New Zealand

Medical Journal

Journal of the New Zealand Medical Association Vol 130 | No 1465 | 10 November 2017

Achieving health equity in Aotearoa: strengthening responsiveness to Māori in health research



Whānau perceptions and experiences of acute rheumatic fever diagnosis for Māori in Northland, New Zealand

Dispensing patterns for antidiabetic agents in New Zealand: are the guidelines being followed?

Remembering the 1918 influenza pandemic: national survey of memorials and scope for enhancing educational value around pandemic preparedness

Overwhelming support for smokefree cars that are carrying children—is the Government listening:

New Zealand **Medical Journal Publication Information**

published by the New Zealand Medical Association

NZMA Chair

Dr Kate Baddock

To contribute to the *NZMJ*, first read: www.nzma.org.nz/journal/contribute

NZMJ Editor

Professor Frank Frizelle

NZMA Communications Manager

Sharon Cuzens

NZMJ Production Editor

Rory Stewart

Other enquiries to:

NZMA

PO Box 156

The Terrace

Wellington 6140

Phone: (04) 472 4741

© NZMA 2017

To subscribe to the NZMJ, email

julie@nzma.org.nz

Subscription to the New Zealand Medical Journal is free and automatic to NZMA members.

Private subscription is available to institutions, to people who are not medical practitioners, and to medical practitioners who live outside New Zealand. Subscription rates are below.

All access to the NZMI is by login and password, but IP access is available to some subscribers.

Read our Conditions of access for subscribers for further information www.nzma.org.nz/journal/subscribe/conditions-of-access

If you are a member or a subscriber and have not yet received your login and password, or wish to receive email alerts, please email: julie@nzma.org.nz

The NZMA also publishes the NZMI Digest. This online magazine is sent out to members and subscribers 10 times a year and contains selected material from the NZMJ, along with all obituaries, summaries of all articles, and other NZMA and health sector news and information.

Subscription rates for 2017

New Zealand subscription rates Overseas subscription rates Individuals* \$306 Individual \$426 Institutions \$530 Institutions \$571 Individual article \$25 Individual article \$25

*NZ individual subscribers must not be doctors (access is via NZMA Membership) New Zealand rates include GST. No GST is included in international rates. Note, subscription for part of a year is available at pro rata rates. Please email julie@nzma.org.nz for more information. Individual articles are available for purchase by emailing nzmj@nzma.org.nz



NZMJDigest

published by the New Zealand Medical Association



NZMJDigest

http://www.nzma.org.nz/publications/nzmjdigest

The NZMA publishes the e-magazine NZMJDigest 10 times a year. It contains news and views from the profession and the NZMA, including the NZMA Chair's editorial, along with highlights from and links to the New Zealand Medical Journal.

Click on the image above to view the latest issue.

We welcome contributions from members and readers. To contribute to the NZMJDigest, please email digest@nzma.org.nz



EDITORIAL

9

Metformin: a golden oldie Helen Lunt, Ben Hudson

ARTICLES

12

Dispensing patterns for antidiabetic agents in New Zealand: are the guidelines being followed? Peter Murray, Hew Norris, Scott Metcalfe, Bryan Betty, Vanessa Young, Bronwyn Locke

19

A review of squamous cell vulvar cancers in Waikato region, New Zealand
Prashanth Hari Dass,
Marion Kuper-Hommel

29

Child morbidity as described by hospital admissions for primary school aged children in Tonga 2009–2013
Fiona Catherine Langridge,

Fiona Catherine Langridge, Sione Vaioleti Hufanga, Malakai Mahunui 'Ofanoa, Toakase Fakakovikaetau, Teuila Mary Percival, Cameron Charles Grant

44

Metabolic monitoring in New Zealand district health board mental health services Aimee Staveley, Ian Soosay, Anthony J O'Brien

53

Remembering the 1918 influenza pandemic: national survey of memorials and scope for enhancing educational value around pandemic preparedness Nick Wilson, Catharine Ferguson, Geoffrey Rice, Michael G Baker, Ben Schrader, Christine Clement, George Thomson

71

Face-to-face versus telephone delivery of the Green Prescription for Māori and New Zealand Europeans with type-2 diabetes mellitus: influence on participation and health outcomes Margaret Williams, Simeon Cairns, David Simmons, Elaine Rush

80

Whānau perceptions and experiences of acute rheumatic fever diagnosis for Māori in Northland, New Zealand Anneka Anderson, Clair Mills, Kyle Eggleton

89

Audit on first seizure presentation to Taranaki Base Hospital: a secondary centre experience Sean Lance, Rajesh Kumar

VIEWPOINT

96

Achieving health equity in
Aotearoa: strengthening
responsiveness to Māori in health
research
Papaarangi Reid, Sarah-Jane Paine,
Elana Curtis, Rhys Jones,
Anneka Anderson, Esther Willing,
Matire Harwood

LETTERS

104

Overwhelming support for smokefree cars that are carrying children—is the Government listening?
Richard Jaine, Richard Edwards,
Jude Ball, Dalice Sim, George Thomson,
R Beaglehole



107

The battle for better nutrition: the role of the escalating fruit and vegetable prices Isaac Amoah, Carolyn Cairncross, Elaine Rush

109

New Zealand's legal action against IQOS postponed, consultation with Big Tobacco follows Marta Rychert

112

Antifungal susceptibility results of vaginal yeast isolates from New Zealand women, 2001–2015 Arthur Morris, Wendy McKinney, Karen Rogers, Sally Roberts, Joshua Freeman

116

Response to Dr Caleb Armstrong: proposed Waikato medical school Ross Lawrenson, Derek Wright, Ayla Thomas

OBITUARY

118

John Samuel Hopkirk David Davidson

METHUSELAH

121

Lithium use in pregnancy and the risk of cardiac malformations

100 YEARS AGO

122

Ambroise Paré, Army Surgeon

ERRATUM

125

Errata



Dispensing patterns for antidiabetic agents in New Zealand: are the guidelines being followed?

Peter Murray, Hew Norris, Scott Metcalfe, Bryan Betty, Vanessa Young, Bronwyn Locke Diabetes is a major health issue in New Zealand. In New Zealand, we have clear guidelines on how to treat diabetes. These guidelines state that if medication is to be started, metformin should be started first, followed by sulfonylureas. This research shows that these guidelines are being well followed in New Zealand, particularly when compared with other countries.

A review of squamous cell vulvar cancers in Waikato region, New Zealand

Prashanth Hari Dass, Marion Kuper-Hommel

We report one of the largest retrospective single-centre review of vulvar cancers in Australasia. Multiple factors including patient choice, tumour location, advanced age, patients' comorbidities and treatment complications have influenced and individualised treatment. Variation in treatment over the course of time, especially in the latter years were observed. Independent of Stage of vulvar cancer, patients with less comorbidities had a better overall survival. Although, treatment was associated with high morbidity, cisplatin chemo-radiotherapy was better tolerated, however this requires validation in larger prospective studies.

Child morbidity as described by hospital admissions for primary school aged children in Tonga 2009–2013

Fiona Catherine Langridge, Sione Vaioleti Hufanga, Malakai Mahunui 'Ofanoa, Toakase Fakakovikaetau, Teuila Mary Percival, Cameron Charles Grant

New Zealand is situated close to the Pacific Islands geographically and has a high Pacific population. Unfortunately, children living in these small but important Pacific nations have not received much attention in regards to their health status. In this paper, 85% of admissions to hospital in Tonga for primary school children were for injury and poisoning, non-respiratory infectious disease, respiratory conditions, abdominal/surgical conditions and dental disease. This information is helpful to inform healthcare priorities for Tonga and other similar countries.

Metabolic monitoring in New Zealand district health board mental health services

Aimee Staveley, Ian Soosay, Anthony J O'Brien

People with mental illness have a 20-year reduction in life expectancy compared to people in the general population. One of the factors contributing to this difference is antipsychotic medication, commonly prescribed for people with severe mental illness. We surveyed the district health boards' mental health services to investigate their policies for monitoring the physical health of people with severe mental illness. We found that most DHBs had policies for monitoring physical health, but also that there was scope for improvement in those policies. Our recommendation is that the Ministry of Health adopt a best practice standard in this area, and require DHBs to report against this standard.



Remembering the 1918 influenza pandemic: national survey of memorials and scope for enhancing educational value around pandemic preparedness

Nick Wilson, Catharine Ferguson, Geoffrey Rice, Michael G Baker, Ben Schrader, Christine Clement, George Thomson

This study aimed to systematically identify physical memorials to the 1918 influenza pandemic in New Zealand. Despite the high impact of the 1918 influenza pandemic in this country (~8,600 deaths), only seven publicly accessible local memorials which referred this pandemic were identified. Another 11 memorials were identified, but these were in private settings or did not refer to the pandemic. There is no national memorial and a marked contrast exists with the number of war memorials (260 times more per 1,000 deaths for one war). There appears to be major scope for enhancing public education around the persisting threat of future pandemics via improved use of physical memorials and linkages to online resources.

Face-to-face versus telephone delivery of the Green Prescription for Māori and New Zealand Europeans with type2 diabetes mellitus: influence on participation and health outcomes

Margaret Williams, Simeon Cairns, David Simmons, Elaine Rush

In Aotearoa/New Zealand, the participation of Māori in the national Green Prescription lifestyle programme is lower than for New Zealand Europeans (Pakeha). A kaupapa Māori informed trial examined, for Māori and Pakeha people newly diagnosed with type 2 diabetes, if face-to-face or telephone delivery of the Green Prescription resulted in better engagement. Near equal participation of both Māori and Pakeha in the trial was achieved with improved health regardless of the mode of delivery, face-to-face or telephone.

Whānau perceptions and experiences of acute rheumatic fever diagnosis for Māori in Northland, New Zealand

Anneka Anderson, Clair Mills, Kyle Eggleton

This study explored Māori whānau experiences of ARF, including their pathways to primary health care and barriers and facilitators for the diagnosis of ARF. This was achieved by interviewing patients with ARF/RHD and their whānau. The study found that barriers to diagnosis were lack of throat swabbing and inappropriate prescription of antibiotics. Access to primary care, having health professionals follow sore throat guidelines, and trust in health professionals facilitated diagnosis. The authors recommend the development of an effective quality improvement strategy for sore throat management, promoting free rapid-response throat swabbing for high-risk populations, and exploring options of self-swabbing to improve ARF services.



Audit on first seizure presentation to Taranaki Base Hospital: a secondary centre experience

Sean Lance, Rajesh Kumar

Seizures are a common symptom that can be caused by a variety of other medical problems. Management of a person after their first seizure should be dictated by their risk to develop further seizures. Adequate investigation and access to care for these patients is essential to help determine this risk and treat them appropriately. Although Taranaki does a fair job, a number of issues could be improved on.

Achieving health equity in Aotearoa: strengthening responsiveness to Māori in health research

Papaarangi Reid, Sarah-Jane Paine, Elana Curtis, Rhys Jones, Anneka Anderson, Esther Willing, Matire Harwood

Responsiveness to Māori reflects the Government's view that health research in New Zealand will contribute to improving Māori health and eliminating health inequities. As recipients of government funding, health researchers have obligations to meet these expectations. In this paper, we consider how an equity approach to responsiveness to Māori can be used by researchers to consider Māori health priorities, develop appropriate relationships with Māori and commit to undertaking research that mitigates rather than extends health inequities.



Metformin: a golden oldie

Helen Lunt, Ben Hudson

etformin has been used in the treatment of type 2 diabetes for 60 years.¹ Past fashions in prescribing have seen metformin come in to and go out of favour. Metformin use has however increased over the last 30 years, as several key publications over that time period have emphasised its efficacy, safety, tolerability and low cost, when compared to alternative therapies.¹ In patients with type 2 diabetes, metformin is now the recommended first-line therapy after exercise and dietary changes.¹

In this edition of the New Zealand Medical Journal, Murray et al discuss trends in New Zealand's recent dispensing patterns of anti-diabetic agents (AAs) in the management of type 2 diabetes, including use of metformin.2 They show that use of metformin as the first dispensed medication for the treatment of type 2 diabetes increased between 2007 and 2016, when metformin prescribed as initial monotherapy reached 85% of prescriptions.² An additional 11% of patients were co-prescribed metformin alongside insulin or a sulphonylurea.2 This co-prescribing is likely to have been in patients presenting with glucose levels that were sufficiently high, that they were unlikely to respond to metformin alone.^{2,3} Murray et al's finding of predominant use of metformin monotherapy as initial choice of AA is a good news story; the available evidence favours metformin as the first line AA given its beneficial effect on cardiovascular (CV) disease risk and weight, and its good safety profile.1

The only country to report rates of initial AA therapy using metformin, that are close to that of New Zealand, is the UK. In their primary care setting, use of metformin as first-line therapy reached 91% in 2013.⁴ There are of course international differences in pharmaceutical availability and reimbursement schedules, as well as differences in study methodologies, that are likely to explain some of the observed international differences in prescribing patterns. Also, neither the current New Zealand study nor the UK study mentioned above,⁴ was

designed to explore the reason(s) why a small minority of patients were not initiated onto metformin, so we do not know if we have reached 'peak metformin' in New Zealand and the UK.

Should New Zealand prescribers aim to use metformin as first-line therapy in virtually all type 2 DM requiring additional treatment over and above lifestyle change? Probably not. It can be surmised that at least some of the patients prescribed alternative AAs would have had a clear contraindication to metformin use, such as renal impairment (eGFR <15ml/min according to recent changes to the New Zealand datasheet for metformin prescribing),^{5,6} and severe hepatic impairment.⁵

Other countries recognise the advantages of metformin and are concerned about their own low rates of metformin initiation. In some countries, prescribers' caution about using metformin partly reflects their attitudes towards 'historical' safety concerns, especially the risk of lactic acidosis in certain patient sub-populations. Reassuringly, the best available current evidence, while admittedly observational in nature, supports use of metformin in patients with stable heart failure and also chronic liver disease. The role of patient-related factors in decision making around choice of initial diabetes therapy in New Zealand is also unknown. Patients rank gastrointestinal (GI) upset as an undesirable side effect of diabetes medications.8 In clinical practice, patients with pre-existing GI upset may make an active decision to commence an agent with a side effect profile that is a better 'fit' with their underlying comorbidities, even with the knowledge that metformin-related GI upset is usually transient and can be managed using an appropriate dosing schedule.3,5

Murray et al cite the bpac^{nz} (Best Practice Advocacy Centre, New Zealand) 2015 diabetes guidelines.³ Guidelines do not remain 'in-date' and relevant forever and there are some emerging areas of clinical uncertainty associated with metformin



initiation that would benefit from further discussion. For example, prolonged metformin use is associated with vitamin B12 deficiency.9 Should a baseline B12 measurement therefore be done at the time of metformin initiation, for comparison with later B12 test results? Also, should metformin or indeed any AA initiation be considered routinely in the very elderly, aged 80 years or more? Actuarial information would suggest that this subgroup of patients is unlikely to live long enough to develop a heavy burden of chronic diabetes complications, even if glycaemic control is allowed to sit above traditional target levels. In these patients the loss of quality-adjusted life years due to medication effects may outweigh any gains achieved through improved glycaemic control.10 At the other end of the age spectrum, current New Zealand registration of metformin restricts use in children,5 whereas some overseas guidelines include dosing schedules specific to children.1

Will a new AA usurp the role of metformin as first-line therapy? The most likely contender in 2017 would be the SGLT2 inhibitors. Several members of this class of AAs appear to confer additional benefits over and above glucose lowering, by offering cardio-protection. SGLT2 inhibitors are cheaper than their injectable competitors, the GLP-1 agonists, but are nevertheless considerably more expensive than metformin. Several SGLT-2 inhibitors are registered for use in New Zealand but none are on the PHARMAC schedule. A definitive evidence-based answer to the clinical question about metformin versus SGLT2 inhibitor use as initial therapy would require a very large randomised controlled trial which included a pharmacoeconomic analysis. Because metformin seems to have its own independent CV risk lowering effects,1 such a trial should include a CV outcomes analysis. It is however unlikely that a pharmaceutical company would

want to fund this type of CV outcomes trial, as they risk demonstrating that their own product shows equivalence, or possibly inferiority, rather than superiority to metformin. Also, such a trial would need to be very large, which means it would also be very expensive. Funding mechanisms that are independent of the pharmaceutical industry would therefore be difficult to identify.

Metformin stands out as one of a handful of medications that have been around for a long time, but whose star continues to shine brightly in evidence-based guidelines, with a growing number of patients being initiated on to this medication. In this high-use setting, a sufficient number of prescribers will initiate metformin in patients with 'historical contraindications' to its use, thereby helping to increase the amount of available data about metformin use, including its safety profile. Analysis of large observational datasets can then help to define metformin's risk-benefit profile in patient subgroups that have traditionally been excluded from using metformin.

In conclusion, metformin is an affordable medication with a well-documented efficacy and tolerability profile. If prescribed according to guidelines, it is safe. Murray et al show that it is being initiated as the main front-line therapy for type 2 diabetes within New Zealand. Their big picture view of adherence to prescribing guidelines looks positive. The next challenge may be to determine the extent to which metformin prescribing follows safety recommendations, such as those provided by Medsafe. It would also be of interest to study prescriber adherence to current New Zealand guidelines and recommendations, not only at the time of metformin initiation but also with regard to longer-term surveillance of individual patients on metformin, who may over time develop new (emerging) contraindications to metformin use.



Competing interests:

Nil.

Author information:

Helen Lunt, Clinical Associate Professor, Department of Medicine, University of Otago, Christchurch, Physician, Diabetes Centre, Canterbury District Health Board, Christchurch; Ben Hudson, Senior Lecturer, Department of General Practice, University of Otago, Christchurch, General Practitioner, Lyttelton Health Centre, Lyttelton.

Corresponding author:

Helen Lunt, Diabetes Physician, Diabetes Centre, 550 Hagely Ave, Christchurch 8011. helen.lunt@cdhb.health.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7391

REFERENCES:

- 1. Bailey CJ. Metformin: historical overview. Diabetologia. 2017; 60:1566–76.
- 2. Murray P, Norris H,
 Metcalfe S, et al. Dispensing
 patterns for antidiabetic
 agents in New Zealand;
 are the guidelines being
 followed? N Z Med J
 2017; 130(1465):12–18.
- 3. Bpac. Managing patients with type 2 diabetes: from lifestyle to insulin. BPJ. 2015; 72:33–42. http://www.bpac.org.nz/BPJ/2015/December/diabetes.aspx Accessed 2 October 2017.
- 4. Sharma M, Nazareth I,
 Petersen I. Trends in
 incidence, prevalence and
 prescribing in type 2 diabetes mellitus between 2000

- and 2013 in primary care: a retrospective cohort study BMJ Open 2016;6:e010210. doi: 10.1136/ bmjopen-2015-010210
- 5. www.medsafe.govt.nz/ medicines (Accessed 11 October 2017).
- 6. Jayathissa S, Dixon P, Bruce R, Reith D. Refining metformin prescribing in New Zealand. N Z Med J 2017; 130(1452):49–53.
- 7. Crowley MJ, Diamantidis CJ, McDuffie JR, et al. Clinical Outcomes of Metformin Use in Populations with Chronic Kidney Disease, Congestive Heart Failure, or Chronic Liver Disease: A Systematic Review. Ann Intern Med. 2017; 166:191–200.

- 3. Purnell TS, Joy S, Little E, et al. Patient Preferences for Noninsulin Diabetes Medications: A Systematic Review. Diabetes Care. 2014; 37:2055–2062.
- 9. Chapman LE, Darling AL, Brown JE. Association between metformin and vitamin B12 deficiency in patients with type 2 diabetes: A systematic review and meta-analysis. Diabetes Metab. 2016; 42:316–27.
- 10. Vijan S, Sussman JB, Yudkin JS, Hayward RA. Effect of patients' risks and preferences on health gains with plasma glucose level lowering in type 2 diabetes mellitus. JAMA Intern Med. 2014; 174:1227–34.



Dispensing patterns for antidiabetic agents in New Zealand: are the guidelines being followed?

Peter Murray, Hew Norris, Scott Metcalfe, Bryan Betty, Vanessa Young, Bronwyn Locke

ABSTRACT

AIMS: Type 2 diabetes mellitus (T2DM) is a significant public health issue in New Zealand. Effective management and glycaemic control is critical for reducing diabetes-related complications. Treatment guidelines are well established in New Zealand. Using dispensing data as a proxy for prescribing data, this paper aims to describe the pattern of first- and second-line antidiabetic agent (AA) dispensing for T2DM in New Zealand and assess adherence with treatment guidelines.

METHODS: Analysis of national dispensing data for AA medications using the Pharmaceutical Collection database from 2007/08 to 2015/16.

RESULTS: Metformin monotherapy remains the most commonly prescribed first-line T2DM medication prescribed, accounting for 85% of initial agents prescribed. Sulfonylureas are the most common second-line agents used, accounting for 70% of all second-line agents.

CONCLUSION: There is a high degree of adherence with the T2DM treatment guidelines in New Zealand.

ype 2 diabetes mellitus (T2DM) is a significant and costly public health issue in New Zealand. Māori and Pacific people bear a disproportionate burden of T2DM-related disease, contributing to ethnic health disparities in New Zealand. Effective management of T2DM is critical for reducing the disease-related complications.

New Zealand guidelines recommend a target HbA1c of 50–55mmol/mol.⁶ T2DM management guidelines support tailoring treatment to the individual, drawing on lifestyle interventions and pharmacological therapies.^{6,7} Antidiabetic agents (AA) available in New Zealand include metformin, sulfonylureas (glibenclamide, gliclazide, glipizide), acarbose, pioglitazone and insulin.^{7,8} The recommended sequence of care in New Zealand is initially utilising lifestyle interventions (eg, exercise, dietary changes) followed by pharmacological therapies. The New Zealand guidelines

currently recommend metformin as a first-line pharmacological agent, followed by the addition of a sulfonylurea if required and, finally, insulin.^{6,7} International guidelines also support the use of metformin as a first-line agent as it is relatively inexpensive, has an established safety profile, provides possible cardiovascular protection and does not lead to weight gain.9 In contrast with the New Zealand recommendations, American and European guidelines support tailoring the choice of second-line therapy to the individual.9 However, many of the agents recommended in these international guidelines are not funded in New Zealand (eg, sodium-glucose cotransporter-2 (SGLT2) inhibitors, dipeptidyl peptidase-4 (DPP-4) inhibitors and glucagon-like peptide-1 (GLP-1) receptor agonists).9

A number of international studies have considered prescribing patterns of AA.¹⁰⁻¹³ Previous New Zealand-based research has



provided a picture of diabetes medicine prescribing and concomitant self-monitoring blood glucose strip usage. 15 However, national patterns of T2DM medication prescribing over time have not been researched in the New Zealand context. Using the national Pharmaceutical Collection database, this analysis describes the pattern of first- and second-line AA dispensing (a proxy for prescribing patterns) for T2DM in New Zealand to assess the degree of adherence by prescribers with treatment guidelines.

Methods

Patients were identified from the Pharmaceutical Collection database who had collected their first dispensing for metformin, sulfonylureas, other funded AA (acarbose and pioglitazone) and/ or insulin during nine financial years (1 July to 30 June) from 2007/08 to 2015/16 where complete patient identifier data was available. The four-year period from 2003/04 to 2006/07, where patient identifier data was less complete, was used to exclude patients from the analysis where they were assumed to have an unknown treatment start date (ie, treatment started prior to the availability of good patient-level data). To try and best identify patients with T2DM (and not those with Type 1 DM), only those who had also collected a dispensing for an AA (at any time from 1 July 2003 to 30 June 2016) were included in the analysis, as an ostensibly

robust administrative proxy measure of true patients with T2DM.

First-line T2DM treatments used in New Zealand

Each patient's earliest diabetes medicine dispensing date was identified. Any diabetes medicine dispensing that occurred within 90 days of that date were grouped together and counted as the patient's first-line T2DM treatment. The 90-day period was chosen as it represents the lifespan of a chronic medicine prescription. As the Pharmaceutical Collection database does not routinely record indication for medicine use, the analysis could not distinguish between dispensing for T2DM and other conditions in which these agents could be used, eg, polycystic ovary syndrome or pre-diabetes. However, it was assumed that T2DM would constitute the vast majority of this group.

Second-line T2DM treatments used in the 2007/08 cohort

The dataset for the patients who started diabetes medicine treatment in 2007/08 (the 2007/08 cohort) was used to investigate second-line treatments. Each patient's second-line treatment was identified in a similar way to their first-line, with all diabetes medicines dispensed within 90 days of starting second-line treatment grouped together. Not all patients in the cohort could be followed up (eg, due to death), hence a discrepancy between the number first starting a T2DM treatment in 2007/08 and those analysed on follow-up.

Table 1: First T2DM medication/s dispensed in patients in New Zealand, 2007/08–2015/16.

Treatment	2007/08	2008/09	2009/10	2010/11	2011/12	2012/13	2013/14	2014/15	2015/16
Metformin	14,143	13,875	14,788	15,651	15,697	15,786	15,646	16,486	16,401
	(80%)	(81%)	(82%)	(84%)	(85%)	(84%)	(84%)	(85%)	(85%)
Metformin/	339 (2%)	384	586	640	640	857	986	994	1,034
insulin		(2%)	(3%)	(3%)	(3%)	(5%)	(5%)	(5%)	(5%)
Metformin/	1,704	1,577	1,489	1,350	1,346	1,351	1,321	1,257	1,162
sulfonylurea	(10%)	(9%)	(8%)	(7%)	(7%)	(7%)	(7%)	(6%)	(6%)
Sulfonylurea	1,359	1,066	945	705	618	472	423	373	331
	(8%)	(6%)	(5%)	(4%)	(3%)	(3%)	(2%)	(2%)	(2%)
Other	225 (1%)	206 (1%)	236 (1%)	261 (1%)	259 (1%)	250 (1%)	263 (1%)	265 (1%)	269 (1%)
Total	17,770	17,108	18,044	18,607	18,560	18,716	18,639	19,375	19,197



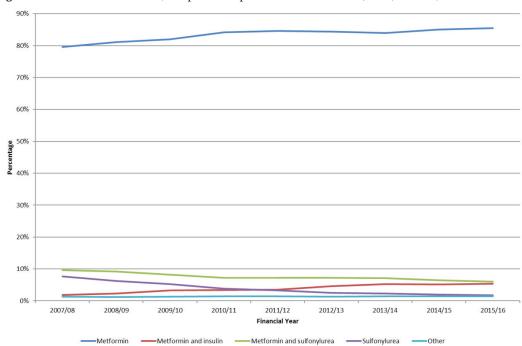


Figure 1: First T2DM medication/s dispensed in patients in New Zealand, 2007/08–2015/16.

Results

First-line T2DM treatments used in New Zealand

From 2007/08-2015/16, a total of 166,016 patients, averaging 18,446 per year, were dispensed their first T2DM treatment (Table 1). Metformin monotherapy was the most commonly dispensed first-line AA in New Zealand (Figure 1). Over time its use as a first line agent increased from 80% in 2007/8 to 85% in 2015/16. Sulfonylurea monotherapy dispensing decreased over the nine years analysed and in 2015/16 accounted for 2% of all first-line dispensing. Dual AA therapy

(metformin and sulfonylurea) dispensing also trended down over time, from 10% in 2007/8 to 6% in 2015/16. However, initial dispensing of both metformin and insulin slightly increased over the period analysed (2% to 5%). Other medicines (eg, acarbose and pioglitazone) and combinations (eg, sulfonylurea plus insulin) accounted for around 1% of first-line dispensing each year. Relative to dispensing patterns in 2007/08, there was a 205% increase in metformin and insulin dispensing, contrasting with 76% and 32% reductions in sulfonylurea and metformin/sulfonylurea dispensing respec-

tively (Figure 2).

250% 200% Percentage change from 2007/08 150% 100% 50% 0% -50% -100% 2007/08 2008/09 2009/10 2010/11 2015/16 2011/12 2012/13 2013/14 2014/15 Metformin -2% 5% 11% 11% 12% 11% 17% 16% Metformin with insulin 0% 13% 73% 89% 89% 153% 191% 193% 205% Metformin with sulfonylurea -21% 0% -7% -13% -21% -21% -22% -26% -32% Sulfonylurea 0% -22% -30% -48% -55% -65% -69% -73% -76% 0% 5% 15% 11% 17%

Figure 2: Changes in first T2DM agent dispensed over time relative to 2007/08.





Sulfonylurea

Metformin

Insulin

Other

Figure 3: Second-line T2DM treatment dispensing for the 2007/8 cohort.

Second-line T2DM treatments used in the 2007/8 cohort

On follow-up, the cohort of patients (N=17,206) who were prescribed their first-line T2DM agent in 2007/2008, 46% (N=7,958) received a second-line agent. Of those who received a second-line therapy, the main agents dispensed as monotherapies were sulfonylureas (70%), followed by insulin (14%), metformin (8%) and other (8%) (Figure 3). Acarbose and pioglitazone were dispensed as second-line agents (as monotherapy or in combination with other agents) in 2.4% and 3.4% of this cohort, respectively. In patients who were dispensed metformin as a first-line agent, 86% were started on a sulfonylurea as a second-line agent (Figure 4).

Discussion

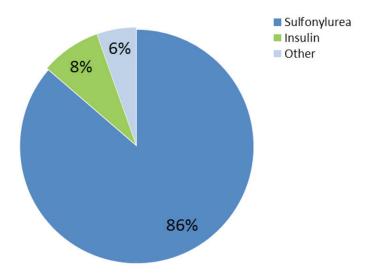
This analysis describes the AA dispensing patterns for T2DM in New Zealand using the national Pharmaceutical Collection database, specifically the choice of first- and second-line agent.

Strengths and limitations

The key strength of this research is that it covers the majority of New Zealand's pharmaceutical dispensing (hence prescribing) patterns and trends at a national level, as the Pharmaceutical Collection dataset includes all government-subsidised dispensing data. This allows for an almost complete picture of dispensing over time.

However, there are a number of limitations to this analysis. Firstly, the Pharmaceutical

Figure 4: Second-line T2DM treatment dispensing for the 2007/8 cohort who were initially dispensed on metformin.





Collection dataset records all dispensing of medications. Dispensing of a T2DM treatment has been used as proxy for patients having true T2DM. This analysis therefore excludes those with T2DM who are not on any medicines and may capture those with impaired glucose tolerance who are treated with diabetic medication.

Secondly, given changes in the dataset over time, the analysis could not reliably extend retrospectively further than 2007/8.

Thirdly, the analysis could not describe the time to transition between first- and second-line treatments to allow assessment of clinical inertia within diabetes management in New Zealand. Further research is warranted into this issue.

Fourthly, the data are dispensing-based, not based on prescription-at-doctor-visit nor patient end-use. Dispensing-based data (rather than prescription-at-doctor-visit or patient end-use) do not capture end-use (ie, whether medicines dispensed are taken by the patient—wastage and suboptimal treatment), nor prescriber intent (since not all prescriptions are necessarily dispensed and captured in the data).

Fifthly, diabetes diagnoses are by inference and will include other conditions where diabetes medicines are used, including polycystic ovary syndrome;¹⁸ however, the numbers of these cases are likely to be relatively small compared with the T2DM population.

Finally, the analysis has not attempted to link dispensing usage with laboratory data such as HbA1c measurements of glycaemic control, nor has it considered the full range of demographic and clinical information—socioeconomic deprivation, region, type of diabetes, macrovascular and microvascular complications, use of other medicines (eg, inhibitors, statins), etc.—to better elucidate key patterns and gaps in the treatment of patients with diabetes.

Implications—international comparison with guidelines adherence

Despite the above limitations, when considering the choice of AA, this is the first analysis we are aware of that has explicitly explored the nationwide dispensing patterns in New Zealand for initial and second-line AAs (hence by implication, prescribing

patterns) over an extended time period. The results indicate that metformin monotherapy has accounted for the majority of all first dispensed T2DM therapies over the nine years studied and currently accounts for 85% of all dispensing. First-line use of sulfonylurea monotherapy has decreased over time. Furthermore, though be it small, there has been a growth in coprescribing of metformin with insulin.

These results indicate high levels of adherence with the national treatment guidelines for T2DM.^{6,7} International studies considering T2DM prescribing patterns have not demonstrated such a high degree of adherence, though guidelines and available treatments can differ across countries. Use of metformin as a first-line agent has ranged from as low as 17% to (a relatively modest) 51%.^{10–14,19–24} Sulfonylurea use as first-line therapy has also ranged from (a still relatively high) 18% to as high as 85%.^{20,22,25,26}

Sulfonylurea monotherapy accounted for 70% of all second-line dispensing (or 86% in those who initially started metformin) for those in the 2007/8 cohort. Again, this demonstrates good adherence with national T2DM treatment guidelines. While concerns have been expressed in the use of sulfonylureas, they remain useful and effective treatments.^{27,28} Internationally for patients initially prescribed metformin, sulfonylureas have been used in 56% or 80% as second-line.29,30 However, the choice of second-line agent can differ across different countries, with a recent study finding dipeptidyl peptidase-4 inhibitors were the most common utilised second-line agent in Japan.²⁴

Future research

This analysis has identified several future research opportunities.

Firstly, there is a need to consider the pattern of AA prescribing by key demographic features (particularly age, gender and ethnicity) to see if there are differences between populations in New Zealand. This is the topic of forthcoming analysis.³¹

Secondly, this analysis could not address the time taken to escalate/add treatments for managing T2DM; this information is critical for addressing the issue of clinical inertia. This topic is attracting increasing attention within the literature, with concerns treatment is not being optimised in patients. ^{16,17} Further research is warranted



into this area in the New Zealand context, particularly if there is differential clinical inertia between ethnic groups.

Conclusions

This analysis of dispensing patterns for AA in New Zealand indicates that there is a high degree of adherence to the T2DM prescribing guidelines. Metformin and

sulfonylureas are the most commonly dispensed first- and second-line agents for T2DM respectively. Further research is warranted into the demographic patterns of AA prescribing, treatment transition timeframes and the issue of clinical inertia (particularly if it is differential across ethnic groups) in managing patients with T2DM in New Zealand.

Competing interests:

The authors are (or were at the time of writing) employees of PHARMAC; the views expressed do not necessarily represent those of PHARMAC.

