victim-blame and to mindfully connect with ceremony, healing and a community of people to foster strength and wellbeing.

Processes dismantling colonisation can be peaceful, entail violent revolt or a mixed approach. Inspired by the revolutionary writings of Fanon, Freire and Said, decolonisation as an international movement has led to self-government for some and increased recognition of Indigenous peoples' rights for others. Such struggles have also resulted in people being harassed, prosecuted and killed in their efforts to achieve social, cultural, political and economic transformation.

The remainder of this paper, informed by conversations within our network, is our emerging allegory which likens the colonial health system in Aotearoa to gorse, and a decolonised Māori-centric health system to a ngahere.

Methodology

In April 2019 health activist group STIR: Stop Institutional Racism hosted an international think tank to explore how to decolonise the public health system in Aotearoa. The gathering was a mixture of Māori and Tauiwi activists, public health practitioners and academics who were committed to strengthening our collective efforts to disrupt institutional racism. The weekend was led by visiting scholar Derek Griffith from the US, with Grant Berghan and Heather Came, then co-chairs of STIR. We worked through a systems change analysis,[12] a preferred method for dealing with complex problems that conventional approaches have proven unable to transform.

The group talked extensively about the current colonial-dominated health system, its administration, and operations. This kōrero (conversation) was informed by decades of Māori and Tauiwi experience working within the health system, engaging in health activism and working within the Academy to generate evidence about how institutional racism manifests within the health system.

Findings

Ngahere

"I am the forest and the forest is me."[13]

Māori have intimate, longstanding, interdependent relationships with the whenua, awa, moana and ngahere in Aotearoa. 'Whenua' also means placenta in Te Reo Māori, and Māori are Tangata Whenua (people of the land), highlighting the attachment of Māori to place. In Māori lore, Tāne Mahuta is the atua (guardian or deity) of the forest and birds, and many life forms in the ngahere, both flora and fauna, are his tamariki (children).

Figure 1: Ngahere.

Photo: Denis Came-Friar.

The ngahere has its own mauri (essence or life-force) and in some areas is home to supernatural beings known as patupaiarehe. These elements influenced how Māori prepared for and conducted themselves in the ngahere. Karakia (incantations, prayers) were conducted and specific locations were avoided. The ngahere was a source of "…spiritual enrichment, cognitive development, reflection and physical health and aesthetic experiences".[13]

The ngahere was a mahinga kai (food gathering place) where birds, freshwater fish, tuna and koura, and plant foods like pikopiko, mauku and tawa berries, were abundant. The ngahere was also an important source of rongoā (Māori medicines) such as kawakawa and kumarahou, timbers for boats and dwellings, and a source of healing.

Māori observed the ngahere and other features of the environment for survival and applied the knowledge gained from the ngahere into all facets of life. For example, when a leader passed away, they were likened to a Tōtara—a native tree known for its strength and height—that had fallen in the forest "Kua hinga te Tōtara i te wao nui a Tāne". Similarly, one could say of someone that was particularly expert in a given field "E kore e mau i a koe, he wae kai pakiaka"—You will not catch the feet accustomed to running among the roots.

The ngahere also represent tribal histories. Māori feel connected to ngahere that contain pūrākau (stories) and where great feats were accomplished, or where tūpuna (ancestors) met and fell in love. The ngahere was and still is an intergenerational mechanism for transferring knowledge about mauri ora across physical, mental, spiritual and collective planes of wellness.

The ngahere has its own mana (prestige) and to trample it is not appropriate. However, for some iwi (tribes) ngahere are the descendants of Tane and therefore tuakana (elder siblings) to human beings. Such peoples are more likely to feel grief and bewilderment at the objectification, exploitation and subsequent annihilation of these kindred communities.

Figure 2: Gorse.

Photo: Denis Came-Friar.

Gorse

The arrival of Pākehā and the subsequent colonisation of Aotearoa led to significant changes in land usage, in keeping with the European philosophy that land was profitable property.[14] Trees were cut for timber at scale and forest was burned to convert to grass, transforming landscapes, devastating waterways and decimating animal and plant populations. Introductions of European plants and animals both deliberate and accidental meant invasive species such as gorse, broom, pine, rats, cats, pigs and deer added to the destruction. Similarly, the arrival of European perspectives on health, health resource distribution and profitability in their health system, severely damaged Māori practices, destroyed their support systems and halved the Māori population by 1900.[15] The focus on 'progress' at the expense and disregard of interconnected and holistic systems (ie, the destruction of ngahere and entire ecosystems because they 'get in the way' of farming and other 'productive' activities) is also reflected in dominant approaches to health. Physical aspects of 'health', or more specifically the absence of illness, have become the focus of most health systems and services, at the expense of social, cultural and spiritual aspects of wellbeing (perhaps due to the commercial potential of the 'illness industry'). While treating and reducing physical illness is also an important part of Māori health aspirations, like the widespread and thorough burning and replacement of ngahere with gorse, the refusal to include Māori views and perspectives in the treatment of physical illness is widespread and opportunities to grow Māori-led solutions up through the gorse of the health system are few.

The European ideology of domination of environments as distinct from Indigenous practices of conservation, balance and sustainability, clashed from the earliest of contacts. Until quite recently, before the emergence of conservation movements, Pākehā discourse undercut the value of environmental resources; we spoke of bush, scrub, swamps and creeks in a manner that marginalised and minimised their value compared to pasture, fields, orchards and plantations. The former were seen as wild, 'waste lands' to be owned, fenced, cleared, tamed and transformed into the latter. It's not difficult to see this same ideology pervading health in Aotearoa, where Māori are portrayed in media and common discourses as being obese, choosing poor diets, having violent relationships, and raising children in the worst of social conditions, while also suggesting that 'new' medicines or 'modern' interventions are the solution to 'Māori problems'.

In 2005 some 29% of Aotearoa remained forested, with 6.3 million hectares of native ngahere remaining.[16] *Not by Wind Ravaged,* a poem by Hōne Tuwhare, speaks of the devastation to the landscape that created the conditions that allowed gorse to thrive.

Deep scarred
not by wind ravaged nor rain
nor the brawling stream
stripped of all save the brief finery
of gorse and broom and standing
sentinel to your bleak loneliness
the tussock grass—
O voiceless land let me echo your desolation.[17]

Gorse is a woody evergreen legume, part of the plant family Fabaceae (see Figure 2), that forms invasive thickets. It is highly flammable and can be used as fuel and is a nitrogen-fixing plant—when it dies it releases nitrogen which helps fertilise the soil. Likewise, the colonised health system is one that has fuelled the flames of political and social outrage many times in the past, with neo-liberal ideology pointing toward the lazy and incompetent Māori and their poor choices, as the source of our collective health woes.

Introduced to Aotearoa in the 1800s to make decorative hedges and wind shelter or fencing for stock and crops, it unexpectedly flourished in our temperate landscape.[18] Price argued planting of hedges was motivated by a European aesthetic of humanising and dominating the landscape. Gorse quickly adapted and became an aggressive invasive species through flowering twice a year here, compared with annual flowering in Europe. The new plantings were an uncontrolled experiment and once established, successfully competed with and displaced native plants. By the 1940s gorse was recognised as a serious noxious weed and by the 1970s some 700,000 hectares were covered in gorse nationwide.[19]

Gorse seeds can lie dormant on the ground for up to 40 years and then can germinate quickly when conditions become favourable. It has an aggressive seed dispersal system which allows for rapid regeneration, while modification of vegetation cover, soil disruption and fire increase seed germination.

"Gorse colonises bare ground… Approximately 6,000–18,000 fertile seeds are produced annually from mature individuals that develop approximately 1,000 flowers per branch… soil seedbank size can exceed 10,000 seeds per m^2."[20]

Millions of dollars each year are invested in attempting to control and contain gorse with inconsistent results. Multiple sustained efforts to contain its spread, including i) chemical, ii) biological controls such as weevils, spider mite, fungi and thrips and controlled burn-offs, and iii) mechanical removal over decades have only achieved partial control.

Gorse has proven resilient to herbicides due to the thick cuticles on its spines which help prevent absorption, similar to the colonial health system's resistance to Māori worldviews, and solutions to current health challenges. Burnt stumps of gorse can readily sprout new growth, and fire can encourage germination of seeds if the temperature is not hot enough or sustained sufficiently. Similarly, when social difficulties arise, racism seems to sprout new seeds, which spreads the ideology throughout a population.

In permaculture terms, gorse acts as a nursery plant so is useful in native bush regeneration. When gorse is young it creates a low protective canopy in which native plant seeds can germinate and grow, enriching the soil by fixing nitrogen. This allows Indigenous plant seedlings to thrive and grow up through the gorse, cutting out its access to light and eventually replacing it. To thrive, gorse requires full sunlight. Similarly, the colonial health system requires constant reinforcement of colonial perspectives and ideals. To regenerate bush, you can clear small areas and plant pioneer species (kanuka, manuka, toetoe or hebe) which are fast-growing, acid soil loving plants. New trees will have eradicated gorse within 10–15 years; a technique that has been used successfully in the Hinewai Reserve on the Banks Peninsula.[21]

Māori have deforested some areas and the evidence from historical sources points to cultivations at a scale that produced surpluses that sustained thriving international trade until the end of the 1850s.[22] Pākehā have over time come to value and

revere pristine forest, shrublands, wetlands and waterways and sought to preserve, protect and restore elements and areas of the ecosphere they have taken over. The weeds, predators and pests along with the human economic and cultural imperatives of colonisation are key threats to the health, diversity and sustainability of ngahere. In our analogy we let 'gorse' stand for the combination of things that colonisation has wrought.

Discussion

The presence of gorse and the compromised state of the ngahere is a symptom of a profound imbalance in the landscape. This ecological imbalance, like the imbalance of ethnic inequities and racism, needs to be corrected to benefit *all* those that live in this whenua. Gorse eradication like the eradication of institutional racism is a wicked problem that needs to be addressed from multiple fronts, using the collective and individual spheres of influence of many within the health system.

Until the ngahere can be restored, and decolonisation occurs interim power sharing arrangements need to be put in place. Came, O'Sullivan, Kidd and McCreanor[23] argued that given the health sector's non-compliance with Te Tiriti o Waitangi, engaging with the WAI 2575 report recommendations is a matter of some urgency. As Wilson,[24] the visionary behind Hinewai Reserve, has argued, gorse can provide an interim protective canopy for shade-loving native plants. Likewise, non-Māori can assist Māori in decolonising the health sector by providing safe environments where Māori health practices and initiatives can be restored, and where Māori health leaders and workers can develop without the racism that is frequently tied to Māori ways of doing things. Any sustained transformation will require time and vision and depend on political will, tenacity and capacity.

In terms of gorse eradication[20] noted that many land managers do not have the time or resources to dedicate to successfully control gorse by traditional means; particularly if the seedbank will be full again in a few seasons. There are those within the health sector that will argue that the sector is underfunded and we don't have the resources to address racism.[25] Leaving aside for now the political question of whether the public health system is underfunded, the costs of inaction in the face of racism for Māori whānau who are disproportionately carrying the burden of disease is simply morally and ethically unacceptable.[26] Like the ngahere struggling to rise above a pervasive gorse canopy, Māori will continue to struggle to achieve good health and flourish without an acknowledgment of the barriers that stifle that progress, and action against the colonial ideology that perpetuates such barriers.

The authors maintain decolonisation work should be a normalised part of the core everyday work of health practitioners, their managers, policy makers and political leaders. For this to occur it is timely to refresh professional competency documents,[27] the *Health Practitioners Competency Assurance Act 2003* [28] and tertiary health curriculum, Ministry of Health and district health board policy and practices.

Broadfield and McHenry[20] have argued when targeting invasive species such as gorse, that it is important to target the root cause of the invasion rather than the symptoms. This aligns well with the arguments put by Came and Griffith[29] that in order to address institutional racism a planned systems change approach is needed. Ad hoc efforts by committed individuals are unlikely to achieve sustained change. Now that institutional racism within the health system is acknowledged,[1,30,31] we need to plan to eradicate it and oftentimes the best way to eradicate the gorse is by nurturing ngahere.

Māori have been actively engaging in restoring ngahere and decolonising the health system,[32] from tikanga and kaupapa driven approaches and initiatives reconnecting to culture and mātauranga Māori (traditional Māori knowledge). 'Mainstream' initiatives have also been redesigned to better align with Māori perspectives, while Māori have been building kaupapa Māori organisations and developing culturally targeted health interventions for decades.[33] Professor Sir Mason Durie[34] has proposed a clear framework and vision for Māori health leadership going forward. After extensive engagement with Māori, Matike Mai Aotearoa[7] have articulated what a Te Tiriti-compliant constitution might look like and challenged Pākehā to engage, while others[35]

have proposed a shift toward traditional beliefs and environmental knowledge as the drivers for health.

Conclusion

We concur with Freire that there are different tasks for the descendants of the colonisers and the colonised. The restoration of ngahere is work that needs to be led by Māori. Pākehā who are used to being in control for the last 170 years, need to surrender and trust Māori intelligence and Māori solutions. As allies, Pākehā can support the rejuvenation of the ngahere by actively taking away things like the gorse that stops the regeneration process. Personally mediated, cultural, historical and institutional racism are fundamental barriers to the achievement of decolonisation. Championing compliance with Te Tiriti o Waitangi is another potentially fruitful contribution.

Competing interests:
Nil.

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REFERENCES:

1. Waitangi Tribunal. (2019). Hauora report on stage one of the health services and outcomes inquiry. Wellington, New Zealand: Author.
2. Williams M, Cram F, Te Huia B, Te Huia T. (2019). WAI 2575: Background history: Scoping report. Wellington, New Zealand: Ministry of Health.
3. Anderson I, Robson B, Connolly M, Al-Yaman F, Bjertness E, King A, Yap L, et al. Indigenous and tribal peoples' health (The Lancet–Lowitja Institute Global Collaboration): a population study. The Lancet, 2016; 338(10040):131–157. doi:10.1016/S0140-6736(16)00345-7
4. Paradies Y. Colonisation, racism and indigenous health. Journal of Population Research, 2016; 33(1):83–96. doi:10.1007/s12546-016-9159-y
5. Moewaka Barnes H, McCreanor T. Colonisation, hauora and whenua in Aotearoa. Journal of the Royal Society of New Zealand, 2019; 49(sup1):19–33. doi:10.1080/03036758.2019.1668439
6. Reid P, Cormack D, Paine S-J. (2019). Colonial histories, racism and health—The experience of Māori and Indigenous peoples. Public Health. doi:10.1016/j.puhe.2019.03.027
7. Matike Mai Aotearoa. (2016). He whakaaro here whakaumu mō Aotearoa. New Zealand: Author.
8. Came H, Baker M, McCreanor T. (In press). Structural racism, constitutional transformation and the New Zealand health sector: Learnings from the Matike Mai Aotearoa report. Journal of Bioethical Inquiry.
9. Smith L. (1999). Decolonizing methodologies: Research and indigenous peoples. Dunedin, New Zealand: University of Otago Press.
10. McGuire-Adams TD, Giles AR. Anishinaabekweg Dibaajimowinan

(Stories) of Decolonization Through Running. Sociology of Sport Journal, 2018; 35(3):207–215. doi:10.1123/ssj.2017-0052

11. Heke I. (2016). Introducing the Atua Matua Māori Health Framework. Unpublished Document, available from: http://toitangata. co. nz/uploads/files/Dr_Ihi_Heke_Atua_Matua_Framework. pdf
12. Stroh D. (2015). Systems thinking for social change. Vermont, USA: Chelsea Green Publishing.
13. Lyver P, Timoti P, Jones C, Richardson S, Tahi B, Greenhalgh S. An indigenous community-based monitoring system for assessing forest health in New Zealand. Biodiversity & Conservation, 2017; 26(13):3183–3212. Retrieved from http://ezproxy.aut.ac.nz/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edb&AN=126197921&site=eds-live
14. Moewaka Barnes H, Eich E, Yessilth S. Colonization, whenua and capitalism: experiences from Aotearoa New Zealand. Continuum: Journal of Media & Cultural Studies, 2018; 32(6):685–697. doi:10.1080/10304312.2018.1525918
15. Waitangi Tribunal. (2011). Ko Aotearoa tenei: A report into claims concerning New Zealand law and policy affecting Māori culture and identity (WAI 262). Wellington, New Zealand: Author.
16. Statistics New Zealand. (2005). Forests and forest products. Retrieved from http://archive.stats.govt.nz/browse_for_stats/environment/environmental-economic-accounts/forests-and-forest-products-2005.aspx
17. Tuwhare H. (1977). No ordinary sun. Dunedin, New Zealand: John McIndoe Ltd.
18. Price L. Hedges and Shelterbelts on the Canterbury Plains, New Zealand: Transformation of an Antipodean Landscape. Annals of the Association of American Geographers, 1993; 83(1):119–140. Retrieved from http://ezproxy.aut.ac.nz/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edsjsr&AN=edsjsr.2569418&site=eds-live
19. Blaschke PM, Hunter GG, Eyles GO, Van Berkel PR. Analysis of New Zealand's vegetation cover using land resource inventory data. New Zealand Journal of Ecology, 1981; 4:1–19. Retrieved from www.jstor.org/stable/24052600
20. Broadfield N, McHenry MT. A World of Gorse: Persistence of Ulex europaeus in Managed Landscapes. Plants (2223-7747), 2019; 8(11):523–544. Retrieved from http://ezproxy.aut.ac.nz/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=edb&AN=139789906&site=eds-live
21. Wilson H, McDonald T, Lamb D. Forest regeneration on Hinewai Reserve, New Zealand: An interview with Hugh Wilson. Ecological Management & Restoration, 2017; 18(2):92–102. doi:10.1111/emr.12261
22. Dawson J. (2007). Conifer–broadleaf forests - Loss of conifer–broadleaf forests. Retrieved from http://www.TeAra.govt.nz/en/interactive/11674/deforestation-of-new-zealand
23. Came H, O'Sullivan D, Kidd J, McCreanor T. The Waitangi Tribunal's WAI 2575 report: Implications for decolonizing health systems. Health and Human Rights, 2020; 22:209–220.
24. Ryan K. (2019). Gorse for the trees: How one man brought back a forest (Interview with Hugh Wilson). Retrieved from http://www.rnz.co.nz/national/programmes/ninetonoon/audio/2018703481/gorse-for-the-trees-how-one-man-brought-back-a-forest
25. Akoorie N. (2018). Underfunding of health system leads to $249m blowout - Health Minister. Retrieved from http://www.nzherald.co.nz/nz/news/article.cfm?c_id=1&objectid=12128639
26. Mills C, Reid P, Vaithianathan R. The cost of child health inequalities in Aotearoa New Zealand: A preliminary scoping study. BMC Public Health, 2012; 12(384). doi:10.1186/1471-2458-12-384
27. Came H, Kidd J, Heke D, McCreanor T. (Under review). Te Tiriti o Waitangi compliance in regulated health practitioner competency documents in Aotearoa: A Critical Tiriti Analysis.
28. Health Practitioners Competence Assurance Act 2003 (S.N.Z No.48.), 2003 S.N.Z No.48.
29. Came, Griffith D. Tackling racism as a "wicked" public health problem: Enabling allies in anti-racism praxis. Social Science & Medicine, 2017; 199:181–188. doi:10.1016/j.socscimed.2017.03.028
30. Health Quality & Safety Commission. (2019). He matapihi ki te kounga o ngā manaakitanga ā-hauora o Aotearoa 2019:

A window on the quality of Aotearoa New Zealand's health care. Wellington, New Zealand: Author.

31. Ministry of Health. (2018). Achieving equity in health outcomes: Highlights of important national and international papers. Wellington, NZ: Author.

32. Emery-Whittington I, Te Maro B. Decolonising occupation: Causing social change to help our ancestors rest and our descendants thrive. New Zealand Journal of Occupational Therapy, 2018; 65(1):12–19. Retrieved from http://ezproxy.aut.ac.nz/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=c-cm&AN=129422879&site=eds-live

33. Gifford H, Batten L, Boulton A, M, C, Cvitanovic L. Delivering on outcomes the experience of Maori health service providers. Policy Quarterly, 2018; 14(2):59–64.

34. Durie M. (2019). Whakaahu whakamua: Decades of Māori advancement. Paper presented at the Toi tu Hauora 2019, Wellington, New Zealand.

35. Warbrick I, Dickson A, Prince R, Heke I. The biopolitics of Māori biomass: towards a new epistemology for Māori health in Aotearoa/New Zealand. Critical Public Health, 2016; 26(4):394–404. doi:10.1080/09581596.2015.1096013

Nurse prescribing in New Zealand—the difference in levels of prescribing explained

Jane Key, Karen Hoare

ABSTRACT

This article discusses the three types of nurse prescriber currently registered in New Zealand (nurse practitioners, registered nurse prescribers (RNP) in primary health and specialty teams and registered nurse prescribers (RNPCH) in community health). It also provides an overview of the evolution of each group, as well as a summary of the current legislation, prescribing restrictions and models of supervision required for each type of prescriber.

New Zealand has been late in implementing nurse prescribing. Towards the end of the 20th century non-medical prescribing was introduced into many westernised countries, notably in the UK, where nurses have been prescribing for decades.[1,2] The situation regarding the late introduction of nurse prescribing in New Zealand, is a curious one. In 2006, there were only five nurse practitioners prescribing in New Zealand (the only group who were eligible to prescribe at the time), which was in part due to objections raised regarding the safety to the public of these professionals and future nurse prescribers.[3] One commentator at that time highlighted that there were more registered nurse prescribers in the UK than there were doctors registered with New Zealand's General Medical Council.[4] Since then, the numbers and levels of nurses prescribing in New Zealand have substantially increased along with other groups of non-medical prescribers such as pharmacists and optometrists.[2] This article explains the evolution and nomenclature of the different levels of nurse prescribing in New Zealand and the legislation under-pinning each of the three levels (see Tables 1 and 3). Additionally, the prerequisites, education, competencies and registration of the three levels are defined along with the intent of each prescriber's role and the clinical contexts. The discussion will be drawn from current New Zealand legislation as well as professional guidelines published by the Nursing Council of New Zealand (NCNZ), who are the responsible agency for setting educational and professional standards for nurses in New Zealand.

Authorised versus designated prescribers

In order to discuss nurse prescribing it is first necessary to clarify two pertinent terms used in the New Zealand legislation; authorised and designated prescribers. Authorised prescribers may *independently* prescribe, supply, sell, administer or arrange for the administration of any medicine that relates to their area of practice.[1] Current authorised prescribers include nurse practitioners, optometrists, practitioners (dentist or medical practitioner), registered midwives or veterinarians.[1] Designated prescribers, on the other hand, may only prescribe from a list of medicines published in the New Zealand Gazette by the Director-General of Health under section 105(5A) of the Medicines Act.[1] Designated prescribers are also expected to prescribe *collaboratively* alongside an authorised prescriber and have limited permission to diagnose (only minor

Table 1: Legislation pertaining to prescribing.

Legislation	Description
Health Practitioners Competence Assurance Act (2003) (HPCA)[3]	The intent of the HPCA aims to protect the public from harm at the hands of healthcare professionals (HCP). It delegates the responsibility for enacting this to Responsible Agencies (RAs) for each profession. The RA for nursing is the Nursing Council of New Zealand (NCNZ). Under the HPCA, the titles of HCP may only be used by those who have met the standards of and are currently registered with the relevant RA.
Medicines Act 1981[1]	Defines the terms medicine, new medicine, prescription medicine and restricted medicine. Regulates medicines, related products and medical devices in New Zealand. It also outlines the legislative framework for prescribing prescription medicines and the groups of health professionals able to prescribe (includes definitions of authorised and designated prescribers).
Medicines Regulations 1984[4]	Outlines the classification of medicines, and lists the medicines in each category. It also regulates the quality, advertising, labelling, production, transport, prescribing and dispensing conditions, licensing, withdrawal, data sheets and includes schedule of medicines. Section 41 outlines the legal requirements for all prescriptions.
Medicines (Standing Order) Regulations 2002[5]	A Standing Order is a generic prescription that allows non-prescribing health professionals to make drug administration decisions as per prescribed criteria. Authorised prescribers can issue and oversee standing orders, designated prescribers cannot.
Medicines (Standing Order) Amendment Regulations 2016	The above regulations were amended by an Order in Council on 11 July 2016 that allowed nurse practitioners and optometrists to issue Standing Orders.
Medicines (Designated Prescriber: Nurse Practitioners) Regulations 2005[6]	Now revoked and replaced section by the Medicines Amendment Act 2013.[7]
Medicines (Designated Prescriber— Registered Nurses Practising in Diabetes Health) Regulations 2011[8]	Now revoked and covered by The Medicines (Designated Prescriber-Registered Nurses) Regulations 2016.[9]
Medicines Amendment Act 2013[7]	Amends the Medicines Act 1981—added nurse practitioners to the list of authorised practitioners who can prescribe medicines that lie within their scope of practice—giving them equivalence to doctors, dentists and midwives.

Table 1: Legislation pertaining to prescribing (continued).

The Medicines (Designated Prescriber-Registered Nurses) Regulations 2016[9]	The purpose of these regulations is: • to authorise registered nurses who meet specified requirements for qualifications, training and competence to be designated prescribers for the purpose of prescribing specified prescription medicines; and • to provide for the qualifications, training and competence requirements; and • to prohibit registered nurses from prescribing specified prescription medicines if they fail to comply with the requirements; and • to make non-compliance with the requirements an offence.
Misuse of Drugs Act 1975[10]	Legislative framework for controlled drugs.
Misuse of Drugs Regulations 1977[11]	Outlines licensing, permissions, restrictions and prescribing of controlled drugs. Allows designated nurse prescribers (primary and specialty care) to prescribe specified controlled drugs from Schedule 1A. Section 29 sets out requirements for controlled drug prescriptions.
Amendment to the Misuse of Drugs Regulations 2014[12]	Designated prescribers may prescribe from Schedule 1A only. Regulation 29 updated requirements for controlled drug prescriptions to include electronic prescriptions (approved).
Misuse of Drugs Amendment Act 2016[13]	Sets out the circumstances under which patients with addictions may be prescribed controlled drugs (generally only for those working in addition services and after specific application).

Table 2: Examples of contexts suitable for nurse prescribers (not an exhaustive list).

Registered nurse with designated prescribing rights (primary health and specialty teams)[2]	**Registered nurse with designated prescribing right (community health)**[22]	**Nurse practitioner**[23]
Primary care or nurse specialist nurse-led clinics (chronic conditions) • Hypertension • Diabetes • Heart failure • Asthma • COPD • Gout • Eczema • Depression • Anxiety • Palliative care Health promotion • Immunisations • Contraception All must have access to an authorised prescriber in order to prescribe. Other areas may be suitable but the list of medicines that can be prescribed may not be pertinent.	• Public health nurses • School nurses • Community health nurses All must have access to an authorised prescriber in order to prescribe.	NPs can diagnose and prescribe independently so they can work anywhere there is service need for the role.

ailments and illnesses, eg, those that can be confirmed with a simple diagnostic test such as a UTI).[2] Current designated prescribers include pharmacist prescribers, dietitian prescribers and RN prescribers.[1] Table 1 lists all New Zealand legislation that pertains to nurse prescribing in New Zealand.

The following section will discuss each of the three types of nurse prescribers registered in New Zealand [nurse practitioners, registered nurse prescribers (rnp) in primary health and specialty teams and registered nurse prescribers (RNPCH) in community health] and Table 3 summarises the legal and prescribing status of the three types of nurse prescriber in New Zealand.

Nurse practitioners—highest level

In 2001, the first nurse practitioners (NPs) were registered with the Nursing Council of New Zealand (NCNZ), some of whom had limited (designated) prescribing rights.[14] The numbers of NPs were slow to increase over the following decade, due in part to the onerous process to register with NCNZ and the lack of job opportunities following registration.[15, 16] However, in the last few years streamlining the registration process along with increased employment opportunities has led to an increase in the numbers of NP registrations. In 2013, the Medicine Amendment Act listed NPs as authorised prescribers, with near identical prescribing rights to doctors and dentists (See Table 1).[7] Currently there are 465 registered NPs (current on 10 June 2020, figures from NCNZ register).

Nurse practitioners are registered nurses who have been conferred with an additional registration by the NCNZ, following completion of an approved clinical Master's degree. The clinical Master's programme must include bioscience, pharmacology, advanced assessment/diagnostic reasoning and a prescribing practicum (300–500 hours of supervised practice).[17] Under the Health Practitioners Competence Assurance Act, NCNZ is responsible for ensuring that only those who are competent to practice independently are registered as NPs.[1,3] NPs are permitted to diagnose and prescribe independently and autonomously; they can procure, supply and administer medications and prescribe any medicines relevant to their population group.[1,4] NPs work as a sole provider or within a team/service and do not require supervision by a medical practitioner, although supervision by a NP or medical practitioner is recommended in their first year of practice. There are no limitations to the type of presentation or disease that NPs can manage. They are required to undergo regular continuing professional development and participate in self and peer review.[18] Responsibility for ensuring competence and patient safety lies with the individual NP and NCNZ.

The intent of the NP role is to provide high-level expert nursing care combined with diagnostic and treatment skills commonly associated with medical practitioners. As clinical leaders, they influence policy, address inequity by improving access to healthcare for all New Zealanders and role model best practice in patient care.[18]

Registered nurse prescribers (RNP) in primary health and specialty teams—middle level

During 2011, registered nurses (RN) specialising in diabetes care were piloted in four sites around New Zealand following a legislation change that gave them limited authority to prescribe.[8] Evaluation of the project described these nurses as providing safe, high-quality prescribing decisions.[19] A further legislation change in 2016 allowed NCNZ to register RN prescribers working in primary care and other specialty areas who had completed a Post-Graduate Diploma, which included a prescribing practicum (150 hours of supervised prescribing practice by an authorised prescriber). Subsequent to the enaction of this new act in 2016, newly registered RNPs working in diabetes care came under the umbrella term of RNPs in primary health and specialty teams. RNPs are described as designated prescribers and the limitations on their prescribing are summarised in Table 3. RNPs work collaboratively with an authorised prescriber and may only prescribe within that collaborative relationship.[1,2,9] RNPs prescribe for a discreet list of conditions and adhere to a specific list of medicines published by the NCNZ.[20] Some of the medicines on this list have been deemed suitable for continuation prescribing (which differs from a repeat prescription as the patient must be assessed face to face and allows for dose adjustments as required).[20]

Table 3: Comparison of nurse prescribers.