Author information:

Peter Murray, Medical Directorate, PHARMAC, Wellington; Hew Norris, Analysis, Corporate Directorate, PHARMAC, Wellington; Scott Metcalfe, Medical Directorate, PHARMAC, Wellington; Bryan Betty, Medical Directorate, PHARMAC, Wellington; Vanessa Young, Operations Directorate, PHARMAC, Wellington; Bronwyn Locke, Engagement and Implementation Directorate, PHARMAC, Wellington.

Corresponding author:

Peter Murray, PHARMAC, 9/40 Mercer Street, Wellington 6011. peter.murray@pharmac.govt.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7413

REFERENCES:

- Ministry of Health. Health Loss in New Zealand 1990–2013: A report from the New Zealand Burden of Diseases, Injuries and Risk Factors Study. Wellington: Ministry of Health.
- 2. Coppell KJ, Mann JI,
 Williams SM, et al.
 Prevalence of diagnosed
 and undiagnosed diabetes
 and prediabetes in New
 Zealand: findings from the
 2008/09 Adult Nutrition
 Survey. N Z Med J. 2013
 Mar 1; 126(1370):23–42.
- 3. Krebs J, Coppell KJ, Cresswell P, et al. Access to diabetes drugs in New Zealand is inadequate. N Z Med J. 2016 Jun 10; 129(1436):6–9.
- 4. Ministry of Health.
 2009. Report on New
 Zealand Cost-of-Illness
 Studies on Long-Term
 Conditions. Wellington:
 Ministry of Health.

- 5. Nathan DM, Cleary PA,
 Backlund JY, et al; Diabetes
 Control and Complications
 Trial/Epidemiology of
 Diabetes Interventions
 and Complications (DCCT/
 EDIC) Study Research
 Group. Intensive diabetes
 treatment and cardiovascular disease in patients
 with type 1 diabetes. N
 Engl J Med. 2005 Dec
 22; 353(25):2643–53.
- New Zealand Guidelines Group. New Zealand Primary Care Handbook 2012. 3rd ed. Wellington: New Zealand Guidelines Group; 2012.
- 7. BPAC. Managing patients with type 2 diabetes: from lifestyle to insulin. Best Practice Journal. 2015:72.
- 8. PHARMAC. New Zealand Pharmaceutical Schedule August 2016. Wellington: PHARMAC.

- 9. Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycemia in type 2 diabetes, 2015: a patient-centered approach: update to a position statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care. 2015 Jan; 38(1):140–9. doi: 10.2337/dc14-2441.
- 10. Chang CH, Jiang YD, Chung CH, et al. National trends in anti-diabetic treatment in Taiwan, 2000–2009. J Formos Med Assoc. 2012 Nov; 111(11):617–24. doi: 10.1016/j.jfma.2012.09.009.
- 11. Desai NR, Shrank WH,
 Fischer MA, et al. Patterns
 of medication initiation in
 newly diagnosed diabetes
 mellitus: quality and cost
 implications. Am J Med.
 2012 Mar; 125(3):302.
 e1–7. doi: 10.1016/j.
 amjmed.2011.07.033.



- 12. Raebel MA, Xu S, Goodrich GK, Schroeder EB, et al. Initial antihyperglycemic drug therapy among 241 327 adults with newly identified diabetes from 2005 through 2010: a surveillance, prevention, and management of diabetes mellitus (SUPREME-DM) study. Ann Pharmacother. 2013 Oct; 47(10):1280–91. doi: 10.1177/1060028013503624.
- 13. Sargen MR, Hoffstad OJ, Wiebe DJ, Margolis DJ. Geographic variation in pharmacotherapy decisions for U.S. Medicare enrollees with diabetes. J Diabetes Complications. 2012 Jul–Aug; 26(4):301–7. doi:10.1016/j. jdiacomp.2012.04.001.
- 14. Geier AS, Wellmann I, Wellmann J, et al. Patterns and determinants of new first-line antihyperglycaemic drug use in patients with type 2 diabetes mellitus. Diabetes Res Clin Pract. 2014 Oct; 106(1):73–80. doi: 10.1016/j. diabres.2014.07.014.
- 15. Metcalfe S, Moodie P, Norris H, Rasiah D. Self-monitoring blood glucose test strip use with diabetes medicines in people with types 1 and 2 diabetes in New Zealand. N Z Med J. 2014; 127(1406):48–62. http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2014/vol-127-no-1406/6371
- 16. Khunti K, Millar-Jones D. Clinical inertia to insulin initiation and intensification in the UK: A focused literature review. Prim Care Diabetes. 2017; 11(1):3–12. doi: 10.1016/j.pcd.2016.09.003. URL: http://www. primary-care-diabetes. com/article/S1751-9918(16)30099-7/ fulltext
- 17. Strain WD, et al. Time to do more: addressing clinical inertia in the management of type 2 diabetes mellitus.

- Diabetes Res Clin Pract. 2014 Sep; 105(3):302–12. doi:10.1016/j. diabres.2014.05.005. URL: http://www. diabetesresearchclinicalpractice.com/article/S0168-8227(14)00219-8/abstract
- 18. Tang T, Lord JM, Norman RJ, et al. Insulin-sensitising drugs (metformin, rosiglitazone, pioglitazone, D-chiro-inositol) for women with polycystic ovary syndrome, oligo amenorrhoea and subfertility. Cochrane Database Syst Rev. 2012; 5:CD003053. doi: 10.1002/14651858. CD003053.pub5
- 19. Stephens JM, Gold KF, Botteman MF. Persistence patterns with oral hypoglycemic agents in type 2 diabetes. JCOM. 2002 Sep; 9(9):491–9.
- 20. Guidoni CM, Borges AP, Freitas OD, Pereira LR. Prescription patterns for diabetes mellitus and therapeutic implications: a population-based analysis. Arq Bras Endocrinol Metabol. 2012 Mar; 56(2):120–7.
- 21. Hassan YA, Mathialagan AM, Awaisu AH, et al. Trend in the use of oral hypoglycemic agents in an outpatient pharmacy department of a tertiary hospital in Malaysia (2003–2006). Asian J Pharm Clin Res. 2009; 2:40–6.
- 22. Boccuzzi SJ, Wogen J, Fox J, et al. Utilization of oral hypoglycemic agents in a drug-insured US population. Diabetes Care. 2001; 24(8):1411–5.
- 23. Clemens KK, Shariff S, Liu K, et al. Trends in Antihyperglycemic Medication Prescriptions and Hypoglycemia in Older Adults: 2002–2013. PloS one. 2015 Sep 3; 10(9):e0137596.
- 24. Tanabe M, Motonaga R, Terawaki Y, et al. Prescription of oral hypoglycemic agents in patients with type-2 diabetes mellitus:

- a retrospective cohort study using a Japanese hospital database. J Diabetes Investig. 2016: Doi: 10.1111/jdi.12567.
- 25. Karter AJ, Moffet HH, Liu J, et al. Achieving good glycemic control: initiation of new antihyperglycemic therapies in patients with type 2 diabetes from the Kaiser Permanente Northern California Diabetes Registry. Am J Manag Care. 2005; 11(4):262.
- 26. Trinacty CM, Adams
 AS, Soumerai SB, et al.
 Racial differences in
 long-term adherence to
 oral antidiabetic drug
 therapy: a longitudinal
 cohort study. BMC Health
 Serv Res. 2009; 9(1):1.
- 27. Abrahamson MJ. Should sulfonylureas remain an acceptable first-line add-on to metformin therapy in patients with type 2 diabetes? Yes, they continue to serve us well!. Diabetes care. 2015; 38(1):166–9.
- 28. Genuth S. Should Sulfonylureas Remain an Acceptable First-Line Add-on to Metformin Therapy in Patients With Type 2 Diabetes? No, It's Time to Move On!. Diabetes Care. 2015; 38(1):170–5.
- 29. Chang YC, Chuang LM, Lin JW, et al. Cardiovascular risks associated with second-line oral antidiabetic agents added to metformin in patients with type 2 diabetes: a nationwide cohort study. Diabet Med. 2015; 32(11):1460–9.
- 30. Morgan CL, Poole CD, Evans M, et al. What next after metformin? A retrospective evaluation of the outcome of second-line, glucose-lowering therapies in people with type 2 diabetes. J Clin Endocrinol Metab. 2012; 97(12):4605–12.
- 31. Murray P, et al. Metformin and sulfonylurea prescribing in New Zealand When are they being started?
 In preparation for the NZ Medical Journal.



A review of squamous cell vulvar cancers in Waikato region, New Zealand

Prashanth Hari Dass, Marion JJ Kuper-Hommel

ABSTRACT

BACKGROUND: Squamous cell vulvar cancers (SCVC) are rare. Although management guidelines have recently been published, New Zealand studies presenting "real world" outcomes are limited.

METHODS: Retrospective single-centre review of SCVC diagnosed between 1 January 2000 and 31 August 2015. Clinical characteristics and outcomes were reviewed.

RESULTS: Among 47 cases reviewed, 38 were ethnically European and 9 Māori. Cases identified as Stage 1 (16), Stage 2 (5), Stage 3 (17), Stage 4 (9). For Stages 1, 2, 3 and 4, (16, 4, 17 and 6) were managed by local excision; (9, 1, 14 and 2) by node dissection and (2, 1, 3 and 5) by chemoradiotherapy respectively. Wound cellulitis (10) and lymphedema (8) were the commonest acute and late complication, respectively. Seven patients were treated with 5-Fluorouracil and Mitomycin, and four received weekly Cisplatin. Grade 3 toxicities seen in five cases treated with 5-Fluorouracil and Mitomycin versus none in the Cisplatin group. No local recurrences observed in patients treated with chemoradiation. Patients with Age Adjusted Charlson Comorbid Index Score (ACCIS) <5 had better overall survival (OS) compared to scores ≥5 (60% versus 41%) with 33 months median follow-up. Five-year OS and disease-free specific survival was 73% and 94% (Stage 1), 40% and 60% (Stage 2), 44% and 59% (Stage 3) and 29% (Stage 4) respectively.

CONCLUSIONS: We present "real world" outcomes of vulvar cancers in this older and comorbid population. Larger, prospective multi-centre studies are proposed.

ulvar cancers account for 0.6% of female cancers in the US.1 In New Zealand, the Ministry of Health registered 70 vulvar cancer cases in 2014 with rates reported to be between 0.2 to 0.3% between 2003 to 2013.2 Surveillance Epidemiology and End Results (SEER) databases between 1973-2004 documented a rise in the incidence of invasive vulvar tumours by 1% per year.3 Risk factors for developing vulvar cancers include human papilloma virus (HPV), accounting for 40% of vulvar cancer cases.4 Squamous cell histology has been reported in up to 95% of vulvar cancers. Most patients present with pruritus followed by vulvar bleeding, discharge, dysuria and pain.5 Presence of inguinal or femoral nodes is the most important prognostic factor for survival.6

Patients with early stage vulvar cancer (Stage 1 and 2) should undergo excision of the primary tumour. Surgical margins

greater than 1cm have been suggested to reduce the risk of local recurrence.⁷ There is growing evidence for sentinel node evaluation in these patients.⁸ Radiation therapy (RT) is offered to patients with close or positive margins (Stage 2) or with inguinal lymph node metastases to reduce risk of locoregional recurrence and improve survival.^{9,10} Patients with locally advanced (Stage 3 and 4a) disease who are surgical candidates should be recommended inguinofemoral lymphadenectomy.¹¹ Chemoradiation or radiation alone is recommended in patients who are not surgical candidates.⁵

In New Zealand, studies providing an update of factors affecting therapeutic management of patients with vulvar cancers are limited. The objectives of our study were to review patient characteristics, treatment choices and outcomes of patients with vulvar cancers in the Waikato Region, New Zealand.



Methods

Retrospective review of all newly diagnosed vulvar cancer cases registered in a regional cancer centre with a catchment population of 720,000 between 1 January 2000 and 31 August 2015. Cases were identified from the databases of the departments of clinical coding, radiation oncology and medical oncology of Waikato Hospital. Using the patient's National Health Index (NHI) number, details of hospital admissions, electronic patient files and individual patient's clinic notes were reviewed and evaluated for completeness of pathological reporting and treatment-related side effects. Data reviewed include ethnicity, age at diagnosis, comorbidities using Age-Adjusted Charlson Comorbidity Index Score (ACCIS), smoking status, body mass index, clinical presentation, primary therapy (surgery, radiation or combined chemoradiation), date of histological diagnosis, staging, complications of primary therapy, recurrences and mortality. For staging, the Federation International Gynaecologic Oncology (FIGO) and TNM classification system were used. ACCIS for each individual patient was calculated, which allowed comparison between cases with regards to treatment, toxicities and outcomes. Patients were reviewed weekly during chemoradiation and 2-3 monthly thereafter. Median follow-up was 33 months. Overall survival was calculated from the date of diagnosis to the date of death from any cause or date last known alive, using the Kaplan Meier method. Health and Disability Ethics Committees (HDEC) review was not required for this study as per Standard Operating Procedures HDEC Version 2.0 August 2014.

In the 15-year period, 60 cases with vulvar cancer were identified from the three different databases. Thirteen cases were excluded; 11 cases had another histological diagnosis, which included vulvar basal cell carcinoma (3), extramammary Paget's disease of the vulva (2), vulvar adenocarcinoma (2), vulvar melanoma (1), leiomyosarcoma (1), metastatic adenoid cystic carcinoma (1) and high-grade neuroendocrine cancer (1). Two cases with vulvar squamous cancer were excluded as they were diagnosed prior to year 2000. This report covers 47 cases of vulvar squamous

cell carcinoma. Between 2000–2004 eleven cases were identified, fourteen cases in 2005–2009 period and eighteen cases in 2010–2014 period.

Results

Forty-seven patients had vulvar squamous cell cancer graded as well differentiated (28%), moderately differentiated (60%) and poorly differentiated (11%). Depth of invasion 1mm or less was seen in four cases versus 33 cases reported having depths of invasion greater than 1mm. Tumour margins (<8mm) were initially identified in 13 cases with seven of the cases requiring re-excision, and among these only three cases had clear margins following re-excision. Depth of invasion, lymphovascular invasion and vulvar intraepithelial neoplasia appeared to be reported in 79%, 68% and 55% respectively. However, p16, p53 and HPV DNA and Ki67 were poorly reported; ie, 21%, 6% and 6% of cases respectively. Patient characteristics are shown in Table 1.

The median age of our patients was 69 years (range 39–94). Eighty-one percent (38 patients) were of European ethnicity and 19% (nine patients) Māori. Thirty-six percent (n=17) of patients were either current smokers or ex-smokers; 45% (n=21) were non-smokers. Smoking status was unknown in 19% (n=9). Median age at presentation for Māori was 57 years versus 76 years for European. Vulvar pruritus, a lump, pain and bleeding were the most common initial symptoms. Thirty-one percent (n=15) of cases had a background of lichen sclerosus. Hypertension, obesity, ischaemic heart disease, diabetes and second malignancies were the top five comorbidities observed among patients (Figure 1). Each patient's ACCIS was evaluated (median score 4).

Over the 15 years, treatment had changed. More formalised imaging, emerging sentinel node evaluation and discussion in multidisciplinary meetings were observed in the latter years. Overall, only 36% of our patients had an initial staging CT scan; 6% had a CT scan at suspected progression and 2% had PET CT scans. Nine percent had an initial MRI scan for evaluation. We observed 54% mortality rate among patients with lymph node involvement versus 39% without lymph node involvement at diagnosis with



 $\textbf{Table 1:} \ \textbf{Vulvar cancer patient demographics and treatment according to stage.}$

Age	Stage	ACCIS	TE	ND	Local rec	Surgical complications	RT intent and dosage	Chemotherapy
39	I	2	Yes	Bilateral	Vulval	Cellulitis and seroma	Curative 50Gy at rec	
47	I	1	Yes	No	Groin	No	Curative 54Gy inguinal + pelvic	
53	I	4	Yes	Bilateral	No	Groin hematoma, cellulitis and lymphedema		
65	I	6	Yes	No	No	No		
83	I	10	Yes	No	No	No		
84	I	5	Yes	No	No	No		
76	I	6	Yes	Bilateral	Vulval	Cellulitis and lymphedema	Curative 49Gy vulva	
40	I	2	Yes	Bilateral	Groin	Lymphedema	Curative 50Gy vulva. RT to bilateral groin at rec	
57	I	2	Yes	Unilateral	No	No	Curative 45Gy vulva	
92	ı	7	Yes	No	No	No		
51	I	2	Yes	No	No	No	Curative 54Gy inguinal + pelvic	5FU mitomycin
55	I	2	Yes	Unilateral	No	No	Curative 50Gy	
69	I	5	Yes	Unilateral	No	No	Curative 45Gy	5FU mitomycin
77	I	7	Yes	No	Vulval	No	Curative 50Gy at rec	
81	I	6	Yes	Bilateral	No	Cellulitis, wound discharge and lymphedema	Curative 45Gy perineum	
64	ı	5	Yes	Bilateral	No	No	Curative 50Gy at rec	
73	II	7	No	No	No	Biopsy complicated by cardiac arrest	Curative 54Gy vulva and groin	
78	П	8	Yes	No	Groin	No	Curative 50Gy +Groin	
91	II	5	Yes	No	No	Delirium post- operatively		
81	II	4	Yes	Bilateral	Groin	No	Palliative + vulva boost 60Gy at rec	
65	П	3	Yes	No	No	No	Curative 54Gy pelvis	5FU mitomycin
60	III	2	Yes	Unilateral	No	No	Curative 50Gy	
83	III	6	Yes	No	No	No	Curative 54Gy	5FU mitomycin
85	III	5	Yes	Unilateral	No	Lymphedema		
63	III	3	Yes	Bilateral	Vulval	Lymphedema	Curative 54Gy at rec	
57	III	2	Yes	No	No	No	Curative 50Gy vulva	
63	III	10	Yes	Bilateral	No	Seroma		
67	III	5	Yes	Bilateral	No	Wound rupture and cellulitis	Curative 45Gy	
82	III	5	Yes	No	No	No	Curative 45Gy pelvis and groin	
84	III	6	Yes	Bilateral	Vulval	Pulmonary embolism	Curative 45Gy	



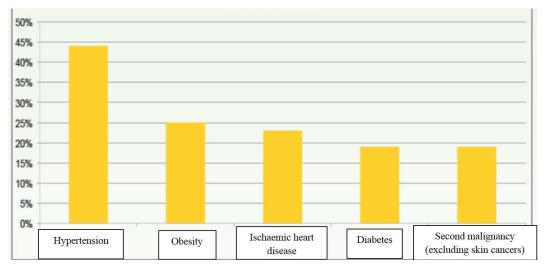
Table 1: Vulvar cancer patient demographics and treatment according to stage (continued).

Age	Stage	ACCIS	TE	ND	Local rec	Surgical complications	RT intent and dosage	Chemotherapy
75	III	4	Yes	Bilateral	Vulval	Cellulitis	Curative 45Gy at Dx, then palliative RT to vulvar	
49	III	1	Yes	Unilateral	No	No	Curative 45Gy groin and pelvis	Cisplatin
79	III	4	Yes	Bilateral	No	Cellulitis, delayed wound healing	Curative 50Gy pelvis and nodes	
87	III	5	Yes	Bilateral	No	Cellulitis, diarrhoea, lymphocele, hematoma	Curative 45Gy groin and vulva, palliative RT lung 20Gy	
76	III	4	Yes	Bilateral	Vulval	No	Curative 50Gy groin at Dx, 50Gy vulva at rec	
50	III	1	Yes	Bilateral	No	No	Curative 54Gy	Cisplatin
76	III	5	Yes	Bilateral	Vulval	No	Curative 61Gy	
81	III	6	Yes	Bilateral	No	Stroke, incontinence, lymphedema		
42	IV	1	No	No	No	No	Curative 59Gy	5FU mitomycin
94	IV	4	Yes	No	No	No	Palliative 56Gy groin	
65	IV	4	Yes	No	No	Cellulitis and wound dehiscence	Palliative RT to L3-5 20G + pelvis and groin	
50	IV	4	Yes	Bilateral	No	Cellulitis and lymphedema	Curative 59Gy pelvis	Cisplatin
64	IV	4	Yes	No	No	No	Palliative 54Gy groin	Cisplatin
70	IV	4	Yes	Bilateral	No	No	Curative 45Gy + brachytherapy 15Gy	5FU mitomycin
79	IV	4	Yes	No	No	No	Curative vulva 54Gy	5FU mitomycin
56	IV	4	No	No	No	No	Palliative 54Gy groin	
84	IV	6	No	No	No	No	Palliative pelvis (RT interrupted due to progression)	

ACCIS: Age Adjusted Charlson Comorbidity Index Score

RT: Radiotherapy TE: Tumore Excised Dx: Diagnosis ND: Node Dissection Rx: Treatment Rec: Recurrence

Figure 1: Top five comorbidities in squamous cell vulvar cancer by percentage (%).





a median follow-up of 33 months. Chemotherapy was only observed to be used from 2007 onwards and not between 2000–2006. Sixty-six percent of cases were discussed in a multidisciplinary meeting (MDM) at initial diagnosis. This is explained by the change in recommendations over the course of time with cases diagnosed prior to 2005 less likely to be discussed in an MDM.

Treatment and complications (refer to Table 1)

Surgery

Patients with early stage disease (Stage 1 and 2) were more likely to undergo local excision of their primary tumour compared to patients with advanced disease (Stage 3 and 4) (94% versus 88%). Lymph node dissections were less likely to be performed in early stage disease versus advanced disease (48% versus 61%). The low rates of node dissection in Stage 1 are due to advanced age and comorbidities; (five patients), negative sentinel node and patient choice (one patient each respectively).

Surgical complications were more common in advanced stage versus early stage (42% versus 33%). The most common acute wound complication was cellulitis (21%). The most common chronic complication was lymphedema (17%). No treatment-related deaths were observed. Only three patients underwent sentinel node (two bilateral and one unilateral) based

management. Among these, one case was positive and lead to inguinal node dissection with positive lymph node involvement. The remaining two negative sentinel node cases have since had no groin recurrences. Eleven percent were deemed inoperable due to extensive tumour infiltration (two patients), and extensive comorbidities (three patients).

Chemoradiotherapy

Eleven patients (23%) received concurrent chemoradiation either in the form of 5 fluorouracil (5FU)-mitomycin (63%, seven patients) or weekly cisplatin (36%, four patients). The median age of patients in 5FU-mitomycin versus cisplatin arms was 69 years and 50 years, respectively. From our small number of patients who received chemotherapy (n=11), we observed that weekly cisplatin was a much better tolerated regime compared to 5FU-mitomycin (Table 2). Comparing between the two chemotherapy arms, ACCIS median for 5FU-mitomycin versus cisplatin were 4 and 2.5, respectively.

Thirty-eight patients (80%) received radiation treatment with either curative (mean 50 Gray, range 45–61 Gray) or palliative intent (which included primaries: vulva and groin; and secondaries: lumbar spine and lung). Radiotherapy infield recurrence was seen in four patients (Table 1). Among the remaining patients who did not receive radiation treatment, four (8.5%) were unfit for treatment due to comorbidities and five (10.6%) had their treatment completed with surgery.

Table 2: Complications observed in the 5FU-mitomycin and cisplatin chemoradiation group by stage and age.

5FU-mi	5FU-mitomycin group			versus cisplatin group			
Stage	Age	Complications in 5FU-mitomycin group		Age	Complications in cisplatin group		
Ib	51	Grade III mucositis and diarrhoea Mallory Weiss tear	IIIb	49	Nil		
Ib	69	PICC line thrombus and cellulitis	IIIc	50	Nil		
П	65	Grade III mucositis	IVA	50	Nil		
Ш	83	Grade III mucositis	IVA	60	Nil		
IV	42	Nil					
IVA	70	Nil					
IVA	79	Vulvitis, UTI and diarrhoea post cycle 1 chemotherapy Hypovolemia and syncope post cycle 2 chemotherapy					



A total of eleven patients with Stage 1 disease received radiotherapy. Seven were administered radiotherapy due to high-risk features such as positive margins (three cases), inoperable (two cases), upstaged (two cases). In the remaining four cases, radiotherapy was administered at cancer recurrence. In three cases that had positive tumour margins, ie, (two had persistent positive tumour margins despite surgical re-excision, and one patient was surgically unfit for re-excision). Two patients' tumours were located close to the perineum or anal verge and thus deemed inoperable. Two cases were upstaged during follow-up postoperatively with inguinal nodes and thus managed with radiotherapy.

Five-year overall survival is shown in Figure 2. Whereas five-year disease free specific survival for Stages 1, 2, 3 and 4 were 94%, 60%, 59% and 29% respectively. The discrepancy between overall survival and disease-specific survival is reflected

by this comorbid cohort of patients. For example, two patients with Stage 1 had died of a second malignancy, and another patient deemed unfit for surgical excision of the primary tumour due to advanced age and comorbidities. A review of Stage 2 patients revealed four of the five patients with advanced age (median age of 78 years). One Stage 2 patient was inoperable and eventually died of ischaemic heart disease. Another Stage 2 patient developed delirium postoperatively and was hence unfit for further adjuvant treatment.

Independent of stage, overall survival outcomes were better for (ACCIS) <5 (60%) versus 41% for scores ≥5 (Figure 3). At the time of data analysis, 22 of the 47 patients had already deceased; with a median time to death from date of diagnosis among deceased patients reported at 23 months. The median follow-up of 33 months (range 3–161 months) was uncensored for death with a mean of 44 months.

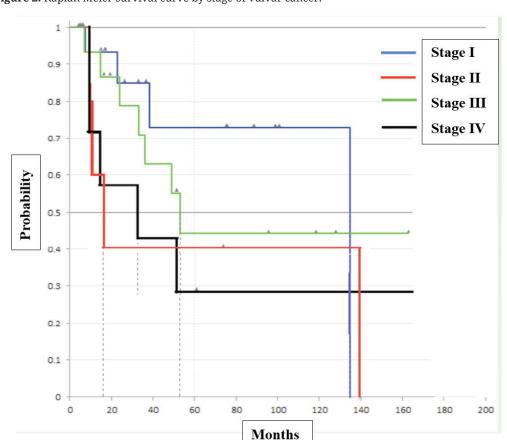


Figure 2: Kaplan Meier survival curve by stage of vulvar cancer.



Alive Deceased

Figure 3: Bar graph showing Age-Adjusted Charlson Comorbid Index Score (ACCIS) versus number of patients alive and deceased.

Discussion

We report one of the largest single-centre reviews of vulvar cancers in Australasia. Multiple factors including patient choice, tumour location, advanced age, comorbidities and treatment complications have influenced and individualised treatment. Variation in treatment over the course of time, such as evolving sentinel node evaluation, advancements in imaging modalities and greater proportion of cases discussed in MDM in the latter years were observed. Although surgical and chemoradiotherapy were associated with high morbidity, we observed that cisplatin radiotherapy was much better tolerated compared to 5FU-mitomycin, however this requires validation in larger prospective studies. Independent of stage, patients with fewer comorbidities had a better overall survival.

Our study showed that Māori tend to present with vulvar tumours younger, with less comorbidities compared to Europeans. Evidence shows that the median life expectancy at birth in New Zealand for non-Māori and Māori is 82 years and 75 years respectively. Wu et al studied vaginal cancers among different races in the US and showed that African, Asian, Pacific Island and older women were more likely to be diagnosed with advanced disease, and these groups had lower five-year relative survival rates than their Caucasian, non-Hispanic, and younger counterparts. Babarinsa et al 4 add

that most patients present late and default during treatment. Our study was too small to evaluate overall survival differences between ethnicities.

The rate of HPV pathological reporting in our centre was poor. Improving HPV reporting will be useful in the future as the presence of "HPV-16 antibodies confer a 5.3-fold risk of vulvar neoplasia. High antibody levels are associated with 20-fold risk, more commonly seen in smokers" probably related to decreased clearance of HPV infection among smokers. ^{4,15} We identified 31% cases had lichen sclerosus prior to diagnosis of vulvar cancer, which suggests the well-known premalignant feature seen more commonly in the elderly. Lichen sclerosus and VIN rates in both ethnicities were similar in our study.

Another predisposing factor of vulvar cancer includes low socioeconomic status and smoking. Thirty-six percent of our patients had prior smoking history. Socioeconomic status was not evaluated in our study. We identified metabolic syndrome to be highly prevalent. At least 20% of our patients had hypertension, diabetes, ischaemic heart disease and obesity. The Metabolic Syndrome and Cancer project identified metabolic syndrome to increase vulvar cancer risk (hazard ratio 1.78). 16

Factors tailoring towards different surgical approaches in the 15-year period include comorbidities, age, depth of invasion and



lateralisation of the tumour. Twelve local recurrences (eight vulvar and four groins) were identified. Although the numbers of patients receiving chemoradiation were small, no local recurrence was observed in our patients who received chemoradiation. A vulvar squamous cancers study in Italy in 502 patients showed that 37% developed recurrences. "Five-year survival rate was 60% for perineal recurrences, 27% for inguinal and pelvic recurrences, 15% for distant recurrences and 14% for multiple recurrences". Factors predicting for risk of recurrences were FIGO stage >2, positive lymph nodes and vascular space invasion, which were all statistically significant. 17,18 The low recurrence rate in our chemoradiotherapy group may be a reflection of selection bias and short follow-up period.

Despite lower rates of infection, wound breakdown and lymphedema observed in our study, surgery is associated with significant morbidity. To address this issue, tertiary centres have been offering sentinel node evaluation. Only 6% of our patients underwent sentinel node evaluation. Chronic lymphedema lasting beyond six months has been described to be worse among those with greater extent of lymphadenectomy, sartorius transposition and adjuvant groin irradiation. Sentinel lymph node biopsy, although not yet the standard of care, is safe and recommended in patients with early stage disease to reduce complications. 6.21

Evidence surrounding the choice of chemotherapy used is based on Phase 2 studies. Moore et al evaluated radiation treatment and weekly cisplatin (40mg/m²) in patients not amenable to surgery. Although 64% complete response was observed, 31% did not complete treatment with a 3.4% death rate, 16% Grade 3 or higher toxicities and 7% refused treatment.22 Another study of 28 patients with locally advanced vulvar cancer that evaluated chemoradiotherapy with 5FU-mitomycin observed an 85% overall response, with vulvar desquamation seen in 93% of patients.23 We observed that weekly cisplatin was much better tolerated compared to 5FU-mitomycin. One may argue that our patients receiving weekly cisplatin were much younger (median 50 years) with a lower ACCIS compared to the 5FU-mitomycin cohort (median 69 years). Older and

more comorbid patients may have been treated with 5FU-mitomycin perhaps due to age selection bias. Data surrounding tolerability and selection criteria of chemotherapy in patients above 65 years of age is scarce. One study assessing chemoradiation using cisplatin and paclitaxel in elderly patients (aged >70 years) with oesophageal cancer showed that 67% completed treatment as scheduled, with the expense of significant haematological toxicity.²⁴

We observed that higher comorbidity score among cancer patients impacted on their overall survival. This finding is supported by a retrospective analysis in vulvar cancer, which showed that Charlson score 2 or greater was associated with a decreased overall survival (hazard ratio 3.03).25 Sogaard et al further highlight that the comorbidities among cancer patients influence the receipt of standard of care, with higher treatment associated complication rate and overall survival.²⁶ Although ACCIS has been demonstrated as a reliable risk measure for perioperative cancer surgery,27 literature on its routine use to optimise care in either vulvar cancer management or multidisciplinary cancer meetings are limited.

The limitations of our study include its small sample size, single centre and retrospective nature. HPV reporting was poor and treatments altered during the time. In advanced disease, chemoradiotherapy demonstrated excellent local control, with cisplatin better tolerated compared to 5FU-mitomycin, requires validation in larger prospective studies and longer follow-up. The impact and importance of expert MDM review is emphasised and recommended. We propose that ACCIS could be incorporated as a tool to reduce treatment morbidity and mortality and may potentially further assist with accurate decision making in vulvar cancer MDMs. This requires further review in prospective larger studies.

Conclusions

We present the "real world" outcomes of a rare malignancy. Treatment is associated with high morbidity in this generally older and comorbid population. Larger prospective multi-centre studies are proposed.



Competing interests:

Nil.

Acknowledgements:

We sincerely thank Dr Ian Kennedy, the staff of Waikato Hospital Oncology Department and Sai Lakshmi Panatt for their valued input during the study.

Author information:

Prashanth Hari Dass, Medical Oncology Clinical Trial and Research Fellow, University of Oxford, United Kingdom; Marion JJ Kuper-Hommel, Medical Oncologist, Lomas Building, Waikato Hospital, Pembroke Street, Hamilton.

Corresponding author:

Dr Prashanth Hari Dass, Medical Oncology Clinical Trial and Research Fellow, University of Oxford, United Kingdom.
pranava108@hotmail.com

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7406

REFERENCES:

- Saraiya M, Watson M, Wu X, et al. Incidence of in situ and invasive vulvar cancer in the US 1998–2003. Cancer Supplement. 2008; 113(S10):2865–2872.
- 2. Ministry of Health. Cancer: New registrations and deaths – series. http:// www.health.govt.nz/ nz-health-statistics/healthstatistics-and-data-sets/ cancer-new-registrationsand-deaths-series
- 3. Bodelen C, Madeleine MM, Voigt LF, Weiss NS. Is the incidence of invasive vulvar cancer increasing in the United States? Cancer Causes Control. 2009; 2099:1779.
- 4. De Vuyst H, Clifford GM, Nascimento MC, et al. Prevalence and type distribution of human papillomavirus in carcinoma and intraepithelial neoplasia of the vulva, vagina and anus: a meta-analysis. Int J Cancer 2009; 124(7):1626–36.
- Alkatout I, Schubert M, Garbrecht N, et al. Vulvar cancer: epidemiology, clinical presentation, and management options. Dovepress. International Journal of Women's Health 2015; 7:305–313.

- Woelber L, Eulenburg C, Choschzick M, et al. Prognostic role of lymph node metastases in vulvar cancer and implications for adjuvant treatment. Int J Gynecol Cancer. 2012 Mar; 22(3):503–8.
- Heaps JM, Fu YS, Montz FJ, et al. Surgical-pathologic variables predictive of local recurrence in squamous cell carcinoma of the vulva. Gynecol Oncol 1990; 38:309–314.
- 8. Te Grootenhuis a NC, Van Der Zee AGJ, Van Doorn BHC, et al. Sentinel nodes in vulvar cancer: Long-term follow-up of the GROningen International Study on Sentinel nodes in Vulvar cancer (GROINSS-V) I. Gynaecologic Oncology Sep 2015.
- 9. Faul CM, Mirmow D, Huang Q, et al. Adjuvant radiation for vulvar carcinoma: Improved local control. Int J Radiat Oncol Biol Phys 1997; 38:381–9.
- **10.** Barnes EA, Thomas G. Integrating radiation into the management of vulvar cancer. Semin Radiat Oncol 2006; 16:168–76.
- **11.** Koh W-J, Greer BE, Abu-Rustum, et al. Vulvar Cancer NCCN Clinical

- Practice Guidelines in Oncology. Version 1.2016. National Comprehensive Cancer Network.
- 12. Marriott L, Sim D.
 Indicators of Inequality
 for Māori and Pacific
 PeopleWORKING PAPERs
 in Public Finance, VictoriaBusiness School, August
 2014. www.victoria.ac.nz/
 sacl/.../WP09_2014_Indicators-of-Inequality.pdf
- 13. Wu X, Matanoski G, Chen VW, et al. Descriptive epidemiology of vaginal cancer incidence and survival by race, ethnicity, and age in the United States. Cancer Supplement. 2008; 113(S10):2873–2882.
- 14. Babarinsa IA, Fakokunde FA, Ogunbiyi JO, et al. Vulvar and vaginal cancers as seen at the University College Hospital, Ibadan, Nigeria. Afr J Med Med Sci. 1999; 28(1–2):77–80.
- 15. Syrjänen K, Shabalova I, Petrovichev N, et al. Smoking is an independent risk factor for oncogenic human papillomavirus (HPV) infections but not for high-grade CIN. Eur J Epidemiol. 2007; 22:723–735.
- **16.** Nagel G1, Concin H, Lukanova, et al. Metabolic syndrome and rare



- gynecological cancers in the metabolic syndrome and cancer project (Me-Can). Ann Oncol. 2011 Jun; 22(6):1339–45. doi: 10.1093/annonc/ mdg597. Epub 2010Oct 21.
- 17. Heaps JM, Fu YS, Montz FJ, et al. Surgical-pathologic variables predictive of local recurrence in squamous cell carcinoma of the vulva. Gynecol Oncol 1990; 38:309.
- 18. Maggino T, Landoni F, Sartori E, et al. Patterns of recurrence in patients with squamous cell carcinoma of the vulva. A multicenter CTF Study. Cancer 2000; 89:116.
- 19. Rouzier R, Haddad B,
 Dubernard G, et al.
 Inguinofemoral dissection
 for carcinoma of the vulva:
 effect of modifications of
 extent and technique on
 morbidity and survival. J
 Am Coll Surg 2003; 196:442.
- 20. Gaarenstroom KN, Kenter GG, Trimbos JB, et al.
 Postoperative complications after vulvectomy and inguinofemoral lymphadenectomy using separate

- groin incisions. Int J Gynecol Cancer 2003; 13:522.
- 21. Van der Zee AG, Oonk MH, De Hullu JA, et al. Sentinel node dissection is safe in the treatment of early-stage vulvar cancer. J Clin Oncol 2008; 26:884.
- 22. Moore D, Ali S, Koh W, et al. (2012) A phase II trial of radiation therapy and weekly cisplatin chemotherapy for the treatment of locally-advanced squamous cell carcinoma of the vulva: a gynaecologic oncology group study. Gynecol Oncol 124: 529–533.
- 23. Tans L, Ansink AC, Mens JW, et al. The role of chemo-radiotherapy in the management of locally advanced carcinoma of the vulva: single institutional experience and review of literature. JWAm J Clin Oncol. 2011 Feb; 34(1):22–6. doi: 10.1097/COC.0b013e3181cae6a1.
- 24. Song T, Zhang X, Fang M, Wu S. Concurrent chemoradiotherapy using paclitaxel plus cisplatin in the treat-

- ment of elderly patients with esophageal cancer Onco Targets Ther. 2015; 8:3087–3094. Published online 2015 Oct 22.
- 25. Ghebre RG, Posthuma R,
 Vogel RI, et al. Effect of age
 and comorbidity of the
 treatment and survival of
 older patient with vulvar
 cancer. Gynecol Oncol.
 2011 Jun 1; 121(3):595–599.
 Published online 2011
 Mar 12. doi: 10.1016/j.
 ygyno.2011.02.005
- 26. Sogaard M, Thomsen RW, Bossen KS, et al. The impact of comorbidity on cancer survival: a review. Clin Epidemiol. 2013; 5(Suppl 1):3–29. Published online 2013 Nov 1. doi: 10.2147/CLEP.S47150
- 27. Chang CM, Yin WY,
 Wei CK, et al. Adjusted
 Age-Adjusted Charlson
 Comorbidity Index Score
 as a risk measure of
 perioperative mortality
 before cancer surgery.
 PLoS One 11(2):e0148076.
 Published online 2016
 Feb 5. doi: 10.1371/
 journal.pone.0148076



Child morbidity as described by hospital admissions for primary school aged children in Tonga 2009–2013

Fiona Catherine Langridge, Sione Vaioleti Hufanga, Malakai Mahunui 'Ofanoa, Toakase Fakakovikaetau, Teuila Mary Percival, Cameron Charles Grant

ABSTRACT

AIMS: To describe inpatient utilisation patterns for primary school aged children in Tonga.

METHODS: We described admissions for children aged 5–11 years to the main hospital in Tonga from January 2009 to December 2013. Rates with 95% confidence intervals (CI) were compared using rate ratios (RR).

RESULTS: There were 1,816 admissions. The average annual admission rate was 20.2/1,000 (95% CI 19.3–21.1). Hospital admission rates were higher in younger than older children (5–7 versus 8–11 years, RR=1.28, 95% CI 1.18–1.41) and in boys than girls (RR=1.52, 95% CI 1.38–1.68). Injury and poisoning (28%), non-respiratory infectious diseases (19%), respiratory conditions (16%), abdominal/surgical conditions (13%) and dental (9%) were the most frequent admission reasons.

A larger proportion of younger versus older children were hospitalised for dental (16% vs 1%, P<0.001) or respiratory conditions (18% vs 14%, P=0.02). A larger proportion of older children were hospitalised for abdominal/surgical conditions (15% vs 11%, P=0.008), other infectious diseases (21% vs 17%, P=0.04), other conditions (10% vs 6%, P<0.001) and cardiac conditions (2% vs 1%, P<0.001).

CONCLUSIONS: In children 5–11 years in Tonga, 85% of admissions were for five groups of conditions. These data inform priority areas for healthcare spending and enable comparisons over time and between different Pacific countries.

The Millennium Development Goals have provided a focus for the global reporting of child health in recent decades¹ by describing child population health using under-five year, neonatal and infant mortality rates.2 The more recently adopted Sustainable Development Goals continue the focus on mortality measures in the under-five age group with the stated goal "By 2030, end preventable deaths of newborns and children under five years of age, with all countries aiming to reduce neonatal mortality to at least as low as 12 per 1,000 live births and under-five mortality to at least as low as 25 per 1,000 live births".3 While this preschool-age focus has been necessary, a

consequence of it has been relative ignorance of the health of children beyond five years of age.⁴

Eighty-four percent of deaths globally in children <5 years old are due to seven causes: neonatal problems, pneumonia, diarrhoea, malaria, measles, human immunodeficiency virus (HIV)/acquired immune deficiency syndrome (AIDS), and injuries.² A range of initiatives have resulted in improvement nutrition, immunisation, newborn health and case management of diseases such as pneumonia and diarrhoea.⁵ As a result, more children are now surviving beyond five years of age.



For primary school aged children in resource-limited countries, both cause of death and burden of disease have been investigated less comprehensively than in the under five-year age group. The data that are available suggest that allergic disease and bronchiectasis are becoming more prevalent in primary school aged children in developing countries. 6,7 Gastrointestinal conditions, including acute, chronic, persistent diarrhoea and constipation, are also common.8,9 The global burden of disease study reports that globally unintentional injuries are the cause of 10-20% of deaths in children aged 5–14 years.¹⁰ Dental caries is a common chronic disease in childhood, 11 estimated to affect 60-90% of school aged children,12 despite being largely preventable.13

Health issues that are prevalent in primary school aged children predict future public health priorities, and social and economic development. Currently, global research in the developing world context in this age group is limited to studies that have focused on specific health issues such as nutrition, ear and hearing health, erspiratory health, parasitic disease, communicable disease, and oral health. Grouped disease to date there does not appear to be any study that has quantified disease burden at a population level in this age group in the developing world setting.

In addition to being limited in scope, studies of health in school-aged children in resource-limited countries has been restricted to the African and Asian regions. In the Pacific region, the 5–12 year-old age group has received minimal attention and the available information is largely anecdotal. Research on the strategies to improve health in this primary school age group is just as scant, although there is growing evidence for school-based interventions. Research

This lack of data is of specific relevance to New Zealand, where 11.3% of the population identify with a Pacific ethnicity and over a third (35.7%) of Pacific peoples were aged under 15 years in the 2013 Census compared to one-fifth (20.4%) of the total population.³¹ Contemporary data on women of child bearing age in New Zealand shows that approximately half of those of Pacific ethnicity were born in the Pacific. Hence,

their health during childhood in the Pacific is likely to impact upon their adult health in New Zealand and the health of their children.³²

In order to identify priority areas for promoting health in primary school aged children in the Pacific, an understanding of the predominant health issues affecting them is required. Our aim in this study was to describe patterns of hospital utilisation for primary school aged children living in Tonga to help begin to understand the epidemiology of primary school aged health in the Pacific region.

Methods

Study setting

We completed our study in Tonga, a South Pacific nation of 170 islands (36 inhabited), with the majority of the population living on the main island of Tongatapu. The total estimated population of Tonga (2011) is $103,252^{33}$ with 38% in the 0–14 age year group. 34

We completed an audit of published literature on child health research in Tonga. This audit identified a small literature generally focused on single health issues or diseases, for example immunisation, 35–37 nutrition, 38–40 rheumatic heart disease, 41–43 respiratory disease 44,45 and oral health. 46

The Tongan healthcare system

In Tonga, health services are free and services are accessible except for those living in the outer islands.³⁴ Primary health care in Tonga is delivered through 14 health centres. There is one central referral hospital (Vaiola Hospital), and three community hospitals located on three of the smaller groups of islands.

Ethics

Ethical approval was granted by the University of Auckland Participants Ethics Committee and the Tonga National Health Ethics and Research Committee.

Study sample

We collected data on all admissions to Vaiola Hospital from January 2009 to December 2013 of children aged 5–11 (inclusive) years. Vaiola is a 200-bed hospital with a dedicated 34-bed paediatric inpatient facility. There is one paediatric consultant, two junior paediatric registrars and two paediatric interns.



Table 1: Groupings and subgroupings of diagnoses for children aged 5-12 admitted to Vaiola Hospital in Tonga 2009-2013.

Respiratory conditions	Injury and poisoning	Abdominal and surgical conditions	Non-respiratory infectious diseases	Chronic conditions	Other conditions	Dental conditions	Cardiac conditions	Neoplasms
Pneumonia	Injury/poisoning	Appendicitis	Skin infection	Disability/	Pain			
Asthma	Open wound	Gastroenteritis	Parasitic infection	chronic	Neurology			
Throat pain/	(unknown cause)	Other abdominal	Fever of unknown	disease	Haematology			
infection		and pelvic	origin	Allergy	Sexual abuse			
Ear infection		conditions	Other infection/		Unspecified			
Other acute		Liver or renal	infectious disease					
upper		disease						
respiratory		General or						
Other		orthopaedic surgery						
respiratory								

The hospital uses a data system called the Tonga Hospital Information System, developed by iSOFT in 2009. The system has admission, discharge and transfer data capabilities together with a disease classification component. Hospital admission and discharge data is collected and coded using ICD10 and ICD10AM disease classification. For each admission, data were extracted that described child gender and age, diagnosis, facility admitted to and length of hospital stay.

Inclusion and exclusion criteria

All inpatient admissions to Vaiola Hospital for children aged 5–11 (inclusive) years from January 2009 to December 2013 were included in this study. We included all admissions and thus potentially included multiple admissions for the same child. Emergency department-only events were excluded.

Data analysis

Discharge diagnosis data were grouped into 27 diagnostic categories based on the most frequent reasons for hospital admission in children aged 0-14 years (neonates excluded) in New Zealand from 2006 to 2010.47 These 27 diagnostic categories were then organised into the following diagnostic groups: respiratory conditions; injury and poisoning; abdominal and surgical conditions; non-respiratory infectious diseases; chronic conditions; other conditions; dental conditions; cardiac conditions; and neoplasms (Table 1). Note that gastroenteritis is included in abdominal and surgical conditions rather than in non-respiratory infectious diseases. For a further breakdown of diagnoses and groupings, see Appendix 1. The data did not include external cause codes, so for the injury and poisoning category it was not possible to

differentiate between intentional and unintentional injury. For this study, open wounds were included in the injury and poisoning group as it was assumed that otherwise the ICD10 code for skin infection would have been used.

Seasons were separated into 'wet and warm' (November to April) and 'dry and cool' (May to October). Admissions were grouped into two age groups: 5–7 and 8–11 years. In Tonga, children attend primary school from age 5 to 11 years (inclusive). To allow for a comparison of younger with older primary school aged children we then divided the sample into two age groups: 5–7 years and 8–11 years.

Sample distribution was described using proportions and means with standard deviations (SD) or medians with interquartile ranges (IQR) depending upon the normality of data distribution. Proportions were compared using the chi square test and means and medians using the t-test and the Wilcoxon rank sum test respectively.

For rate calculations, the population and projected population of 5-11 year-olds as defined at the 2006 and 2011 national census were used as the denominator. Of note, the national census in 2011 had a slightly lower actual count than the projected population growth from 2006. There was no explanation for this from the 2011 census report. Hospital admission rates by season, age, gender, diagnostic group and admission facility were described. Rates were compared using rate ratios (RRs) and 95% confidence intervals (CI). Statistical analysis was completed using SAS version 9.3 (SAS Institute, Cary, NC, US) and Stats-Direct version 2.7.9 (Altrincham, Cheshire, UK) software.



Table 2: Hospital admission rates for children aged 5–11 years (inclusive) admitted to hospital in Tonga from 2009 to 2013 by season, age and gender.

Variable	n (%, 95% confidence interval)	Population [†]	Rate per 1,000 (95% CI)	Rate ratio (95% CI)				
Year of admission								
2009	305 (17, 15–19)	18,036	16.9 (15.1–18.9)	1.00				
2010	328 (18, 16–20)	18,125	18.1 (16.2–20.1)	1.07 (0.91–1.25)				
2011	346 (19, 17–21)	17,813	19.4 (17.4–21.6)	1.15 (0.98–1.34)				
2012	415 (23, 21–25)	17,915	23.2 (21.0–25.4)	1.37 (1.18–1.59)				
2013	422 (23, 21–25)	17,990	23.5 (21.3–25.8)	1.39 (1.19–1.61)				
Season								
Wet and warm (November to April)	924 (51, 49–54)	89,879	10.3 (9.6–11.0)	1.00				
Dry and cool (May to October)	892 (49, 47–51)	89,879	9.9 (9.3–10.6)	0.96 (0.88-0.97)				
Age in years								
5	375 (21, 19–23)	13,178	28.5 (25.7–31.4)	1.00				
6	308 (17, 15–19)	13,125	23.5 (20.9–26.2)	0.82 (0.71–0.96)				
7	226 (12, 11–14)	13,030	17.3 (15.2–19.7)	0.61 (0.51-0.72)				
8	218 (12, 11–14)	12,899	16.9 (14.7–19.3)	0.59 (0.50-0.70)				
9	227 (13, 11–14)	12,736	17.8 (15.6–20.2)	0.63 (0.53-0.74)				
10	228 (13, 11–14)	12,549	18.2 (15.9–20.7)	0.64 (0.54–0.75)				
11	234 (13, 11–14)	12,362	18.9 (16.6–21.5)	0.67 (0.56-0.79)				
Age group in years								
5–7	909 (50, 48–52)	39,333	23.1 (21.6–24.6)	1.00				
8-11	907 (50, 48–52)	50,546	17.9 (16.8–19.1)	0.78 (0.71-0.85)				
Gender								
Female	681 (37, 35–40)	42,910	15.9 (14.7–17.1)	1.00				
Male	1,135 (63, 60–65)	46,969	24.1 (22.8–25.6)	1.52 (1.38–1.68)				

[†]Based upon 2006 and 2011 census.

Results

Over the study interval there were 1,816 admissions of children aged 5–11 years to Vaiola Hospital, giving an average annual admission rate of 20.2/1,000 (95% CI 19.3–21.1). In comparison with 2009, the admission rate was greater in 2012 (RR=1.37) and 2013 (RR=1.39). Admission rates were higher in younger than older children (5–7 versus 8–11 years, RR=1.28) and in boys compared with girls (RR=1.52), and varied

by season (dry and cool vs wet and warm, RR=0.96) (Table 2).

Of the 1,816 admissions, 1,474 (85%) were due to diagnoses that placed them in one of five diagnostic groups: injury and poisoning (28%), non-respiratory infectious diseases (19%), respiratory conditions (16%), abdominal, gastrointestinal and surgical conditions (13%) and dental conditions (9%). The median length of stay was three days (IQR 2–6) (Table 3).



Table 3: Diagnostic groups and length of stay for children aged 5–11 years admitted to hospital in Tonga from 2009–2013.

Diagnostic group	n (% of all admissions, 95% CI†)	Rate per 1,000 [‡] (95% CI)
Injury and poisoning	485 (28, 27–30)	5.6 (5.2-6.2)
Injury/poisoning	377 (22)	4.4 (4.0-4.9)
Open wound (unknown cause)	108 (6)	1.3 (1.0-1.5)
Non-respiratory infectious diseases	328 (19, 17-21)	3.8 (3.4-4.3)
Other infection/infectious disease	209 (12)	2.4 (2.1–2.8)
Skin infection	97 (6)	1.1 (0.9–1.4)
Fever of unknown origin	18 (1)	0.2 (0.1–0.3)
Parasitic infection	4 (0)	0.05 (0.01-0.1)
Respiratory conditions	282 (16, 14–18)	3.3 (2.9–3.7)
Pneumonia	126 (7)	1.5 (1.2–1.7)
Asthma	56 (3)	0.7 (0.5–0.8)
Other respiratory	47 (3)	0.5 (0.4–0.7)
Throat pain/infection	25 (1)	0.3 (0.2–0.4)
Ear infection	22 (1)	0.3 (0.2-0.4)
Other acute upper respiratory	6 (0)	0.07 (0.02-0.1)
Abdominal and surgical conditions	228 (13, 11–15)	2.7 (2.3-3.0)
General or orthopaedic surgery	98 (6)	1.1 (0.9–1.3)
Other abdominal and pelvic conditions	61 (3)	0.7 (0.5–0.9)
Appendicitis	43 (2)	0.5 (0.2–0.3)
Gastroenteritis	23 (1)	0.3 (0.1–0.4)
Liver or renal disease	3 (0)	0.03 (0.007–0.09)
Dental conditions	151 (9, 7–10)	1.7 (1.4-2.0)
Other conditions	142 (8, 7-9)	1.6 (1.3-1.9)
Unspecified	60 (3)	0.7 (0.5–0.9)
Neurology	43 (2)	0.5 (0.3–0.6)
Pain	26 (1)	0.3 (0.2-0.4)
Haematology	12 (1)	0.1 (0.07-0.2)
Sexual abuse	1 (0)	0.01 (0.003-0.06)
Neoplasms	51 (3, 2-4)	0.7 (0.5-0.9)
Cardiac conditions	49 (3, 2-4)	0.5 (0.4–0.7)
Chronic conditions	33 (2, 1-3)	0.3 (0.2-0.5)
Disability/chronic disease	26 (1)	0.3 (0.2–0.4)
Allergy	7 (0)	0.07 (0.03–0.2)
	median (IQR)	
Length of stay in days	3 (2-6)	

 $^{^{\}dagger}\text{CI}$ confidence interval.

[‡]Assuming a total population of 85,915 based on 2011 Census data.



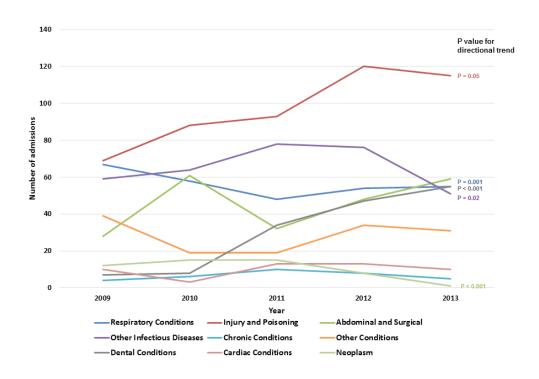


Figure 1: Admissions to Vaiola Hospital from 2009–2013 for children aged 5–11 years.

Between 2009 and 2013, year-to-year variability was evident in the frequency of several of the diagnostic groups: respiratory conditions (P=0.005); abdominal and surgical conditions (P<0.001); other infectious diseases (P=0.02); other conditions (P=0.004); dental conditions (P<0.001) and neoplasms (P=0.001). Directional trends were present for respiratory conditions (P=0.001), injury and poisoning (P=0.047), other infectious disease (P=0.02), dental conditions (P<0.001) and neoplasms (P<0.001) (Figure 1). The proportion of hospital admissions per year due to respiratory conditions (P=0.001), other infectious diseases (P=0.02) and neoplasms decreased (P<0.001). The proportion of hospital admissions per year for injury and poisoning (P=0.047), and dental conditions increased (P<0.001).

The number of admissions by diagnostic group varied by age (Figure 2). A larger proportion of younger (5–7 years) versus older (8–12 years) children were admitted for treatment of dental (16% vs 1%, P<0.001) or respiratory conditions (18% vs 14%, P=0.02). A larger proportion of older children were admitted for abdominal and surgical conditions (15% vs 11%, P=0.008), other

infectious diseases (21% vs 17%, P=0.04), other conditions (10% vs 6%, P<0.001) and cardiac conditions (2% vs 1%, P<0.001).

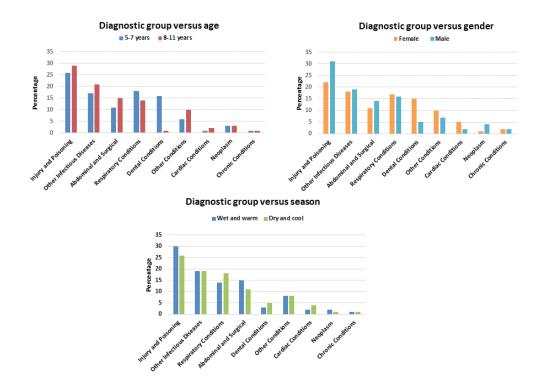
The number of admissions by diagnostic group varied with gender (Figure 2). A larger proportion of boys than girls had a hospital admission for injury or poisoning (31% vs 22%, P<0.001) or neoplasm (4% vs 1%, P<0.001). A larger proportion of girls than boys had a hospital admission for a dental (15% vs 5%, P<0.001) or a cardiac condition (5% vs 2%, P<0.001).

Admissions by diagnostic group varied by season (Figure 2). A larger proportion of the hospital admissions in the dry/cool season were for respiratory (18% vs 14%, P=0.013) or dental conditions (5% vs 3%, P<0.001). A larger proportion of the hospital admissions in the wet/warm season were due to injury and poisoning (30% vs 26%, P=0.04) or for abdominal and surgical conditions (15% vs 11%, P=0.006).

There were six in-hospital deaths over the five-year study period in the age group 5–11 (inclusive) years, three due to neoplasms and one each due to encephalopathy, sepsis and hepatic failure.



Figure 2: Admissions to Vaiola Hospital from 2009–2013 for children by diagnostic group versus age, gender and season of admission.



Discussion

Our study of hospital admissions from 2009–2013 of children in Tonga aged 5–11 years showed an average annual hospitalisation rate of 20.2/1,000, with injury and poisoning, infectious diseases, respiratory conditions, abdominal, gastrointestinal and surgical conditions and dental conditions accounting for 85% of hospital admissions. Hospitalisation rates decreased with increasing age. A larger proportion of hospital admissions among younger children (5–7 years old) were for dental or respiratory conditions and a larger proportion of hospital admissions among older children (8-11 years old) were for abdominal and surgical conditions, other infectious diseases and cardiac conditions.

This is the first study that has quantified population disease burden in this age group in the Pacific. The primary school age group is important because it is an age when diseases such as asthma, rheumatic heart disease and obesity, which cause long-term morbidity, can first manifest clinically. This age group is also one for whom health care is facilitated by the potential ease of access created by school-based delivery of healthcare.

Our intention in this first study was simply to describe the prevalent causes of disease burden and how these varied by demographic characteristics. As such, our study has a number of limitations. As we restricted our analysis to hospital admission data we were unable to describe health status or to consider the many important health issues that are infrequent causes of hospital admission. Thus our study provides no information, for example, about nutritional status, vision and hearing; nor any understanding of emotional, psychological, social, cognitive or behavioural wellbeing. Nor do our data allow for any consideration of the status of the child's environment, including family, culture, economics, school and home environment. It does however provide baseline information and point of focus to inform future investigations and policy development.

It is important to acknowledge that due to external cause coding not being available, it was not possible to identify if an injury was intentional or unintentional. A number of child advocates in Tonga have highlighted the need for more work to be done on preventing child maltreatment, as well as improving the processes in place for managing and recording it. A recent



study on violence against women in Tonga placed the nation among the highest in the world for levels of physical violence against women by non-partners. ^{48,49} Another study comparing three Pacific Island countries reported that Tongan children expressed the highest levels of violence against them, including in the criminal justice system, schools and home. ⁵⁰

In low-resource countries such as Tonga, many children do not access hospital care regardless of the severity of their disease. This is certainly the case for acute lower respiratory infection in developing countries where most deaths take place outside hospitals.51 Hospital admission data is consequently likely to underestimate the burden of disease. In global burden of disease studies, hospital discharge data is only one of nine different data sources used to show burden of disease and years lived with disability. These studies also use systematic reviews for disease sequelae, reports to governments, population-based disease registries, antenatal clinics, outpatient data, household surveys, re-analysis of cohort studies and indirect prevalence studies.52 The hospital data presented therefore needs to be interpreted with some caution, however this should not be a deterrent to presenting this part of the picture, particularly in countries where there is such a lack of baseline information.

The Tongan Hospital Information System enabled us to use electronic data to describe relationships between demographics and hospital utilisation. The use of ICD discharge codes and the creation of a smaller number of diagnostic groupings enabled comparisons over time and between population subgroups in this study, which can also be used with subsequent comparisons to different time intervals and other Pacific nations.

The temporal trends over the relatively short time interval of this study need to be interpreted with caution. The reduction in proportion of hospital admissions per year due to respiratory conditions and infectious diseases could be due to improved management of these conditions in the community and/or better preventative measures to combat such diseases. The increase in accident and injury admissions potentially indicates an area where more focused preventive strategies may be

needed. The dramatic increase in dental admissions between 2010 and 2011 may be contributed by changes in documentation methods within the dental department. The larger number of younger children admitted to the dental department does indicate though the fragility of the primary dentition in this age group. The finding that younger children were at increased risk of admission with respiratory issues and that boys were at increased risk of admission with injury and poisoning is consistent with the global literature. 53,54

Our findings appear consistent with the small number of reports from other countries of the prevalent health issues in this age group. Lozano et al10 studied age-specific mortality across 21 global regions (Tonga and other Pacific countries were included as Oceania) between 1980 and 2010 and found that in the 5-14 year old age group, infectious diseases, HIV/tuberculosis, injuries and some cancers prevailed, with mortality rates low in this age group. In a study investigating health expenditure due to multiple chronic diseases in the 0-17 year age group in the US, Zhong et al55 found the most prevalent chronic conditions were asthma/ chronic obstructive pulmonary disease, allergic rhinitis and behavioural problems. In Australia, a study of children's wellbeing in their middle years (8-14 years) found a significant proportion of children had low wellbeing, and particularly those considered to be marginalised.4 This marginalised group included those defined as "culturally and linguistically diverse", which would include migrants from Pacific Islands.

The data presented here are the first to describe child health in the primary school age group in Tonga. Our findings complement data reported in 2010 about 12-15 year-old children from 24 high schools in Tonga, which participated in a global school-based health survey. In this older age group, 59% were overweight and 21% obese. Among students that had ever used drugs, approximately two-thirds (68%) were using drugs before the age of 14 years. More than one-third (36%) of students had attempted suicide one or more times in the past 12 months. Bullying was a concern for 66% of students. Only 27% reported parents and guardians understanding their problems. Violence was significant with 49% reporting



physical fights, 51% being attacked and 63% seriously injured in the past 12 months.⁵⁶ These prevalent concerning health issues highlight the need to determine earlier childhood predictors of adolescent health.⁵⁷

This description of disease in primary school aged children requiring inpatient hospital care makes an important contribution to child health research in Tonga and the rest of the Pacific. These data and analyses provide a contemporary description that can be used to inform priority areas for healthcare spending and to enable comparisons over time and between different countries in the Pacific region. From these data the key areas to target for health prevention strategies are injury and poisoning, infectious diseases, respiratory conditions, abdominal and surgical and dental conditions. While this study cannot determine the best way to target these particular issues, it does establish a baseline description of disease burden against which the effectiveness of interventions can be measured.

This report of morbidity and mortality data represents the first phase of a more comprehensive description of the health status of children in Tonga. Our findings have helped to inform other projects in progress, which will allow a broader and more detailed description of the contemporary health status and challenges to health faced by children growing up in Tonga.

The global state of primary school children's health in resource-limited countries and the conditions which affect this age group remain relatively undefined. Although under five-year mortality rates do show the condition of overall society due to the vulnerability of this age group, they do not encompass the whole of child wellbeing. The current lack of global focus on the Pacific region and on the primary school age group in particular provides a challenge worth rectifying.



Appendix 1: The nine diagnostic groups and the diagnostic categories within each of these groups.

Respiratory conditions					_
Asthma Asthma unspecified Status asthmaticus	Bacterial/viral/other pneumonia Lobar pneumonia Pneumonia unspecified Bronchiectasis Influenza with pneumonia	Acute URTI Acute obstructive laryngitis	Throat pain/ infection Acute tonsillitis Chronic tonsillitis Hypertrophy of tonsils	Ear infection Chronic mucoid otitis media Acute suppurative otitis media Impacted cerumen Polyp of middle ear Periauricular sinus and cyst Disease of inner ear unspecified	Other respiratory Other disorders of lung Bronchopneumonia Pleural effusion Acute lower respiratory infection Obstructive sleep apnoe
Injury and poisoning					,
Ingestion of noxious substance Ciguatera fish poisoning Concussion Dislocation Contusion Abrasion Crush injury Fracture Foreign body Abrasion Burn Unspecified injury Traumatic amputation Sprain Open wound Traumatic pneumothorax Non-fatal drowning Muscle/tendon injury	Open wound Open wound in different parts of the	anatomy			
Abdominal or surgical conditions					
General surgery/orthopaedic surgery Ectopic testis Hypospadias Cleft palate Inguinal hernia Meckel's diverticulum Circumcision Intestinal adhesions Undescended testicle Removal of fixation plate Slipped upper femoral epiphysis Juvenile osteochondrosis	Abdominal/pelvic pain/ constipation Constipation Unspecified abdominal pain	Gastroenteritis Diarrhoea and gastroenteritis Acute amoebic dysentery Nausea and vomiting	Liver disease Abscess of liver Acute and subacute hepatic failure	Appendicitis Acute appendicitis Unspecified appendicitis Other appendicitis	
Non-respiratory infectious diseases		,			
Skin infection Abscess Furuncle and carbuncle Local infection skin Cellulitis Granulomatous Impetigo	Parasitic infection Angiostrongyliasis Balantidiasis Brucellosis	Fever of unknown origin Febrile convulsions Persistent fever Fever unspecified	Other infection/infection lines of Viral infection unspection unspection unspective Cystitis Meningococcal menioste Osteomyelitis Acute disseminated Infective myositis Pyogenic arthritis Bacterial meningitis Shigellosis Typhoid fever Urinary tract infection Meningococcemia Sepsis unspecified Keratitis Zoster Nonspecific lymphana Acute periodontitis Pneumococcal menioviral encephalitis Orchitis epididymitis Agranulocytosis Mastoiditis Sialoadenitis Meningitis unspecific Acute lymphadenitis Tubulo-interstitial in Varicella	ecified ingitis encephalitis on denitis ingitis s	



ARTICLE

Appendix 1: The nine diagnostic groups and the diagnostic categories within each of these groups (continued).

Chronic disease						
	All	I	T			
Disability/chronic disease Systemic lupus erythematosus Mental disorder Congenital deformity Nephritic syndrome Deformity Cerebral palsy Polyarthritis Schizophrenia Juvenile rheumatoid arthritis Communicating hydrocephalus Osteoporosis	Allergy Allergic purpura Anaphylactic shock					
Other conditions						
Haematology Sideropenic dysphagia Epistaxis Aplastic anaemia Idiopathic thrombocytopenic purpura Coagulation defect	Unspecified Syncope and collapse Inflammatory condition of the jaw Disorder of male genital organs Hypoglycaemia unspecified Retention of urine Malaise and fatigue Other shoulder lesions Orthostatic hypotension Hydrocele unspecified	Sexual abuse	Pain Acute pain Headache Chronic regional pain syndrome Cramp and spasm Low back pain Myalgia Unspecified pain Cervicalgia	Neurology Nerve root and plexus disorder Epilepsy Unspecified convulsions Anoxic brain damage Autonomic nervous system disorde	r	
Cardiac conditions						
Cardiac Rheumatic mitral insufficiency Scarlett fever Congenital malformation of heart Supraventricular tachycardia Patent ductus arteriosus Acute rheumatic heart disease Rheumatic fever Palpitations Rheumatic chorea Unspecified atrial septal defect Obstructive hypertrophic cardiomyopat Aortic valve insufficiency Rheumatic mitral insufficiency Rheumatic heart disease unspecified	thy					
Teeth						
Teeth Dental caries unspecified Hereditary disturbance of tooth structu Disorder of teeth and support structure Developmental odontogenic cysts						
Neoplasm						
Neoplasm Hodgkin disease unspecified Malignant neoplasm Acute myeloid leukaemia Acute lymphoblastic leukaemia						



Competing interests:

Mrs Langridge reports grants from University of Auckland during the conduct of the study; grants from New Zealand Optometric Vision Research Foundation, grants from The Oticon Foundation, non-financial support from The Ranchhod Foundation, outside the submitted work. MO is part of the supervision team for this PhD candidate (main author).