	Registered nurse with designated prescribing rights (primary health and specialty teams)	**Registered nurse with designated prescribing right (community health)**	**Nurse practitioner**
Education	Post-Graduate Diploma (including RN prescribing practicum)	Completion of an approved work-based learning package	Clinical Master's degree in advanced nursing practice (including NP prescribing practicum)
Type of prescriber	**Designated prescriber**	**Designated prescriber**	**Authorised prescriber**
Conditions they can prescribe for?	The specific common and long-term conditions nurses can prescribe for include **diabetes** and related conditions, **hypertension**, **respiratory diseases** including asthma and COPD, anxiety, **depression**, **heart failur**e, **gout**, **palliative care**, **contraception**, **vaccines**, **common skin conditions** and **infections**. Any diagnostic uncertainty must be discussed with or referred to an authorised prescriber.	They may prescribe where the diagnosis has already been made (eg, rheumatic fever secondary prevention), where the diagnosis is relatively uncomplicated (eg, determined through laboratory testing) or for minor ailments or illnesses. Any diagnostic uncertainty must be discussed with or referred to an authorised prescriber.	Able to independently assess, diagnose and prescribe for a **population group or context**. May work autonomously or within a healthcare organisation. Consults with health professional colleagues when relevant. There are **no** limitations to conditions that may be prescribed for. NPs are expected to use their professional and clinical judgement about presentations and patients that are outside their level of knowledge and skillset.
Model of prescribing	**Collaborative prescribing**	**Collaborative prescribing**	**Independent/autonomous prescribing**
What medicines can they prescribe?	May only prescribe from the published medicines list for registered nurse prescribers in primary and specialty care from NCNZ.[20] Some restrictions related to route, form and context have been included in the list.	May only prescribe from the published medicines list for registered nurse prescribers in community health from NCNZ.[24] Some restrictions related to route, form, duration and context have been included in the list.	May prescribe any medicines within their scope of practice, knowledge and competence.
Can they issue repeat prescriptions?	Only after face-to-face assessment (if covered by the medicines list). A small number of medications are deemed suitable for continuation prescribing in the list (where dose adjustments may be necessary) but the RN prescriber must assess the patient face-to-face. These medicines must be initiated by an authorised prescriber.	Only after face-to-face assessment (if covered by the medicines list). Continuation prescribing for Valaciclovir only (but the RN prescriber must assess the patient face-to-face). and this medication must be initiated by an authorised prescriber.	Yes. Provided they have sufficient knowledge about the patient's history and current status to do this safely.
Can they prescribe controlled drugs?	A registered prescriber may prescribe from a limited schedule (1A) of controlled drugs to a patient under their care for a period of seven days ONLY.[11] (Additional prescribing can be granted by NCNZ (upon application) to those working in addition services.)	No	Yes. Same as medical practitioner.

Table 3: Comparison of nurse prescribers (continued).

	Registered nurse with designated prescribing rights (primary health and specialty teams)	Registered nurse with designated prescribing right (community health)	Nurse practitioner
What duration of treatment may be prescribed?	Schedule 1A controlled drugs for seven days.[11] Up to three months' supply of other prescription, restricted medicines, pharmacy-only medicines from the medicines list (unless otherwise stated). Up to six months' supply of an oral contraceptive.[20]	The medicines list for nurse prescribers in community health limits many medications to a single dose or course.	Same as medical practitioner.
Prescribe unapproved medicines?	May only prescribe from the published *Medicines List.*[20] Unapproved medicines are not included in this list. A small number of medicines that are commonly prescribed for unapproved uses have been included in this list.	May only prescribe from the published *Medicines List*.[24] Unapproved medicines are not included in this list.	May prescribe any medicines relevant to their areas of practice. They prescribe within their scope of practice, knowledge and competence.[25] *Currently, section 29 medications cannot be dispensed by a pharmacist unless prescribed by a medical practitioner.*
***ISSUE* Standing Orders?**	Designated prescribers are not permitted to issue standing orders	Designated prescribers are not permitted to issue standing orders	Yes
Able to issue verbal orders for medicines?	No	No	Yes
Order diagnostic tests?	It is expected that RN prescribers are able to order tests that will inform their prescribing.	Yes, limited to their prescribing, eg, wound, throat swabs.	Yes

The intent of the RNP role, is to prescribe within an existing or pre-determined diagnosis, although NCNZ does allow for RNPs to make simple diagnoses such as urinary tract and skin infections.[2] However, RNPs are not expected to demonstrate the same diagnostic skills as medical and nurse practitioners and are required to have oversight from an authorised prescriber who is readily accessible to examine the patient if required.[21] While there is an associated workload for authorised prescribers to supervise RNPs, it is arguably more satisfying than overseeing standing orders. There are clear expectations in terms of governance, audit, ongoing education requirements and peer review for workplaces who employ RNPs.[2] Other restrictions to RNP prescribing are described in Table 3.[2] As of 31 March 2020, there were 59 diabetes nurse prescribers and 213 primary health and speciality teams nurse prescribers registered with NCNZ.

Registered nurse prescribing in community health (RNPCH)—lowest level

In 2019, a third group of nurse prescribers were created; RN prescribers in community health (RNPC). They are also classed as designated prescribers and registered by NCNZ following successful completion of a workplace toolkit.[9,22] The list of medicines they can prescribe from is very limited and the duration of the prescription is for a single dose or course.[24] Like RNPs, RNPCs must work and prescribe collaboratively with and be supervised by authorised prescribers.

The intent of this role is to address inequity in primary care provision and to promote population health by providing access to care and expediting treatment of conditions such as group A streptococcal pharyngitis or impetigo.[22] As with

RNPs, these prescribers are not expected to diagnose anything other than simple ailments. As of 31 March 2020, there were 60 community nurse prescribers registered with nursing council.

To allow further comparisons and clarification, Table 2 summarises some appropriate contexts for each type of prescriber and Table 3 summarises the main differences between the three groups.

Discussion

NPs have the same autonomous diagnosing and prescribing rights as medical practitioners, which allows them to work flexibly and independently in any number of contexts. They can diagnose and treat all first presentations of patients within their knowledge and skillset and do not require medical oversight. In addition, they are expert nurses with the associated knowledge and skills. Despite these attributes NPs still face barriers to employment and restrictions in some practice settings.

Arguably the numbers registered are not commensurate with the needs of the New Zealand population, particularly in primary healthcare.[15] RNPs are well placed to run nurse-led clinics for chronic conditions and some specialty services where the diagnosis is already established and the medicines list they prescribe from is pertinent. Utilising them to see purely first presentations is not impossible but requires RNPs to discuss all but the simplest of cases with an authorised prescriber. RNPCs can prescribe limited medications for simple conditions in uncomplicated patients. Both RNPs and RNCPs require an authorised prescriber to be freely available or to work in tandem with them. The added supervisory burden to the authorised prescriber must be factored into the service delivery model and resourcing. It should also be noted that this model places the accountability for the *diagnosis* of all discussed patients with the supervising authorised prescriber, whereas the prescribing accountability remains with the RN prescriber.

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Nil.

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REFERENCES:

1. Medicines Act 1981, Stat. 118 (NZ). http://www.legislation.govt.nz/act/public/1981/0118/latest/DLM53790.html (accessed 28 July 2020).
2. Nursing Council of New Zealand. Preparation and guidance for employers and registered nurses prescribing in primary health and specialty teams. 2020.
3. Health Practitioners Competence Assurance Act 2003, Stat. 118 (NZ). http://www.legislation.govt.nz/act/public/2003/0048/latest/DLM203312.html (accessed 28 July 2020).

4. Medicines Regulations 1984, Stat. 143 (NZ). http://www.legislation.govt.nz/regulation/public/1984/0143/latest/DLM95668.html (accessed 28 July 2020).
5. Medicines (Standing Order) Regulations 2002, Stat. 373 (NZ). http://www.legislation.govt.nz/regulation/public/2002/0373/10.0/DLM170107.html (accessed 28 July 2020).
6. Medicines (Designated Prescriber: Nurse Practitioners) Regulations 2005, Stat. 266 (NZ). http://www.legislation.govt.nz/regulation/public/2005/0266/latest/whole.html (accessed 28 July 2020).
7. Medicines Amendment Act 2013, Stat. 141 (NZ). http://www.legislation.govt.nz/act/public/2013/0141/latest/DLM4096106.html (accessed 28 July 2020).
8. Medicines (Designated Prescriber-Registered Nurses Practising in Diabetes Health) Regulations 2011, Stat. 54 (NZ). http://www.legislation.govt.nz/regulation/public/2011/0054/latest/DLM3589235.html (accessed 28 July 2020).
9. Medicines (Designated Prescriber-Registered Nurses) Regulations 2016, Stat. 140 (NZ). http://www.legislation.govt.nz/regulation/public/2016/0140/11.0/DLM6870521.html (accessed 28 July 2020).
10. Misuse of Drugs Act 1975, Stat. 116 (NZ). http://www.legislation.govt.nz/act/public/1975/0116/latest/DLM436101.html (accessed 28 July 2020).
11. Misuse of Drugs Regulations 1977, Stat. 37 (NZ). http://www.legislation.govt.nz/regulation/public/1977/0037/latest/whole.html (accessed 28 July 2020).
12. Amendment to the Misuse of Drugs Regulations 2014, Stat. 199 (NZ). http://www.legislation.govt.nz/regulation/public/2014/0199/latest/whole.html (accessed 28 July 2020).
13. Misuse of Drugs Amendment Act 2016, Stat. 80 (NZ). http://www.legislation.govt.nz/act/public/2016/0080/11.0/DLM6984401.html (accessed 28 July 2020).
14. Ministry of Health, Nursing Council of New Zealand, DHBNZ, NPACNZ. Nurse Practitioners: A healthy future for New Zealand. 2009.
15. Carryer J, Adams S. Nurse practitioners as a solution to transformative and sustainable health services in primary health care: A qualitative exploratory study. Collegian. 2017; 24:525–31.
16. Hoare K, Francis K, Millls J. Reflective thought in memos to demonstrate advanced nursing practice in New Zealand. Reflective Practice. 2012; 13:13–25.
17. Nursing Council of New Zealand. Education programme standards for the nurse practitioner scope of practice. 2017.
18. Nursing Council of New Zealand. Nurse Practitioner Scope of Practice Guidelines for Applicants. 2019.
19. Wilkinson J, Carryer J, Adams J. Evaluation of a diabetes nurse specialist prescribing project. Journal of Clinical Nursing. 2014; 23:2355–66.
20. Nursing Council of New Zealand. Medicines list for registered nurse prescribing in primary health and specialty teams. 2018.
21. Nursing Council of New Zealand. Scope of practice for registered nurses. Edition., cited June 3 2020]. Available from: http://www.nursingcouncil.org.nz/Public/Nursing/Scopes_of_practice/Registered_Nurse/NCNZ/nursing-section/Registered_nurse.aspx
22. Nursing Council of New Zealand. Guideline for registered nurses prescribing in community health (managed roll out 2019). 2019.
23. Nursing Council of New Zealand. Nurse Practitioner Scope of practice for nurse practitioners. Edition., cited June 3 2020].Available from: https://www.nursingcouncil.org.nz/Public/Nursing/Scopes_of_practice/Nurse_practitioner/NCNZ/nursing-section/Nurse_practitioner.aspx
24. Nursing Council of New Zealand. Medicines list for registered nurse prescribing in community health (Managed Rollout 2019). 2019.
25. Medsafe. Compliance: Use of Unapproved Medicines and Unapproved Use of Medicines which includes advice on meeting the Health and Disability Services Code of Consumer Rights. 2014.

The case for a bicultural dementia prevalence study in Aotearoa New Zealand

Sarah Cullum, Makarena Dudley, Ngaire Kerse

As the world's population ages, the prevalence of dementia is projected to increase from the current 50 million to 130 million in 2050.[1] The present cost of dementia is over one trillion US dollars and is expected to double in 10 years.[1] In response, the World Health Organization (WHO) has declared dementia a global public health priority[2] and has called on all 194 member states to produce a national dementia plan or strategy for 2017–2025.[3]

In Aotearoa New Zealand, there are estimated to be currently over 60,000 people living with dementia and this number is projected to reach 170,000 by 2050.[4] The annual national cost of dementia is estimated to be $1.7 billion NZD, and projected to increase to $2.7 billion NZD by 2030.[4] The Ministry of Health has acknowledged the major economic challenge of dementia but, to date, there has been no planning to address the rapidly increasing future demands of dementia on our health and social care systems,[5] nor the psychological and economic consequences on whānau and families living with dementia. This is partly due to the fact that the figures given above are only estimates, extrapolated from other countries' dementia prevalence data[4] because there has never been a dementia prevalence study in Aotearoa New Zealand. The Government requires accurate New Zealand-specific data to inform its spending and policy decisions, which would only be available from a carefully conducted epidemiological survey.

Aotearoa New Zealand is officially recognised as a bicultural (Māori and non-Māori) nation. In recognition of the Treaty of Waitangi, attention to equity in health and social services is mandated, and this applies as much to dementia as it does to other health conditions. Globally, there is increasing recognition that dementia outcomes differ across different communities, thus research designed for different cultures is required rather than a 'one size fits all' approach.[6] For that reason we present in this viewpoint the justification for a bicultural dementia prevalence study in Aotearoa New Zealand.

Evidence that dementia may be different for Māori

The sparse research evidence that is available suggests that Māori may present with dementia up to 10 years earlier than NZ Europeans.[7] This might be expected as recent studies have found that a considerable proportion of dementia is attributable to modifiable risk factors such as diabetes, hypertension and obesity,[8] risk factors that are more common and present earlier in Māori.[9,10] The ongoing impact of colonisation and its sequelae contribute to an increased risk of dementia for Māori.[11] Socioeconomic disadvantage, such as less access to education and healthcare, and discrimination are more prevalent in Māori communities.[12,13] These socioeconomic risk factors are also linked to dementia and will increase the likelihood of negative outcomes for Māori.[14] On the other hand, despite higher levels of comorbidity, Māori with dementia presenting at a memory service in South Auckland had a lower age-adjusted risk of mortality compared to NZ Europeans,[15] which suggests the possibility of a different aetiology that might be responsive to different and potentially more effective treatment options. These questions can only be answered by a community-based dementia prevalence study.

The impact of dementia on Māori whānau (families) is also significant. Recent research[16–18] and interRAI data[19] suggest that care arrangements and caregiver input are disproportionately higher in Māori

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whānau. The current societal structure where economic success dominates, coupled with poor access to, and culturally inappropriate, public services for kaumātua (Māori elders) means that traditional care practices, where the person is cared for at home, add to whānau burden as family members forgo paid work. A national dementia prevalence study would document potential disparity for Māori and provide evidence for the impact of dementia on whānau, which will inform the future development of culturally responsive services.

Measuring dementia in a bicultural prevalence study

The largest epidemiological study of dementia in Māori was conducted as part of the *Life and Living in Advanced Age*, a Cohort Study in New Zealand (LILACS NZ).[20] LiLACS NZ engaged over 400 Māori aged 80–90 and 500 non-Māori aged 85 years in 2010 and has actively followed up study participants for five years. Careful validation of the dementia screening tool, in this case the 3MS[21] showed that a different cut point was needed for Māori, as the screening tool developed for NZ European populations overestimated the likelihood of dementia in Māori.[22] To allow equal comparison across groups, a national dementia prevalence study would therefore require diagnostic assessment tools that are both scientifically robust and not biased by culture. Such tools have been used in cross-country comparison of dementia prevalence worldwide. The most frequently used tool is the 10/66 dementia protocol,[23] which is considered to be the global gold standard for comparative dementia epidemiology. It is a dementia diagnostic assessment tool that is relatively unbiased by language or culture, and therefore can be adapted for use in communities outside of the UK where it was developed.[24] The 10/66 dementia protocol takes approximately 90 minutes to administer and includes an interview with the main participant and an informant (the main participant's co-resident or main caregiver) to assess care arrangements, caregiver burden and the economic cost to the family. These are sensitive issues in some cultures and consequently the tool requires adaptation, translation and revalidation for each cultural group. Studies to develop and validate the tool have taken place in Latin America, China, India, Nigeria and South Africa and more recently in higher income countries such as Singapore. It has achieved excellent results against a gold standard diagnosis: sensitivity (94%) and specificity (97% in high education controls and 94% in low education controls).[24]

A bicultural prevalence study ensures inclusion of dementia-related outcomes that incorporate values that are important to Māori communities. Recent research contributes to this body of knowledge. The study 'Kaumātuatanga ō Te Roro (The Ageing Brain)' conducted 17 focus groups with 223 kaumātua (Māori elders) throughout Aotearoa New Zealand and the findings demonstrated that Māori understanding of mate wareware (dementia) includes its effect on the wairua (spiritual dimension) of Māori.[16] The roles of aroha (love, compassion), manākitanga (hospitality, kindness, generosity, support, caring) and cultural activities are important elements of care. The output of this research is being used to develop a Māori-responsive assessment tool for the clinical diagnosis of dementia that includes the assessment of wairua, aroha, manākitanga and cultural roles.

A proposed bicultural dementia prevalence study in Aotearoa New Zealand: aims and methods

The aim of a community-based study in Aotearoa New Zealand would be to measure the extent of dementia in the older population and its associated health and sociodemographic risk factors, plus its impact of dementia on individuals, their families and larger society. The methods for a bicultural national dementia prevalence study would require two parallel arms of data collection, one for Māori and one for non-Māori. Both arms would include the following elements in common to ensure comparability across groups: (i) a culturally unbiased diagnostic assessment, (ii) sampling procedures that ensure adequate representation of Māori, and (iii) robust engagement with Māori and non-Māori communities to ensure successful recruitment to the study. In addition, the Māori arm would require a Māori-centred approach that would incorporate a combination of mātauranga Māori (Māori knowledge) and western science

knowledge.[25] Kaupapa Māori methodology, a unique approach to research that reflects the philosophies, values and practices of Māori, would inform all aspects of engagement with Māori.[26] There would be Māori leadership and collaborative and consultative input from Māori groups to ensure Māori aspirations and outcomes are central to the research.

A culturally unbiased diagnostic assessment: development and testing

A bicultural dementia prevalence study will require a culturally unbiased diagnostic assessment that can be used to accurately compare findings across both Māori and non-Māori arms of the study. The 10/66 dementia protocol has been demonstrated to be a suitable instrument but, as it was developed in the UK, would need to be adapted for use in Māori and translated into te reo Māori. This would involve an iterative review process with refinement by a Māori advisory panel (including a bilingual dementia specialist) to confirm cultural acceptability, conceptual validity and tolerability. The adapted tool would then be piloted in a sample of Māori families to ensure that it is acceptable, before testing its diagnostic accuracy. The diagnostic accuracy of the Māori-adapted version of the 10/66 dementia protocol would be blindly tested against a 'gold standard' dementia assessment in Māori with and without dementia, and their whānau. Once demonstrated to have validity it could be used in the proposed dementia prevalence study.

The subcomponents of the English version of the 10/66 dementia protocol have been used in multiple research studies in the UK but not in NZ Europeans. Accordingly, we intend to also evaluate the cultural appropriateness and acceptability of the original English version in a planned feasibility study that will include community-dwelling NZ Europeans. The feedback we receive from NZ Europeans and other major New Zealand ethnic groups will help inform a future dementia prevalence study.

Sampling procedures that ensure adequate representation of Māori

The sampling method for a representative population-based sample would be similar to the methods used in the New Zealand Mental Health Study Te Rau Hinengaro.[27] This consisted of a mesh-block sampling frame (the smallest unit for which Statistics NZ has demographic information, comprising approximately 60–100 people) and door-to-door knocking for recruitment. Under ideal circumstances the prevalence study would be conducted in several different geographical areas based on socioeconomic deprivation indices to ensure adequate representation of Māori and non-Māori from all socioeconomic backgrounds, in addition to sampling in both urban and rural areas of North and South Island. This would ensure that sufficient Māori are included to allow equal sampling power for accurate estimations of prevalence. Census data for the selected areas could be used to calculate the probability of finding dementia cases in adults aged 65 years or older, and then oversampling for Māori to ensure adequate representation. As an example of sample size calculation, we used census data to discover that approximately 6,800 Māori and 31,000 NZ European people aged 65 and over were living in South Auckland at the time of the 2013 census. Based on a probable 10/66 dementia prevalence of 10%, we estimated that sample sizes of approximately 750 Māori and 850 NZ European people aged 65 or over would be required in South Auckland to generate prevalence estimates with an acceptable degree of certainty.

Dementia presents up to 10 years earlier in Māori compared to NZ Europeans[7] therefore, it would be preferable to extend our cohort to include 55–65 year olds. However this would double the cost of the study because there as many Māori aged 55–64 years as there are >65 years old and the prevalence of dementia in the younger age group is lower. An alternative option would be to establish a separately funded younger cohort that would enable thorough investigation of dementia incidence and risk factors in these populations.

Community engagement and recruitment

Communities would need to be involved in design of the project to encourage ownership of the project by the community it aims to benefit. Dementia is still a misunderstood disorder so engagement with local

communities to provide education would be essential. Activities such as dementia roadshows at community venues allow the audience to fully interact, ask questions and learn about dementia; this approach raises awareness and interest in the study itself. These could be held at sites that serve older people from both non-Māori and/or Māori communities such as local marae, churches and organisations providing services for older people. Community groups can co-design the best ways to connect with local families and whānau using different strategies such as traditional media, social media and community activities. In addition, we intend to work with relevant NGOs such as Alzheimers New Zealand, Dementia New Zealand, Age Concern, as well as newly formed National Māori Dementia Advisory Group, organisations that have agreed to support the study.

The recruitment of participants to a dementia prevalence study will involve door-knocking in pre-selected areas to establish ages and ethnicities of people over 65 years living in each household, and to determine whether the household would be willing to have a researcher return to conduct the 10/66 interview. Effective recruitment at this stage of the study will be crucial for the success of the intended prevalence study and the generalisability of its findings, therefore it is essential that cultural safety is observed. Māori door-knockers and interviewers will be required in accordance with kaupapa Māori methodology. A key concept of cultural safety is the recognition of the unequal distribution of power inherent in relationships.[28] The use of ethnic-matched door-knockers is essential to safeguard the cultural safety of the public who may feel less empowered with a person who is not of the same ethnic background.

Māori-specific measures and methods

Māori philosophy is based on a holistic approach to health and wellbeing. Te Whare Tapa Wha is one such model that encompasses four cornerstones of health including wairua (spirituality), whānau (family), hinengaro (mind) and tinana (body), and provides an appropriate framework for understanding dementia from a Maori perspective.[29] In research studies, Māori researchers must be involved at all stages, from research leadership, through study design and conduct. Processes of the study are governed by tikanga such as karakia (prayer) to start and finish meetings, whakawhanaungatanga (relationship building) when initially meeting potential participants, and manākitanga (caring of participants). The project is overseen and guided by a Rōpu Kaitiaki (Māori guidance group) consisting of kaumātua (Māori elders) who will oversee the cultural safety of the research and play an integral role in facilitating community relationships.

Impact of the findings of a bicultural dementia prevalence study in Aotearoa New Zealand

This viewpoint argues the need for and describes the groundwork required to conduct a bicultural dementia prevalence study in Aotearoa New Zealand. The study findings would describe the current extent and impact of dementia for Māori and non-Māori families, highlighting any potential disparities across ethnic groups, with each group containing enough participants for ethnic specific analyses. Furthermore, the findings would inform the development of interventions that would hopefully make a positive difference to Māori and non-Māori families living with dementia.

Culturally appropriate service provision

Considerable work is needed to address disparities in health outcomes for Māori at all levels.[30,31] Accurate information is needed to understand inequity, and develop Māori-specific responses.[32,33] In recent years the focus of dementia research has shifted from cure to prevention and care, and particularly support for carers.[8] To support people with dementia in New Zealand, we need to support the families and whānau that look after them. One successful strategy in the UK, STrAtegies for RelaTives (START), uses psychological therapies to develop individually tailored and cost-effective coping strategies for carers of people with dementia. START reduces anxiety and depressive symptoms of carers for at least six years.[34] To date, these approaches have not been considered, adapted or implemented in whānau living in Aotearoa New Zealand,

nor have therapeutic and care models been developed by Māori for Māori. A well-designed bicultural dementia prevalence study would provide data to start this process and address the government goals of reducing inequalities in health and social outcomes.

Cost of dementia for Māori and non-Māori

The inclusion of survey questions asking about the direct and indirect costs of care could provide valuable data regarding the current cost of providing support for people living with dementia in Aotearoa New Zealand. Findings may highlight potential inequities in care provision and cost, not only for health and social care services but also for families and whānau. The prevalence of dementia is predicted to triple in the next three decades. Simulation modelling will help to estimate the costs of providing culturally appropriate services to families and whānau living with dementia, and to develop a model projecting financial and organisational demands under different assumptions of dementia prevalence, care pathways and service models.

Non-Māori living in Aotearoa

In addition to Māori and NZ Europeans, numbers of older people are rising rapidly in other ethnic groups living in Aotearoa, in particular Pasifika, Chinese and Indian,[4] with an associated increase in dementia prevalence in these populations. Consequently, there is a need for more epidemiological information about dementia in these groups too. Our intention is to adapt the instruments and methods described above to enable adequate representation of Pasifika, Chinese and Indian older people, as well as Māori and NZ Europeans in future dementia prevalence studies.

Conclusion

A bicultural dementia prevalence study in Aotearoa New Zealand would provide population-based data and projected costs of dementia for Māori and non-Māori. Health inequities for Māori will be described, and data will be available to begin Māori-informed and developed responses. The data will help inform the Ministry of Health in responding to identified needs with culturally appropriate dementia services, as well as improving public awareness and reducing stigma. If successful, the methods could be extended to other non-Māori communities. This will strengthen development of an up-to-date national dementia plan for Aotearoa New Zealand.

Competing interests:
Nil.

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REFERENCES:

1. Prince M, Wimo A, Guerchet M, et al. World Alzheimer Report 2015: the global impact of dementia, an analysis of prevalence, incidence, costs and trends. London: Alzheimer's Disease International. London; 2015.
2. World Health Organisation. Dementia: a public health priority. United Kingdom; 2012.
3. World Health Organisation. Global action plan on the public health response to dementia 2017-2025. Geneva: World Health Organization; 2017.
4. Deloitte Access Economics. 2017. Updated Dementia Economic Impact Report 2016, New Zealand. Alzheimers New Zealand.
5. http://www.alzheimers.org.nz/getattachment/Our-voice/Policy-documents-and-submissions/Alzheimers-NZ-Advice-for-Budget-2019-(1).pdf (Accessed 05/05/2019)
6. Brayne C, Miller B. Dementia and aging populations—A global priority for contextualized research and health policy. PLOS Med. 2017; 14(3):e1002275.
7. Cullum S, Mullin K, Zeng I, et al. Do community-dwelling Māori and Pacific peoples present with dementia at a younger age and at a later stage compared with NZ Europeans? Int J Geriatr Psychiatry. 2018; 33(8):1098–1104.
8. Livingston G, Sommerlad A, Orgeta V, et al. Dementia prevention, intervention, and care. Lancet. 2017; 390:2673–734.
9. Joshy G, Simmons D. Epidemiology of diabetes in New Zealand: revisit to a changing landscape. N Z Med J. 2006; 119:U1999.
10. Feigin VL, Krishnamurthi RV, Barker-Collo S, et al. 30-Year Trends in Stroke Rates and Outcome in Auckland, New Zealand (1981–2012): A Multi-Ethnic Population-Based Series of Studies. PLoS. 2015; 10(8):1–28.9.
11. Reid P, Cormack D, Paine SJ. Colonial histories, racism and health - the experience of Māori and Indigenous peoples. Public Health, 2019; 172:119–124. http://dx.doi.org/10.1016/j.puhe.2019.03.027
12. Harris R, Tobias M, Jeffreys M, et al. Effects of self-reported racial discrimination and deprivation on Māori health and inequalities in New Zealand: cross-sectional study. Lancet, 2006; 367:2005–2009.
13. Cormack D, Harris R, Stanley J. Investigating the Relationship between Socially-Assigned Ethnicity, Racial Discrimination and Health Advantage in New Zealand. PLoS ONE, 2013; 8(12):e84039. http://dx.doi.org/10.1371/journal.pone.0084039
14. Curtis E, Harwood M, Riddell T, et al. Access and society as determinants of ischaemic heart disease in indigenous populations. Heart, lung & circulation, 2010; 19(5–6):316–324. http://dx.doi.org/10.1016/j.hlc.2010.04.129
15. Cullum S, Varghese C, Coomarasamy C, et al. Predictors of mortality in Māori, Pacific Island and European patients diagnosed with dementia at a New Zealand Memory Service. Int J Geriatr Psychiatry. 2020; 35(5):516–524. doi: 10.1002/gps.5266. Epub 2020 Feb 19.
16. Dudley M, Menzies O, Elder H, et al. (2019) Mate wareware: Understanding 'dementia' from a Māori perspective. NZ Med J, 2018; 132(1503):66–74.
17. Gott M, et al. 'No matter what the cost': a qualitative study of the financial costs faced by family and whānau caregivers within a palliative care context. Palliat Med. 2015a; 29(6):518–28.
18. Moeke-Maxwell T., Mason K., Toohey F., Dudley J. (2019) Pou Aroha: An Indigenous Perspective of Māori Palliative Care, Aotearoa New Zealand. In: MacLeod R., Van den Block L. (eds) Textbook of Palliative Care. Springer, Cham. https://doi.org/10.1007/978-3-319-77740-5_121
19. InterRAI NZ Annual Report 2016–17. New Zealand 2017. http://www.interrai.co.nz/assets/Documents/Publications-and-Reports/Annual-Report-2016-17-web-version.pdf (Accessed 24/04/2020)
20. Kerse N, Teh R, Moyes S, et al (2015). Cohort Profile: Te Puawaitanga o Nga Tapuwae Kia Ora Tonu, Life and Living in Advanced Age: a Cohort Study in New Zealand (LiLACS NZ). International Journal of Epidemiology, 2015; 44(6):1823–1832. 10.1093/ije/dyv103
21. Jones T, Schinka J, Vanderploeg R, et al. 3MS normative data for the elderly. Archives of Clinical Neuropsychology, 2001; 17(2):171–177.
22. Zawaly K, Moyes S, Wood P, et al. Diagnostic accuracy of a global cognitive screen for Māori and non-Māori octogenarians. Alzheimer's and Dementia: Translational Research and Clinical Interventions, 2019; 5:542–552. 10.1016/j.trci.2019.08.006

23. Prince M, Ferri CP, Acosta D, et al. The protocols for the 10/66 dementia research group population-based research programme. BMC Public Health. 2007; 7:165.