Acknowledgements:

The authors would like to thank the Ministry of Health in Tonga. We acknowledge NZAID, The Oticon Foundation, NZOVRF and the Ranchhod Foundation for funding support. FL is supported by The University of Auckland Doctoral Scholarship. The authors have no competing interests to declare.

Author information:

Fiona Catherine Langridge, PhD Candidate, Departments of Pacific Health, Paediatrics: Child and Youth Health, The University of Auckland, Auckland; Sione Vaioleti Hufanga, Biostatistician, Biostatistics Department, Chief Information Officer, Ministry of Health, Tonga; Malakai Mahunui 'Ofanoa, Senior Lecturer, Departments of Pacific Health; Toakase Fakakovikaetau, Paediatrician, Paediatrics Department, Vaiola Hospital, Nuku'alofa, Tonga; Teuila Mary Percival, Paediatrician and Senior Lecturer, Departments of Pacific Health; Cameron Charles Grant, Paediatrician and Professor, Paediatrics: Child and Youth Health, The University of Auckland, Auckland, Starship Children's Hospital, Auckland.

Corresponding author:

Fiona Langridge, Department of Pacific Health, Faculty of Medicine and Health Sciences, University of Auckland, Private Bag 92019, Auckland.

f.langridge@auckland.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7407

REFERENCES:

- 1. Duke T, Kado JH, Auto J, et al. Closing the gaps in child health in the Pacific: An achievable goal in the next 20 years. J Paediatr Child Health. 2015; 51:54–60.
- 2. Denno D. Global child health. Pediatr Rev. 2011; 32:e25–e38.
- 3. United Nations. The sustainable development goal report 2016; New York: United Nations 2016. Available from: http://unstats. un.org/sdgs/report/2016/ The%20Sustainable%20 Development%20Goals%20 Report%202016.pdf accessed 10 August 2017.
- Redmond G, Skattebol
 J, Saunders P, et al. Are
 the kids alright? Young
 Australians in their middle
 years: Final report of the
 Australian Child Wellbeing
 Project, Flinders University,
- University of New South Wales and Australian Council for Educational Research. Flinders University, University of New South Wales and Australian Council for Educational Research, 2016. Available from: http://australianchildwellbeing.com.au/sites/default/files/uploads/ACWP_Final_Report_2016_Full.pdf accessed 10 August 2017.
- 5. Bryce J, Terreri N, Victora CG, et al. Countdown to 2015: tracking intervention coverage for child survival. Lancet. 2006; 368:1067–1076.
- Karadag B, Karakoc F, Ersu R, et al. Non-cystic-fibrosis bronchiectasis in children: a persisting problem in developing countries. Respiration. 2005; 72:233–238.

- 7. Prescott SL, Pawankar R, Allen KJ, et al. A global survey of changing patterns of food allergy burden in children. World Allergy Organ J. 2013; 6:21.
- 8. Thapar N, Sanderson IR. Diarrhoea in children: an interface between developing and developed countries. Lancet. 2004; 363:641–653.
- Wald A, Sigurdsson L. Quality of life in children and adults with constipation.
 Best Pract Res Clin Gastroenterol, 2011; 25:19–27.
- 10. Lozano R, Naghavi M, Foreman K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2013; 380:2095–2128.



- 11. Jackson SL, Vann Jr WF, Kotch JB, et al. Impact of poor oral health on children's school attendance and performance. Am J Public Health. 2011; 101:1900–1906.
- 12. Petersen PE, Bourgeois D, Ogawa H, et al. The global burden of oral diseases and risks to oral health. Bull World Health Organ. 2005; 83:661–669.
- Parker EJ, Jamieson LM, Broughton J, et al. The oral health of Indigenous children: A review of four nations. J Paediatr Child Health. 2010; 46:483–486.
- 14. Gore FM, Bloem PJ, Patton GC, et al. Global burden of disease in young people aged 10–24 years: a systematic analysis. Lancet. 2011; 377:2093–2102.
- 15. Asiegbu UV, Asiegbu OG,
 Onyire BN, et al. Assessment of gross malnutrition
 among primary school
 children using body mass
 index as an assessment
 tool in abakaliki metropolis
 of Ebonyi State, SouthEast Nigeria. Niger J Clin
 Pract. 2017; 20:693–699.
- 16. Ukaegbe OC, Umedum NG, Chime EN, Orji FT. Assessment of common otolaryngological diseases among children in rural primary schools in south eastern Nigeria. Int J Pediatr Otorhinolaryngol. 2016; 89:169–172.
- 17. Gharaibeh NS. Effects of indoor air pollution on lung function of primary school children in Jordan. Ann Trop Paediatr. 1996; 16:97–102.
- 18. Odinaka KK, Nwolisa EC, Mbanefo F, et al. Prevalence and Pattern of Soil-Transmitted Helminthic Infection among Primary School Children in a Rural Community in Imo State, Nigeria. J Trop Med. 2015; 2015;349–439.

- 19. Kombich JJ, Muchai PC, Tukei P, Borus PK. Rubella seroprevalence among primary and pre- primary school pupils at Moi's Bridge location, Uasin Gishu District, Kenya. BMC Public Health 2009; 9:269.
- 20. Klepp KI, Ndeki SS, Seha AM, et al. AIDS education for primary school children in Tanzania: an evaluation study. AIDS. 1994; 8:1157–1162.
- 21. Gajanana A, Thenmozhi V, Samuel PP, Reuben R. A community-based study of subclinical flavivirus infections in children in an area of Tamil Nadu, India, where Japanese encephalitis is endemic. Bull World Health Organ. 1995; 73:237–244.
- 22. Ayanniyi AA, Mahmoud AO, Olatunji FO. Causes and prevalence of ocular morbidity among primary school children in Ilorin, Nigeria. Niger J Clin Pract. 2010; 13:248–253.
- 23. Sarvamangala K, Koujalgi MB, Manjunath TP.
 Prevalence of anemia, morbidity and school absenteeism among lower primary school children of Davangere city. Indian J Public Health Res Dev. 2014; 5:220–225.
- 24. Thapa KB, Okalidou A,
 Anastasiadou S. Teachers'
 screening estimations of
 speech-language impairments in primary school
 children in Nepal. Int J
 Lang Commun Disord.
 2016; 51:310–327.
- 25. Rabbani MG, Hossain MM. Behaviour disorders in urban primary school children in Dhaka, Bangladesh. Public Health. 1999; 113:233–236.
- 26. Narwaria YS, Saksena DN. Prevalence of dental fluorosis among primary school children in rural areas of Karera Block, Madhya Pradesh. Indian J Pediatr. 2013; 80:718–720.

- 27. Percival T, Langridge F, Stowers L. Keeping promises, measuring results: implications for maternal and child health in the pacific. Health information Services Knowledge Hub working paper series. Queensland: School of Population Health, University of Queensland, AusAID. 2013 (26). Available from: http://www.researchgate. net/profile/Teuila_Percival/ publication/237084244_ Keeping_promises_measuring_results_the_Pacific_Maternal_and_Child_ Health Indicators Project/ links/00b4953c84c336c6da000000/ Keeping-promises-measuring-results-the-Pacific-Maternal-and-Child-Health-Indicators-Project.pdf accessed 10 August 2017.
- 28. Gray S, Lennon D, Anderson P, et al. Nurse-led school-based clinics for skin infections and rheumatic fever prevention: results from a pilot study in South Auckland. N Z Med J. 2013 Apr 19; 126:53–61.
- 29. Lennon D, Stewart J,
 Farrell E, et al. Schoolbased prevention of acute
 rheumatic fever: a group
 randomized trial in New
 Zealand. Pediatr Infect
 Dis J. 2009; 28:787–794.
- 30. Anderson P, King J,
 Moss M, et al. Nurse-led
 school-based clinics for
 rheumatic fever prevention and skin infection
 management: evaluation
 of Mana Kidz programme
 in Counties Manukau. NZ
 Med J. 2016; 129:36–45.
- 31. New Zealand Ministry of Health. Tagata Pasifika in New Zealand. 2014; Available at: http://www.health.govt.nz/our-work/populations/pacific-health/tagata-pasifika-new-zealand accessed 02/10, 2017.
- **32.** Morton SM, Atatoa Carr PE, Grant CC, et al. Cohort profile: growing up in New



- Zealand. Int J Epidemiol. 2013; 42(1):65–75.
- 33. Tonga Department of
 Statistics. Census Statistics
 2011. 2016; Available at:
 http://tonga.prism.spc.
 int/#population-statistics-including-administrative-information-and-statistical-tabulation-of-the-2011
 accessed 5 November 2016.
- 34. World Health Organisation. Tonga: A Country Profile. 2011. Available from: http://www.wpro.who. int/countries/ton/33TONpro2011_finaldraft.pdf accessed 10 August 2017.
- 35. Russell FM, Fakakovi T,
 Paasi S, et al. Reduction
 of meningitis and impact
 on under-5 pneumonia
 after introducing the Hib
 vaccine in the Kingdom of
 Tonga. Ann Trop Paediatr. 2009; 29:111–117.
- 36. Danielsson N, Fakakovikaetau T, Szegedi E.
 Improved immunization practices reduce childhood hepatitis B infection in Tonga. Vaccine. 2009; 27:4462–4467.
- **37.** Lutui F, Grant CC, Best E, et al. Invasive Pneumococcal Disease in Children in Tonga. Pediatr Infect Dis J. 2017; 36:239–240.
- 38. Schultz J, Utter J, Mathews L, et al. The Pacific OPIC project (Obesity Prevention in Communities): action plans and interventions. Pac Health Dialog. 2007; 14:147–153.
- 39. Swinburn B, Pryor J,
 McCabe M, et al. The
 Pacific OPIC project
 (Obesity Prevention in
 Communities)-objectives
 and designs. Pac Health
 Dialog. 2007; 14:139–146.

- **40.** Fotu K, Moodie M, Mavoa H, et al. Process evaluation of a community-based adolescent obesity prevention project in Tonga. BMC Public Health. 2011; 11:284.
- 41. Finau SA, Taylor L.
 Rheumatic heart disease and school screening:
 Initiatives at an isolated hospital in Tonga. Med J
 Aust. 1988; 148:563–567.
- 42. Carapetis JR, Hardy M, Fakakovikaetau T, Taib R, et al. Evaluation of a screening protocol using auscultation and portable echocardiography to detect asymptomatic rheumatic heart disease in Tongan schoolchildren. Nat Clin Pract Cardiovasc Med. 2008; 5:411–417.
- 43. Poole-Wilson PA, Seth S. Rheumatic fever: The potential advantages of technology. Nat Clin Pract Cardiovasc Med. 2008; 5:426–427.
- 44. Foliaki S, Annesi-Maesano I, Daniel R, et al. Prevalence of symptoms of childhood asthma, allergic rhinoconjunctivitis and eczema in the Pacific: The International Study of Asthma and Allergies in Childhood (ISAAC). Allergy Eur J Allergy Clin Immunol. 2007; 62:259–264.
- 45. Foliaki S, Fakakovikaetau T, D'Souza W, et al. Reduction in asthma morbidity following a community-based asthma self-management programme in Tonga. Int J Tuberc Lung Dis. 2009; 13:142–147.
- **46.** Hoffman MP, Cutress TW, Tomiki S. Prevalence of developmental defects of enamel in children in

- the Kingdom of Tonga. N Z Dent J. 1988; 84:7–10.
- 47. Craig L, Adams J, Oben G, et al. The Health Status of Children and Young People in New Zealand. Dunedin: New Zealand Child and Youth Epidemiology Service, University of Otago; 2013. Available from: http://www.otago.ac.nz/nzcyes/otago086007.pdf accessed on 10 August 2017.
- 48. Jansen H, Johansson-Fua S, Hafoka-Blake B, 'Ilolahia GR. National Study on Domestic Violence against Women in Tonga Nofo 'a kainga. Kingdom of Tonga: Ma`a Fafine mo e Famili Inc. 2012. Available from: http://www.pacificwomen.org/wp-content/uploads/tongavaw-report-final-20121.pdf accessed 10 August 2017
- 49. McLean A. Corporal
 Punishment of Children
 in Tonga–A Violation of
 Constitutional Rights.
 Asia-Pacific Journal on
 Human Rights and the
 Law. 2014; 15:73–118.
- 50. Smith BJ, Phongsavan P,
 Bampton D, et al. Intentional injury reported
 by young people in
 the Federated States of
 Micronesia, Kingdom of
 Tonga and Vanuatu. BMC
 Public Health. 2008: 8:145.
- 51. Nair H, Simões EA, Rudan I, et al. Global and regional burden of hospital admissions for severe acute lower respiratory infections in young children in 2010: a systematic analysis. Lancet. 2013; 381:1380–1390.
- **52.** Vos T, Flaxman AD, Naghavi M, et al. Years lived with



- disability (YLDs) for 1,160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2013; 380:2163–2196.
- 53. Morrongiello BA, Rennie H. Why do boys engage in more risk taking than girls? The role of attributions, beliefs, and risk appraisals. J Pediatr Psychol. 1998; 23:33–43.
- **54.** Walker CLF, Rudan I, Liu L, et al. Global burden of childhood pneumonia and diarrhoea. Lancet. 2013; 381:1405–1416.
- 55. Zhong W, Finnie DM, Shah ND, et al. Effect of multiple chronic diseases on health care expenditures in childhood. J Prim Care Community Health. 2015 Jan;6:2–9.
- **56.** World Health Organisation. Tonga - 2010 Global Schoolbased Student Health
- Survey. Kingdom of Tonga: World Health Organisation. 2012. Available from: http:// www.who.int/chp/gshs/ GSHS_Tonga_2010_Report. pdf?ua=1 accessed 10 August 2017.
- 57. Kessler RC, Berglund P, Demler O, et al. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005; 62:593–602.



Metabolic monitoring in New Zealand district health board mental health services

Aimee Staveley, Ian Soosay, Anthony J O'Brien

ABSTRACT

AIM: To audit New Zealand district health boards' (DHBs) metabolic monitoring policies in relation to consumers prescribed second-generation antipsychotic medications using a best practice guideline.

METHODS: Metabolic monitoring policies from DHBs and one private clinic were analysed in relation to a best practice standard developed from the current literature and published guidelines relevant to metabolic syndrome.

RESULTS: Fourteen of New Zealand's 20 DHBs currently have metabolic monitoring policies for consumers prescribed antipsychotic medication. Two of those policies are consistent with the literature-based guideline. Eight policies include actions to be taken when consumers meet criteria for metabolic syndrome. Four DHBs have systems for measuring their rates of metabolic monitoring. There is no consensus on who is clinically responsible for metabolic monitoring.

CONCLUSIONS: Metabolic monitoring by mental health services in New Zealand reflects international experience that current levels of monitoring are low and policies are not always in place. Collaboration across the mental health and primary care sectors together with the adoption of a consensus guideline is needed to improve rates of monitoring and reduce current rates of physical health morbidities.

haracterised by central obesity, dyslipidaemia, hypertension and insulin resistance,¹ metabolic syndrome is a major public health issue that affects a diverse range of population groups.² Compared to the general population, metabolic syndrome increases an individual's risk of developing cardiovascular disease by two times within the next 5–10 years and increases the risk of developing type 2 diabetes mellitus five times.²

Rates of morbidity and premature death are elevated for most mental health disorders, including depression, bipolar disorder, addictions and personality disorder.³ People who have a severe mental illness experience worse physical health outcomes than the general population.⁴ In New Zealand, mental health consumers experience over twice the risk of premature death compared to the general

population and people with psychotic illnesses experience a three times higher risk of premature death.5 The life expectancy of a person with a severe mental illness is estimated to be 20 years less than the general population,6 and this mortality gap is reported to be increasing.7 Factors contributing to this disparity include a higher incidence of risk factors for chronic diseases, adverse effects of psychotropic medications, higher rates of unnatural death and reduced access to healthcare. 4,8 In mental health consumers experiencing severe mental illnesses, modifiable risk factors contributing to this difference in life expectancy include higher rates of obesity, smoking, alcohol and drug misuse, poor nutrition and lower rates of exercise. 9,10 These factors contribute to the increased prevalence of metabolic syndrome and consequently cardiovascular disease and type 2 diabetes mellitus. 11,12 The prevalence



of metabolic syndrome within this population group is also around two to three times higher than in the general population¹⁰ and even higher again in people experiencing schizophrenia.¹²

Second-generation antipsychotic agents are the mainstay medical treatment for people with serious mental illnesses such as schizophrenia and are also prescribed off label for other disorders in both children and adults.1 Second-generation antipsychotic agents may be preferred over first-generation antipsychotics due to the decreased risk of extrapyramidal side effects and reduced rates of relapse.1 Although the exact mechanisms are unclear, second-generation antipsychotics can contribute to metabolic complications and are associated with weight gain, increased triglyceride levels, decreased high-density-lipoprotein (HDL) levels and impaired glucose metabolism.^{1,6} It is important to note that independent of antipsychotic medications, severe mental illnesses such as schizophrenia are associated with other risk factors for the development of metabolic syndrome, such as obesity, smoking and lack of exercise.9,10

There is consensus across the literature that consumers prescribed antipsychotic agents should be closely monitored and screened for metabolic syndrome. However, the best model of monitoring and frequency with which it should be taking place is still unclear. In addition, there is a lack of worldwide consensus for the diagnostic criteria of metabolic syndrome, resulting in differences in estimates of prevalence and difficulties in comparing data across populations.2 Internationally, metabolic monitoring practices for mental health consumers prescribed antipsychotic medications have been found to be inadequate. 13,14,15 A recent Australian study examining the attitudes of prescribing psychiatrists found that although 80% of psychiatrists surveyed felt that metabolic monitoring was their responsibility, the rates of metabolic screening were below 50%.16 The New Zealand Metabolic Working Group Initiative published guidelines in 2008 for monitoring for metabolic syndrome in mental health service users, especially those taking second-generation antipsychotic agents. 17 The 2008 guideline did not recommend who should be clinically responsible for metabolic monitoring or how rates of monitoring should

recorded, although it did suggest that this responsibility belongs with the prescriber. There have been no further guidelines released for the New Zealand population that align with the most recent evidence and therefore New Zealand has no national standards or guidance for metabolic screening within mental health and addiction services. A recently published guideline for the treatment of schizophrenia¹⁸ provides general recommendations for physical health monitoring but not a specific recommendation about metabolic syndrome. To date there has been no published studies considering the guidelines used and the rates of metabolic screening within New Zealand mental health services.

Considering the worsening physical health outcomes for mental health consumers,7 we sought to investigate policies for monitoring metabolic parameters for people with serious mental disorders treated with second-generation antipsychotic medications throughout New Zealand DHBs.

Methods

Metabolic screening policies and guidelines related to health consumers on second-generation antipsychotics were requested from each of the 20 DHBs. Emails were sent with requests for the policies and available information about the rates of monitoring. Policies were analysed using as an audit tool, a best practice standard developed following a review of the literature. The definition of metabolic syndrome agreed by the researchers was the 2009 'harmonised' definition developed by international cardiac and metabolic health organisations,2 and is the most recent available. The harmonised definition includes five factors: waist circumference, blood pressure, fasting triglycerdes, fasting HDL cholesterol and fasting plasma glucose. Abnormalities in three of the five factors must be present to meet the criteria for metabolic syndrome. Cut-offs have been established for each factor, with the exception of waist circumference, which varies according to ethnicity. Criteria for the diagnosis of metabolic syndrome are shown in Table 1. In addition to the five components of metabolic syndrome, we included frequency of monitoring in the best practice guideline.



Table 1: Criteria for metabolic syndrome.[¥]

- 1. The health consumer must have at least three of the following risk factors:
- · Waist circumference with ethnicity-specific values
- European
 - Female ≥80cm, Male≥94 cm
- South Asians, Chinese, Japanese, Ethnic South and Central Americans
 - Female ≥80cm, Male ≥90cm
- No current data for other groups including Māori or Pacific Island people. These groups should use European values until more research is performed.
- 2. Systolic blood pressure of ≥130mmHg or diastolic blood pressure of ≥85mmHg
- 3. Fasting triglycerides ≥1.7mmol/L
- 4. Fasting HDL-cholesterol <1.03mmol/L for males and <1.29mmol/L for females
- Fasting plasma glucose ≥5.6mmol/L

*Source: Alberti KG, Eckel RH, Grundy SM, et al.3

Recommendations for frequency of monitoring were developed from the literature, considering both practicality and time available to DHB mental health and primary care staff. The frequencies were based on the recently released British Association for Psychopharmacology guidelines19 and an Australian guideline published by Waterreus and Laugharne.13 Additional features incorporated into the best practice guideline were a definition of metabolic syndrome, a statement of interventions for when metabolic syndrome was detected, a statement about collaboration with primary care, identification of the clinician/s responsible for monitoring and a process for auditing the DHB's rate of monitoring. Policies were read independently by two researchers and discussed to reach consensus about their content.

Results

We received a 100% response rate from the DHBs. Fourteen (70%) of DHB mental health services have a metabolic monitoring policy. Additionally, the policy from Ashburn Clinic (a private psychiatric facility) was also included in the study. Many of the policies were embedded within broader physical health polices which were not specific to metabolic syndrome. There was considerable variability in the size (number of pages) of policies, and their clarity when being implemented by clinicians. There was also considerable variability in the extent of information about metabolic syndrome contained in the policies.

Metabolic syndrome

Five of the 14 policies reviewed included monitoring for each of the five components of metabolic syndrome. Of these five policies, two included a complete set of cut-off values. In most cases where cut-off values were given, they were at variance with those of the harmonised standard. For each of the components of metabolic syndrome, policies had a varied range of recommended frequency of measurement. All 14 policies included measuring waist circumference with two policies using the parameters recommended by the International Diabetes Federation. Seven of the policies included cut-off values for waist circumference although frequency of measurement was different in each policy. All policies included measurement of blood pressure with six including cut-off values. All but one DHB included frequency of blood pressure monitoring. Thirteen of the 14 policies included the measurement of fasting plasma glucose with six policies including parameters for blood glucose levels. Thirteen recommended the measurement of triglycerides with eight specifying parameters. Finally, nine of the 14 policies included HDL-C levels with six specifying cut-off values. Six DHBs included LDL/HDL ratio, as recommended in the 2008 New Zealand guideline but not included in the 2009 harmonised guideline. One DHB policy included the use of a cardiovascular disease software tool PREDICT to measure risk.

Three policies included a definition of metabolic syndrome. Two policies defined



metabolic syndrome according to the International Diabetes Federation criteria and one DHB defined metabolic syndrome per the World Health Organization criteria. One policy included a system for the diagnosis of metabolic syndrome (three or more of impaired glucose metabolism, increased HDL-C, increased blood pressure or increased waist measurement). No policy specified what to do if metabolic syndrome was detected, although several policies contained recommendations relating to specific parameters.

Interventions

Eight of the 15 policies included actions that should take place if abnormal results are shown. Of these, five referred to liaison with or referral to a general practitioner. Three policies mentioned considering the use of metformin to moderate the effects of antipsychotic-induced weight gain. All the polices mentioning interventions discussed education surrounding reducing risk factors by promoting healthy lifestyles, although none mentioned specific health promotion interventions. Examples included healthy eating and physical exercise. One DHB also stated that referral to an occupational therapist, dietitian or other community service as an intervention for someone whose cardiovascular disease risk was high.

Collaboration with primary healthcare

Eleven of the 15 policies referred to collaboration with primary healthcare services. Six made reference to mental health workers ensuring that service users were actively encouraged and assisted to enrol with a primary healthcare provider. Many of the policies ensured that the GP was notified of commencement of antipsychotic medication, metabolic status, current interventions, other investigations and recommended follow-up by written or other means.

Clinician responsible for monitoring

The area of clinical responsibility for monitoring showed the greatest variability. Eleven policies made a statement about this aspect, with responsibility variously assigned to the prescriber, the psychiatrist, general practitioners, registered nurses, key workers and case managers. In one

case all DHB clinical staff were assigned responsibility for monitoring and in several cases there was shared responsibility. Four policies made no statement about who held clinical responsibility for monitoring.

Auditing procedure

Four of the 15 policies included reference to auditing with intervals of either six months or 12 months specified. Of the four policies with audit procedures, two specified an annual random sample of 10 cases with a detailed description of the audit process.

Discussion

This is the first study to analyse the New Zealand DHB policies on metabolic monitoring for consumers prescribed antipsychotic medication. Fourteen DHBs and Ashburn Clinic have metabolic monitoring policies. The proportion of DHBs having metabolic monitoring policies (70%) is slightly higher than the 55% figure reported in Australia,16 but against that it needs to be noted that only two of the policies were consistent with our literature-based guideline. There is limited consensus on what constitutes a comprehensive metabolic screening policy and thus the calibre of monitoring varies widely across DHBs. When measured against a best practice standard, few policies included all necessary components such as cut-off values, a definition of metabolic syndrome and actions to be taken with consumers meeting criteria for metabolic syndrome. Policies also show a large amount of variation in frequency of monitoring and aspects such as clinical responsibility and collaboration with primary care. No DHBs have auditing procedures that would allow them to determine what percentage of mental health consumers have their metabolic status assessed. These findings are consistent with international research showing low levels of monitoring and lack of consensus on responses to metabolic syndrome¹⁵ and on what to monitor, what cut-off values should be used and when monitoring should occur.8

We have proposed a best practice guideline (see Appendix) combining the 'harmonised' definition of metabolic syndrome developed by Alberti et al,² with recommendations for frequency of monitoring and other components based on our review of international literature.



Our proposed model does not cover every contingency. People taking antipsychotic agents for the first time or children and adolescents with psychotic disorders are at higher risk of metabolic complications and consequently these groups should be monitored more closely for changes in metabolic status.1 This study only looked at physical health measures in relation to metabolic syndrome; it did not consider the place of metabolic monitoring within the overall physical health of mental health consumers. We also did not consider potential for other adverse effects of clozapine, or problems associated with lithium. Our guideline does not take into account age, smoking status or personal or family health history and is not intended to substitute for cardiovascular risk assessment as recommended in the New Zealand Primary Care Handbook.²⁰ Rather than attempt to establish a model that would cover every variable, we aimed to produce a standard that is both practicable in terms of resources, and within the skillset of mental health and primary care nurses. In addition to recording metabolic parameters, our proposed model would incorporate evidence-based interventions aimed at preventing weight gain and improving cardiovascular health. Non-pharmacological interventions include nutrition and physical exercise education,^{21,22} and physical exercise alone.23 Metformin has also been suggested as a useful intervention²⁴ for reducing metabolic abnormalities.

A prominent theme in the literature is the lack of consensus about whether responsibility for metabolic monitoring of people with mental illness belongs with mental health services or with primary care.25 Internationally, models of primary care differ significantly in coverage, skill mix and payment models, and any New Zealand guidance must be tailored to our primary care system. Even within New Zealand, there are different models and degrees of integration of primary care and mental health services, and any implementation of the guideline will need to accommodate regional variation and local service models. However, the literature is clear that closer integration of primary care and mental health is needed.26 with a growing volume of research suggesting that more integrated approaches—with professionals working

more closely together—has the potential to improve outcomes and reduce costs.²⁷ In the case of metabolic syndrome, the best practice guideline should be seen as applying to consumers wherever they are receiving their healthcare and continuing in times of transition from one healthcare provider to another.

One response to the issue of metabolic syndrome is the development of the role of 'cardio metabolic nurses specialist', a role dedicated to metabolic monitoring and interventions to reduce cardiometabolic risk.²⁸ However, there is currently little evidence to demonstrate the effectiveness of this role and it carries the disadvantage that metabolic syndrome may be seen as a specialist area of clinical practice, rather than a component of the role of every clinician working with people taking antipsychotic medication. An English trial of a community nurse-led cardiovascular screening programme for health consumers with severe mental illnesses showed an increase in people being screened and proved superior to other interventions tested.²⁹ A recent Australian study reported increases in metabolic monitoring following the location of a general practitioner clinic attached to a mental health service.30

A recent systematic review of screening practices for people treated with antipsychotic medication concluded that despite availability of guidelines, rates of screening are low.26 There is clearly a need to translate guidelines into practice. Qualitative research experiences and expectations of physical healthcare show that service users expect clinicians to work proactively with them in this area, especially if their motivation appears low.³¹ We suggest that in addition to screening there needs to be an emphasis on evidence-based interventions for those at risk. The best practice guideline suggested in this study could be implemented in both mental health and primary care services, the major aim being that all consumers at risk will be monitored, with an identified clinician managing the monitoring process. Consumers moving between services would maintain the same metabolic monitoring record in secondary mental health and primary care. Responsibility for maintaining the record could move between primary care and mental health services.



Clinicians would ensure that the necessary tests are ordered, clinical assessments undertaken and interventions provided as indicated. In some cases, for example with consumers having routine cardiovascular assessment, metabolic monitoring might involve extracting laboratory results and information from clinical records rather than arranging additional appointments specifically for the purposes of metabolic monitoring. Maintaining a shared electronic record would help facilitate the sharing of information. Any such model will need to address attitudinal and skill barriers identified in the literature³² but would address the common finding of disagreement over who is responsible for monitoring.²⁵ The model would need to involve collaboration and liaison with all health professionals, including psychiatrists, general practitioners, pharmacists, nurses, key workers and support workers. In some regions the cost of access to primary care is a barrier and initiatives such as the funded visits provided by Tairawhiti DHB is one way of addressing this issue.33 (http://www.tepou. co.nz/news/improving-access-to-primarycare-in-tairawhiti/628)

Need for Māori and Pacific parameters

Although the Alberti et al 'harmonised' statement on metabolic syndrome includes waistline parameters for various ethnic groups, it does not provide waistline parameters for Māori and Pacific people. A New Zealand cross sectional study found the prevalence of metabolic syndrome for Māori was 32% and Pacific 39% compared to 16% for the general population. ³⁴ Similar differences in obesity were found in the most recent New Zealand Health Survey. ³⁵ Future development of our guideline needs to consider whether waistline parameters are needed that are specific to these groups.

National monitoring

During the process of data collection many DHB staff expressed interest in the outcome of the research as a means of providing guidance on metabolic monitoring. We received many requests for results and recommendations for the study to assist DHBs in reviewing their metabolic monitoring policies. Based on these requests, and after considering the many literature reports of low levels of monitoring, it is our recommendation that this guideline is considered as a basis for a nationally standardised monitoring programme which is subject to audit and reporting to the Ministry of Health. Implementation would need to be managed jointly between DHB mental health services and primary care services, with the aim of continuity of monitoring across points of service transition.

Conclusion

The physical health status of people with mental illness is an issue of critical significance that needs to be a priority area for the mental health and primary care sectors. Metabolic monitoring for consumers taking antipsychotic medication offers one strategy of identifying those at increased risk of adverse cardiovascular events and diabetes. Our best practice standard could be developed into a practical tool for DHBs and the primary care sector. An inter-sector model of monitoring could see the implementation of more systematic monitoring of metabolic status for people with mental illness. The five-part model of monitoring, together with recommended actions, and collaboration between the mental health and primary care sectors and a system of measuring and reporting on adherence to the model, has the potential to increase levels of monitoring and to improve the health status of people with mental illness. There is a need for further research on effectiveness of service delivery models.



Appendix

Appendix 1: Metabolic monitoring best practice standard.

	Frequency	Parameters	Interventions
Waist circumference	Baseline Monthly for first 3 months 6 months 9 months Annually	European: Female ≥80cm Male ≥95cm South Asians, Chinese, Japanese, South and Central Americans: Female ≥80cm Male ≥90cm No current norms for other groups. These groups should use the European values until more research becomes available.	Should be a recommendation or pathway to follow if an abnormality is detected.
Fasting plasma glucose	Baseline 3 months 6 months 9 months Annually	≥5.6mmol/L	Should be a recommendation or pathway to follow if an abnormality is detected.
Blood pressure	Baseline 3 months 6 months 9 months Annually	Systolic ≥130mmHg and/or Diastolic ≥85mmHg	Should be a recommendation or pathway to follow if an abnormality is detected.
Fasting lipids (triglyceride)	Baseline 3 months 6 months 9 months Annually	≥1.7mmol/L	Should be a recommendation or pathway to follow if an abnormality is detected.
Fasting lipids (HDL-cholesterol)	Baseline 3 months 6 months 9 months Annually	Female <1.29mmol/L Male <1.03mmol/L	Should be a recommendation or pathway to follow if an abnormality is detected.

Values outside the normal range for three or more of these parameters indicates metabolic syndrome. The person should receive evidence-based interventions aimed at restoring metabolic health. Consideration should be given to medication review. The health record should include a statement of the consumer's involvement with their primary care provider. This could include ensuring the mental health consumer is enrolled with a primary care provider or ensuring clear communication with the general

practitioner about the commencement of psychotropic medication, monitoring needed and expectations of the role of each health professional in caring for the health consumer. The policy should clearly state who is responsible for monitoring and the roles of each person involved should be clearly described. Lastly, the policy should include a way for DHBs to audit the monitoring process to determine the rate of adherence to the best practice standard for metabolic screening.



Competing interests:

Aimee Staveley reports grants from The University of Auckland Faculty of Medical and Health Sciences during the conduct of the study. Dr O'Brien reports summer scholarship from University of Auckland during the conduct of the study.

Acknowledgements:

This research was partly funded by a grant from the University of Auckland Faculty of Medical and Health Sciences. We are indebted to the service users and clinicians who advised on various aspects of the research, and to the DHB staff who provided metabolic monitoring policies.

Author information:

Aimee Staveley, Research Student, School of Nursing, University of Auckland, Auckland; Ian Soosay, Honorary Academic, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Anthony J O'Brien, Senior Lecturer, School of Nursing, University of Auckland, Auckland.

Corresponding author:

Anthony J O'Brien, Senior Lecturer, School of Nursing, University of Auckland, Private Bag 92019, Auckland.

a.obrien@auckland.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7408

REFERENCES:

- 1. De Hert M, Detraux J, Van Winkel R, et al. Metabolic and cardiovascular adverse effects associated with antipsychotic drugs. Nat Rev Endocrinol 2012; 8(2):114–26.
- Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association: World Heart Federation: International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation 2009; 120(16):1640-5.
- Harris EC, Barraclough B. Excess mortality of mental disorder. Br J Psychiatry. 1998; 173(1):11–53.
- 4. Thornicroft G. Physical health disparities and mental illness: the scandal of premature mortality. Br J Psychiatry 2011; 199(6):441–2.