24. Prince MJ. The 10/66 dementia research group - 10 years on. Indian J Psychiatry. 2009;51 Suppl 1:S8–S15.

25. Cunningham C. A framework for addressing Māori knowledge in research, science and technology. Pacific Health Dialog, 2000; 7(1):62–69.

26. Hudson M, Milne M, Reynolds P, Russell K, Smith B. (2010). Te ara tika. Guidelines for Māori research ethics: a framework for researchers and ethics committee members.

27. Wells JE, Oakley Browne MA, Scott KM, et al. Te Rau Hinengaro: the New Zealand Mental Health Survey: overview of methods and findings. Aust N Z J Psychiatry. 2006; 40:835–44.

28. Richardson S, Williams T. Why is cultural safety essential in health care. Med Law, 2007; 26(4):699–707.

29. Durie M. (1982). Te Whare Tapa Whā model. In Paper delivered at Hui Taumata and shared as part of training to the New Zealand Psychologists Conference Palmerston North, New Zealand, March 10th.

30. Cormack D, Reid P, Kukutai T. Indigenous data and health: critical approaches to 'race'/ethnicity and Indigenous data governance. Public Health, 2019; 172:116–118. http://dx.doi.org/10.1016/j.puhe.2019.03.026

31. Curtis E, Jones R, Tipene-Leach D, et al. Why cultural safety rather than cultural competency is required to achieve health equity: a literature review and recommended definition. International Journal for Equity in Health, 2019;18(1):174. http://dx.doi.org/10.1186/s12939-019-1082-3

32. Reid P, Paine SJ, Curtis E, et al. Achieving health equity in Aotearoa: strengthening responsiveness to Māori in health research. New Zealand Medical Journal, 2017; 130(1465):96–103.

33. Reid P, Paine SJ, Te Ao B, et al. Estimating the economic costs of ethnic health inequities: Protocol for a prevalence-based cost-of-illness study in New Zealand (2003–2014). BMJ Open, 2018; 8(6):e020763.

34. Livingston G, Manela M, O'Keeffe A, et al. Clinical effectiveness of the START (STrAtegies for RelaTives) psychological intervention for family carers and the effects on the cost of care for people with dementia: 6-year follow-up of a randomised controlled trial. Br J Psychiatry. 2019; 216(1):35–42.

A rare presentation of Eagle syndrome

Hemisha Patel, James Sanders, Matthew Phillips, Aideen NI Mhuineachain, Julian White

Eagle syndrome was first described by Otolaryngologist Watt W Eagle in 1937 as a set of symptoms associated with an elongated styloid process.[1] It is divided into two main presentations, the first being classic Eagle syndrome with symptoms including odynophagia, otalgia and a foreign body sensation when swallowing, and the stylocarotid form, presenting with neurological symptoms such as visual loss, motor weakness and transient ischaemic attack (TIA), or stroke owing to compression, with possible ensuing dissection, of the internal carotid artery (ICA).[2]

A 39-year-old male was admitted to our stroke unit with blurred vision, right-sided weakness and dysarthria. He was otherwise fit and well, and on no regular medications. He had a CT brain on arrival, which was normal, followed by a carotid ultrasound, which revealed a distally occluded left ICA. An MRI brain showed numerous small ischaemic strokes in the left frontal and parietal lobes, confined to the left anterior and middle cerebral artery territories (Figure 1). A CT angiography of the neck vessels revealed bilateral ICA dissections as well as bilaterally elongated styloid processes of 48mm (Figure 2). Given his age, lack of risk factors and absence of any alternative stroke etiology despite an exhaustive workup, the diagnosis of bilateral carotid artery dissections secondary to Eagle syndrome was made.

Following discussion at a multidisciplinary meeting with neurologists and ENT surgeons present, the patient had an external approach resection of his right styloid process. A total of 30mm of the styloid process, as well as the calcified stylohyoid ligament, was removed (Figure 3). The left styloid process was resected six months following initial surgery, and at the subsequent six-month stroke clinic follow-up, a repeat CT angiography revealed complete recanalisation of the occluded left ICA.

Figure 1: Diffusion weighted MRI scan demonstrating areas of cortical infarct following carotid artery compression.

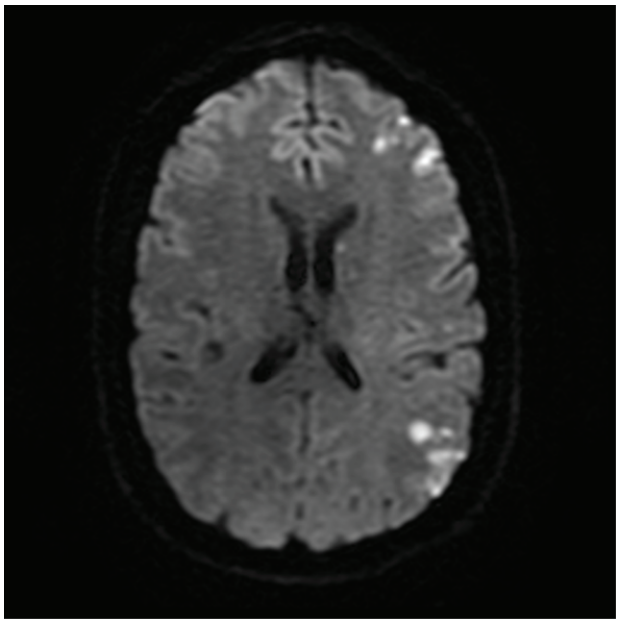

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Figure 2: CT reconstruction scan representing bilateral elongated styloid process.

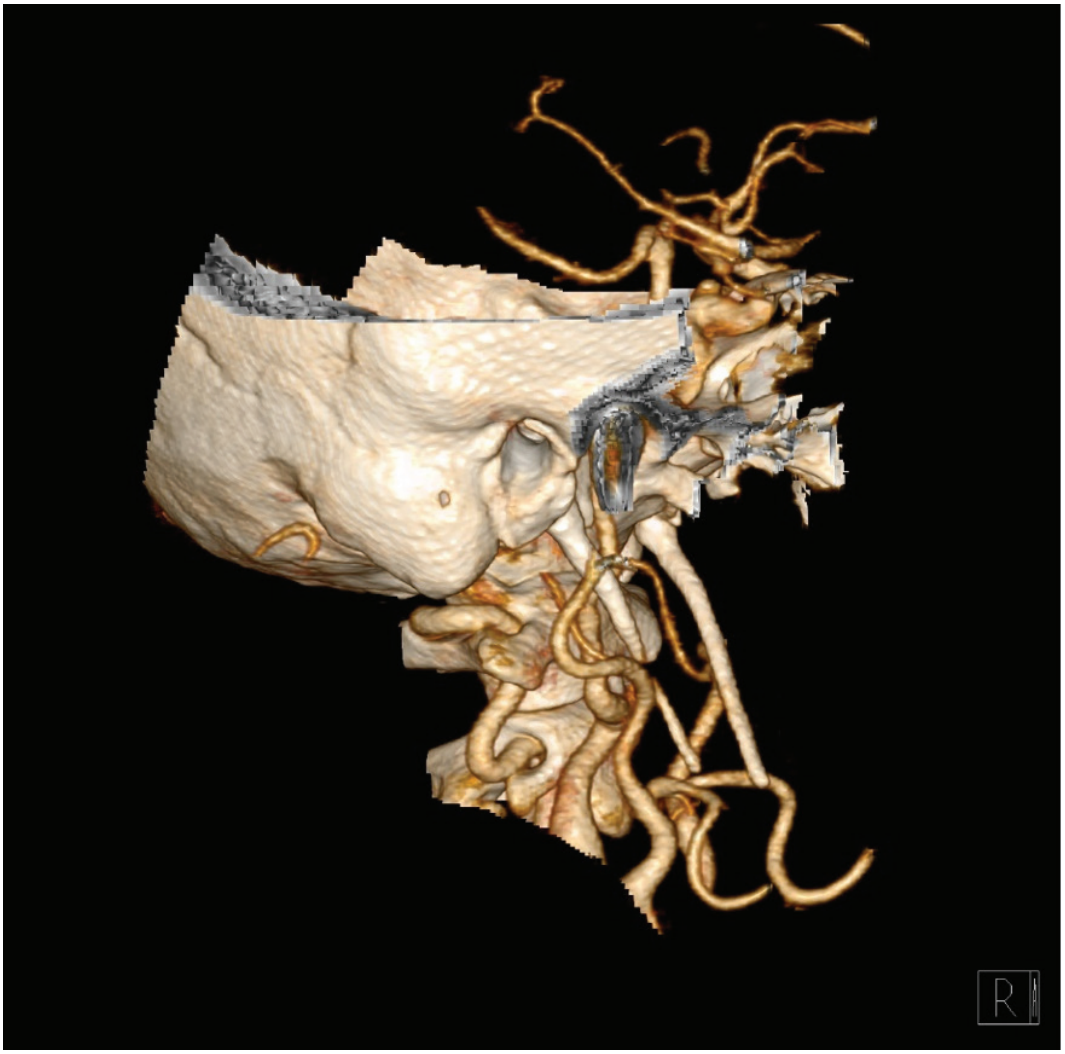
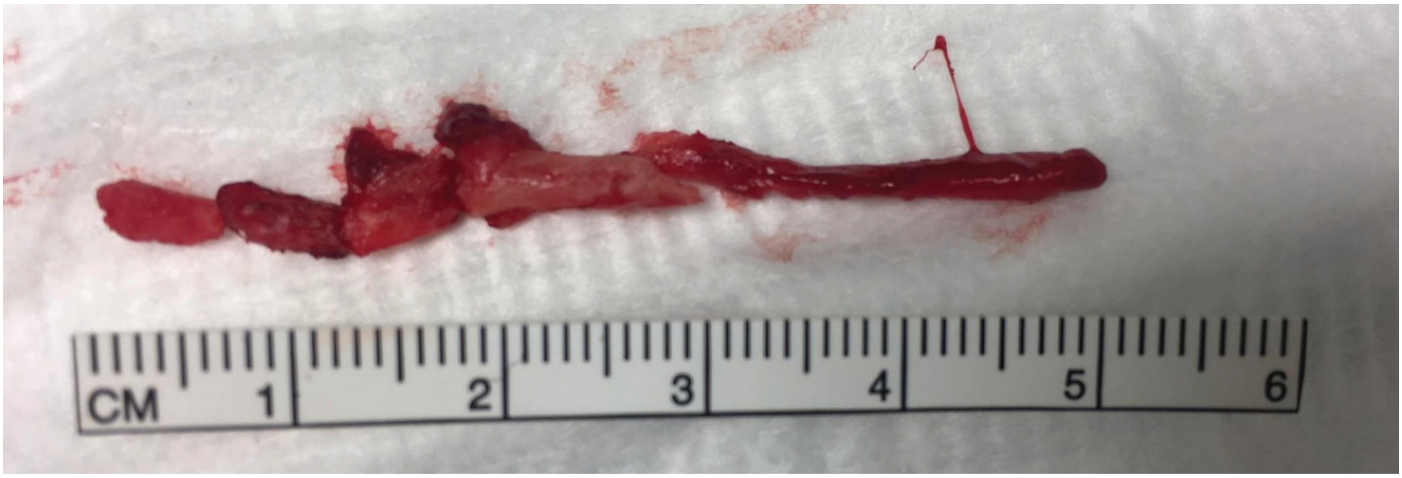


Figure 3: 30mm stylohyoid bone removed in fragments with associated calcified stylohyoid ligament.



Eagle described an elongated styloid process as greater than 30mm.[3] The diagnosis of Eagle syndrome is based on history and examination. The gold standard for diagnosis is a CT scan, which provides better bone definition and valuable information regarding surrounding structures, particularly regarding the length and angulation of the styloid processes.[4]

Eagle syndrome may be managed conservatively or surgically. Conservative management is reserved for patients with mild symptoms or strong contraindications to surgery, and consists of simple analgesics such as non-steroidal anti-inflammatory drugs, corticosteroid injections, anticonvulsants and antidepressants.[5]

The definitive treatment for Eagle syndrome is surgery, which may be performed via the intraoral approach or the cervical approach. The intraoral approach involves a tonsillectomy followed by careful blunt dissection and fracturing of the styloid process. Advantages include no external scar and the short procedure time. Disadvantages include incomplete exposure of the styloid process, poor exposure to control bleeding given the proximity to the carotid artery and pharyngeal venous plexus, infection and airway oedema.[5] Therefore, bilaterally elongated styloid processes are a relative contraindication to this approach.

In contrast, the external cervical approach involves an oblique incision from below the angle of the mandible. Dissection is carried out until the styloid process is palpated and removed. Advantages include better exposure of the surrounding vessels and nerves. Disadvantages include an external scar, longer operating time, and risk of damage to the marginal mandibular branch of the facial nerve.[5]

Clinicians must be aware of the potential association between an elongated styloid process and carotid artery dissection in patients presenting with a TIA or stroke. Given that Eagle syndrome is often treatable, its prompt recognition may confer a significant benefit to the patient by preventing further ischaemic neurological events.

Competing interests:
Nil.

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REFERENCES:

1. Eagle W. ELONGATED STYLOID PROCESSES: Report of Two Cases. Archives of Otolaryngology - Head and Neck Surgery, 1937; 25(5):584–587.
2. Eagle W. ELONGATED STYLOID PROCESS: Further Observations and a New Syndrome. Archives of Otolaryngology - Head and Neck Surgery, 1948; 47(5):630–640.
3. Badhey A, Jategaonkar A, Anglin Kovacs A, Kadakia S, De Deyn P, Ducic Y, Schantz S, Shin E. Eagle syndrome: A comprehensive review. Clinical Neurology and Neurosurgery, 2017; 159:34–38.
4. Subedi R, Dean R, Baronos S, Dhamoon A. (2017). Carotid artery dissection: a rare complication of Eagle syndrome. BMJ Case Reports, p.bcr2016218184.
5. Thoenissen P, Bittermann G, Schmelzeisen R, Oshima T, Fretwurst T. Eagle's syndrome—A non-perceived differential diagnosis of temporomandibular disorder. International Journal of Surgery Case Reports. 2015; 15:123–126. doi:10.1016/j.ijscr.2015.08.036.

Multi-territory infarcts caused by intracranial giant cell arteritis

Karim M Mahawish, Pietro Cariga

Giant cell arteritis (GCA) is the most common form of vasculitis in adults. It is characterised by a pan-arteritis of medium to large-sized arteries. Despite major recent advances in the treatment of GCA, the diagnosis is challenging. Symptoms include headache (in two thirds of subjects), scalp tenderness and jaw claudication. As sudden permanent visual loss occurs in 8–30%, and stroke in 3–10% of patients, GCA should be considered a medical emergency.[1]

Case report

A 65-year-old woman presented with a one-week history of dysphasia and right hemiparesis. C-reactive protein (72mg/L) and platelet count (799x10^9/L) were elevated. Magnetic resonance imaging (MRI) of the brain demonstrated bilateral acute ischaemic infarcts (Figure 1) and distal left internal carotid critical stenosis (Figure 2A). Other tests including vasculitis auto-antibodies, anti-phospholipid antibodies, syphilis, human immune virus, Janus Kinase 2 mutation and cerebrospinal fluid analysis were unremarkable. Transthoracic echocardiogram and 72 hours of telemetry did not demonstrate any abnormalities.

Three days later, her speech improved and with prompting the patient admitted recent jaw claudication. Temporal arteries were found to be non-tender and non-pulsatile. Ultrasound of the temporal arteries showed low resistive waveform, however no halo sign. Temporal artery biopsy (TAB) demonstrated a heavy lymphocytic infiltrate within the external elastic lamina. GCA was diagnosed and she was commenced on

Figure 1: Diffusion weighted MRI brain with B1000 sequence (left) and apparent diffusion coefficient (right) demonstrating areas of restricted diffusion in the left anterior cerebral artery, left parietal area and in the right hemisphere borderzone middle cerebral/anterior cerebral artery territory.

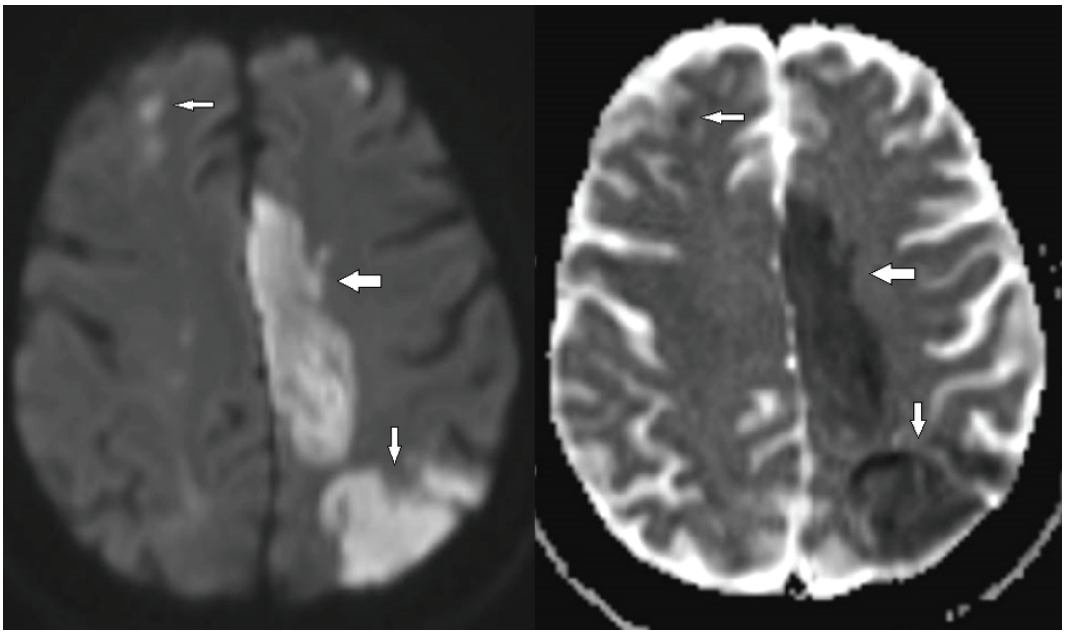
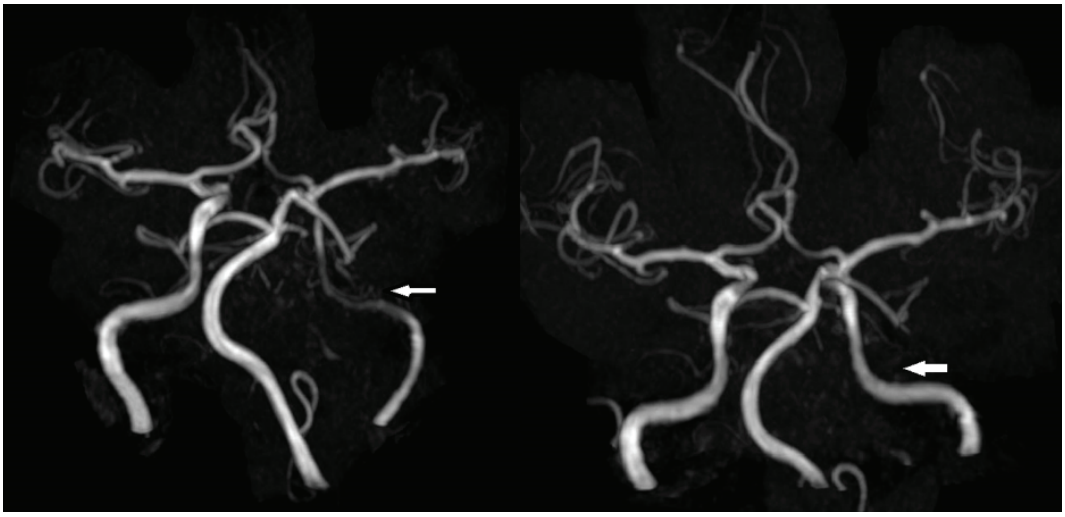


Figure 2A (left): Time of flight MR angiography demonstrating critical stenosis of the distal left internal carotid artery. 2B (right): Follow-up MR angiography following high dose corticosteroids demonstrating improved caliber of the left internal carotid artery and distal branches of the anterior and middle cerebral arteries.



intravenous methylprednisolone 1g daily for three days, then oral prednisone with tapering dose. Follow-up MRI two weeks later demonstrated improved caliber of the internal carotid artery (Figure 2B) and more prominent distal branches of the middle and anterior cerebral arteries. The C-reactive protein and platelet count normalised within 5 and 50 days of treatment respectively. We speculate that hypoperfusion and a pro-inflammatory state were responsible for the strokes.

Discussion

GCA predominantly affects individuals over 50 years of age, particularly Caucasian females who have a 1% lifetime risk, twice that of men.[1] Previously termed 'temporal arteritis', GCA may present with intracranial and/or extra-cranial arterial involvement, or even proximal disease (aorta and associated branches). It is likely that both genetic and environmental factors initiate an inflammatory cascade leading to GCA.[2]

Constitutional symptoms may be present. Cranial manifestations include temporal cutaneous hyperalgesia, jaw or tongue claudication and prominent, beaded or irregular temporal artery with a decreased pulse. Large vessel manifestations include aortitis, limb claudication and aneurysms. Strokes occur due to the hypercoagulable proinflammatory state, vessel wall inflammation and co-existing atherosclerosis causing vessel occlusion and/or embolisation of inflammatory thrombi.

The majority of patients have thrombocythemia and elevated acute phase reactants, however the latter may be normal in 1–10%.[1]

Though temporal artery biopsy is considered the gold standard diagnostic test, false-negative rates of up to 40% have been reported.[3] This may be due to sampling errors (eg, inadequate biopsy length), the presence of skip lesions and the duration of glucocorticoid treatment.

Non-invasive imaging of involved arteries with colour Duplex Ultrasonography, 3-Tesla MRI or in the case of extra-cranial GCA, 18F-fluorodeoxyglucose positron emission tomography (18F-FDG-PET) are now featuring more prominently as fist line investigations in the diagnosis of GCA. These tests demonstrate good sensitivity and specificity for GCA compared with positive TAB.[4–6]

GCA is treated with high-dose glucocorticoids as first-line therapy; however, long-term toxicity is common. The addition of methotrexate reduces relapse rates and glucocorticoid requirements.[1] Tocilizumab is an IL-6 receptor antagonist which reduces relapse rates and glucocorticoid requirements; however, is not licensed in New Zealand.

In our patient, intracranial GCA progressed insidiously until she experienced hypoperfusion strokes due to poor flow through inflamed vessels. The diagnosis required a high index of suspicion and additional tests to reach the diagnosis. While the predominant left hemisphere infarcts resulted from the severely stenosed left internal carotid, the right hemisphere infarcts were the likely result of stenosed distal branches of the middle and anterior cerebral arteries resulting in watershed or borderzone infarcts. The follow-up MRI demonstrates improved flow in these vessels.

In summary, features of GCA should be specifically sought from patients presenting with acute ischaemic stroke as symptoms may be insidious and it is associated with significant morbidity. Diagnosis can be challenging, however effective treatments are available.

Competing interests:
Nil.

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REFERENCES:

1. Mollan SP, Paemeleire K, Versijpt J, et al. European Headache Federation recommendations for neurologists managing giant cell arteritis. J Headache Pain. 2020 Mar 17; 21(1):28.
2. Mackie SL, Taylor JC, Haroon-Rashid L, et al. Association of HLA-DRB1 amino acid residues with giant cell arteritis: genetic association study, meta-analysis and geo-epidemiological investigation. Arthritis Res Ther. 2015; 17:195.
3. Allsop CJ, Gallagher PJ. Temporal artery biopsy in giant-cell arteritis: a reappraisal. Am J Surg Pathol. 1981; 5:317–23.
4. Rinagel M, Chatelus E, Jousse-Joulin S, et al. Diagnostic Performance of Temporal Artery Ultrasound for the Diagnosis of Giant Cell Arteritis: A Systematic Review and Meta-Analysis of the Literature. Autoimmun Rev. 2019; 18:56–61.
5. Rhéaume M, Rebello R, Pagnoux C, et al. High-resolution magnetic resonance imaging of scalp arteries for the diagnosis of giant cell arteritis: results of a prospective cohort study. Arthritis Rheumatol. 2017; 69:161–8.
6. Lee YH, Choi SJ, Ji JD, Song GG. Diagnostic Accuracy of 18F-FDG PET or PET/CT for Large Vessel Vasculitis: A Meta-Analysis. Z Rheumatol. 2016; 75:924–31.

Physician heal thyself: observations on trigeminal neuralgia

Denis Friedlander

In 2008, age 76 years, I developed left sided lancinating facial pains, perhaps three quarters to one second apart and in three- to four-second epochs, followed by a pause of a few seconds until the next paroxysm. Any speech, oral stimulation or merely a change in facial expression was sufficient to trigger pain—I dared not grimace.

After a few days in one of the severer periods of consuming paracetamol and tramadol, I became desperate with 90 minutes of almost continuous spasms. My daughter, with whom I live, called an ambulance. En route to hospital I inhaled deeply the nitrous oxide/oxygen blend, in the manner of a woman in established labour. As we arrived at the emergency room, the pain ceased and I decided to return home, given a lengthy wait for medical attention.

For the next decade I suffered to a greater or lesser extent with trigeminal neuralgia (TN). I was never completely free of the affliction, at best occasional bouts, at worst, periods of two or three weeks when I needed to lie down and use strong analgesics. I could not touch or wash my left face, swim, blow my nose or pull shirts on over my head. To shave, wash my hair or clean my teeth needed great care. Pain was most prevalent in the first hour or two of the morning, particularly when straining to pass stool. One of the attacks 'to live in infamy' occurred with a piece of chocolate in my mouth, with severe split-second flashes of neuralgia at three per second. I had to rush to the bathroom to rinse my mouth for immediate relief. It seemed sweet taste was involved although I thought taste went via the seventh cranial nerve—perhaps not always.

I saw a neurologist, but his drug treatments were poorly effective. Carbamazepine did not provide the anticipated relief and I felt agitated on this but gave it a good trial. Gabapentin up to 600mg four times daily, as much as I could tolerate, reduced the severity of bouts modestly but not the frequency. Baclofen 60mg a day did not help. Amitriptyline 20mg caused an anxiety state after three weeks. Clonazepam 0.5mg knocked me out for six hours, but the drug-induced rest was helpful in acute situations. I did my own research and was soon watching a microvascular decompression on YouTube. I saw a neurosurgeon, but he was reluctant to operate; I was too old.

The pain was brutal and, during severe bouts, I was best to lie supine, relax and try to stay calm. I seldom had the TN pain during sleeping hours (provided I kept off my left side). The cardiologist in me correlated this lack of TN pain with a lower nocturnal blood pressure (BP) and again the highest BPs in the first hours after rising as documented on my 24hr BP record, with the worst period of pain. I was taking treatment for mild hypertension (Felodipine 5mg and a diuretic) but considered whether more rigorous BP control might be useful. I decided to escalate treatment, achieving a mean systolic pressure of 70mmHg at night and 90–100mmHg in the day by adding candesartan, eventually at a high dose of 32mg. I was surprised that I could tolerate a systolic pressure in the 90s while up and at work with so little symptomatic hypotension but kept a careful watch on my BP with this approach to self-management. My pain was then much better controlled—not perfect, but for several years I was much more comfortable, could work and I had not enjoyed such sustained relief until my BP was this low.

I am now 88 years old. Four years ago, the TN returned despite my low BP. The usual trigger point for the pain was either lateral

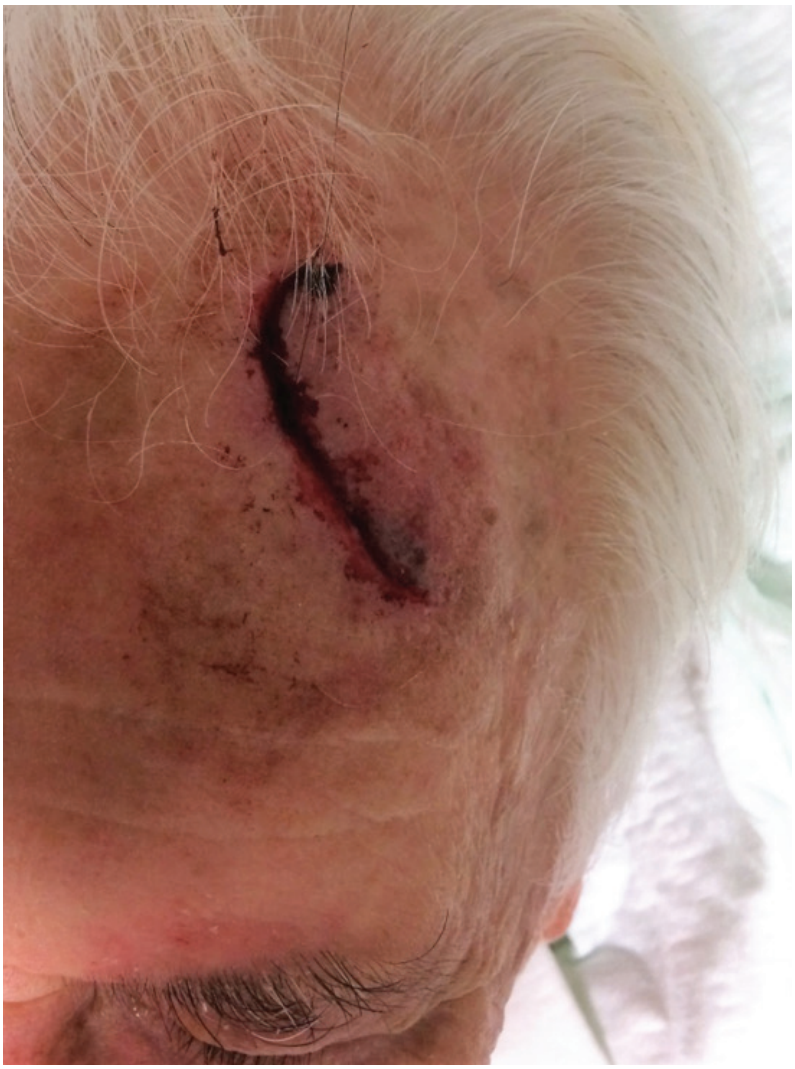
to the left upper lip, less commonly in the scalp, near the hairline. A second neurologist was consulted. Mexiletine produced ataxia; prednisone was unhelpful; oxcarbazepine had a slight benefit but made me groggy. A repeat MRI showed a vascular impingement of the fifth nerve root but did not change a different surgeon's recommendation—still too old.