- 5. Cunningham R, Sarfati D, Peterson D, et al. Premature mortality in adults using New Zealand psychiatric services. N Z Med J. 2014; 127(1394).
- Brown S, Kim M, Mitchell C, Inskip H. Twenty-five-year mortality of a community cohort with schizophrenia. Br J Psychiatry. 2010; 196(2):116–2.
- 7. Saha S, Chant D, McGrath J. A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time? Arch Gen Psychiatry 2007; 64(10):1123–31.
- 8. Cohn TA, Sernyak MJ.

 Metabolic monitoring
 for patients treated with
 antipsychotic medications. Can J Psychiatry.
 2006; 51(8):492–501
- Chesney E, Goodwin GM, Fazel S. Risks of all-cause and suicide mortality in mental disorders: a meta-review. World Psychiatry 2014; 13(2):153–160.
- **10.** Riordan HJ, Antonini P, Murphy MF. Second generation antipsy-

- chotics and metabolic syndrome in patients with schizophrenia: risk factors, monitoring, and healthcare implications. Am Health Drug Benefits 2014; 4(5):292.
- 11. De Hert M, Dekker J, Wood D, et al. Cardiovascular disease and diabetes in people with severe mental illness position statement from the European Psychiatric Association (EPA), supported by the European Association for the Study of Diabetes (EASD) and the European Society of Cardiology (ESC). Eur Psychiatry 2009; 24(6):412–24.
- 12. De Hert M, Correll CU, Bobes J, et al. Physical illness in patients with severe mental disorders. I. Prevalence, impact of medications and disparities in health care. World Psychiatry. 2011; 10(1):52–77.
- 13. Waterreus AJ, Laugharne J. Screening for the metabolic syndrome in patients receiving antipsychotic treatment: a proposed algorithm. Med J Aust. 2009; 190(4):185–9.



- 14. De Hert M, Vancampfort D, Correll CU, et al. Guidelines for screening and monitoring of cardiometabolic risk in schizophrenia: systematic evaluation. Br J Psychiatry. 2011; 199(2):99–105.
- 15. Barnes TR, Paton C, Cavanagh MR, et al. A UK audit of screening for the metabolic side effects of antipsychotics in community patients. Schizophr Bull. 2007; 33(6):1397–403.
- 16. Laugharne J, Waterreus
 AJ, Castle DJ, Dragovic
 M. Screening for the
 metabolic syndrome in
 Australia: a national survey
 of psychiatrists' attitudes
 and reported practice in
 patients prescribed antipsychotic drugs. Australas
 Psychiatry. 2016; 24(1):62–6.
- 17. New Zealand Mental
 Health Metabolic Working
 Group. Intervention guide
 for mental health service
 users on antipsychotic
 therapy. Auckland: Janssen
 Cilag NZ Ltd. 2008
- 18. Galletly C, Castle D, Dark F. Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the management of schizophrenia and related disorders. Aust NZ J Psychiatry 2016; 50(5):1–117.
- 19. Cooper SJ, Reynolds GP, Barnes TR, et al. BAP guidelines on the management of weight gain, metabolic disturbances and cardiovascular risk associated with psychosis and antipsychotic drug treatment. J Psychopharmacol 2016; 30(8):717–48.
- 20. Ministry of Health (2013). New Zealand Primary Care Handbook 2012 (updated 2013): Cardiovascular Disease Risk Assessment
- 21. Evans S, Newton R,
 Higgins S. Nutritional
 intervention to prevent
 weight gain in patients
 commenced on olanzapine:
 a randomized controlled
 trial. Aust NZ J Psychiatry
 2005; 39(6):479–486.

- 22. Yarborough BJH, Leo MC, Stumbo S, Perrin NA, Green CA. STRIDE: a randomized trial of a lifestyle intervention to promote weight loss among individuals taking antipsychotic medications. BMC Psychiatry 2013; 13(1):238.
- 23. Poulin MJ, Chaput JP,
 Simard V, Vincent P,
 Bernier J, Gauthier Y,
 et al. Management of
 antipsychotic-induced
 weight gain: prospective
 naturalistic study of the
 effectiveness of a supervised exercise programme.
 Aust NZ J Psychiatry
 2007; 41(12):980–989.
- 24. Ehret M, Goethe J, Lanosa M, Coleman CI. The effect of metformin on anthropometrics and insulin resistance in patients receiving second generation antipsychotic agents: a meta-analysis. J Clin Psychiat 2010; 71(10):1286–1292.
- 25. Ward T, Wynaden D, Heslop K. Who is responsible for metabolic screening for mental health clients taking antipsychotic medications.

 Int J Ment Health Nurs.
 2017 Epub ahead of print doi: 10.1111/inm.12309
- 26. Mitchell A, Delaffon V, Vancampfort D, et al. Guideline concordant monitoring of metabolic risk in people treated with antipsychotic medication: systematic review and meta-analysis of screening practices. Psychol Med. 2012; 42(01):125–47.
- 27. Naylor CP, McDaid M, Knapp D, Fossey M, Gallea A. 2012. Long term conditions and mental health: The cost of co-morbidities. London: The Kings Fund.
- 28. Happell B, Stanton R, Platania-Phung C, McKenna B, Scott D. The cardiometabolic health nurse: Physical health behaviour outcomes from a randomised controlled trial. Issues Ment Health Nurs 2014; 35(10):768–775.

- 29. Osborn DP, Nazareth
 I, Wright CA, King MB.
 Impact of a nurse-led
 intervention to improve
 screening for cardiovascular risk factors in
 people with severe mental
 illnesses. Phase-two cluster
 randomised feasibility
 trial of community mental
 health teams. BMC Health
 Serv Res. 2010; 10(1):61.
- 30. Coates D, Woodford P,
 Higgins O, Grover D.
 (2017). Evaluation of a
 general practitioner-led
 cardiometabolic clinic:
 Physical health profile
 and treatment outcomes
 for clients on clozapine.
 Int J Ment Health Nurs.
 2017 Epub ahead of print.
 doi: 10.1111/inm.12321
- 31. Young SJ, Praskova A,
 Hayward N, Patterson
 S. (2016). Attending to
 physical health in mental
 health services in Australia:
 a qualitative study of
 service users' experiences
 and expectations. Health
 Social Care Community
 2016; 25(2):605–611.
- 32. Robson D, Haddad M,
 Gray R, Gournay K. Mental
 health nursing and physical
 health care: A cross-sectional study of nurses' attitudes,
 practice, and perceived
 training needs for the
 physical health care of
 people with severe mental
 illness. Int J Ment Health
 Nurs 2013; 22(5):409–417.
- 33. Improving access to primary care in Tairawhiti. Retrieved 19 May 2017 from www. tepou.co.nz/news/improving-access-to-primary-care-in-tairawhiti/628
- 34. Gentles D, Metcalf P, Dyall L, Sundborn G, Schaaf D, Black P, et al. Metabolic syndrome prevalence in a multicultural population in Auckland, New Zealand. NZ Med J 2017; (Online) 120(1248).
- 35. Ministry of Health. Annual Update of Key Results 2015/16: New Zealand Health Survey; Wellington: Ministry of Health 2016.



Remembering the 1918 influenza pandemic: national survey of memorials and scope for enhancing educational value around pandemic preparedness

Nick Wilson, Catharine Ferguson, Geoffrey Rice, Michael G Baker, Ben Schrader, Christine Clement, George Thomson

ABSTRACT

AIM: To systematically identify physical memorials to the 1918 influenza pandemic in an entire country.

METHODS: Internet searches, contact with local historians and field expeditions were conducted.

RESULTS: Despite the high impact of the 1918 influenza pandemic in New Zealand (~8,600 deaths), only seven publicly accessible local memorials which referred this pandemic were identified. Another 11 memorials were identified, but these were in private settings or did not refer to the pandemic. There is no national memorial and a marked contrast exists with the number of war memorials (260 times more per 1,000 deaths for one war), and for 20 smaller mass fatality events (one of which has eight memorials alone). The current educational value of these pandemic memorials is likely to be minimal since only three are in cities, there is a lack of supporting signage and there are no links to online resources.

CONCLUSIONS: Despite the major impact of the 1918 influenza pandemic in New Zealand, publicly accessible memorials were found to be rare. This was in marked contrast to other disaster-related memorials and particularly to war memorials. There appears to be major scope for enhancing public education around the persisting threat of future pandemics via improved use of physical memorials and linkages to online resources.

Pandemics of influenza and other infectious diseases remain a serious global threat, requiring ongoing preparations by all countries. Such preparations include wide-ranging core capacities for surveillance and response. There is also increasing emphasis on preventing emergence of new microbial threats with approaches such as the Global Health Security Agenda. However, there might be decades between future pandemics, particularly those of the scale seen in the 1918 influenza pandemic. Consequently, this threat can fade from public and

official memories. Without clear reminders of the potentially massive effects of such pandemics, there may be little impetus to ensure preparatory efforts and proportionate resourcing.

Memorials might be one way to ensure that a society's collective recollection of the threat of influenza and other pandemics remains. Physical memorials can provide a locality for civic rituals (such as gatherings at anniversary events), and for visits by school groups and others. Memorials can also be used to enhance awareness around



social capital and civil responsibility—in this case by potentially providing examples and narratives of health workers and volunteers who worked to reduce the impact of the pandemic. Some memorials (such as the National War Memorial in New Zealand) even offer educational programmes that interpret the function and meanings of the memorial to visitors.

New Zealand provides a good case study for considering the issues around the memorialisation of pandemic influenza, as it is a relatively small country and there is fairly detailed national documentation of memorials on official websites. It is also a country where there might be at least some modest awareness of the impact of the 1918 influenza pandemic, owing to the major scale of the impact, especially for the Māori population as detailed in a book⁴ and on a popular official history website.⁵ As with most other countries, New Zealand has experienced three notable influenza pandemics since 1900; in 1918 (8,573 deaths),4 in 1957-59 (179 deaths in 19576 with even higher excess deaths in 19597) and in 2009 (49 deaths8). The 1968 pandemic reached New Zealand9 but we found no data on excess mortality estimates.

Methods

Searches to identify memorials

We aimed to identify memorials for multiple pandemic deaths or for individual health workers involved in responding to the pandemic. We conducted internet searches using Google and Google Images with search terms covering all of the following terms: "New Zealand", "influenza", "Spanish flu" and "memorial/monument/ obelisk/statue/plaque/cemetery". Searches were conducted in December 2015. Also examined were: a New Zealand online encyclopaedia (http://www.teara.govt.nz/en), an online history site (http://www.nzhistory.net.nz/) and an online list of national monuments.¹⁰

New Zealand has an online "Historic Heritage" list of historic sites, which includes selected memorials and cemeteries (http://www.heritage.org.nz/the-list). This database was also searched with the terms: "influenza", "epidemic" and "pandemic".

To determine if the gravestones of any individuals provided linkages to memorials, we also searched the online Cenotaph database¹¹ (keyword searches: "influenza", "epidemic", "pandemic"). Specific books on memorials in New Zealand were also examined.¹²⁻¹⁴

Local networks and memorial site visits

We utilised informal local history networks to identify potential additional memorials in the Northland Region (which has a relatively high population of Māori who suffered disproportionately in the pandemic⁴). For selected memorials we conducted site visits to document accessibility, to take photographs and ascertain any associated signage in the vicinity. We also determined if publicly accessible memorials were visible on Google Street View.

Comparisons with other memorials

For comparisons with other New Zealand-based mass fatality events, we selected events with 20 or more fatalities since 1900 (as per a recent study¹⁵). To identify any memorials associated with these events, we used the data from an official history website, ¹⁶ supplemented with internet searches, to see if memorial data were lacking at the primary data source (using the search terms: "memorial", "monument" and "plaque"). For comparisons with war memorials we used the following official website: http://www.nzhistory.net.nz/map/memorials-register-map.

Results

Memorials related to the pandemic

A total of seven publicly accessible memorials that referred to multiple pandemic deaths or health worker responder deaths from the pandemic were identified (Table 1). Even so, several of these cannot be considered to be fully pandemic-related in that: (i) they also relate to the life work of the named individual (eg, the two memorials to Dr Cruickshank including Figure 1); and (ii) also relate to other causes of death (ie, for soldiers the Featherston Camp memorial and for the nurses memorialised at the two chapels listed in Table 1).

For none of these seven memorials was there evidence of signage in the vicinity

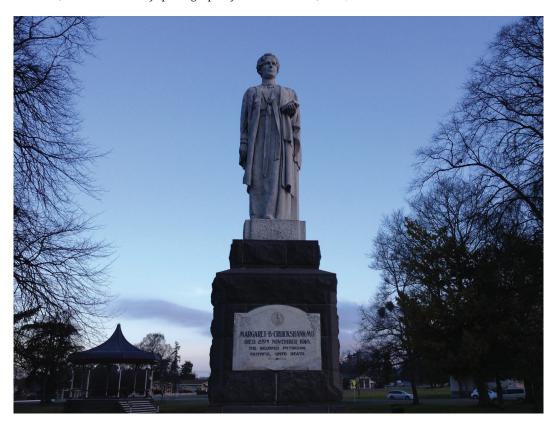


Table 1: The seven identified publicly accessible memorials relating to multiple deaths from the 1918 influenza pandemic deaths (or health worker responders) in New Zealand and which specifically mention "influenza" (organised by north to south location within the country).

Memorial type	Location	Further details (see the Appendix for additional details and selected photographs)
North Island		
Granite memorial to the 1,128 citizens of Auckland who died in the pandemic (with many in unmarked graves)	Waikumete Cemetery, Glen Eden, Auckland	The granite memorial erected in 1988 specifically states: "victims of the 1918 influenza epidemic" and "Particular respect is paid to doctors, nurses, citizen volunteers whose selfless efforts to aid the sick resulted in their own untimely deaths." Of note is that the memorial location is not shown on any signage at the cemetery or in an online map of the cemetery, but it is mentioned on the Waikumete Cemetery's official website.
Headstone on grave raised by public subscriptions for the nurse Jessie Linton	Shortland Historic Cemetery, Thames	The inscription on the white marble gravestone states: "after untiring devotion to duty during the epidemic of 1918 she fell a victim". The site visit indicated that the wording is becoming illegible due to corrosion of the lettering (Figure A, Appendix).
The Featherston Military Camp memorial obelisk and adjacent memorial wall.	Featherston Cemetery, Featherston	The notice beside the memorial wall briefly refers to the "Spanish influenza" epidemic killing soldiers training at the Featherston military camp. The obelisk refers to those who died in the camp—but not to influenza. Both the obelisk and the memorial wall are also for others who died in the camp (eg, from accidents or other diseases or injuries), though pandemic influenza was the major cause as per the November 1918 dates of death on the gravestones and other work. ²³ (Figure B, Appendix).
South Island		
Marble statue of Dr Margaret Cruickshank with information board	Seddon Square, Waimate, South Canterbury (Figure 1)	Dr Cruickshank was New Zealand's first registered woman doctor, and she died in the 1918 pandemic while caring for her patients. The inscription on the three metre high statue reads: "The Beloved Physician/Faithful unto Death". The information board beside the statue does refer to the pandemic and her death during it. The creation of this memorial may also reflect her multiple healthcare contributions to the community. Nevertheless, in 2007 the New Zealand Ministry of Health named a pandemic preparedness exercise "Exercise Cruickshank" in recognition of her work during the 1918 influenza pandemic (http://www.nzhistory.net.nz/people/margaret-cruickshank).
Grave of Dr Margaret Cruickshank and associated information board	Waimate Old Cemetery, McNamaras Road, Waimate, South Canterbury	The information board with a map is just inside the entrance of the Old Cemetery. It provides information about Dr Cruickshank's work in the influenza pandemic "providing services well beyond expected of a doctor" and her death from it. The inscription on her prominent gravestone does not refer to influenza. See other details in the row above.
Christchurch Nurses' Memorial Chapel	Christchurch Hospital site, Riccarton Avenue, Christchurch	This memorial is multi-purpose in that it is for two of the nurses who died in the pandemic and it is New Zealand's only dedicated memorial chapel to women who died in any war. It is publicly accessible in principle, but has not been so in recent years due to repairs from earthquake damage sustained in 2011.
Plaque for nurses who died (in a chapel)	Dunedin Hospital, Dunedin	The plaque is for the "Dunedin Hospital Trained Nurses" who died in the war or from the "influenza epidemic" in the Otago Region, or in a "troopship epidemic" (ie, on the Tahiti troopship ²⁴). Also listed are the names of four Voluntary Aid Detachment workers who died in the epidemic. See also Figure G in the Appendix.



Figure 1: Statue of Dr Margaret Cruickshank, a physician who died in the 1918 influenza pandemic, Waimate, South Canterbury (photograph by the lead author, 2016).



directing visitors to their location, including the one at Waikumete Cemetery for the 1,128 people who died from the pandemic in Auckland. None of the publicly accessible memorials were specifically for Māori and only three were in cities: at Waikumete in Auckland, the Chapel in Christchurch and the Chapel in Dunedin. There was also fairly limited information on the memorials relating to the pandemic, with only two having information boards (excluding the Chapel in Christchurch which has been closed for some time due to earthquake damage and could not be visited). The role of health workers and/or citizen volunteers during the pandemic was explicit in five of the memorials: at Waikumete, to the nurse Jessie Linton, the two memorials to Dr Cruickshank and to nurses in the Dunedin Chapel. None of the memorial sites had documentation that linked to any related website resources about the pandemic or which linked all the influenza pandemic

memorials together in a thematic manner. Six of the seven memorials were identifiable based on internet searches and five had some aspects of the memorial that were visible on Google Street View.

Another 11 memorials for mass deaths or deaths of health worker responders were pandemic-associated but were either in private settings or did not specifically mention the pandemic (Table 2). Nine of these had specific relevance to Māori, and all nine of these were located in the North Island. These memorials to Māori do not have routine public accessibility, as they are in urupā (cemeteries) or are on marae, which are private spaces. Marae are areas with buildings and grounds which are the cultural and social focus for an iwi (tribe), hapū (sub-tribe) or whānau (family). Of these 11 additional memorials, seven had some information about them on the internet.



Table 2: Additional memorials with a relationship to the 1918 influenza pandemic in New Zealand, but which are either in private settings or do not specifically refer to the pandemic (organised by north to south location within the country).

Memorial type	Location	Further details
North Island		
Stone memorial with plaque	Te Aute Urupā (cemetery), Te Tii Mangonui, Bay of Islands, Northland, (private setting)	The translation of the korero on the memorial from Te Reo Māori (Māori language) is as follows: "In memory Of those of our extended family who died In the great flu during the years 1918–1935 Rest peacefully in the Lord" (translation courtesy of Te Huranga Hohaia of the Te Tii Community). The time period covers those who died in the pandemic and those who were considered to have died in subsequent years from its effects. Of note is that urupā are private settings with no public access. See Figure C in the Appendix.
Stone memorial to local Māori who died in the pandemic	Beside the "Church of Our Lady of the Assumption" at Motukaraka Point, Hokianga Harbour, Northland	The memorial inscription is in Te Reo Māori and has the names of eight individuals who died in the pandemic (ages 31 to 73 years). It specifically includes the word "influenza" in English language. The official heritage listing is Category 1 and the site is now part of the adjacent marae (a private setting).
Memorial tablet in a meeting house (Porowini)	Otiria Marae (west of Kawakawa) Northland (private setting)	The meeting house of this marae (Porowini), is reported to have a memorial table on one wall. It has "a list of the names of 28 Ngāti Hine men, women and children who died of influenza between 20 November 1918 and 3 January 1919". (http://www.nzhistory.net.nz/media/photo/otiria-marae-memorials).
Three memorials: common graves with headstones	Tapikitu Urupā, Omanaia Churchyard, Omanaia, Hokianga, Northland, (private setting)	One gravestone lists the names of 22 family members who died in November and December 1918. Another is for six family members with dates in 1919 and 1920 (possibly reflecting the subsequent pandemic waves). The third headstone has part of the inscription that specifically refers to the epidemic (in English language): "In memory of those family members who passed away during the flu epidemic in 1918". This latter memorial is associated with a 15-metre-long area with unmarked graves. See Figure D in the Appendix for one of the three memorials.
A carved wooden Māori cenotaph	Te Ihingarangi Marae, Waimiha, north of Taumarunui, (private setting)	The cenotaph was designed and carved by Tene Waitere of Ngāti Tarāwhai. A marae is a private setting and so this memorial is not accessible to the public. Four colour photos are in an online image gallery (http://maorimaps.com/te/main-map#url=/te/full_marae/waimiha-te-ihingarangi).
A carved wooden Māori cenotaph	Te Kōura Marae, north of Taumarunui, (private setting)	The cenotaph was designed and carved by Tene Waitere of Ngāti Tarāwhai (with similar features to the one at Te Ihingarangi Marae, detailed in the row directly above). A photograph from 1920 is online (http://www.nzhistory.net.nz/media/photo/maorimemorial-influenza-pandemic) and a more recent colour photo also (the first image at: http://www.maorimaps.com/main-map#url=/full_marae/te-k%C5%8Dura).
A granite obelisk	Te Reinga Marae, Wairoa, northern Hawkes' Bay (private setting)	The obelisk has text in Te Reo Māori that can be paraphrased in English language as: "These are the people of the tribe who died in the epidemic that they called the flu in the year of our Lord 1918." A photograph and additional details are at: http://www.nzhistory.net.nz/media/photo/te-reinga-marae-influenza-memorial
South Island		
Marble statue of Dr Charles Little	Waikari Hospital grounds, Waikari, North Canterbury	The inscription says "to commemorate a life of devotion and self-sacrifice" but does not mention the pandemic. This monument may reflect Dr Little's role in providing care during the pandemic (from which he died) but also his prior healthcare work to the community "for close on forty years". See also Figure E in the Appendix.
Obelisk to Dr Little (the same doctor as per the above row)	Rutherford Reserve, Mountainview Rd, Culverden, North Canterbury	The inscription says "he gave his life for others" but does not mention the pandemic. See also Figure F in the Appendix.



Table 3: New Zealand war memorials, sourced from New Zealand History Online¹⁶ with a comparison to the 1918 influenza pandemic (data from this study).

War/s, pandemic	Memorials throughout NZ* (N)	Deaths in this war/ pandemic	Calculated memorials per 1,000 deaths
NZ Wars (1845–1872)	68	Approximately 3,000	22.7
South African War (1899–1902)	49	230	213.0
Both World Wars** (1914–19, 1939–45)	941	First World War=18,058 (including war-related deaths up to 31 August 1921); Second World War=11,928; Total=29,986	31.4
1918 influenza pandemic	7 (see Table 1)	Using 8,573 ⁴ (but possibly higher given new estimates of deaths in the military, albeit with some of these outside of New Zealand at the time ²⁵)	0.8

^{*}In addition, there are multiple museum displays related to all these and other wars involving the New Zealand population, eg, in Auckland's War Memorial Museum, and the "National Army Museum" in Waiouru.

Comparison with memorials to other mass fatality events

The physical memorial status was ascertained for non-epidemic/non-pandemic mass fatality events for the period 1900 to 2015 in New Zealand where there were 20+fatalities (n=20 events with 1,414 deaths, Table A3). It was found that there are memorials in existence or currently being constructed for 80% (16/20) of these events. Indeed, for one of these events there are at least eight memorials associated with it: the Mt Erebus aircraft crash. Several other such events had multiple memorials to them and in two cases they have dedicated sections in New Zealand museums (an earthquake and a shipwreck—see Appendix).

It was also apparent that the list of "disasters" at an official New Zealand history website¹⁷ includes no epidemics or pandemics at all. That is, it ignores the influenza pandemics of: 1918,⁴ 1957–1959,^{6,7} 1968 and of 2009.⁸ It also makes no mention of other post-1900 epidemics such as the 53 deaths in a 1913 smallpox epidemic, up to 173 deaths per year in various poliomyelitis epidemics and up to 375 deaths per year in various measles epidemics.¹⁸ This research also failed to identify any physical memorials to any of these other pandemics or epidemics.

Comparison with war memorials

The ratio of the number of publicly accessible memorials to deaths in various wars is shown in Table 3. This ratio was 261 times higher for the South African War than the pandemic (213 vs 0.8 memorials per 1,000 deaths). For the two World Wars together (since memorials to these are often combined), the respective value was 38 times higher.

Discussion

Main findings and interpretation

This study found only seven publicly accessible memorials that referred to the 1918 influenza pandemic in New Zealand. The comparison with the much greater number of war memorials and memorials other disaster events (with the latter comprising a much lower total mortality burden) was particularly stark. Such a difference is further compounded when considering the estimated NZD 122 million (around USD 79 million) spent on Second World War memorials by the New Zealand Government (in 2011 dollars), 19 and the NZD 120 million spent on a national war memorial park²⁰ that was completed in 2015. Many reasons might explain these differences, but possibilities include: (i) the



 $^{^{**}}$ Combined for both wars since the same memorial is often used for both wars in the New Zealand context.

timing of the 1918 pandemic at the end of the First World War (when New Zealand society was still highly disrupted from the impact of this war); (ii) the perception of these pandemic deaths being less "heroic" than military deaths; and (iii) the limited understanding around this time of potential lessons for the future arising from the pandemic experience. For example, some citizens who promoted the construction of various memorials to preventable causes (eg, to train crash disasters and also war memorials) might have believed that memorialising the dead and the disaster event might provide lessons for prevention. But in the early part of the 20th century the epidemiology of pandemic influenza and the potential for preventing and controlling it was still poorly understood scientifically and in popular culture. Other reasons may also apply given the complex psychological and sociological processes involved in memorialisation processes, particularly as detailed for war memorials. 13,21,22

The findings of this study also suggest that the current educational value of these publicly accessible pandemic memorials is likely to be constrained by: (i) only being in three cities; (ii) the limited signage and information on the memorials relating to the influenza pandemic; and (iii) the complete lack of any website resources that link these memorials together and to additional website-based information about pandemics. This situation suggests major scope for enhancing the presence and use of such memorials if a society wishes to better remember the large impacts of the 1918 pandemic and consider the implications for future pandemic preparedness. These impacts include the large numbers dying within months, the social and economic impacts of these sudden deaths, and the disproportionate effects on particular populations—in this case on Māori.6 While future influenza pandemics might only be preventable to some extent, it is very likely the appropriate preparedness could reduce the scale of the impact of a pandemic (hence the support for such preparations by the World Health Organization¹).

No published studies on 1918 pandemic influenza memorials in other countries were identified, and our internet searches for New Zealand memorials only rarely incidentally identified such memorials in other countries (eg, the Lueg Monument in Switzerland to 54 Bern cavalrymen who died in the pandemic). Further research could more systematically determine the presence of such memorials in both combatant and non-combatant nations at this time.

Strengths and limitations of this study

This study benefited from New Zealand being a relatively small country in which the 1918 influenza pandemic has at least some level of local documentation in a popular book4 and on a popular New Zealand history website.5 Furthermore, it is a country for which national collections of memorial data have been assembled online. But there are limitations in that some additional memorials may not have been identified, especially if these are in private places such as marae, urupā or consist only of a plaque inside a building that is not documented in any book or website. For example, we are recipients of oral history reports concerning three other possible pandemic-associated sites, but for which no written documentation has yet been identified (Table A2). More detailed involvement of local historians throughout the country may be required to capture data on all such pandemic-associated sites.

Options for enhancing the educational utility of pandemic-related memorials

If a society wished to enhance the long-term educational value of memorials concerning pandemic influenza and its future threat, a range of options exist, as suggested by the New Zealand situation:

- Upgrading existing publicly accessible physical memorials to include more information about the 1918 pandemic. Also, symbols (eg, quick response codes) on each memorial or on related signage could allow users with smartphones to gain additional information immediately. Signage around the locality could also make the memorials easier to find. In some cases, existing memorials could benefit from restoration work (Figure A).
- Increasing the number of memorials is an option, especially in cemeteries where there are unmarked graves of those who died in the pandemic. An example of such unmarked graves



is at Andersons Bay Cemetery in the City of Dunedin. Funds could be made available to relevant local authorities that wished to identify sites not yet covered by any memorial. For example, this could include Māori tribal (iwi) authorities who might wish to memorialise additional sites where the pandemic had a notable impact.

- Producing a national memorial (physical or online) that listed all the doctors, nurses and other health workers who died in the pandemic. This could even be done for all the victims, since a comprehensive online memorial exists for all those dying in the wars New Zealand has been involved in (the Cenotaph¹¹).
- Integrating links to all the memorials into one official website that has a detailed set of information about the pandemic, its local impacts and a discussion of future pandemic threats. An integrated memorial could also contribute materials to a "traveling museum exhibition" that could tour the country.

A deadline for working on these options could be the 100th anniversary of the 1918 pandemic, with the key organising agencies being the government ones concerned about heritage and health. Ideally however, a range of other parties might be involved in such planning including local government, local heritage and health groups, and representatives of indigenous populations who have suffered disproportionately in past pandemics.

Conclusions

Despite the major impact of the 1918 influenza pandemic in New Zealand (~8,600 deaths), only seven publicly accessible memorials that refer to it were identified in this study. This was in marked contrast to other disaster-related memorials and particularly to war memorials. Furthermore, the current educational value of these pandemic memorials is likely to be very limited for a range of reasons such as remote location and limited signage. There appears to be major scope for enhancing public education around the persisting threat of future pandemics via improved use of physical memorials and linkages to online resources.



Appendix: Additional detail on the identified memorials and selected photographs

Table A1: Additional details on the identified publicly accessible memorials relating to the 1918 influenza pandemic in New Zealand which specifically mention "influenza" (organised by north to south location within the country).

Memorial type	Location	Links to image/s of the memorial	Further details
North Island	1		
Granite memorial to the 1,128 citizens of Auckland who died in the pandemic (with many in unmarked graves).	Waikumete Cemetery, Glen Eden, Auckland	For a photograph and details of the inscription see: https://ohnoinfluenza.files.wordpress.com/2013/06/6504329169_8c-877c59f4_z.jpg	The granite memorial erected in 1988 specifically states: "victims of the 1918 influenza epidemic" and "Particular respect is paid to doctors, nurses, citizen volunteers whose selfless efforts to aid the sick resulted in their own untimely deaths." The memorial is situated at one end of a mowed lawn above unmarked graves (section E on "Eucalyptus Avenue", with the entrance on Waikumete Road being the nearest). Of note is that the memorial location is not shown on any signage at the cemetery or in an online map of the cemetery, but it is mentioned on the Waikumete Cemetery's official website. From the satellite "Earth" view on Google Street View, it is possible to see the impressions of the separate unmarked graves (eg, https://www.google.co.nz/maps/place/Eucalyptus+Ave,+Glen+Eden,+Auckland+0602/@-36.9079602,174.6486759,153m/data=!3m1!1e3!4m2!3m1!1s0x6d0d4177ed5c9c43:0xfe8f9fc261323bac)
Headstone on grave raised by public subscriptions for the nurse Jessie Linton.	Shortland Historic Cemetery, Thames	The third image at this website shows the gravestone: http://www.thetreasury.org.nz/Flu.htm. See also Figure A in this Appendix.	The inscription on the white marble gravestone states: "after untiring devotion to duty during the epidemic of 1918 she fell a victim" The site visit indicated that the wording is becoming illegible due to corrosion of the lettering (Figure A). The site is plot 3465, approximately 30 meters behind and to the right of the main cemetery sign (and a few meters in front of a grave with a cross on the top of it). This site is approximately opposite from 103B Danby Street, Thames, and is the most predominantly white headstone in the centre of this Google Street View image: https://www.google.co.nz/maps/@-37.1424328,175.557212,3a,27. 8y,60.41h,90.48t/data=!3m6!1e1!3m4!1sqR5F8E6KOm6bL45Cy_lqlQ!2e0!7i13312!8i6656!6m1 !1e1. An adjacent plot (3464) is for Amy Ritchie (the wife of a local doctor) who also died in the pandemic.
The Featherston Military Camp memorial obelisk and adjacent memorial wall.	Featherston Cemetery, Featherston	Obelisk and wall: http:// www.nzhistory.net.nz/media/ photo/featherston-cemetery- war-memorial See also Figure B in this Appendix.	The notice beside the memorial wall briefly refers to the "Spanish influenza" epidemic killing soldiers training at the Featherston Military Camp. The obelisk refers to those who died in the camp but does not use the word "influenza". Both the obelisk and the memorial wall are also for others who died in the camp (eg, from other diseases and injuries), though pandemic influenza was the major cause as per the November 1918 dates of death on the grave headstones and as detailed in other work (see Sertsou et al, <i>Theor Biol Med Model</i> 2006; 3:38). The wall was also constructed "to celebrate the 30 th anniversary of the twinning of Featherstor with Messines, Belgium". Although not visible on Google Street View, the memorial site is visible on "Earth" view on the western corner of the cemetery: https://www.google.co.nz/maps/search/featherston+cemetery/@-41.1220637,175.3142862,306m/data=!3m1!1e3. None of the individual gravestones refer to influenza or the epidemic.
South Island	I.		
Marble statue of Dr Margaret Cruickshank with information board.	Seddon Square, Waimate, South Canterbury	Contemporary colour photograph: http://www.teara.govt.nz/en/photograph/32564/statue-of-margaret-cruick-shank-waimate See also Figure C in this Appendix.	Dr Cruickshank was New Zealand's first registered woman doctor, and she died in the 1918 influenza epidemic while caring for her patients. The inscription on the three-metre high statue reads: "The Beloved Physician/Faithful unto Death" but does not mention influenza or the pandemic. However, the information board beside the statue does refer to the pandemic and her death during it. The creation of this memorial may also reflect her multiple healthcare contributions to the community. Nevertheless, in 2007 the New Zealand Ministry of Health named a pandemic preparedness exercise "Exercise Cruickshank" in recognition of her work during the 1918 influenza pandemic (http://www.nzhistory.net.nz/people/margaret-cruickshank). The statue is visible on Google Street View (at 125 Queen St, Waimate): https://www.google.co.nz/maps/@-44.7352367,171.0449472,3a,21.3y,119.91h,88. 63t/data=!3m6!1e1!3m4!1sEvoCocP3Z83bcl79BAeKdA!2e0!7i13312!8i6656 Other information about Dr Cruickshank (including her role in the pandemic) is in the Waimate Museum. Also in this museum is a brass plaque from the maternity ward at the former Waimate Hospital (this ward was named in her honour in 1948).



Table A1: Additional details on the identified publicly accessible memorials relating to the 1918 influenza pandemic in New Zealand which specifically mention "influenza" (organised by north to south location within the country) (continued).