Then it happened, the accidental cure of my TN. Two years ago, I lay down after an evening meal on my bed watching TV and went to sleep. I woke at 11pm with some numbness right leg and when I stood up, the limb was limp. I fell, striking my forehead with extreme force on a wooden stool, about 20cm off the floor, without loss of consciousness. Thinking I may have had a stroke but then realizing it was a flaccid weakness—it will recover. But I realized I was bleeding from the scalp, where my TN trigger point was located at the time (Figure 1). My daughter, who knew I could not tolerate any sensory stimulus to this area, was reluctant to stem the bleeding but this was essential and to my surprise did not cause pain. In hospital, seven stitches were necessary.

For two years I have had no TN pain, other than after vigorously blowing my nose with a slight hint of "be careful" warning. I was able to reduce my BP treatment. I could now tolerate all stimuli to my face, even swim and wash, and perform my ablutions without fear of the 'tic doloreaux'.

My understanding of TN is that it is usually related to a vascular compression of the trigeminal nerve root near the

Figure 1: The result of the fall.



brainstem by a stiff and tortuous artery. Arterial pulsations gradually lead to demyelination of the sensory fibres in the nerve root, leading to short circuiting of electrical impulses from these fibres to neighbouring unmyelinated pain nerve fibres. Thus a touch or stretch sensory receptor starting an electrical signal to indicate touch or stretch arrives at the brain in a group of pain fibres and the subject feels pain, not touch or stretch.

Hypertension is a predisposing factor for arterial tortuosity and treatment may have reduced the pressure on my trigeminal root. Epidemiological data provides some support for HT as a risk factor for TN,[1] but its reduction doesn't seem to have been trialed as a possible treatment. I would suggest that the possibility of treatment by BP reduction could do with further study. It seemed to work in me.

What might have happened with the blow to the head? My head was in free fall for 1.5 metres and was brought to a stop in 0.005 metres (the depth of my skin laceration and small bone give) so the negative G force on the contents of my head would be very high. (by using the formula $V^2=U^2+2as$ on the above figures gives a decelerating force of around 300G!). The resulting shock waves in head contents (compression and stretching) and Newton's first law on the more dense artery and also the brain stem would reset the nerve/artery relationship, the kinked artery being far less fixed than the nerve root. The artery was moved sufficiently away from the nerve root and stayed away.

Although such drastic treatment of the head is not available for treatment, there may be less dramatic ways of applying G forces to the head or pressure waves in the CSF that could be considered as the basis for a possible curative treatment. But whatever the mechanism, the fall and sudden deceleration injury does seem to have cured my neuralgia.

Alas, as if this were not enough for one physician, I have developed motor neuron disease—hence the third neurologist, who encouraged me to contribute these observations.

Competing interests:
Nil.
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REFERENCES:

1. Pan SL, Yen MF, Chiu YH, et al. Increased risk of trigeminal neuralgia after hypertension: A population-based study. Neurology. 2011; 77(17):1605–1610.

Uptake and outcome of a community-based healthy lifestyle intervention for preschoolers identified with obesity: an audit of the Whānau Pakari preschool programme

Tami L Cave, Lisa E Wynter, Cervantée EK Wild, José GB Derraik, Esther J Willing, Paul L Hofman, Yvonne C Anderson

Childhood obesity in Aotearoa/New Zealand remains challenging to address. Eleven percent of children aged 2–14 years are affected by obesity, with Māori (16%), Pacific (28%) and children from most socioeconomically deprived areas (20%) more likely to be affected.[1] Although prevalence has declined slightly in four-year-old children between 2010 and 2016,[2] estimates suggest over 8,400 children were affected by obesity in 2015/16, reaffirming the need for ongoing action.[2]

The Raising Healthy Kids target was implemented in July 2016 as part of the New Zealand Government's childhood obesity plan.[3] The target aimed for 95% of children identified with obesity at the B4 School Check (B4SC) to be offered a referral to a health professional, for clinical assessment and support from a family-based nutrition, activity and lifestyle intervention by December 2017.[3] Due to the target's reporting focus on initial referral rather than follow-up and referral outcome, little information regarding uptake or outcome of intervention programmes has been collected for preschoolers referred under the target nationally.

In Taranaki, children identified at the B4SC with obesity from July 2016 were referred to Whānau Pakari—a multidisciplinary, family-centred assessment and intervention programme supporting children with obesity aged 4–16 years. The programme offers comprehensive home-based assessments every six months for one year, as well as additional regular group sessions. A randomised clinical trial (RCT) embedded within Whānau Pakari (recruitment January 2012–August 2014) showed modest improvements in body mass index (BMI) standard deviation score (SDS) in both an assessment-and-weekly sessions model (intervention) and an assessment-and-advice model (control) at 12 months, with high initial engagement for Māori and children from the most deprived quintile of households.[4] Attendance was assessed as key to outcome, with a doubling of reduction in BMI SDS at 12 months (which persisted to 24 months) for those attending ≥70% of the group sessions in the intervention model.[5]

There are currently no national data on uptake of childhood obesity programmes by preschool children referred from the B4SC, and limited regional data. Therefore, the objectives of this audit were firstly, to determine what proportion of referred preschool children engaged with the Whānau Pakari preschool programme, completing at least a baseline assessment (ie, what was the *uptake* of preschoolers referred). Second, for those children who did engage, to determine the *effect* of the

Whānau Pakari preschool programme on BMI SDS after six and 12 months. The preschool programme responded to 'real-world' needs of families, offering home-based assessments at baseline, six and 12 months, alongside weekly or fortnightly sessions specifically for preschoolers (dependent on whether older siblings within the family were already attending the weekly session programme, and family and/or session availability).

A total of 143 children aged 4–5 years were referred from the B4SC to the Whānau Pakari preschool programme between July 2016 and March 2019. Of those referred, the families of 75 children (52%) engaged with the service, the families of 67 children (47%) declined any involvement when contacted, and the family of one child was excluded for not meeting eligibility criteria. No demographic differences were observed between groups (Table 1).

Among children who engaged with the Whānau Pakari preschool programme, 38 completed the six-month assessment (51%) and 24 (32%) completed the 12-month assessment. At the time of undertaking the audit, 10 (13%) children had self-discharged as caregivers reported having successfully made healthy lifestyle changes.

Among participants who completed the assessments, there was no overall change in BMI SDS from baseline at six months [-0.09 SDS (95% CI -0.23, 0.05); p=0.18] or at 12 months [-0.04 SDS (95% CI -0.29, 0.20); p=0.73]. Nonetheless, a BMI SDS reduction was observed in 55% (n=21) and 42% (n=10) of participants at six and 12 months respectively, with the remaining children displaying an increase in BMI SDS.

Participation for Māori remained similar to that for NZ European children both at six months (40% vs 42%, respectively) and 12 months (42% vs 42%, respectively). In

Table 1: Demographic characteristics of participants who engaged or declined to engage with the Whānau Pakari preschool programme.

		Engaged	Declined to engage	p-value
n		75	67	
Female		34 (45%)	36 (54%)	0.32
Age (years)†		4.5±0.2	4.5±0.2	0.27
BMI SDS†		2.68±0.62*	2.50±0.63	0.10
Ethnicity§	Māori	31 (41%)	22 (33%)	0.26
	NZ European	34 (45%)	41 (61%)	
	Pacific	3 (4%)	3 (4%)	
	Asian	4 (5%)	1 (1%)	
	Other European	2 (3%)	-	
	Latin American/Hispanic	1 (1%)	-	
Deprivation quintiles‡	1 (least deprived)	6 (8%)	6 (9%)	0.50
	2	8 (11%)	14 (21%)	
	3	19 (25%)	14 (21%)	
	4	22 (29%)	15 (22%)	
	5 (most deprived)	20 (27%)	18 (27%)	

BMI SDS, body mass index standard deviation score.
Age and BMI SDS data are means ± standard deviations; remaining data are n (%).
†Age and BMI data at point of referral.
*Referral height and weight not available for one child.
§Prioritised ethnic group.
‡Quintiles of household deprivation based on the NZ Deprivation Index 2013.[6]

addition, participation for children from the most deprived quintile of households remained relatively high at 29% and 25% at six and 12 months, respectively (vs 15% background population rate).[7] Despite the Raising Healthy Kids target being an initiative driven by the Ministry of Health, reporting of referral outcomes (including uptake of weight management support) has been largely overlooked in this process, making it difficult to interpret a 52% uptake into the Whānau Pakari preschool programme. More than half of preschoolers referred engaged with the support service available, receiving a comprehensive assessment focused on addressing weight-related comorbidities and screening of wider aspects of wellbeing. However, a substantial proportion of families declined to engage after the B4SC referral. Preliminary results from an evaluation of South Island children identified at the B4SC with obesity between 2016 and 2017 suggested low uptake of community interventions, yet this was not quantified.[8] Overall, these findings are notable, given recommendations relating to early intervention, and the prevalence of weight-related comorbidities identified in older New Zealand children with obesity.[9,10] To maximise uptake of family-based interventions for this age group, it is essential to understand the reasons why caregivers decline weight-related support for their preschoolers. Focus groups are underway to explore caregivers' views relating to the referral process and uptake into such programmes.

Uptake of the Whānau Pakari preschool programme for Māori was comparable to that of NZ European children, mirroring previous results.[4] However, in contrast to the Whānau Pakari RCT that found a lower rate of continued participation among Māori, participation at six and 12 months remained similar for both groups. This indicates that Māori preschoolers in Taranaki are receiving relatively equitable access to weight-related assessments, as well as investigation of weight-related comorbidities once on the programme. This is important given the disproportionate prevalence of obesity for Māori children, and the population demographics for children aged under five years in the region, with 83% identifying as European and 33% as Māori.[11]

While no change in BMI SDS was observed overall at six or 12 months from baseline, these findings are difficult to interpret given the small sample size, high entry BMI SDS, and loss of those who self-discharged reporting to have made healthy lifestyle changes. Importantly, the lack of an effect was not surprising due to the programme's focus on achieving persistent healthy lifestyle change rather than weight reduction per se. Given this approach, the primacy of BMI SDS as the key outcome measure of this audit may be questioned. However, this raises the tension that exists for intervention programmes when navigating high-level drivers for action against obesity that differ to the drivers at a community level. Nevertheless, it cannot be ignored that a reduction in BMI SDS is still key to a reduction in weight-related comorbidities over time for children affected by obesity. Additionally, international population data show that BMI SDS increases between four and five years of age in those identified with overweight or obesity.[12] While this is not an ideal counterfactual for this cohort, the lack of an overall increase in BMI SDS in this cohort may be more encouraging than results imply.

In conclusion, the Whānau Pakari preschool programme provided an appropriate assessment *and* intervention solution for the Raising Healthy Kids target, embedded within a pre-existing model addressing obesity across the paediatric lifecourse. Understanding ways to enhance programme uptake from the referral and improvements in BMI SDS are areas of further research.

Competing interests:
Nil.

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REFERENCES:

1. Ministry of Health. Annual update of key results 2018/19: New Zealand Health Survey. Wellington: Ministry of Health; 2019.
2. Shackleton N, Milne BJ, Audas R, et al. Improving rates of overweight, obesity and extreme obesity in New Zealand 4-year-old children in 2010–2016. Pediatr Obes. 2018; 13(12):766–777.
3. Ministry of Health. Children and young people living well and staying well: New Zealand childhood obesity programme baseline report 2016/17. Wellington: Ministry of Health; 2017.
4. Anderson YC, Wynter LE, Grant CC, et al. A novel home-based intervention for child and adolescent obesity: the results of the Whanau Pakari randomized controlled trial. Obesity. 2017; 25(11):1965–1973.
5. Anderson Y, Wynter L, O'Sullivan N, et al. Two-year outcomes of Whānau Pakari, a multi-disciplinary assessment and intervention for children and adolescents with weight issues: a randomized clinical trial. Pediatr Obes. 2020; e12693.
6. National Map Data Service. NZ Deprivation Index 2013. Updated July 07, 2017. http://koordinates.com/from/data.nationalmap.co.nz/layer/87297/. Accessed June 22, 2019.
7. Atkinson J, Salmond C, Crampton P. NZDep2013 Index of Deprivation. http://www.otago.ac.nz/wellington/otago069936.pdf. Published 2014. Accessed November 16, 2019.
8. Dainty G, Reith D, Taylor B. Kids BMI study - Evaluation of community interventions for pre-schoolers with high BMI in the South Island (abstract). In: Paediatric Society of New Zealand 70th Annual Scientific Meeting

Conference Handbook. Paediatric Society of New Zealand; 2018:76.

9. Wen LM, Rissel C, He G. The effect of early life factors and early interventions on childhood overweight and obesity 2016. J Obes. 2017; 2017:3642818.
10. Anderson YC, Wynter LE, Treves KF, et al. Prevalence of comorbidities in obese New Zealand children and adolescents at enrolment in a community-based obesity programme. J Paediatr Child Health. 2016; 52(12):1099–1105.
11. Statistics New Zealand. Census data 2018: Age and sex by ethnic group (grouped total responses), for census usually resident population counts. http://nzdotstat.stats.govt.nz/wbos/Index.aspx?DataSetCode=TABLECODE8277#. Accessed April 14, 2020.
12. Geserick M, Vogel M, Gausche R, et al. Acceleration of BMI in early childhood and risk of sustained obesity. N Engl J Med. 2018; 379(14):1303–1312.

Richard Keith Pears

26 June 1932–18 September 2020

Richard Keith Pears, the middle son of Doris and James Pears was born on 26 June 1932 at the Lumsden Maternity Hospital. The Pears family lived on a sheep station called Dunrobin Valley in the shadow of the Takitimu Mountains in Northern Southland. He died in Wanaka on 18 September 2020 in rest home care at the age of 88. He had an elder brother Kenneth and a younger brother Rob. Richard attended school locally until he was 11 and then it was to Cathedral Grammar, followed by Christ's College in Christchurch. Home visits were few and far between. Otago University followed in Dunedin where he boarded at Selwyn College and gained entrance to medical school graduating in 1956.

He was married to Betty Watters at the same time and so started a very significant and supportive partnership over some 62 years. Betty died in January 2019. Betty was the daughter of Dr Watters in Gore, Richard eventually taking over his practice. This however was not before he had completed his residency years in Invercargill and Christchurch, and then travelled overseas to the Edinburgh Royal Infirmary where he completed his Diploma in Anaesthetics. He also gained his Diploma in Obstetrics while in the UK.

Richard had prepared himself well for work as a GP in a country town where there was a local hospital with a resident surgeon needing help with anaesthesia, and a large obstetric practice. Richard was at first practising from home but he did not accept this as ideal especially for the family and also if a model of teamwork and colleagueship was to be established in GP practice, where after-hours work could be rostered and skills shared within a team relationship. To this end the Gore Medical Centre was opened in the late 60s and it was the first medical centre in New Zealand. It became the vanguard for other very singular initiatives in general practice throughout the country.

The most significant development was that of pioneering a training programme for general practitioners, quite a remarkable and almost controversial initiative in those days, and done in tandem with and following the initiative of the renowned Dr Eric Elder from Tuatapere. Their thesis was that general practice was as much a specialty as any other medical discipline and that "sorting out the muddle in your head and finding a new way of working was the essence of reorientating hospital-trained GPs". The Southland venture stimulated activity throughout the rest of the country.