Memorial type	Location	Links to image/s of the memorial	Further details
Grave of Dr Margaret Cruickshank and associated information board.	Waimate Old Cemetery, McNamaras Road, Waimate, South Canterbury	Photograph of the gravestone on a cemetery brochure: https://www.waimatedc. govt.nz/data/assets/ pdf_file/0008/23849/CEME- TERY-BROCHUREpdf	The information board with a map is just inside the entrance of the Old Cemetery. It provides information about Dr Cruickshank's work during the influenza pandemic "providing services well beyond expected of a doctor" and her death from it. The inscription on her prominent gravestone does not refer to influenza. See other details in the row above.
Christchurch Nurses' Memorial Chapel.	Christchurch Hospital site, Riccarton Avenue, Christchurch	Outside: http://www.nzhis- tory.net.nz/media/photo/ christchurch-nurses-memori- al-chapel Internal photograph: http:// christchurchcitylibraries. com/Heritage/Photos/Disc13/ IMG0036.jpg	This memorial is multi-purpose in that it is for two of the nurses who died in the 1918 influenza pandemic (Grace Beswick and Hilda Hooker [http://my.christchurchcitylibraries.com/1918-influenza-epidemic-how-christchurch-coped/] and it is New Zealand's only memorial chapel to women who died in any war. It is an architecturally significant building and is a Category 1 Historic Place (http://www.heritage.org.nz/the-list/details/1851). It has not been accessible to the public for some time due to repairs from earthquake damage. Further details are on the Chapel's website (http://cnmc.org.nz/). The Chapel is visible on Google Street View: https://www.google.co.nz/maps/@-43.5343579,172.6242029,3a,90y,44.99h,90.58t/data=!3m6!1e1!3m4!1saDLD6doXcvoOJ6lvMOMX9Q!2e0!7i13312!8i6656!6m1!1e1
Plaque for nurses who died.	Chapel in Dunedin Hospital	Nil online. See also Figure G in this Appendix.	The plaque is for the "Dunedin Hospital Trained Nurses who Died on Active Service", including four who died from the "influenza epidemic" in the Otago Region, and another one who died in a "troopship epidemic" (ie, on the Tahiti troopship, which travelled from New Zealand to the UK [Summers et al, Emerg Infect Dis. 16:1931–1937). Also listed are four VAD workers who died in the epidemic (these were Voluntary Aid Detachment workers who assisted nurses).

Other possible memorials to the 1918 influenza pandemic

Our searches included the following possible memorials (Table A2), but we lack sufficient information to be sure about any link with the 1918 pandemic. Further historical research may be required to clarify any such associations.

Table A2: Possible sites with a relationship to the 1918 influenza pandemic but for which further research is needed to clarify.

Site	Location	Further details
Large, possibly common, grave with a metal fence surrounding it.	St James Church, Kerikeri, Northland	A single oral history report provided to one of us by a former worker at the churchyard (Mr Derek Moon) was that a large grave was a common grave for 1918 influenza pandemic victims. The location of the grave and its level of weathering is compatible with an age of around 1918 but no other documentation has been identified to date (eg, in the Anglican Archives).
Common grave.	Maketu Cemetery, Bay of Plenty	Oral history reports provided to one of us are that an unmarked concrete tomb is a common grave for 1918 influenza pandemic victims, but no specific documentation could be identified.
Memorial row of trees.	Rissington Cemetery, Rissington, Hawkes Bay	A single oral history report provided to two of us from a former resident of the area (Mrs Joan Hamlin) was that the row of oak trees beside the Rissington cemetery were planted as a memorial to 1918 pandemic victims and to the hard work of the wife of a local station owner in caring for the sick. However, the site visit indicated no relevant plaque or signage at the cemetery and no online information could be identified.



Table A3: Presence of physical memorials for all the sudden mass fatality events occurring from 1900 to 2015 with 20 or more fatalities (Wilson et al, Aust NZJ Public Health, e-publication 28 February 2017) (for events occurring within current New Zealand territory, including the Exclusive Economic Zone, and ordered by descending number of deaths).

Sudden mass fatality event (excluding more drawn out epidemics and pandemics)	Year	Deaths	Any plaque or monument*
Hawke's Bay earthquake (using the total from the memorial, though further research is underway to better clarify this).	1931	258	Yes—multiple***
Crash of Air New Zealand Flight TE901 into Mt Erebus, Ross Dependency, Antarctica. (Within study scope since NZ has a territorial claim on this part of Antarctica).	1979	257	Yes—eight**
Canterbury earthquake (February 2011).	2011	185	Yes—multiple
Tangiwai rail crash related to a lahar from volcanic activity which destroyed a railway bridge.	1953	151	Yes—multiple
Sinking of the SS <i>Penguin</i> near Wellington in "heavy seas" (noting that some online information uses an incorrect "75" deaths).	1909	72	Yes—multiple
Cyclone Giselle and sinking of the TEV <i>Wahine</i> near Wellington (51 immediate deaths from the sinking, two delayed deaths from injuries, and three killed in the storm on the mainland).	1968	56	Yes—multiple#
Featherston Prisoner-of-War Camp riot (deaths in Japanese prisoners-of-war and one guard).	1943	49	Yes
Sinking of the SS <i>Elingamite</i> off the Three Kings Islands.	1902	45	No
Ralph's Mine explosion in Huntly.	1914	43	Yes
Ballantyne's store fire in Christchurch.	1947	41	Yes
Seacliff Mental Hospital fire (north of Dunedin).	1942	37	No
Sinking of the MV <i>Kaitawa</i> near Cape Reinga in "heavy seas".	1966	29	Yes
Pike River Mine explosions (northwest of Greymouth).	2010	29	Yes
Sinking of the <i>Wimmera</i> after striking German mines during the First World War (north of Cape Maria van Diemen, Northland).	1918	26	No
Sinking of the <i>Manchester</i> (Tasman Sea, near Cape Farewell).	1912	25	No
Sinking of the <i>Loch Long</i> off the Chatham Islands.	1902	24	Yes
Crash of NZ National Airways Corp. Flight 441 (Kaimai Ranges).	1963	23	Yes—multiple
Sinking of the <i>Ranui</i> off Mount Maunganui in a "violent sea".	1950	22	Yes
Kopuawhara flash flood destroying a railway work camp (during construction of the Napier-Gisborne Railway line).	1938	21	Yes
Railway crash at Hyde (Otago).	1943	21	Yes
	•	Total= 1,414	80% have memorials (16/20

^{*} If the presence of a memorial was not apparent in the documentation of the primary data sources (http://www.nzhistory.net.nz/map/ memorials-register-map), then internet searches were conducted using the search terms: "memorial", "monument" and "plaque".

** There are eight memorials listed at: http://www.nzhistory.net.nz/media/photo/memorial-cross-mount-erebus but not all may have a

[#] The "Museum of Wellington City and Sea" has a permanent commemorative exhibition to the Wahine sinking.



plaque or monument.

*** The MTG Hawke's Bay Museum Tai Ahuriri in Napier has dedicated a gallery to this earthquake.

Figure A: Gravestone of the nurse Jessie Linton erected by the people of Thames after her death in the 1918 influenza pandemic, but now showing scope for renovation work (photograph by Nick Wilson, 2015).



Figure B: Obelisk style memorial to military personnel who died in the Featherston Camp, mainly from pandemic influenza in 1918 (photograph by Nick Wilson, 2015).





Figure C: Memorial to Māori victims of the 1918 pandemic at the Te Aute Urupā (cemetery), Te Tii Mangonui, Bay of Islands, Northland (photograph by Catharine Ferguson, 2016; with permission from the kaumātua at Te Tii).



Figure D: One of three common grave headstones to Māori victims of the 1918 influenza pandemic at Tapikitu Urupā, Omanaia Churchyard, Omanaia, Northland (photograph by Catharine Ferguson, 2016; with permission from the local kaumātua).





Figure E: Statue to Dr Charles Little, a physician who died in the pandemic, outside Waikari Hospital, North Canterbury (photograph by Geoffrey Rice, 2016).





Figure F: Memorial to Dr Charles Little, a physician who died in the pandemic, at Culverden, North Canterbury (photograph by Geoffrey Rice, 2016).





Figure G: Memorial plaque to nurses who died in the pandemic (and the First World War) at the Chapel in Dunedin Hospital (photograph by Nick Wilson, 2016).





Competing interests:

Nil.

Acknowledgements:

The authors gratefully acknowledge those who helped in Northland to identify memorials and permit photographs to be taken: (i) Te Huranga Hohaia and the kaumātua of the Te Tii Mangonui Community, Bay of Islands, Northland; and (ii) Steve Morunga and Cecelia Morunga of Omanaia, Hokianga, Northland. We also thank Imelda Bargas (Ministry of Culture and Heritage), Aidan Challis and Stuart Park for their helpful assistance.

Author information:

Nick Wilson, Professor, University of Otago, Wellington; Catharine Ferguson, Kerikeri,
Northland; Geoffrey Rice, Professor, University of Canterbury, Christchurch;
Michael G Baker, Professor, University of Otago, Wellington; Ben Schrader, Wellington;
Christine Clement, Te Puke, Bay of Plenty; George Thomson, Associate Professor, University
of Otago, Wellington.

Corresponding author:

Professor Nick Wilson, University of Otago, Wellington. nick.wilson@otago.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7409

REFERENCES:

- World Health Organization. Pandemic influenza risk management: WHO interim guidance. Geneva: World Health Organization, 2013.
- 2. MacDonald G, Moen A, St Louis M. The national inventory of core capabilities for pandemic influenza preparedness and response: an instrument for planning and evaluation. Influenza Other Respir Viruses 2014; 8:189–93.
- 3. Frieden T, Tappero J, Dowell S, Hien N, Guillaume F, Aceng J. Safer countries through global health security. Lancet 2014; 383:764–6.
- Rice G. Black November: The 1918 influenza pandemic in New Zealand. Christchurch: Canterbury University Press, 2005.
- 5. Ministry for Culture and Heritage. The 1918 flu pandemic. Updated 28 October 2014. http://www.nzhistory.net.nz/culture/influenza-pandemic-1918

- 6. Wilson N, Telfar Barnard L, Summers J, Shanks G, Baker M. Differential mortality by ethnicity in 3 influenza pandemics over a century, New Zealand. Emerg Infect Dis 2012; 18:71–77.
- 7. Viboud C, Simonsen L, Fuentes R, Flores J, Miller MA, Chowell G. Global Mortality Impact of the 1957–1959 Influenza Pandemic. J Infect Dis 2016; 213:738–45.
- 8. Wilson N, Summers JA,
 Baker MG. The 2009 influenza pandemic: a review of
 the strengths and weaknesses of the health sector
 response in New Zealand.
 N Z Med J 2012; 125:54–66.
- 9. Pereira MS, Schild GC. An antigenic variant of the Hong Kong-68 influenza A 2 virus. J Hyg (Lond) 1971; 69:99–103.
- Ministry for Culture and Heritage. A list of national monuments.
 March 2015. http:// www.mch.govt.nz/

- nz-identity-heritage/national-monuments-war-graves/ list-national-monuments
- 11. Auckland War Memorial Museum. Cenotaph database. http://muse.aucklandmuseum.com/databases/Cenotaph/locations.aspx?
- **12.** Aberhart L. Anzac: Victoria University Press, 2014.
- 13. Maclean C, Phillips J.
 The sorrow and the
 pride: New Zealand war
 memorials: Historical
 Branch, GP Books, 1990.
- 14. Bargas I, Shoebridge T. New Zealand's First World War Heritage. Auckland: Exisle Publishing, 2015.
- 15. Wilson N, Morales A, Guy N, Thomson G. Marked decline of sudden mass fatality events in New Zealand for the 1900 to 2015 period: The basic epidemiology.

 Aust N Z J Public Health 2017; 41:275–279.
- **16.** Ministry for Culture and Heritage. 'NZ memorials register map'.



- Updated 20 December 2012 Memorials. http://www. nzhistory.net.nz/map/ memorials-register-map
- 17. Ministry for Culture and Heritage. New Zealand disasters timeline. Updated 3 September 2015. http://www.nzhistory.net.nz/culture/new-zealand-disasters/timeline
- 18. Maclean F. Challenge for health: A history of public health in New Zealand. Wellington: Government Printer, 1964.
- 19. Phillips J. 'Memorials and monuments Memorials to the centennial and the Second World War', Te Ara the Encyclopedia of New Zealand, updated 13 July 2012. http://www.

- TeAra.govt.nz/en/memorials-and-monuments/ page-6
- 20. Forbes M. National
 War Memorial takes
 shape. Stuff. 3 October
 2013. http://www.
 stuff.co.nz/dominion-post/news/9240092/
 National-War-Memorial-takes-shape
- 21. Winter J. Sites of Memory, Sites of Mourning: The Great War in European Cultural History: Cambridge University Press, 1998.
- 22. Alderman D, Dwyer O. 'Memorials and Monuments', Academia, 2009. http://www.academia. edu/6091282?Memorias_and_Monuments

- 23. Sertsou G, Wilson N, Baker M, Nelson P, Roberts MG. Key transmission parameters of an institutional outbreak during the 1918 influenza pandemic estimated by mathematical modelling. Theor Biol Med Model 2006; 3:38.
- 24. Summers JA, Wilson N, Baker MG, Shanks GD. Mortality risk factors for pandemic influenza on New Zealand troop ship, 1918. Emerg Infect Dis 2010; 16:1931–7.
- 25. Summers JA, Shanks GD, Baker MG, Wilson N. Severe impact of the 1918–19 pandemic influenza in a national military force. N Z Med J 2013; 126(1378):36–47.



Face-to-face versus telephone delivery of the Green Prescription for Māori and New Zealand Europeans with type-2 diabetes mellitus: influence on participation and health outcomes

Margaret Williams, Simeon Cairns, David Simmons, Elaine Rush

ABSTRACT

AIM: In Aotearoa/New Zealand, the proportion of Māori who participate in the national Green Prescription lifestyle programme is lower than for New Zealand Europeans. We compared the uptake and effectiveness of two modes of Green Prescription delivery: face-to-face and telephone among both Māori and New Zealand Europeans.

METHOD: Sixty-eight Māori and 70 New Zealand Europeans with type-2 diabetes participated in this six-month randomised trial of the two modes of delivery. Recruitment integrated an explicitly Māori culturally sensitive approach. All participants received lifestyle intervention. Anthropometry, blood lipids and glycated haemoglobin were measured before and after the intervention.

RESULTS: The face-to-face approach (first meeting) yielded 100% uptake into the programme among both Māori and New Zealand Europeans. At six months there were overall reductions in weight (1.8; [95 CI%, 0.6, 2.9kg]), waist circumference (3.7 [2.6, 4.8cm]), and total cholesterol (0.6 [0.3, 0.9mmol/l]) and glycated haemoglobin (3.1 [-0.2, 6.7mmol/mol]). There were no significant differences by mode of delivery, ethnicity or gender.

CONCLUSION: The Green Prescription programme resulted in small but clinically favourable improvements in health outcomes for type-2 diabetes patients, regardless of the mode of delivery for both Māori and New Zealand Europeans.

mproving blood glucose, lipids, weight and blood pressure reduces complications among patients with type-2 diabetes mellitus. 1,2 Such improvements can be realised through increased physical activity, healthy eating and appropriate pharmacological treatment. 3,4 In Aotearoa/New Zealand a number of lifestyle interventions exist,5 including the national Green

Prescription (GRx) health service, which is usually delivered through a regional sports trust. Individuals referred to the national GRx programme receive a three-month service, including four telephone calls, mailed support material with tailored support and advice from a GRx facilitator about the recommended quality and quantity of physical activity and food. ^{6,7}.



The prevalence of type-2 diabetes among Māori, the indigenous people of Aotearoa/ New Zealand, is twice that of New Zealand Europeans, and is associated with a greater risk of diabetes complications.8 Clearly, there is a need to understand better how health outcomes for Māori can be enhanced after diabetes has been diagnosed⁵ and one approach includes improving uptake of physical activity. Māori have a low participation rate in the GRx service (ie, <16%),7,8,9 and there is a need to improve their uptake into this programme. There are two steps needed for participation in an effective health service—the first is to gain trust to assist the entry of the individual into the service, and the second is to have good adherence during the service itself. It has been suggested that Māori prefer faceto-face delivery over telephone delivery, especially for strengthening mutual trust and understanding. 10,11,12

Therefore, the aim of this study was to compare the effect of face-to-face and telephone modes of delivery of the national GRx programme on participation and health outcomes (including glycaemic control, blood lipid profile, anthropometric and cardiovascular risk factors) on Māori and Europeans newly diagnosed with type-2 diabetes.

Methods

This was an open-label randomised trial (ACTRN1261000165088) undertaken through Sport Waikato, a regional sports trust that serves the Waikato province of Aotearoa/New Zealand. Waikato spans 21,220 km² and includes one metropolitan city and 10 small rural/semi-rural towns. In general, the relative socioeconomic status is low, particularly for Māori.¹³

Study design: kaupapa Māori research

A kaupapa Māori framework/research ethics were utilised^{10,11,12,} in an attempt to improve engagement in the GRx health service for both Māori and New Zealand Europeans. This approach included the integration of Māori culture, principles and values, knowledge and language into the communication,^{11,14} underpinned by the Treaty of Waitangi principles of participation, partnership and protection.¹⁵

Traditionally, such approaches require faceto-face engagement promoting whakamana (empowerment).11-13 For Māori these are achieved through whānaungātanga (strengthening mutual relationships), manaakitanga (enhancing the integrity of the person) and pātaka mātauranga (sharing knowledge that leads to understanding and responsibility). Therefore an understanding of the individual in their community needs to be considered as part of the communication with participants and for improving health literacy holistically. 10,12 A reference group of health professionals and Māori/Iwi leaders was formed to guide and implement this trial, provide opportunities, establish working relationships that would reach potential participants and develop a kaupapa Māori GRx working manual for Sport Waikato. In the planning stages, meetings with the reference group occurred monthly while fortnightly meetings occurred with the Sport Waikato team, including the Māori GRx facilitator employed for the delivery of this trial. Utilising a kaupapa Māori approach to research and health literacy focuses on respectful relationship with participants, their families and community. Ethical approval was provided by the Northern Y ethics committee, reference number, NTY/07/12/137. The study recruitment period occurred over an 18-month period from November 2008 until February 2010.

Recruitment and participation

Initial contact occurred between a patient and primary care provider (ie, general practitioner and/or practice nurse) who assessed their suitability for referral to the GRx trial for Sport Waikato. Patients who were on insulin therapy or likely to receive insulin therapy or dialysis treatment in the next 12 months, had ambulatory problems or conditions that would prevent participation in physical activity, were excluded. Eligible participants were then invited to participate in the study by Sport Waikato (Figure 1). Subsequently, referrals were excluded prior to the trial because their contact details were invalid, incorrectly identified as eligible, which included other ethnicities, or who declined participation. The selected patients received a first face-to-face meeting with a female Māori researcher (MHW) who explained why and how the GRx programme



may benefit his or her health and management of type-2 diabetes. This was a key feature of the informed consent process. Written informed consent to participate was then obtained and baseline health outcome measures recorded.

An independent administrator subsequently used an electronically generated randomisation schema, stratified by ethnic group, to assign participants randomly to receive either face-to-face or telephone modes of delivery. Figure 1 shows the CONSORT diagram.

Figure 1: Recruitment, randomisation and participation at baseline and after six months of the GRx research study.



 ${\sf NZE} = {\sf New Zeal} \ {\sf Zeal} \ {\sf and Europeans}. \ {\sf Transient} = {\sf no forwarding contact details}.$



Health outcome measures

Anthropometric measures were obtained in triplicate for: height (to nearest 0.5cm) without shoes using a portable stadiometer (PE87 portable stadiometer Mentone Educational, Moorabbin, Victoria, Australia); body weight (to nearest 0.1kg) in light clothing and without shoes (Wedderburn Electronic Scale 0-150kg, Auckland, NZ); standing waist circumference obtained at the lateral mid-point between the lower rib and the iliac crest (to nearest 0.5cm). Mean values including body mass index were calculated for each participant. Systolic and diastolic blood pressure, and heart rate were recorded after a minimum sitting rest period of 5 min using an Omron IntelliSense Automatic Blood Pressure Monitor (Kyoto, Japan). Biochemical tests were undertaken by Path Lab Waikato Limited, an NZS/ISO 15189:2007 accredited laboratory (International Accreditation New Zealand) and included HbA_{1c}, (mmol glycated Hb/mol total Hb), and blood lipids (total cholesterol, highdensity lipoprotein, low-density lipoprotein, triglyceride concentrations [mmol/L]).

The New Zealand physical activity questionnaire short form (NZPAQSF) was administered with assistance as required. These measures were obtained close to the baseline and trial termination time points. Some participants had incomplete assessments despite attempts to obtain these data.

Delivery of the GRx intervention

Participants received monthly one-on-one support for six months by either the face-to-face or telephone approach. A Māori GRx facilitator trained in nutrition, physical activity and motivational interviewing delivered the GRx programme. The same information was communicated regardless of the delivery approach. In the first session, a physical exercise and healthy eating plan following Ministry of Health healthy eating guidelines¹⁸ was negotiated, and support

materials were provided either by post or in person. The physical activity plans incorporated walking, swimming, weight training and also utilised common activities such as washing clothes, vacuuming or gardening. Participants set achievable goals for increasing incidental physical activity and to consume healthier foods for the next month. For the face-to-face delivery the session time with the GRx facilitator was 15–60 min in a setting agreed to by the participant. In most cases this was the home of the participant.

Statistical analysis

Data are presented as mean, standard deviation and range for continuous variables. Categorical variables are reported as both frequency and percentages. Participation differences were assessed using the Fisher test. Differences are compared using paired *t*-test (baseline to completion) or unpaired *t*-test (between groups). Differences between the groups were also shown with the 95% CI for the difference of means. Statistical analyses were performed using IBM SPSS Statistics version 22 (IBM Corporation, Armonk, New York).

Results

Figure 1 shows that of the 1,755 referred to the Sport Waikato GRx programme, 210 were referred into the trial, of whom 138 were eligible, contactable and agreeable to being referred into the trial. All of the 138 attendees to the first face-to-face GRx information meeting gave informed consent to participate (49% Māori, 62% women). Of these, 64% (88/138) actively participated to the cessation of the six-month trial. Drop-out at six months was greater for Māori (49%, 33/68) than for New Zealand Europeans (24%, 17/70 (p=0.04)). Attrition for the faceto-face and telephone approach were 31% (22/70) and 41% (28/68) respectively overall (p=0.509), and 41% (15/36) and 56% (18/32) respectively among Māori (p=0.529).



Table 1: Baseline health outcome characteristics of patients newly diagnosed with type-2 diabetes distinguished by ethnicity and gender.

	Māori (n=68)		New Zealand Europeans (n=70)			
Measure	Women	Men	Women	Men		
	(n=39)	(n=29)	(n=47)	(n=23)		
Age (yr)	53±10	56±12	57±13	62±13		
	[35, 74]	[35, 80]	[30, 83]	[38, 86]		
Body weight	106.9±27.2	117.4±21.0	96.8±23.7	112.2±25.4		
(kg)	[57.5, 185.9]	[72.8, 157.0]	[47.6, 152.9]	[71.2, 159.3]		
Waist	125.5±18.7	125.6±16.1	116.1±16.4	126.3±17.4		
circumference	[90.7, 167.6]	[86.6, 157.0]	[78.0, 141.9]	[92.5, 164.4]		
(cm)						
Height (cm)	160.3±6.5	172.8±6.1	159.2±6.8	173.8±6.7		
	[146.0, 172.0]	[157.4, 185.3]	[143.5, 177.6]	[163.3, 186.1]		
BMI (kg/m²)	41.5±9.9	39.3±6.5	38.2±9.6	36.9±6.9		
	[24.1, 67.6]	[25.5, 50.4]	[20.7, 70.2]	[26.0, 51.6]		
Systolic BP	142±19	138±19	138±20	139±17		
(mmHg)	[102, 179]	[106, 172]	[107, 183]	[107, 183]		
Diastolic BP	88±12	86±12	80±13	80±10		
(mmHg)	[68, 122]	[54, 113]	[54, 112]	[61, 98]		
Resting heart	74±11	72±12	72±13	70±11		
rate	[53, 94]	[51, 101]	[49, 100]	[52, 91]		
(beat/min)						
HbA_{1c}	58.4±21.2 (n=36)	72.3±23.9 (n=19)	68.9±21.8 (n=22)	68.2±23.9 (n=15)		
(mmol/mol)	[38.8, 121.9]	[41.0, 121.9]	[38.8, 125.2]	[46.5, 118.6]		
Physical activity						
Briskly walking	13 {0, 42}	20 {0, 40}	20 {0,40}	10 {0, 30}		
{min/wk}	a30 {8, 33}	°30 {16, 99}	a30 {15, 60}	^a 38 {16, 330}		
Moderate	75 {0, 54}	195 {0, 375}	90 {0, 180}	40 {0, 210}		
activity	°0 {0, 285}	a90 {0, 225}	^a 105 {0, 225}	a90 {0, 427}		
{min/wk}						

Data are mean values ± standard deviation; range [minimum, maximum]; n = number of participants; BMI = body

mass index; BP = blood pressure; HbA $_{1c}$ = glycated haemoglobin. Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; Q_3 , the 75th percentile at baseline and a Median $\{Q_1, Q_3\} = Q_1$, the 25th percentile; $\{Q_1, Q_2\} = \{Q_1, Q_3\} = \{Q_$



Table 2: Influence of the GRx programme on health outcome measures distinguished by mode of delivery.

	All		Face-to-face			Telephone			
Measure	Baseline	6 months	Mean difference	Baseline	6 months	Mean difference	Baseline	6 months	Mean difference
Body weight (kg)	107.0±24.8	105.2±24.8	-1.8	108.2±25.3	106.2±24.4	-2.0	105.4±25.7	103.9±25.4	-1.5
	(n=88)	(p=0.03)	(-0.6, -2.9)	(n=48)	(p=0.033)	(-0.2, -3.8)	(n=40)	(p=0.031)	(-0.1, -2.9)
Waist circumference (cm)	122.8±17.9 (n=86)	119.1±17.5 (p<0.001)	-3.7 (-2.6, -4.8)	123.1±18.9 (n=47)	119.4±17.9 (p<0.001)	-3.7 (-2.0, -5.4)	122.4±17.0 (n=39)	118.7±17.1 (p<0.001)	-3.8 (-2.4, -5.1)
HbA _{1c} , (mmol/mol)	65.7±23.6	62.6±19.9	-3.1	65.6±24.5	60.8±18.9	-4.8	65.7±23.0	64.5±21.0	-1.2
	(n=64)	(<i>p</i> =0.069)	(0.2, -6.7)	(n=34)	(p=0.059)	(0.2, -9.8)	(n=30)	(<i>p</i> =0.599)	(3.4, -5.8)
TC (mmol/L)	5.3±1.7	4.7±1.4	-0.6	5.4±1.8	4.9±1.7	-0.6	5.1±1.5	4.5±1.0	-0.6
	(n=57)	(p<0.0001)	(-0.3, -0.9)	(n=33)	(<i>p</i> =0.002)	(-0.2, -0.9)	(n=24)	(<i>p</i> =0.022)	(-0.1, -1.2)
HDL (mmol/L)	1.0±0.4 (n=59)	1.0±0.3 (p=0.66)	0.02 (0.11,-0.07)	0.9±0.3 (n=33)	1.0±0.4 (p=0.049)	0.1 (0.1, 0.0)	1.1±0.5 (n=26)	1.0±0.2 (p=0.578)	0 (0.1, -0.2)
TC/HDL	6.3±3.6	5.9±3.0	-0.4	6.8 ± 4.1	6.2±3.5	-0.6	5.5±2.7	5.4±2.1	-0.1
	(n=57)	(<i>p</i> =0.064)	(0.0, -0.8)	(n=33)	(<i>p</i> =0.048)	(0.0, -1.1)	(n=24)	(<i>p</i> =0.729)	(0.4, -0.6)
LDL (mmol/L)	2.8±1.0 (n=54)	2.7±0.9 (p=0.326)	-0.1 (0.1, -0.3)	2.7±0.8 (n=31)	2.7±0.9 (p=0.93)	0 (0.2, -0.2)	2.9±1.2 (n=23)	2.7±0.8 (p=0.262)	-0.2 (0.2, -0.7)
Triglycerides	2.4±2.0	2.1±1.7	-0.3	2.3±1.6	2.0±1.6	-0.3	2.5±2.5	2.2±1.8	-0.3
(mmol/L)	(n=60)	(p=0.076)	(0.0, -0.7)	(n=33)	(p=0.023)	(0, -0.6)	(n=27)	(p=0.36)	(0.4, -1.1)
Systolic BP (mmHg)	139±19 (n=82)	139±19 (p=0.967)	0 (4, -4)	143±20 (n=44)	143±22 (p=0.935)	0 (6, -6)	134±15 (n=39)	134±15 (p=0.967)	0 (5, -5)
Diastolic BP	83±13	83±14	-2	85±16	83±14	-2	80±9	79±12	-1
(mmHg)	(n=82)	(p=0.680)	(1, -4)	(n=44)	(p=0.193)	(1, -6)	(n=38)	(<i>p</i> =0.476)	(2, -4)

Data are mean values \pm standard deviation; 95% confidence interval of the difference (in brackets); n = number of participants; BP = blood pressure; HbA_{1c} = glycated haemoglobin. TC=total cholesterol, HDL= high density lipoproteins, TC/HDL = total cholesterol/high density lipid cholesterol ratio, LDL= low density lipoproteins. Data are shown only when both baseline and 6 months values were obtained for each participant.

Baseline characteristics for all participants are shown in Table 1. After six months intervention (Table 2), there were significant reductions in weight, waist circumference and total cholesterol, but no significant differences in change of body weight or waist circumference according to mode of delivery. When data from both modes of delivery were combined, (Table 2) there was a 1.8kg reduction in body weight and 3.7cm reduction in waist circumference. The HbA_{1c} concentration fell by 3.1mmol/mol across the 64 participants with data. This effect was dominated by a lowered HbA_{1c} in patients who displayed a poorer glucose regulation at baseline, ie, HbA_{1c} >80mmol/mol. This small effect was slightly, but insignificantly, greater with the face-to-face than telephone approach. The GRx trial was associated with a significant but small reduction in total plasma cholesterol concentration. Changes in plasma high-density lipoproteins and triglyceride concentrations did not differ

significantly with mode of delivery (Table 2). There was no overall influence of the GRx intervention on arterial blood pressure (Table 2). No significant differences were found across ethnicity or gender.

The NZPAQSF data revealed that overall at baseline the individual's total time spent in physical activity was extremely low (Table 1). The majority of participants (90%) did not undertake the recommended 30 min per day of brisk walking on five or more days per week. Many participants (36%) reported that they did not do any brisk walking or physical activity over the previous six months, while 20% reported no moderate or vigorous physical activity. Between baseline and the cessation of the six months GRx trial. the proportion of participants who were completely inactive (ie, no time at least brisk walking) halved (Table 1). Overall there was no statistically significant change in time spent walking following the treatment.



Discussion

This is the first study to assess the effectiveness of the national GRx health service after six months of treatment among Māori and New Zealand Europeans newly diagnosed with type-2 diabetes. The major findings were that: the face-to-face first meeting, used as the GRx information session, yielded a 100% uptake into the study and the service; and overall the six months GRx trial was associated with clinically favourable albeit small reductions in weight, waist circumference and total cholesterol concentrations, without notable differences attributed to mode of delivery.

Participation

A key question addressed in this study was whether the face-to-face approach was associated with greater benefit than the telephone support for both Māori and New Zealand Europeans, especially in relation to participation. This notion derived from preliminary work, 19 which indicated that Māori preferred such an approach. The initial face-to-face information meeting prompted every patient that attended to sign up as a participant in the GRx trial (ie, 100% uptake). This exceeded recruitment numbers in earlier studies.^{7,8} Moreover, a concern identified in a previous national GRx health service survey was the paucity (<4%) of Māori participants.7 In the present study this was improved with nearly half of the participants being Māori. We attribute this success to the kaupapa Māori research and service approach, although we did not directly compare it with the telephone approach, as employed previously. 19 Despite this excellent uptake and regardless of the mode of delivery of the GRx programme there was still higher attrition of Māori than European at six months. Arguably, the very group that would have been useful to interview about attrition are those that did not complete the GRx intervention to provide insights and understanding of the barriers to participation and behavior changes.

Health outcome measures

The reductions in weight, waist circumference, total cholesterol for both interventions, would both be associated with improvements in quality of life and morbidity. The waist circumference reduction is particularly welcome, as it

likely reflects a reduction in intra-abdominal fat. 20,21 The decrease in HbA $_{\rm 1c}$ seen in the present study although not statistically significant, is an important factor associated with reductions in diabetes complications in newly diagnosed type-2 diabetes 22,23 and for cardiovascular disease. 23,24,25

One question arising from these health outcome measures is why the beneficial effects were small. Firstly, the NZPAQSF data demonstrated that the participants as a whole were extremely physically inactive (Table 1). This aspect probably contributed to the participants being heavier than those in many other studies (also BMI ≥40kg/ m² compared to 26-34kg/m²), had raised diastolic blood pressures and elevated HbA₁₀. For several of these participants the GRx trial increased their levels of physical activity (especially from nothing to some brisk walking), but overall there was little change. One plausible explanation for these smaller health outcome changes may simply be the modest increase in physical activity levels achieved by the participants. This is not unexpected given that the physical characteristics along with sedentary lifestyle of these participants at baseline is likely to restrict them embarking on much physical exercise. Moreover, low intensity dynamic exercise (ie, walking and/or resistance training) has been shown to improve the blood lipid profile and physical characteristics. 25,26 Thus, resistance training combined with aerobic training is noted to improve glycaemic control. Secondly, direct monitoring of food choice changes did not occur. However, participant self-reports about positive nutritional changes were anecdotally reported by the facilitators and in-participant interviews, but the reliability of these reports is unknown. Thirdly, some participants were possibly on medications that may have promoted body weight gain to confound any effects of the increased physical activity levels by participants.^{27,28}

Strengths and weaknesses

Strengths include a high proportion of Māori, probably due to the recruitment approaches used, the complete success in translating referral (once contact made) into participation and the clear study design. A further strength is that the lipid and glycated haemoglobin measures were accessed from patient records from the general practi-



tioner rather than increasing the participant burden. The major weaknesses are the small number recruited overall, the high drop-out and that participant satisfaction is based on anecdote. The trial had intended to recruit 70 Māori and 70 New Zealand Europeans into each intervention and the final number was half this. This was due to a lower than expected eligibility rate and the limited recruitment period. The drop-out rate was largely due to the mobility of the population, not active withdrawal.

Recommendations

The first interaction in GRx delivery should be face-to-face to improve uptake to participate (particularly among Māori), but the subsequent delivery can involve either face-to-face or telephone approaches. To make more conclusions that are robust on the relative merits of the different modes of delivery on adherence requires

an expansion of the sample size or use of a meta-analytical approach across multiple studies. Cost effectiveness should also be explored. While face-to-face delivery requires more facilitator time related to travel and organisation, there may be those that should be targeted for this more time-consuming approach (eg, those dropping out of the telephone approach). Larger improvements may occur with greater increases in physical activity associated with a longer duration of programme coupled with, and/or greater emphasis on food choices. Weekly or fortnightly GRx facilitator support rather than once a month might also improve response. In conclusion, the GRx health service appears to be equally beneficial for both Māori and New Zealand Europeans with newly diagnosed type-2 diabetes through either mode of delivery, face-to-face or telephone.

Competing interests:

Nil.

Acknowledgements:

The authors wish to acknowledge Ministry of Health New Zealand for funding the delivery component of the GRx health service, and Sport Waikato for employing the GRx kaiwhakahaere (facilitators) to deliver the GRx programme.

Author information:

Margaret Williams, School of Public Health and Psychosocial Studies, Auckland University of Technology, Auckland; Simeon Cairns, School of Sport and Recreation, Auckland University of Technology, Auckland; David Simmons, School of Medicine, Western Sydney University, Australia; Elaine Rush, Centre for Child Health Research, Auckland University of Technology, Auckland.

Corresponding author:

Dr Margaret Williams, School of Public Health and Psychosocial Studies, Auckland University of Technology, 640 Great South Road, Manukau, Auckland 2025.

marwilli@aut.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7410

REFERENCES:

- 1. Inzucchi SE, Bergenstal RM, Buse JB, et al. Management of hyperglycaemia in type 2 diabetes: a patient-centered approach. Position statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetologia. 2012; 55(6):1577–96.
- Joshy G, Simmons D. The epidemiology of diabetes in New Zealand: Revisit to a changing landscape. The New Zealand Medical Journal. 2006; 119(1235).
- 3. Minet L, Moller S, Vach W, et al. Mediating the effect of self-care management intervention in type 2 diabetes: a meta-anal-
- ysis of 47 randomised controlled trials. Patient education and counselling. 2010; 80(1):29–41.
- Woods K. A focus on nutrition: Key findings of the 2008/09 New Zealand adult nutrition survey.
 Wellington, New Zealand: Ministry of Health; 2011.



- Ministry of Health. The Health of New Zealand Adults 2011/12: Key findings of the New Zealand Health Survey. Wellington: Ministry of Health; 2012.
- 6. Elley CR, Kerse N, Arroll B, et al. Cost effectiveness of physical activity counselling in general practice. The New Zealand Medical Journal. 2004; 117(1207):1–15.
- Johnson M, Wood A.
 Green prescription patient survey 2015 report.
 Wellington, New Zealand:
 Ministry of Health 2015
 Contract No.: #4603-00.
- 8. Atlantis E, Joshy G,
 Williams M, Simmons D.
 Diabetes among Māori and
 other ethnic groups in New
 Zealand in: Diabetes Mellitus in Developing Countries
 and Underserved Communities: Springer; 2016.
- 9. Pringle R. Health and physical activity promotion: A qualitative examination of the effect of receiving a Green Prescription (GRx). Hamilton, New Zealand: University of Waikato. Wilf Malcolm Institute of Educational Research; 2008.
- **10.** Mane J. Kaupapa Māori: A community approach. MAI Review. 2009; 3(1):1–9.
- 11. Hudson M, Milne M,
 Reynolds P, et al. Te Ara
 Tika: Guidelines for
 researchers on health
 research involving
 Māori. Auckland: Health
 Research Council; 2010.
- 12. Kerr S, Penney L, Moewaka-Barnes H, McCreanor M. Kaupapa Māori action research to improve heart disease services in Aotearoa, New Zealand. Ethn Health. 2010:1–17.
- 13. Quick-Stats about Māori [Internet]. 2006. Available from: http://www.stats.govt.nz/census
- **14.** Smith LT. Decolonizing Methodologies: Research

- and Indigenous Peoples: Zed Books; 2012.
- 15. Wyeth EH, Derrett S, Hokowhitu B, et al. Rangatiratanga and Oritetanga: responses to the Treaty of Waitangi in a New Zealand study. Ethn Health. 2010; 15(3):303–16.
- 16. McLean G, Tobias M. The New Zealand Physical Activity Questionnaires. Report on the validation and use of the NZPAQ-LF and NZPAQ-SF self-report physical activity survey instruments. 2004.
- 17. Moy KL, Scragg RK,
 McLean G, Carr H. The
 New Zealand Physical
 Activity Questionnaires:
 validation by heart rate
 monitoring in a multiethnic
 population. Journal of
 Physical Activity & Health.
 2008; 5 Suppl 1:S45–61.
- 18. Ministry of Health. Food and nutrition guidelines for healthy adults. A background paper. Wellington: Author; 2003.
- 19. Williams MH, Rush E, Crook N, Simmons D. Te Rongoä Käkäriki: Green Prescription (GRx) programme: Green Prescription health service among Mäori in the Waikato and Ngäti Tüwharetoa rohe. MAI JOURNAL. 2015; 4(2):16.
- 20. Despres JP. Targeting abdominal obesity and the metabolic syndrome to manage cardiovascular disease risk. Heart. 2009; 95(13):1118–24.
- 21. Rush EC, Crook N,
 Simmons D. Optimal waist
 cutpoint for screening
 for dysglycaemia and
 metabolic risk: evidence
 from a Māori cohort. The
 British journal of nutrition.
 2009: 102(5):786–91.
- **22.** UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose

- control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. Lancet. 1998; 352(9131):837–53.
- 23. Holman RR, Paul SK, Bethel MA, et al. 10-Year Follow-up of Intensive Glucose Control in Type 2 Diabetes. New England Journal of Medicine. 2008; 359(15):1577–89.
- 24. Wing RR, Bolin P, Brancati FL, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med. 2013; 369(2):145–54.
- 25. Andrews RC, Cooper AR, Montgomery AA, et al. Diet or diet plus physical activity versus usual care in patients with newly diagnosed type 2 diabetes: the Early ACTID randomised controlled trial. Lancet. 2011; 378(9786):129–39.
- 26. Foley L, Maddison R, Jones Z, et al. Comparison of two modes of delivery of an exercise prescription scheme. The New Zealand Medical Journal. 2011; 124(1338):44–54.
- 27. Elley CR, Kenealy T, Robinson E, et al. Cardiovascular risk management of different ethnic groups with type 2 diabetes in primary care in New Zealand. Diabetes Research Clinical Practice. 2008; 79(3):468–73.
- 28. Coppell KJ, Kataoka
 M, Williams SM, et al.
 Nutritional intervention
 in patients with type 2
 diabetes who are hyperglycaemic despite optimised
 drug treatment-Lifestyle
 Over and Above Drugs in
 Diabetes (LOADD) study:
 randomised controlled
 trial. Bmj. 2010; 341c3337.



Whānau perceptions and experiences of acute rheumatic fever diagnosis for Māori in Northland, New Zealand

Anneka Anderson, Clair Mills, Kyle Eggleton

ABSTRACT

AIM: In New Zealand, acute rheumatic fever (ARF) remains a significant health problem with persistent ethnic inequities. Māori children 5–15 years of age in Northland have some of the highest ARF rates nationally. This study explored Māori whānau experiences of ARF, including pathways to primary healthcare and barriers and facilitators for diagnosis of ARF.

METHODS: The study applied a qualitative kaupapa Māori approach including eight whānau, two individual interviews and participant observations with 36 participants.

RESULTS: Barriers to accessing primary healthcare included: geographic distance, unavailability of appointments, cost, poor trust and rapport between health providers and whānau. Good rapport, communication and trust with health professionals facilitated utilisation of services.

Barriers to diagnosis were lack of throat swabbing and inappropriate prescription of antibiotics. Access to primary care, having health professionals follow sore throat guidelines and trust in health professionals facilitated diagnosis.

CONCLUSION: Health services could better support ARF diagnosis through the development of an effective quality improvement strategy for sore throat management, promoting free rapid response throat swabbing for high-risk populations, and exploring options of self-swabbing. Training and evaluation targeted at rapport building should also be established for health professionals to facilitate primary healthcare utilisation.

cute rheumatic fever (ARF) is a preventable inflammatory disease that can develop after pharyngitis caused by group A streptococcus (GAS) bacteria.¹ The most severe sequela is rheumatic heart disease (RHD) with mitral and/or atrial valve damage, which may require cardiac surgery and valve replacement.¹ Prevention of ARF requires early effective treatment of GAS pharyngitis with an appropriate antibiotic.² Both ARF and RHD remain significant causes of morbidity and mortality in New Zealand.³.4

ARF rates in New Zealand began to increase in the 1980s and have remained high until 2013/14.⁵ Rates of ARF in New Zealand are highest in Māori and Pasifika

children between the ages of 5–14 years.^{3,4} Incidence rates reported from 1996 to 2005 are 8.0 per 100,000 for Māori, 16.6 for Pasifika and 0.8 for New Zealand Europeans.⁶ Recent studies show these disparities are widening with increasing incidence of ARF among 5–15 year old Māori and Pasifika children.^{1,4}

Socioeconomic deprivation and household crowding are known to be associated with ARF in New Zealand.^{1,4} Most ARF cases occur within the most deprived regions of New Zealand, with the highest rates seen in Northland, South Auckland, the Bay of Plenty and Gisborne.⁴



In 2011, Northland District Health Board (NDHB) reported that 95% of ARF cases within the DHB were Māori children. Rates of ARF among Māori children in Northland, calculated from 2002–2011, were some of the highest in the country with rates of 78/100,000 per year in Māori children between 5–15 years of age compared to 4.6/100,000 per year in non-Māori.

There are few published evaluations to date of primary prevention programmes for ARF in New Zealand. However, international research supports application of targeted primary prevention within high-risk ARF areas such as Northland.9 Since 2011, the government has funded primary ARF prevention programmes in high-risk areas of New Zealand, but secondary prevention programmes focusing on secondary antibiotic prophylaxis for known ARF cases remain "the backbone of disease control".6 Apart from published audits of secondary prevention registers,10,11 little research has been undertaken to understand the perspective or experience of the person with ARF/RHD and their whānau (family group).12 Additionally, although access to healthcare has been cited as a probable cause of ARF disparities,4,11 there has been no published research into this specifically.

The project aimed to address these gaps in ARF/RHD research by exploring Māori experiences of ARF/RHD, including their pathways to primary healthcare and key barriers and facilitators for the diagnosis of ARF.

Methods

The study applied a qualitative Kaupapa Māori research design, including participant observations, whānau and individual semi-structured interviews with Māori who resided in Northland at the time of the research. Ethics approval was received from the University of Auckland Human Participants Ethics Committee in 2013.

Kaupapa Māori research

Kaupapa Māori research (KMR) has been described as a critical framework that gives meaning to the life of Māori and analyses unequal relations of power that influence Māori wellbeing. It is a methodology that is controlled by Māori to benefit Māori.¹³⁻¹⁵

These elements of KMR allow it to operate as an empowering lens that places Māori at the centre of the study and rejects cultural deficit explanations. ^{13,16}

Data collection

Participants included people who self-identified as Māori, had ARF and/or RHD and had received ARF/RHD treatment in Northland. Participants also included consenting whānau of people with ARF/RHD. Participants were recruited with the support of NDHB public health nurses (PHNs) and were identified from the NDHB Rheumatic Fever Prophylaxis Register.

KMR approaches to interactions with participants were undertaken, which focused on whakawhanaungatanga (relationship building). Whanaungatanga was established through whakapapa (family) connections and following tikanga and kawa (customs and protocols), including karakia (prayers), kai (food) and koha (acknowledgements).

Participant observation is a method that allows for first-hand accounts of people's lived experiences.¹⁷ Participant observations were undertaken with whānau for up to three days in their homes, workplaces and at community events. Data was collected in a field journal by the researcher, transcribed and analysed as described below.

Eight whānau interviews and two individual interviews were undertaken with participants. Interviews involved openended questions based around key research topics allowing for in-depth narratives. ¹⁸ Interviews were audio recorded and held in participants' homes, workplaces and community centres.

Data analysis

Data were transcribed and entered into an NVivo 10 software programme. A general inductive approach was used for data analysis. ¹⁹ Independent coding was undertaken by three researchers (two identified as Māori), then triangulated for internal validity.

Results

Participants

There were 36 participants in our study (Table 1).



Table 1: Research participants.

Whānau	Pseudonym	Sex	Age*	ARF/RHD status
1	Erena	Female	Adult	None—whānau member
	Mikaere	Male	Youth	ARF/RHD
	Hinenui	Female	Child	Suspected ARF—whānau member
2	Matire	Female	Adult	RHD
3	Rangimarie	Female	Youth	ARF
4	Mere	Female	Adult	None—whānau member
	Hone	Male	Child	ARF
	Aroha	Female	Youth	None—whānau member
	Marama	Female	Child	None—whānau member
	Tane	Male	Adult	None—whānau member
5	Huhanna	Female	Adult	None—whānau member
	Romana	Male	Child	ARF
	Hemi	Male	Adult	None—whānau member
	Maata	Female	Adult	None—whānau member
6	Manaia	Female	Adult	None—whānau member
	Kiri	Female	Child	ARF
	Moana	Female	Adult	None—whānau member
	Rawiri	Male	Adult	None—whānau member
	Roimata	Female	Child	None—whānau member
	Puti	Female	Child	None—whānau member
	Ana	Female	Adult	None—whānau member
	Tui	Female	Adult	None—whānau member
7	Tia	Female	Adult	RHD—whānau member
	Ariana	Female	Child	ARF
8	Wikitoria	Female	Adult	None—whānau member
	Tamatea	Male	Youth	ARF
	Ataahua	Female	Adult	None—whānau member
	Marika	Female	Child	None—whānau member
	Ahorangi	Female	Child	None—whānau member
	Ngaio	Female	Child	None—whānau member
	Manu	Male	Child	None—whānau member
9	Kahurangi	Female	Adult	Childhood ARF—whānau member
	Anaru	Male	Adult	None—whānau member
	Hohepa	Male	Child	ARF
10	Anahera	Female	Adult	None—whānau member
	Ngaire	Female	Child	ARF

^{*}Adult (>25 years), Youth (16–24 years), Child (<15 years).



The study identified many barriers and facilitators to accessing primary healthcare services and for the timely diagnosis of RF. These barriers related to direct and indirect costs of healthcare services, and healthcare professionals not establishing positive relationships and communication with whānau.

Good rapport, communication and trust between health professionals and whānau, along with following sore throat guidelines, facilitated utilisation of services and diagnosis.

Accessing primary healthcare Direct and indirect costs of care

Not all whānau were able to access medical care for their children when needed. Barriers to access included not being able to get appointments with general practice clinics, direct economic costs and indirect costs such as not being able to get time of work, not having access to transport, not being able to afford petrol for vehicles and geographic distance, as explained by Matire:

Yip not very often I could go out so yeah it wasn't something that my mother could just take me to the doctors, cause my mother was working, my father was working, yeah so not very often I got to go to see a doctor.

Some whānau chose to go directly to a hospital emergency department rather than seek primary care. These decisions were based on direct economic costs, their belief that it was a quicker option, and that they would most likely be referred to hospital anyway. Wikitoria explained why she adopted this strategy:

I know if I go to the doctors [GP] they're going to send me up there [hospital] anyway so I may as well just go straight up there. So I just went straight up there and, and sat there for six hours waiting for his [son's] turn.

Healthcare professionals' relationships with whānau

Healthcare professionals' attitudes and ability to create whanaungatanga (relationships) with whānau influenced whānau engagement and utilisation of health services. Whānau reported having negative experiences within primary care services that created mistrust of general practitioners (GPs). Participants described feeling as though they were inferior and were discrim-

inated against. Whānau felt their doctors judged them by where they lived and how they looked, did not listen to them, and were dismissive of their experiences and questions. Erena's narrative below describes such an interaction:

I think it's the brushing off like, "you've just got the flu" you know? It's like you're made to feel you're a bit bloody second class citizen, like that sort of sort attitude, like we don't count... I would say to him [GP] questions like "ah doctor X do you think she needs to see a specialist?" [He'll reply] "who is the qualified doctor here?" that sort of crap.

Utilisation of sore throat guidelines in primary care services

Whānau commonly cited concerns about lack of throat swabbing and under prescription of antibiotics as barriers to trusting GPs. Participants were aware that sore throats could lead to rheumatic fever and should be swabbed by health professionals. However, many participants described situations when they had presented with sore throats, asked for throat swabs and had not been given them. Whānau claimed that unless they had "pushed" they would not have had a throat swab taken, as described by Erena and her son Mikaere:

Mikaere: Yeah [the doctor] just said I had the flu, sent me home on some different drugs.

Erena: Paracetamol sent him home um, [the GP said] "see how it goes over a couple of days". But he [GP] never took a throat swab.

Mikaere: Nah he just brushed it off...

Erena: And we're aggressive, we're really aggressive when we go into the doctor's.

Mikaere: We have to be aggressive with these fellas.

Erena: But what happens if it's a whānau that isn't as experienced or aggressive as us? What happens to them? You know and their kids go undiagnosed because they're taking what the doctors are saying as law, and it's terrible care.

Facilitators to accessing primary healthcare services

Factors that facilitated access to primary healthcare and positive experiences of services were whanaungatanga, communication and trust with primary healthcare staff. Huhana stated that the relationship



between her whānau and their GP was critical in their utilisation of primary care services:

Doctor X is our GP now because he's done really well with our son, when we see him on the street [the GP says] "how's Romana [her son]?" You know? It's become like a personal relationship now, where he asks about him [Romana] and he cares about him.

Diagnosis of ARF

Delays in diagnosis

Diagnosis of ARF (from time first taken to a GP) for participants varied between an immediate diagnosis at primary care to a four-month delay and eventual diagnosis in hospital. Two RHD participants explained that their past ARF went undiagnosed until they presented with RHD symptoms. Another two children were only diagnosed with ARF when they presented at hospital with other illnesses. Delays in diagnosis occurred even when whānau suspected their children had ARF and voiced their concerns to GPs. Kahurangi had ARF as a child and her eldest son had also been diagnosed with ARF. When her youngest son began exhibiting rheumatic fever symptoms she took him to her GP several times, but as she described, he was misdiagnosed on each occasion:

A good four years before that [diagnosis of ARF] I had a fair idea that Hohepa [son] had rheumatic fever and I had been into the clinic a couple of times and I had explained to them that I was sure it was rheumatics because of the symptoms he was getting but the doc, the nurses at the clinic were telling me he had rheumatism arthritis and I was adamant that it was rheumatics because I had dealt with the symptoms before with my eldest son but they kept putting it off and kept telling me it was rheumatism arthritis and then yeah, couple years later he's diagnosed with rheumatics... I think about three times I had gone in there with him... they kept telling me it was rheumatism arthritis.

Two other whānau felt that delays in the diagnosis of their children was due to lack of knowledge and awareness of ARF by healthcare professionals, as Mikaere stated:

Because they [hospital doctors] had never seen it [ARF] before, there was only one doctor that's what I'm saying, only one doctor who knew his stuff. All the rest they were just practicing on me basically... they didn't know what they were up to.

Facilitators of diagnosis of ARF

Access to primary care, healthcare providers' knowledge and appropriate action of sore throat management were the key three factors that facilitated the diagnosis of rheumatic fever for participants. Trusting GPs to have an understanding of ARF and provide expected sore throat management influenced participants' health-seeking behaviours. Anahera's narrative illustrates positive interactions between whānau and GPs:

They [GPs] always take swabs, every time, like even with my moko [grandchild], he's two in March, even when I hear him cough, poor Dr X (laughs). I'm pretty sure he's [grandchild] too young to get rheumatics but oh he's off to the doctor to get swabbed, my poor moko you know? He, [the] doctor goes "I'm pretty sure it's not [rheumatic fever]" and I go "I don't care, I want it swabbed".

Discussion

KMR has been described by Linda Smith²⁰ as a decolonising tool to understand, analyse and address structural inequities experienced by Māori in New Zealand. Applying this critical framework enabled a non-deficit, whānau-centred approach to be undertaken through a Māori lens and worldviews. This approach highlighted how systemic structural failures of New Zealand's health system perpetuate inequitable outcomes in ARF experiences for Māori. Barriers whānau faced accessing primary health services in Northland included geographic distance, unavailability of appointments, lack of access to transport and childcare, direct and indirect costs of services, and lack of trust in health professionals due to poor management and whakawhanaungatanga by GPs. These barriers are consistent with health literature in New Zealand,^{21–23} indicating these are persistent issues not yet addressed through health policy or systematic change.

Direct and indirect costs of health services are recognised access barriers for Māori,²²⁻²⁵ and can influence delays in diagnosis of ARF/RHD. From October 2014, doctor visits and prescription medicines were free for patients under 13 years of age in Northland. From July 2015, this government initiative was extended nationally.²⁶ Despite this initiative, there are still notable inequities



in access between Māori and non-Māori due to direct and indirect costs.²⁷ The Ministry of Health 2015/16 update of the New Zealand health survey²⁷ reported that almost one quarter (24%) of children had experienced unmet need for primary healthcare during the past 12 months due to direct costs, lack of childcare, lack of transport and unavailability of health appointments. There were also significant ethnic differences reported after adjusting for age and sex differences, with Māori children 1.3 times more likely not to have accessed primary healthcare when they needed it than non-Māori children. A sub analysis of the Northland data of the New Zealand Health Survey 2011-1428 revealed similar trends with 24% of Northland children reporting unmet need for GP services over the last 12 months and citing similar barriers to seeking GP care. These inequities demonstrate the complexity of issues influencing access to healthcare. Rather than relying on a single intervention to target direct cost, a multi-pronged approach that can address multiple barriers could be a more effective strategy.

Not being able to obtain timely GP appointments is an increasingly common barrier to accessing healthcare in New Zealand. 21,29,30,31 Initiatives such as walk-in clinic appointment systems and flexible operational hours^{29,31,32} have been proposed to counter this issue. However, there are structural barriers in implementing such innovative services, mostly due to funding mechanisms of general practice and the private business model that predominates in New Zealand.33 Given the health impact and complexity of this issue, further research is needed to explore the influence of existing primary care models on health, and to investigate alternative models.

Barriers to ARF diagnosis identified in the study were the lack of throat swabbing and inappropriate prescription of antibiotics. Many whānau were not given throat swabs or prescriptions for antibiotics even when presenting to GPs with sore throats, requesting throat swabs and disclosing histories of rheumatic fever. These experiences indicate negative perceptions by whānau of GPs within Northland and influenced whānau decisions to bypass primary care services in favour of secondary care.

Best practice guidelines for sore throat management in New Zealand² recommend that throat swabs are undertaken and patients are started on appropriate antibiotic treatment if the patient is deemed at 'high risk' of developing rheumatic fever. All whānau members who presented to GPs with sore throats in this study would have met at least two of these high-risk criteria, demonstrating that sore throat guidelines are not consistently followed in general practice within Northland. These findings are supported by a recent study undertaken assessing adherence of school-based sore throat programmes and GPs in Northland to national guidelines for the management of laboratory-proven GAS.34 The study found that one in five children presenting to general practices with positive throats swabs did not receive treatment regimens recommended by the guidelines.

A number of reviews show that guidelines often only have moderate effectiveness in improving clinical outcomes or changing process of care.^{35–37} However, evidence suggests that an effective quality improvement strategy including: audit and feedback, computerised advice, point of care reminders, practice facilitation, educational outreach and processes for patient review and follow-up can overcome health provider barriers.³⁸ Given the experiences of whānau in this study, we recommended that such a strategy be developed.

Our findings support other ways of increasing access for whānau to high-quality sore throat management, including the 'rapid response' free at point of care access currently being implemented for children at high risk in Northland in pharmacies and schools, and potentially other community venues. Recent research suggests that self-swabbing or swabbing by parents/caregivers is non-inferior to swabbing by health professionals for GAS detection. Self-swabbing may overcome some barriers whānau experience in accessing primary care. The feasibility of implementing self-swabbing requires further research.

One of the greatest facilitators of positive experiences for whānau within Northland's primary healthcare system was health providers' ability to establish trust and whanaungatanga with whānau. The impact



of good rapport, communication and trust with health professionals has been well established in New Zealand, with greater rapport promoting increased patient satisfaction. ^{24,25,40} Declining confidence and trust in GPs was reported in the 2015/16 New Zealand Health Survey with adults who reported no confidence and trust at all in their GP increasing from 2.1% in the 2011/12 Health Survey to 3.4% in 2015/16 Health survey. ²⁶ Training and evaluation targeted at rapport building should be established for health professionals to facilitate healthcare utilisation and merits further research.

This study provided a whānau-centred context to ARF/RHD research in New Zealand, demonstrating how experiences and narratives of those who suffer

from disease are important. Presenting patient^{21,25,30,32} and whānau voices⁴¹ within health contexts is an approach not yet utilised within ARF/RHD literature.

The study included a relatively small sample size, and therefore may not represent the diversity of whānau experiences. Selection bias may have also occurred during recruitment.

Despite these potential weaknesses, the research provides a beginning point to inform ARF/RHD prevention approaches in Northland from a qualitative, KMR methodology. This framework can be applied to future research looking at experiences of ARF/RHD in New Zealand to provide a fuller understanding of these challenging issues.

Competing interests:

Nil.

Acknowledgements:

The authors would like to express their gratitude to all of the whānau who were involved in the research. The team also acknowledges the huge contribution and support offered to the study from the Public Health Nurses, Kaunihera o Kaumātua o Te Poutokomanawa of NDHB. Finally, we would like to thank our research funder—The University of Auckland.

Author information:

Anneka Anderson, Lecturer, Te Kupenga Hauora Māori, University of Auckland, Auckland; Clair Mills, Medical Director, Medecins Sans Frontieres (MSF), Paris, France; Kyle Eggleton, General Practitioner, Ki A Ora Ngātiwai and Te Kupenga Hauora Māori, University of Auckland, Auckland.

Corresponding author:

Anneka Anderson, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Private Bag 92019, Auckland 1142.

a.anderson@auckland.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7411

REFERENCES:

- Milne RJ, Lennon DR, Stewart JM, Vander Hoorn S, et al. Incidence of acute rheumatic fever in New Zealand children and youth. J Paediatr Child Health. 2012; 48(8):685–691.
- 2. Lennon D, Peat B, Kerdemelidis M, Sharpe N, et al. New Zealand Guidelines for Rheumatic Fever Group A Streptococcal Sore Throat Management
- Guideline: 2014 Update. Auckland: Heart Foundation New Zealand; 2014.
- Jaine R, Baker M, Venugopal K. Epidemiology of acute rheumatic fever in New Zealand 1996–2005.
 J Paediatr Child Health. 2008; 44(10):564–71.
- Webb R, Wilson N.
 Rheumatic fever in New
 Zealand. J Paediatr Child
 Health. 2013; 49(3):179–184.
- 5. Ministry of Health. Progress on the Better Public Services rheumatic fever target. Wellington: Ministry of Health. Available at http://www.health.govt.nz/about-ministry/whatwe-do/strategic-direction/better-public-services/progress-better-public-services-rheumatic-fever-target [verified at 27 April 2017].



- Jaine R, Baker M, Venugopal K. Acute Rheumatic Fever associated with household crowding in a developed country. Pediatr Infect Dis J. 2011; 30(4):315–319.
- 7. Northland District Health Board. Rheumatic Fever Prevention Plan 2013-2017. Whangarei: Northland District Health Board; 2013.
- 8. Robin A, Mills C, Lennon D, Tuck R. The Epidemiology of Rheumatic fever in Northland, 2002–2011. NZ Med J. 2013; 126(1373):46–52.
- 9. Kerdemedlidis M, Lennon DR, Arroll B, Peat B, et al. The primary prevention of rheumatic fever. J Paediatr Child Health. 2010; 46:534–48.
- 10. Grayson S, Horsburgh M, Lennon D. 2006. An Auckland regional audit of the nurse-led rheumatic fever secondary prophylaxis programme. NZ Med J. 119(1243):51–57.
- 11. Atatoa-Carr P, Bell
 A, Lennon DR. Acute
 rheumatic fever in the
 Waikato District Health
 Board region of New
 Zealand: 1998–2004. NZ
 Med J. 2008; 121(1285):96.
- 12. New Zealand Guidelines Group. RapidE: rheumatic fever. A systematic review of the literature on health literacy, overcrowding and rheumatic fever. Wellington: Ministry of Health, 2011.
- 13. Walker S, Eketone A, Gibbs A. 2006. An exploration of Kaupapa Māori research, its principles, processes and applications. Int J Soc Res Methodol. 9(4):331–344.
- 14. Mahuika R. Kaupapa Māori Theory is critical and anti-colonial. MAI Review. 2008; 3(4):1–16.
- **15.** Smith GH. Protecting and respecting indigenous

- knowledge. In Battiste M editor. Reclaiming Indigenous voice and vision. Canada: UBC Press. 2000. p.209–224.
- **16.** Barnes HM. Kaupapa Māori: Explaining the ordinary. Pacific Dialog. 2000; 7(1):13–16.
- 17. Emerson RM, Fretz RI, Shaw LL. Writing Ethnographic Fieldnotes. Chicago: The University of Chicago Press; 1995.
- 18. Angrosino MV. Doing Cultural Anthropology: Projects for Ethnographic Data Collection. Illinois: Waveland Press Inc; 2002.
- Thomas D. A general inductive approach for analysing evaluation data. AM J Evaluation. 2006; 27(2):237–246.
- 20. Smith LT. Keeping a decolonising agenda to the forefront. In Hutchings J, Lee-Morgan J, editors. Decolonisation in Aotearoa: Education, Research and Practice. Wellington: NZCER Press; 2016. P.ix-x.
- 21. Anderson A. Understanding migrants' primary health care utilisation in New Zealand through an ethnographic approach. Divers Health Soc Care. 2008; 5(4):291–301.
- 22. Crengle S, Lay-Yee R, Davis P, Pearson J. A comparison of Māori and non-Māori patient visits to doctors:
 The National Primary Medical Care Survey (NatMedCa) 2001/02. Report 6. Wellington: Ministry of Health/Manatū Hauora.
- 23. Jatrana S, Crampton P. Primary health care in New Zealand who has access? Health Policy. 2009; 93(2009):1–10.
- 24. Jansen P, Smith K. Māori experiences of primary health care: breaking down barriers. NZ Fam Prac, 2006; 33(5):298–300.

- 25. Kerr S, Penney L,
 Moewaka Barnes H,
 McCreanor T. Kaupapa
 Māori Action Research
 to improve heart disease
 services in Aotearoa, New
 Zealand. Ethn Health.
 2010; 15(1):15–31.
- 26. Ministry of Health.

 Zero fees for under 13s.

 Wellington: Ministry of
 Health; 2017. Available
 at http://www.health.
 govt.nz/our-work/
 primary-health-care/
 primary-health-caresubsidies-and-services/
 zero-fees-under-13s
 [Verified 13 January 2017].
- 27. Ministry of Health. Annual Update of Key Results 2015/16: New Zealand Health Survey. Wellington: Ministry of Health; 2016.
- 28. Rumball-Smith J. NZ
 Health Survey 2011-2014,
 Te Tai Tokerau. Whangarei: Northland District
 Health Board; 2015.
- 29. Ludeke M, Puni R, Cook L, Pasene M, Abel G, et al. Access to general practice for Pacific peoples: a place for cultural competency. J Prim Health Care. 2012; 4(2):123–130.
- **30.** Lee R, North N. Bariiers to Māori sole mothers' primary health care access. J Prim Health Care. 2013: 5(4):315–321.
- 31. Eggleton K, Penney L, Moore J. Measuring doctor appointment availability in Northland general practice. J Prim Health Care. 2017; 9(1):56–61.
- 32. Slater T, Matheson A,
 Davies C, Tavite H, et al.
 'It's whanaungatanga
 and all that kind of stuff'.
 Māori cancer patients'
 experiences of health
 services. J Prim Health
 Care. 2013: 5(4):308–314.
- **33.** Finlayson MP, Sheridan NF, Cumming JM, Fowler S. The impact of funding changes



- on the implementation of primary health care policy. Prim Health Care Res Dev. 2012; 13(2):120–9.
- 34. Shetty A, Mills C, Eggleton K. Primary care management of group A streptococcal pharyngitis in Northland. J Prim Health Care. 2014; 6(3):189–194.
- 35. Lugtenberg M, Burgers JS, Westert GP. Effects of evidence-based clinical practice guidelines on quality of care: A systematic review. Qual Saf Health Care.2009; 18(5):385–92.
- **36.** Grimshaw JM, Freemantle N, Wallace S, Russell I,

- et al. Developing and implementing clinical practice guidelines. Qual Health Care. 1995; 4:55–64.
- 37. Grimshaw JM, Russell IT. Effect of clinical guidelines on medical practice: A systematic review of rigorous evaluations. Lancet. 1993; 342(8883): 1317–1322.
- 38. Irwin R, Stokes T, Marshall T. Practice-level quality improvement interventions in primary care: a review of systematic reviews. Prim Care Res Dev. 2015; 16(6):556–577.
- **39.** Murray MA, Schulz LA, Furst JW, Homme JH, et

- al. Equal performance of self-collected and health care worker-collected pharyngeal swabs for group a streptococcus testing by PCR. J Clin Microbiol. 2015; 53(2):573–8.
- **40.** Jansen P, Smith K. Māori experiences of primary health care: Breaking down the barriers. NZ Fam Physician. 2006; 33(5):289–300.
- 41. Jones B, Ingham T, Davies C, Cram F. Whānau Tuatahi: Māori community partnership research using a kaupapa Māori methodology. MAI review. 2010; 3:1–14.



Audit on first seizure presentation to Taranaki Base Hospital: a secondary centre experience

Sean Lance, Rajesh Kumar

ABSTRACT

BACKGROUND: Management of first seizure should be based on treating the underlying cause and tailoring investigations to identify those patients at high risk of recurrence.