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Richard was one of the earliest "host" general practitioners. This was in itself a huge undertaking, not only in the hosting, but in convincing the Royal NZ College of GPs to come on board and help with the immense amount of academic initiative and associated research needed in setting up a path to Fellowship, with examinations, in parallel with consistent and regular reviews over time for GP registrars attached to practices like the Gore Medical Centre.

Richard directed the Southland Scheme until 1980 and then moved to Christchurch where he was the Canterbury Regional Director until he retired in 1996. Looking back, these times were a "ferment" coming out of Southland… "of all places"… indeed it was the explosion of general practice into a specialty. There were lots of bureaucratic hurdles, and some quite rough spots in the late 70s and early 80s. It was eventually picked up by the universities and became a programme of quality and was renamed the Family Medicine Training Programme: FMTP. Richard was granted an MSD Fellowship to the US in 1977 to further his studies in this area and likewise even when close to retirement he helped set up a family medicine programme in Riyadh in Saudi Arabia.[1]

As if that was not enough to take up his time, Richard was one of the individuals who initiated the concept of the Gore and District Counselling Centre, a creative and much needed response to the mental health needs of the Eastern/Northern Southland and West Otago communities. Opening in 1977, the Centre continues to successfully provide counselling, psychotherapy, supervision and mental health education.

Richard was a very compassionate man who loved and supported Betty fully, a great listener according to his children (and his colleagues) with a lot of personal and spiritual depth. He had great patience, was seldom angry, and his judgment well considered. He was a lay cannon in the Christchurch Cathedral. He loved the hills and the family's hut up the North Etal Creek on the Dunrobin Valley estate… a wild but welcoming place, with snow on the river flats in winter and the warm nor-westers in summer. He loved wandering, stick in hand. Richard is survived by his younger brother, Rob, his daughters, Rebecca and Anna, and sons Andrew and Richard.

As an old practice partner of Richard's it has been a privilege to write this obituary on behalf of Richard, his medical colleagues and the family.

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I wish to acknowledge the help of Anna, Rebecca, Andrew and Richard as well as Dr Peter Fettes, a practice partner of Richard during those Gore years, and Margaret Pullar, Past Director of the Gore and District Counselling Centre.

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REFERENCES:

1. Anyon P, Rainey H. The Amoeba, the Snail, and the Octopus….a History of General Practice Vocational Training in NZ". RNZCGP 2001 e-book publication.

Hidden Dangers

By GORDON MACDONALD, M.D.

Everyone familiar with medical literature is aware of the, fact that surgeons who operate upon the genital organs of either sex are liable to attack, but this is particularly so in the case of males. Like most men in practice in one locality for many years, several such cases have come under my observation, both in my own practice and that of others. For the benefit of some of the junior members I may relate a few incidents that occurred to myself.

The first was that of a young man who had contracted gonorrhoea several times, with subsequent strictures. I had no dealings with him in any of his gonorrhoeal attacks, but he came to me to be relieved of his strictures. This involved him in time, exposure, suffering, and expense, with much food for reflection. One night in his own home he became exasperated, seized his razor, and amputated his penis close to the pubis. Next morning I found him in one of my beds in the hospital. In a sane moment he said, "Thank your stars, doctor, that I did not come to see you last night, for I was desperate and ready for any foolish act."

The next case was that of a young man suffering from varicocele. I knew him well, but was not his medical adviser. He had agreed to operation, and asked his surgeon to get me to give him chloroform. This I did, but had neither part nor say in the operation. Judge of my surprise when a few months afterwards he came to me in a state of great excitement, declaring that we had ruined his life, as he was in constant pain, and had more or less lost the power of sexual intercourse. I examined him carefully and found the testis shrunk to a mere nothing. This was probably due to some injury to the cord or inclusion of it in the ligature or the pressure due to the varicocele. He vowed vengeance, and extracted it in his own fashion to his own satisfaction.

The third case was that of a middle-aged man of a religious, nervous, almost fanatical disposition. I had known him casually for a few years, but had no dealings with him. One day in a state of great misery he consulted me about an abscess in the left testicle, with more or less hernia testis. I told him the right procedure was to have the organ removed, and that he had better consider the matter. Then he said that was the very advice his family doctor had given him, but he was no surgeon, and would I operate? This I did with the aid of his usual adviser. He made a rapid recovery and expressed gratitude to be rid of so painful an encumbrance. Some six months afterwards he came to me and said he was suffering intense pain in the scar and stump, and that the cure was ten times worse than the disease. So far as the parts were concerned things were quite normal, nor did he show any signs of pain in them while his attention was diverted. I at once recognised that I had to deal with a possibly desperate man, and was upon my guard. During the next three years he paid me several visits, and always the same tale of pain, sleeplessness, and loss of sexual power, with a veiled desire for revenge upon his tormentors. One day he entered my waiting-room, and as he was the only one present the conversation went on there. After the usual formalities he said, "Are you a Christian, doctor?," to which I replied, "Yes, and I hope you are one also." During this short chat I noticed him trying to pull his hand out of his overcoat pocket, as if it contained something. I at once said, "Leave it there," and with this a revolver fell upon the floor. He was too nervous to execute his purpose, and left, never to return.

Turning, now, to women, I was frequently consulted by a farmer's wife about chronic leucorrhoea, ovarian pains, and excessive child-bearing. She was more or less a physical wreck, and I could do little for her. On one occasion I was called to see her at her home. I made a vaginal examination and found a small ovarian tumour. There was some interregnum in the hospital so far as gynaecology was concerned, so l took her into the female surgical ward and operated

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myself. We removed the tumour and ovary after the usual preparation. When operating I noticed that the uterus was unusually large, although she declared that she had menstruated a few days before admission. She made a good recovery and was much improved in health by the rest, good food, and nursing. Seven months afterwards she was delivered of a full-grown child, to which all our interference made no difference. Years rolled on and back she came, loudly denouncing me, saying I had "spayed her" (removed ovaries), and her husband would not look at her. She pestered me for a year or two and abused and vilified me to her entire satisfaction. Her case is recorded in the pages of the N.Z. MEDICAL JOURNAL.

The next two cases came together. They were women whom I recommended to the hospital for operation because of ovarian tumours. Both of them were about thirty, and married, and in each case both ovaries were removed. In the hospital I had no dealings with them, as they were in the hands of the gynaecologist. They made good recoveries, but for several years afterwards they complained bitterly of indescribable feelings and desires and of being unsexed and ruined. They hurled every sort of vile motive and epithet at both the operator and myself and vowed every manner of vengeance and reprisal. In men one finds that their minds run towards some form of violence, while women resort to that old and many-edged weapon that can pour forth honey or poison at will. It is somewhat disconcerting to be thus abused and vilified while doing one's best to relieve the suffering of one's fellow creatures, and so long as one acts honestly and to the best of one's abilities one always has the satisfaction of having done the right thing. There is, however, another philosophy in the world, namely, that of the man who says, "Well, I generally find that those for whom I do least thank me most. Choose ye, therefore, which leader ye shall follow."

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Proceedings of the Health Research Society of Canterbury's Emerging Researcher Awards 2019

Understanding the interaction between maternal tobacco use during pregnancy and adult offspring with conduct disorder

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Aims

Metastable epialleles (MEs) are described as loci at which epigenetic regulation is established during *in utero* development and maintained throughout life. We know that maternal tobacco smoking during pregnancy can alter offspring DNA methylation. Furthermore, associations between maternal smoking and offspring conduct disorder has been observed. Our aim is to provide a molecular link between maternal smoking and conduct disorder in offspring.

Methods

Both exposed to maternal tobacco smoking *in utero* and individuals who were not were selected from the Christchurch Health and Development longitudinal study. This also included groups of individuals with and without conduct disorder. Bisulfite-based Amplicon Sequencing (BSAS) was used to investigate DNA methylation differences and potential MEs between the different groups.

Results

A novel gene, *GRIN2b,* which is expressed during *in utero* development and declines post birth, displays differential DNA methylation in response to maternal tobacco exposure in offspring adults with conduct disorder.

Conclusions

This research shows DNA methylation providing an aetiology of the observed link between maternal smoking and childhood/adolescent conduct disorder, which provides new insights into the mechanisms involved in the detrimental outcomes associated with *in utero* tobacco smoke exposure.

An exploratory patient study assessing CYP450 enzyme function in women receiving chemotherapy for breast cancer

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Aims

Obese breast cancer patients respond less well to chemotherapy. Chemotherapy drugs are metabolised by cytochrome P450 (CYP450) enzymes. Inflammatory cytokines inhibit expression and activity of CYP450 enzymes, altering drug metabolism. This study (HDEC:16/CEN/116) investigated whether obesity-related cytokines are associated with alterations in CYP450 activity in women receiving chemotherapy for breast cancer.

Methods

Phenotypic activity of CYP450 enzymes (CYP1A2, CYP2C9, CYP2C19, CYP2D6 and CYP3A4) were assessed using the 'Inje' probe drug cocktail and mass spectrometry. Inflammatory cytokines were quantified in patient serum samples using immunoassays. Voluntary physical activity was recorded on FitBit One® devices.

Results

This study recruited 12 women (n=7, BMI<30; n=5, BMI≥30), aged 40–68 years. Serum B-cell activating factor (BAFF), growth and differentiation factor 15 (GDF-15) and monocyte chemoattractant protein 1 (MCP-1) increased, and interleukin 10 (IL-10) decreased during chemotherapy. As serum concentrations of MCP-1 increased, the activity of CYP3A4 decreased. Daily step counts decreased early in chemotherapy. However, alterations in cytokine concentrations were not dependent on differences in obesity or physical activity.

Conclusions

This study provides preliminary evidence that circulating inflammatory cytokines may influence CYP450-mediated chemotherapy metabolism, and validates feasibility of the 'Inje' cocktail to measure CYP450 activity in patients receiving chemotherapy for breast cancer.

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Variable expression quantitative trait loci analysis of breast cancer risk variants

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Aim

Genome wide association studies in breast cancer have identified more than 180 risk variants. A major challenge has been to understand the functional consequences of these variants. Studies to date have utilised expression quantitative trait loci (eQTL) to identify candidate susceptibility genes at risk loci. We propose that variable expression quantitative trait loci (veQTL) will provide additional information and identify novel candidate breast cancer susceptibility genes.

Methods

RNA-sequencing and genotype data from 635 samples was acquired from the Genotype-Tissue Expression (GTEx) Common Fund Project. Tissue-specific veQTL and eQTL analysis was performed using breast cancer risk variants from four tissue types: breast, kidney, lung and ovary.

Results

Seventy veQTL identified 60 candidate genes associated with breast cancer risk variants in breast tissue. These were enriched for genes involved in C21-steroid biosynthesis and extracellular structure process. Notably, individuals homozygous for the risk allele of rs11075995 were associated with expression variability of four genes (*STAR, CYP17A1, CYP11B1* and *HSD3B2*) involved in the conversion of cholesterol into steroids, a potential mechanism of cancer risk.

Conclusion

Tissue-specific veQTL analysis identified novel candidate breast cancer susceptibility genes, including those associated with the C21-steroid biosynthesis pathway.

Ocular gene therapy protects against retinal degeneration and vision loss in sheep with CLN5 Batten disease

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Aims

To test if ocular gene therapy can preserve retinal structure and function. Neuronal ceroid lipofuscinoses (NCL; Batten disease) are a group of inherited neurodegenerative diseases primarily affecting children. A common feature is progressive loss of vision. Brain-directed gene therapy in sheep models of CLN5 and CLN6 NCL can halt disease progression, however treated animals still lose their sight.

Methods

We performed intravitreal injections of self-complementary AAV9 vectors containing either CLN5 or CLN6 into three-month-old *CLN5*$^{-/-}$ or *CLN6*$^{-/-}$ sheep. Electroretinography (ERG) was performed monthly following treatment and retinal histology was assessed post-mortem.

Results

ERG b-wave amplitudes were normalised in the treated eyes compared with the untreated eyes in *CLN5*$^{-/-}$ animals up to 18 months of age. ERG amplitudes in both eyes of *CLN6*$^{-/-}$ animals declined with age, however the treated eye maintained higher amplitudes. Post-mortem analyses revealed significant attenuation of retinal atrophy and storage body accumulation in the treated eye compared with the untreated eye in both *CLN5*$^{-/-}$ and *CLN6*$^{-/-}$ animals.

Conclusions

The single administration of AAV9.CLN5 can successfully ameliorate retinal deficits in *CLN5*$^{-/-}$ sheep. Therefore combining ocular and brain-directed gene therapies presents a promising treatment strategy for future trials aiming to halt clinical progression in CLN5 NCL.

Circulating myeloperoxidase is elevated in septic shock and is associated with systemic organ failure and mortality in critically ill patients

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Aims

This study aimed to determine if the oxidant-generating enzyme myeloperoxidase is elevated in critically ill patients and if elevated levels are associated with adverse patient outcomes.

Methods

Myeloperoxidase was measured by ELISA in a cohort of 44 critically ill patients and 44 healthy controls. Intensive care mortality prediction scores (SOFA, APACHE III) and patient mortality were obtained from clinical notes. Routine blood measures of organ dysfunction were assessed and cell-free DNA was detected using fluorescence staining.

Results

Myeloperoxidase was significantly higher in critically ill patients than healthy controls, and was elevated in septic shock relative to non-septic patients. Myeloperoxidase correlated significantly with SOFA scores in the critically ill patients, and with markers of tissue dysfunction and injury such as lactate, alanine transferase and cell-free DNA. Hospital mortality for the whole cohort was 27%; mortality in the high APACHE III subgroup was 38%, and when combined with higher than mean myeloperoxidase, mortality increased to 71%.

Conclusions

Myeloperoxidase is associated with markers of tissue injury and systemic organ failure, particularly in septic patients. The enzyme is also associated

with mortality in patients with higher APACHE III scores, and thus has potential as a diagnostic marker to improve mortality prediction.

Examining the links between community water fluoridation, area-level deprivation and childhood dental ambulatory sensitive hospitalisations: nationwide pooled evidence from New Zealand

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Aim

This study examines the association between community water fluoridation (CWF) and dental ambulatory sensitive hospitalisations (ASH) and the moderating effect of CWF on the association between area-level deprivation and dental ASH.

Method

Dental ASH conditions, ie, dental caries and diseases of pulp/periapical tissues, age, gender and meshblock were extracted from pooled (2011 to 2017) cross-sectional data on children aged 0–4 and 5–12 from the National Minimum Dataset. Dental ASH rates for children aged 0–4 and 5–12 (/1,000) were calculated for census area units (CAU). CWF was obtained for 2011 and 2016 from the Institute of Environmental Science and Research. Multilevel negative binomial models investigated associations between area-level deprivation, dental ASH rate, and moderation by CWF status.

Result

Findings show that relative to CWF in both 2011 and 2016, no CWF was associated with increased dental ASH rates in children aged 0–4 (IRR=1.17 [1.06–1.29]; and aged 5–12 (IRR=1.18 [1.08–1.29]). The association between CWF and dental ASH rates was more pronounced for children within the most deprived areas in children aged 0–4.

Conclusion

Our findings suggest that variation in CWF is associated with structural inequalities in oral health outcomes for children in New Zealand.

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