AIM: To establish the incidence of first seizure presentation to Taranaki Base Hospital and investigate the management of these patients.

METHOD: A retrospective audit was performed identifying patients presenting to Taranaki Base Hospital from 1 January 2015 to 31 December 2015 with a first seizure.

RESULTS: Thirty-seven patients presented with their first seizure with 50% found to have an easily reversible precipitant. Forty-three percent had a history of previous brain insult and 52% had an abnormality identified on neuroimaging. Only 14% received formal neurology follow-up and only 8% had electroencephalography. Forty-three percent received chronic antiepileptic drug therapy and 27% had a recurrent seizure within 12 months. Only 43% had documented driving advice.

CONCLUSIONS: The incidence of first seizure presentation to Taranaki Base Hospital is similar to worldwide data. In general, patients receive basic investigations in keeping with international guidelines. This audit has helped to identify a number of areas to address with the current service provision, including ways to improve access to important investigations and ways to develop a guideline to standardise care.

eizures are a common symptom encountered in emergency departments regularly. Seizures may represent a diagnosis of epilepsy but can also be a symptom of a wide range of medical illnesses, along with medication or substance effects.

Traditionally, the diagnosis of epilepsy relied on the patient having at least two unprovoked seizures more than 24 hours apart. This definition was revised in 2014 by the International League against Epilepsy (ILAE) to include:

- those patients with a single unprovoked seizure and a probability of further seizures similar to the general recurrence risk after two unprovoked seizures (ie, at least 60%) occurring over the next 10 years;
- and those patients with a diagnosis of an epilepsy syndrome.¹

Seizures are dangerous and life-threatening so establishing the cause or making a diagnosis of epilepsy with the appropriate management thereafter is essential. There are also significant ongoing social and occupational implications associated with the diagnosis (or lack thereof).

Worldwide incidence of a single unprovoked seizure is approximately 23 to 61 per 100,000 per year.² Approximately 8–10% of the population will have a seizure in their lifetime with 2–3% of them developing epilepsy.³

After an unprovoked first seizure, recurrence (without treatment) is estimated to be 21–45% after two years with the highest risk being immediately after the initial seizure. A variety of factors increase this risk, including electroencephalography (EEG) with epileptiform abnormalities,



previous central nervous system (CNS) insult (eg, stroke, brain tumour, head injury) or abnormal CNS imaging.⁴

A guideline from the American Academy of Neurology (AAN) from 2007 outlines the standard of care for investigating a patient with an unprovoked first seizure. This is also reviewed in the Journal of the American Medical Association in 2016. They recommend:

- All patients should have neuroimaging—up to 30% have potentially significant abnormalities detected^{3,5}
- Outpatient EEG has a yield for epileptiform abnormalities of 29%^{3,5}
- Routine screening for metabolic abnormalities (eg, hyponatraemia) and drug intoxication has been proposed and invariably occurs, but there is a lack of evidence as to its utility. Likewise, lumbar puncture is not recommended as a routine investigation ^{3,5}

Following a subsequent unprovoked seizure, the risk of recurrence is substantially higher (57% by one year and 73% by four years). In these patients, it is well established that antiepileptic drug (AED) therapy is beneficial in terms of reducing seizure recurrence, inducing remission and improving quality of life.

Management following a first unprovoked seizure is somewhat less clear.

The AAN guidelines on management of an unprovoked first seizure in adults from 2015 reflect this. These guidelines identified a number of patient groups at higher risk of recurrence after an unprovoked first seizure compared to those without:

- prior brain insult RR 2.55 (95% CI 1.44–4.51)
- patients with epileptiform EEG abnormalities RR 2.16 (95% CI 1.07–4.38)
- patients with abnormal brain imaging RR 2.44 (95% CI 1.09–5.44) 4

The guidelines highlight that immediate AED therapy significantly reduces the risk of recurrent seizure; however, this is not accompanied by improved rate of seizure remission in the long term (>3 years) or an improvement in quality of life. Additionally, 7–31% of patients experience side effects from AEDs. ⁴

These guidelines mirror the shift in focus of epilepsy diagnosis to the more practical definition from the ILAE where those patients with unprovoked first seizures who are judged to have a risk of recurrence of more than 60% (ie, similar to the 57% recurrence risk after a second seizure) warrant upfront and immediate AEDs.^{1,4}

Additional recommendation made from the National Institute of Clinical Excellence (NICE) guidelines from 2012 state that all adults presenting with a first seizure should be seen by a specialist in epilepsy as soon as possible.⁶

In New Zealand, a recent study by Joshi et al in 2015 identified a disparity in the access to care between patients in the Wellington region attributed to the hospital they presented to—either Wellington hospital with an established tertiary level neurology department, or Hutt hospital, which has neurology care provided by visiting neurologists from Wellington.7 They found that patients presenting with seizures were much more likely to be referred to the neurology service if they presented to Wellington hospital (52%) compared to Hutt hospital (13.4%). This difference was even more marked when examining for first seizure presentation where 63% were referred to neurology from Wellington, whereas only 9.8% were referred from Hutt hospital.7

Taranaki DHB (Taranaki Base Hospital (TBH) and Hawera Hospital), serves a population of 118,110 over a very wide area (7,948 km²). The Taranaki population is slightly older than the New Zealand population as a whole (36.9% over age 50 compared to 33.9%) with a higher proportion of Māori and lower proportion of Pacific Islanders compared to the rest of New Zealand (see Table 1).8 Taranaki Base Hospital is a secondary level hospital in New Plymouth with tertiary care being provided by a number of different DHBs dependent on the specialty. Tertiary level neurology care is provided by Auckland DHB with visiting neurologists from attending approximately once per month, usually for two days of clinics which tend to be severely overbooked. There is no EEG service in Taranaki and patients travel to either Waikato or Manawatu for this. Acute inpatient care is provided by general physicians.



Anecdotal experience indicates that the management of patients presenting with first seizure to Taranaki DHB is inconsistent and not in keeping with international guidelines. With ever-increasing modernisation of healthcare delivery by ways such as telemedicine, geographical constraints should no longer detriment patients' care.

This audit will establish a baseline set of data to identify problematic areas and ways in which to bring the care of patients presenting with first seizures into line with international guidelines.

Aims

The aim of this audit was to investigate the current incidence of first seizures presenting to Taranaki Base Hospital (TBH). Additionally, the audit aimed to investigate the management of these patients and compare to current guidelines. We also assessed the rates of documentation of safety advice and events and suggest areas for improvement and development.

Methods

This study was designed as a retrospective audit over a 12-month period from 1 January 2015 to 31 December 2015.

A list of NHI numbers were obtained from patients over this period who presented

Figure 1: Selection process.

to TBH with the diagnosis of "seizure", "convulsion" or "epilepsy".

Following this, the investigator examined the electronic records (discharge summaries, admission notes, results) and the hard copy referral letters and medical records. Data were collected on a standard Excel spreadsheet.

Inclusion and exclusion criteria were defined:

- Inclusion
 - Age >16
 - Documented history consistent with seizure—as determined by the primary investigator
- Exclusion
 - Age <16
 - Previous seizure
 - Non seizure

Results

One hundred and twenty-five patients were included in the initial data collection. Twenty-two patients (18%) were excluded as they were <16 years old, 31 (25%) excluded because it was deemed not to be a seizure (most commonly syncope), and 35 (28%) were excluded because it was not a first seizure. This left 37 patients (30%) included for further investigation.

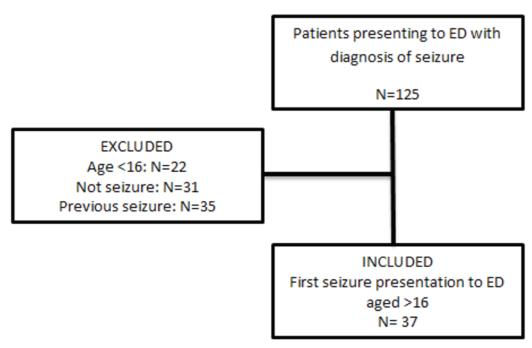




Table 1: Demographic data.8

Ethnicity	N (%)	DHB data (%)	NZ data (%)	
NZ European and other	26 (70.3)	79.9	77.7	
Māori	11 (29.7)	18.9	15.8	
Pacific Island	0 (0)	1.2	6.5	

There was a roughly 2:1 split between males (62%, n=23) and females (38%, n=14). Mean age was 57.5 years, median was 58 years, with a standard deviation of 23.5 and interquartile range of 38.

Forty-three percent (n=16) had documented evidence of prior brain insult, including stroke (n=5), dementia (n=4), intracranial lesions (n=3), head injury (n=3) and previous encephalitis (n=1).

Potential reversible precipitants for seizures were found in 50% of patients:

- 11% (n=4) had seizures after exposure to drugs known to lower seizure threshold either in overdose or newly prescribed
- 14% (n=5) had seizures caused by illicit substances
- 22% (n=8) had seizures related to alcohol (five intoxicated and three in withdrawal)
- 3% (n=1) had a seizure associated with fever (negative lumbar puncture)

Most patients were recorded to have had generalised convulsive seizures (71%). Eighteen percent had focal seizures and in 11% the seizure type was unknown.

All patients received basic bloods including full blood count, renal function and electrolytes.

Neuroimaging was performed in the majority (97%) of patients. Most (92%) had CT; all were performed acutely on the day of presentation. MRI was done 32% of patients: in two cases instead of CT, and in addition to CT in 10 patients. Ten were done as inpatients with the average wait for MRI of 2.4 days (range 0–7 days), and two were as outpatients with the average wait of 12.5 days (range 11–14 days).

Of those that had neuroimaging, 52% had abnormal scans. Strokes (43%), atrophy (29%) and masses (29%) made up the abnormalities seen on CT. Strokes (38%) and masses (38%) were the most common abnormality.

mality on MRI. In those patients that had both modalities, 30% had their findings only demonstrated with MRI.

Table 2:

Manage	Management			
None	None			
Medical	Medical therapy			
• Acı	ıte	16 (70)		
•	Benzodiazepines	10		
•	IV antiepileptic	6		
	 Phenytoin 	4		
	 Sodium Valproate 	2		
• Chi	onic antiepileptic therapy	16 (70)		
•	Phenytoin	4 (25)		
•	Sodium Valproate	4 (25)		
•	Levetiracetam	3 (19)		
•	Lamotrigine	2 (13)		
•	Carbamazepine	1 (6)		
•	Levetiracetam + Lamotrigine	2 (13)		
Acute m	nedical admission	29 (78)		
Neurolo	Neurology follow-up			
EEG	3 (8)			
Driving	16 (43)			
Recurre	10 (27)			

Thirty-eight percent (n=14) of patients did not receive any medical therapy for their seizures. Table 2 details the treatments received by those patients that did.

Management was predominantly inpatient-based with 78% admitted under general medicine.

Fourteen percent (n=5) of patients had formal neurology follow-up. The mean wait time for follow-up was four months. Only 8% (n=3) were referred for EEG, of which one was abnormal.

Less than half (48%) of the total study population had specific driving advice documented.



Discussion

This audit was designed to investigate the incidence of first seizure presentation to the emergency department at Taranaki Base Hospital and to establish how these patients are managed.

The incidence is similar to worldwide data: approximately three per 10,000 per year.

The population group is small and also only captures those patients who present to ED, therefore missing those not presenting through this pathway.

The approach to these patients by medical staff was fairly standard. Although not endorsed by guidelines, all patients had routine bloods.

Most patients (97%) had neuroimaging with the majority having CT. This is close to the goal of neuroimaging of all patients with first seizure as suggested by the AAN guidelines.⁵ Fifty-two percent had abnormal neuroimaging, which is in keeping with the findings from the AAN guidelines, which comment on an average yield for an abnormal finding of 15%, but with a wide range from 1-57%.5 The wide range would be expected given the significant potential for difference in interpretation of the imaging and findings. MRI was used more sparingly and not surprisingly had a higher sensitivity with 30% of patients having significant findings only identified on MRI. All CTs were completed acutely via the ED. The wait time to MRI for these patients is excellent (2.4 days for inpatient and 12.5 days for outpatient). Given that most of the patients were admitted, it is promising that these patients receive their investigations promptly. 12.5 days for an outpatient MRI is also impressive, but note must be made of the small sample size (n=2) very likely resulting in an underestimate of the true time period.

The majority (78%) were admitted to the general medical service for further investigation and management; however, 30% did not require any acute medical therapy and so may be more appropriate for outpatient management, which could be facilitated by development of a clear guideline.

Forty-three percent of patients (n=16) received ongoing medical therapy with AEDs. Most (88%) were with single agents. Phenytoin was the equal most commonly

prescribed antiepileptic for ongoing medical therapy. This may reflect the high use of phenytoin in the acute setting (two-thirds of patients being treated with IV antiepileptics received IV phenytoin), which arguably is even more concerning with the increased risk of side effects and safer alternatives. This likely also reflects clinicians' familiarity with the drug given its long history of use and non-epilepsy specialists providing the bulk of the care.

The percentage that received ongoing AEDs is similar to both the percentages of patients with previous brain insults (43%) and abnormal imaging (52%), which suggests that there is an appropriate assessment of seizure recurrence risk with the resources available. Additionally, of those with abnormal neuroimaging, 76% (n=13) went on to receive chronic AED therapy, highlighting the influence and importance of this investigation.

Of the 19% (n=7) of patients identified as having unprovoked seizures with no significant history and normal neuroimaging, only one went on to have an EEG. AAN guidelines suggest EEG as a routine investigation following a first seizure as it can provide additional prognostic information to justify further AED therapy.5 The lack of a local EEG facility presents a significant barrier for these patients and clinicians may be less inclined to request the investigation due to these resource constraints. Additionally, only 14% of patients received any follow-up through the neurology service. Again, this represents a significant barrier related to the service in Taranaki as there are only visiting neurologists who are already overbooked. Clearly a local epilepsy specialist would help to address this problem, but other considerations should be made for novel ways to address this problem such as telemedicine and virtual clinics from the already established visiting neurologists.

The New Zealand Transport Associations guidelines state that patients with a solitary seizure should be managed in the same manner as those with established epilepsy with a stand-down period for 12 months (that can be reduced to six months if endorsed by specialist) unless there is exceptional circumstances such as a clearly identified provoking cause for the seizure. 9 Regardless of seizure aetiology, only 48%



had documented advice regarding driving either in the clinical notes or the discharge summary. Documentation of premorbid driving status was globally absent. When excluding those patients who died in hospital and those presumed not to drive (ie, were rest home residents), 35% of the total still lacked driving documentation.

There are a number of limitations to this audit. A retrospective design means that collection of data relies solely on adequate documentation introducing information bias. There is potential for investigator error and bias when collecting the data, which was done by examination of old written and electronic records. Inclusion/exclusion of patients was especially vulnerable to error—both from the initial treating clinician and also the interpretation of the information in the notes as determined by a single investigator. Of those excluded, they had alternative diagnoses but it is very difficult to corroborate and confirm these retrospectively. Importantly, none of these patients were subsequently diagnosed with seizures in the next 12 months in Taranaki. Another example is with regard to driving advice and its documentation—the findings may reflect poor documentation by clinical staff rather than a true finding.

Conclusions and recommendations

First seizure presentations to Taranaki Base Hospital occur at a similar rate to worldwide data. Initial investigation and management is broadly in keeping with current guidelines, but there are areas in which to improve patient care.

This audit can help in the development of a specific pathway for management of a patient with a first seizure with clear guidelines on investigations both in the acute phase and following discharge from hospital. Dedicated education sessions to promote this proposed pathway will help to familiarise staff with its use and serve as a forum to troubleshoot any initial or ongoing issues.

Specific aspects to address would be to ensure all patients have neuroimaging and particularly important is identification of those high-risk patients and consideration of commencing antiepileptic therapy when appropriate. Generating an evidence-based guideline for choice of antiepileptic therapy would also be an easy way to simplify management and ensure appropriate care.

Streamlining of the referral process for EEG and aiming to remove the perceived barriers to its request is important going forward, as is referral to the neurology service. This may come in the form of virtual clinics or consideration of other alternatives such as combining their neurological consultation with their EEG consultation in another hospital. Development of a subspecialty interest in epilepsy (ideally in general neurology) by a general physician should also be encouraged and facilitated in order to further reduce the load on the visiting specialists.

Improvement of documentation of driving advice is very important going forward, with the recommendation for an information sheet detailing safety factors and other information about seizures to be developed along with a way to document its receipt by the patient (one option is to have a removable sticker on the information sheets that is then transferred to the patient's notes).

Further audit following the implementation of these changes is essential. Ideally this would be best done in a prospective manner in order to improve and eliminate some of the aforementioned limitations involved.



Competing interests:

Nil.

Author information:

Sean Lance, Medical Registrar, Taranaki Base Hospital, New Plymouth; Rajesh Kumar, General Physician, Taranaki Base Hospital, New Plymouth.

Corresponding author:

Sean Lance, Department of Medicine, Capital & Coast District Health Board, Wellington. sean.lance@ccdhb.org.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7412

REFERENCES:

- Fisher RS, et al. A practical clinical definition of epilepsy. Epilepsia 2014; 55(4):475–82.
- 2. Hauser W, Beghi E. First seizure definitions and worldwide incidence and mortality. Epilepsia 2008; 49(1):8–12.
- 3. Gavvala JR, Schuele SU. New-Onset Seizure in Adults and Adolescents. A Review. JAMA 2016; 316(24):2657–2668.
- 4. Krumholz A, et al.
 Evidence-based guideline: Management of an
 unprovoked first seizure
 in adults. Report of the
 Guideline Development
 Subcommittee of the American Academy of Neurology
 and the American Epilepsy

- Society. Neurology 2015; 84(16):1705–1713.
- 5. Krumholz A, et al. Practice Parameter: Evaluating an apparent unprovoked first seizure in adults (an evidence-based review). Report of the Quality Standards Subcommittee of the American Academy of Neurology and the American Epilepsy Society. Neurology 2007; 69(21):1996–2007.
- G. National Institute of Clinical Excellence (NICE) guidelines 2012 (last updated Feb 2016). Epilepsies: diagnosis and management. Guidance and guidelines. NICE online. Available at http://www.nice.org.uk/guidance/cg137. Accessed May 2017.

- Joshi, et al. Inequities in provision of seizure care across the Wellington region. NZMJ 2015; 128(1417):30–35.
- 8. Ministry of Health New Zealand website. Population of Taranaki DHB. Ministry of Health NZ. Available at http://www.health.govt.nz/new-zealand-health-system/my-dhb/taranaki-dhb/population-taranaki-dhb. Accessed May 2017.
- Medical aspects of fitness to drive: A guide for health practitioners. New Zealand Transport Agency. Published June 2014. Accessed 16th August 2016. www.nzta.govt.nz/assets/ resources/medical-aspects/ Medical-aspects-2014.pdf



Achieving health equity in Aotearoa: strengthening responsiveness to Māori in health research

Papaarangi Reid, Sarah-Jane Paine, Elana Curtis, Rhys Jones, Anneka Anderson, Esther Willing, Matire Harwood

ABSTRACT

Excellent health research is essential for good health outcomes, services and systems. Health research should also build towards equity and in doing so ensure that no one is left behind. As recipients of government funding, researchers are increasingly required to demonstrate an understanding of their delegated responsibilities to undertake research that has the potential to address Māori health needs and priorities. These requirements form the basis of responsiveness to Māori in health research, and several research institutions have implemented systems to support their organisational approach to this endeavour. However, many health researchers have a narrow view of responsiveness to Māori and how it might be relevant to their work. In this viewpoint paper we provide an overview of existing frameworks that can be used to develop thinking and positioning in relation to the Treaty of Waitangi and responsiveness to Māori. We also describe an equity-based approach to responsiveness to Māori and highlight four key areas that require careful consideration, namely: (1) relevance to Māori; (2) Māori as participants; (3) promoting the Māori voice, and; (4) human tissue. Finally, we argue for greater engagement with responsiveness to Māori activities as part of our commitment to achieving equitable health outcomes.

ealth research has an extensive reach into health practice from evidence-based medicine and clinical trials through to systems monitoring and data reporting. As a result, health professionals are required to adhere to the policies, protocols and ethical parameters associated with research in Aotearoa New Zealand. Inherent within these processes are responsibilities for and responsiveness to Māori health development.

What is responsiveness to Māori?

Responsiveness to Māori reflects the Government's view that health research conducted in New Zealand should contribute to improving Māori health and eliminating health inequities. Researchers must therefore consider how their processes can better reflect Māori health needs and priorities. Responsiveness to Māori recognises the Government's accountabilities under the Treaty of Waitangi, which flow on to research organisations

receiving government funding. The Crown expects these accountabilities to be made transparent and they are explicit in administration agreements between research funders and providers.

Health researchers are required to demonstrate an understanding of these delegated responsibilities, including whether the research:

- is a strategic priority for Māori;
- makes the most of opportunities to inform the elimination of ethnic inequities;
- incorporates traditional or contemporary Māori processes;
- supports Māori development, including workforce development;
- team has any explicit relationships with Māori, and;
- actively protects Māori rights, including cultural and intellectual property rights.



Health researchers must also consider a range of Māori expectations,^{4–7} including:

- that researchers respect and uphold the Treaty of Waitangi;
- that the research will impact positively on Māori and improve Māori health:
- that Māori rights and interests, including Māori ethical principles, are best protected through Māori involvement in research governance;
- that researchers will invest in research processes that facilitate greater communication and transparency; and,
- that accountability to Māori is demonstrated through sound reporting mechanisms and consultation-to-dissemination pathways.

Approaching responsiveness to Māori in health research

A number of 'Responsiveness to Māori' frameworks are available to health researchers such as those used by the Waitangi Tribunal and the Ministry of Health (Table 1). Both position the Treaty of Waitangi at the forefront of health research in New Zealand with the Waitangi Tribunal emphasising the Crown's role in upholding and protecting Māori rights and the delegation of these responsibilities to health researchers funded from government agencies. In addition, some iwi have developed their own frameworks and criteria for assessment of research to be conducted within their regions and/or with their people (eg, Ngati Porou Hauora and Ngai Tahu Research). Regardless of the source, frameworks are most effective for

Table 1: Summary of Treaty of Waitangi frameworks and responsiveness to Māori.

Framework	Principles	Application to responsiveness to Māori in research
Waitangi Tribunal	Partnership	The Treaty requires each party to act with the utmost good faith towards the other. It includes the duty to consult with Māori and obtain the full, free and informed consent.
Treaty Principles	Reciprocity	The partnership is reciprocal for mutual advantage and benefit.
Timespies	Autonomy	The Crown guaranteed to protect Māori autonomy in recognition of the promises of kawanatanga and tino rangatiratanga, including Māori rights to determine Māori processes and priorities.
	Active protection	The Crown's duty to protect Māori rights and interests. The duty is not passive but active and requires honourable conduct, full consultation and, where appropriate, decision-making by those whose interests are to be protected.
	Options	Māori have options stemming from both traditional/customary practices and modern possibilities.
	Mutual benefit	The Treaty was signed for mutual benefit and Māori were to retain resources to ensure the colonisation of New Zealand was not detrimental.
	Equity	The obligations that require the Crown to act fairly so that Māori were/are not disadvantaged. Where Māori have been disadvantaged, the Crown is required to take active measures to restore the balance.
	Equal treatment	Requires the Crown to act fairly between Māori groups.
	Redress	Where the Crown has acted in breach of its obligations and Māori have suffered prejudice, the Crown has a clear duty to set matters right. In respect of historical grievances, this usually requires compromise on both sides and redress should not create a fresh injustice.
Ministry of Health—He Korowai Oranga	Partnership	Working with Māori individuals and communities to develop strategies for Māori health gain and access to appropriate services.
	Participation	Requires Māori involvement in all levels of the health and disability sector from delivery to planning and decision-making.
	Protection	Involves the Crown working to ensure Māori health equity and safeguarding Māori cultural concepts, values and practices.

Sourced from:

http://www.nph.org.nz/our-services/research-and-evaluation/

http://www.ngaitahuresearch.co.nz/about/

http://www.waitangitribunal.govt.nz/treaty-of-waitangi/principles-of-the-treaty/



responsiveness to Māori if they are incorporated in a comprehensive manner.

An equity-based approach to responsiveness to Māori

Responsiveness to Māori in research is not new^{8,9} and many institutions have implemented systems to support their organisational approach. Others promote equity as a starting point for responsiveness to Māori as this focus requires researchers to consider Māori health priorities based on inequities, develop appropriate relationships with Māori and commit to undertaking research that mitigates rather than extends health inequities. An equity-based approach encourages health researchers to consider responsiveness to Māori in relation to four main areas:

1. Relevance to Māori

Research that seeks to improve Māori health and reduce inequities is a Government priority.¹ Thus, researchers need to establish whether the topic is important for Māori health and/or whether inequities exist. Opportunities to enhance relevance to Māori include:

a. Consultation with Māori

Consultation with Māori is a fundamental obligation of Treaty responsiveness, and many researchers engage in this process. The Treaty Principles focus on quality relationships with Māori and acting with the utmost good faith. Researchers ought to consider and reflect on all of the different layers of research relationships they have with Māori, including as colleagues, students, advisors, partners, governors and participants. Consultation requires respectful information sharing and dialogue; it is not a one-way conversation or an opportunity for researchers to tell Māori what they want or need. Furthermore, consultation is very context-specific, thus some projects will require more in-depth consultation strategies than others.10

b. Dissemination

This goes hand-in-hand with consultation. It closes the consultation loop and as such it is an important standard of 'good faith'. Ideally, the project should be part of the development of a research relationship and the feeding back of results provides an opportunity to discuss further action. Dissemination to a broader Māori audience

should be considered as part of the consultation process, and worked towards as part of the research.

c. Enabling relationships with Māori individuals and communities

Good relationships can be mutually beneficial and enabling to both researchers and Māori. Ideally researchers should invest in and start this process during the conception of a research project and well in advance of research deadlines. Successful interactions happen when researchers engage in genuine, respectful and mutual relationships with Māori, and when common goals are enunciated, processes agreed and resources shared.

d. Māori health research workforce development

Addressing ethnic inequities in the health research workforce is a strategic priority across the sector. Researchers should take opportunities to contribute to Māori health research workforce development by actively recruiting Māori students, researchers and support staff, and ensure that these individuals are supervised and mentored in a culturally safe environment.

e. Theoretical space

The advancement of Kaupapa Māori Theory (KMT) and Research (KMR)12 has drawn many Māori researchers into this developing and contested theoretical space. 13 The term KMR often signals Māori-led research that has a series of philosophical aims, including promoting Māori at the centre of the inquiry, developing research questions that Māori partners have signalled are important, appropriate sampling, utilising Māori processes where appropriate, resisting 'victim-blame' analyses, partnering with Māori with aligned objectives, Māori health research workforce development and contributing to the elimination of ethnic inequities.14 Other Māori researchers may use the terms KMR and KMT but focus primarily on Māori knowledge and traditional processes. It is important to note that KMR can encompass a broad range of epistemologies so researchers using KMR should reference their philosophical aims, objectives and theoretical positioning.

Non-Māori research teams should consider ways to support Māori research staff and students as they grow their theoretical iden-



tities and research capabilities. Not all Māori researchers agree to their work being classified as KMR. Non-Māori researchers may wish to familiarise themselves with KMT and KMR when partnering with KM researchers. The terms Kaupapa Māori-consistent or Kaupapa Māori-partnered research have been used for projects led by non-Māori but aligning with KM objectives.

2. Māori as participants

Health researchers should familiarise themselves with the concepts of Māori ethnicity, ancestry and descent and consider the relative strengths and limitations of each variable in relation to particular research questions. A range of tools are available for measurement of these constructs within the health sector.

a. Ethnicity

Ethnicity is a socio-demographic variable that is routinely collected across national health datasets to quite high levels of completeness. Because of this, ethnicity data in New Zealand are strong by international standards. However, it is important to carefully consider what we are measuring when using ethnicity as a variable. Ethnicity is a social construct.15 It is not about how we look or act or what others think. It is not the same as ancestry or descent but rather it is about self-identifying the social group or groups with whom we affiliate and therefore how we might live our lives and experience society.16 Ethnicity is not fixed and people may change their ethnicity at different times of their lives.

b. Ethnicity data standards

Ethnicity should be collected using the standard ethnicity question that is used in the NZ Census and most official datasets.¹⁷ Failure to use the standard question introduces uncertainty into the research analysis and impacts on the comparability of data.¹⁶

c. Māori ancestry and descent

The Māori descent question in the New Zealand Census simply asks if one is descended from a New Zealand Māori, and for some research questions a family history or genealogy may be more relevant. This information should be gathered directly from the participant(s). Whakapapa (genealogy) information is considered by many to be tapu (sacred) and there may be restric-

tions on how this information is gathered, stored, used and governed. Ethnicity data is an inappropriate proxy for descent as a small proportion of people who identify Māori ethnicity do not report Māori ancestry and a larger proportion of those who report Māori ancestry do not identify Māori ethnicity. In the 2013 Census, 0.8% of people who reported Māori ethnicity did not report Māori descent. In contrast, 16.1% of those who reported Māori descent did not identify Māori ethnicity. ¹⁹

3. Promoting Māori voice

The Treaty guarantees that the Crown will act in such a way that Māori will not be disadvantaged, and if disadvantage is demonstrated, the Crown will take measures to correct the imbalance. The Māori population is 16% of the total New Zealand population, and few researchers think about the impact of a numerically minority voice on policy and programmes generated from research, especially the impact on further inequity and marginalisation. A random population sample will often contain fewer than 15% Māori, so the dominant 'voice' generated largely tells the 'story' of non-Māori: their strengths, risks, needs and preferred ways of being. The Māori 'story' could be very different. Researchers should be aware of this in the construction of their research. Promoting Māori voice is relevant to both qualitative and quantitative studies.

a. Qualitative research

If ethnic inequities exist in the research topic, it is important that priority be given to the group with the inequity—their 'voice' should be heard and their reality understood. A project that prioritises Māori 'voice' may require additional consideration, planning and perhaps staffing/supervision, but will add significantly to research impact and utility (eg,^{20–22}).

b. Quantitative research

Equal explanatory power²³ means that research has either prioritised Māori participation in quantitative research or is constructed so that the Māori sample is equally powered to answer the research question in simple and/or complex analyses (eg,²⁴). It is not 'over-sampling' Māori, rather it is appropriate sampling and respect for the Māori 'voice'. Constructing a sample with



equal power to answer the research question for Māori as well as non-Māori will provide multiple opportunities for dissemination.

c. Data analysis

Researchers should be wary of common errors made when analysing Māori data. If Māori data are different, do not assume that the 'difference' lies within Māori (bodies, culture or behaviours). This tendency to 'victim-blame' peoples is called 'deficit theorising'²⁵ and shows superficial knowledge of the determinants of health and health inequities.²⁶ Instead, consideration should be given to the structural or system-level factors likely to be involved (eg^{27,28}).

4. Human tissue

The term human tissue covers all physical samples, regardless of size (eg, blood samples, tissue biopsies and cells, molecules and genetic profiles) or source (eg, commercial cell lines, pathological specimens, research samples and those from tissue collections or biobanks). No matter the source, Māori, and indeed many New Zealanders, consider human tissue to be tapu, meaning it comes with a set of restrictions. These restrictions are usually managed by informed consent processes and the formal information made available to prospective participants, including:

- Agreed parameters surrounding the use of human tissue including possible future use;
- Agreement on storage, management and governance of samples. Many samples are now stored for future use that may extend beyond the career, or indeed life of the primary investigator or project. Samples may also be requested by international research partners. Thus, it is critical to consider who has governance over the future decision-making in respect of samples and the data generated by them;
- Processes for return or destruction of samples;
- Feedback to participants or their whānau on pertinent health information obtained from the samples.^{4,29}

a. Genetic samples

In addition to the issues noted above, researchers who collect human tissue for the specific intention of, or potential for, genetic analysis must also consider the following:

 Genetic material not only provides information about the donor, but

- also information about whānau of the donor. Because of this, there is growing interest in obtaining whānau consent in addition to individual consent. While not current practice, researchers planning to take samples for genetic analysis should consider 'future proofing' their samples by incorporating family into the consent process. Although there is no 'best practice' yet for gaining whānau permission, at the very least, researchers should note whether other 'genetic relatives' were consulted during the process of informed consent and whether their permissions were also gained.
- Some researchers consider the physical sample and the data generated from human tissue as different. Usually significant consideration is given to the ethical and secure storage, management and sometimes governance of the genetic material without similar attention given to the data it generates. Good research practice ought to include due consideration to the governance and secure storage of an individual's tissue and generated data. Although this is not current practice we urge researchers to plan for this in future projects.
- Genetic samples are often sent overseas for sequencing or analysis by collaborators or commercial companies. Research teams need to consider how they will maintain their Treaty responsibilities once the samples are outside New Zealand's jurisdiction. The likelihood of genetic material or data leaving New Zealand, now or in the future, should be reflected in the researcher's governance plan and outlined as part of the informed consent process.

b. Data

Issues surrounding ownership and guardianship of research datasets have become more urgent with the growth of 'big data' and international collaborative research. Once integrated into large datasets, it is unclear how Māori data will be treated in terms of groupings, analyses and interpretations. Significant work on 'data sovereignty' by indigenous researchers here and overseas is underway,³⁰ so researchers should stay abreast of developments.



c. Working with genetically modified organisms

Many New Zealanders, including Māori, are concerned about the use of genetically modified organisms including in research. The Hazardous Substances and New Organisms (HSNO) Act 1996 requires that the principles of the Treaty of Waitangi are considered in applications. Because of this obligation, it can be important to acknowledge this concern and note relevant accreditation and regulation of laboratory facilities.

d. The special case of transgenic animals and xenotransplantation

The Royal Commission on Genetic Modification (2001) noted that a number of concerns were raised by Māori (and other New Zealanders) to xenotransplantation and transgenic animals. The Commission noted that there were research benefits to these

technologies but recommended strict regulation.³¹ Researchers should demonstrate an understanding of the range of views held by Māori and describe how the research will be conducted in accordance with appropriate standards and regulation.

Conclusion

All health researchers in New Zealand should be accountable to our delegated responsibilities under the Treaty of Waitangi and be able to enact issues of responsiveness to Māori. This paper proposes key elements to consider in this respect. In addition, researchers will need to consider what the standards of excellent practice will be in the future, especially as they train junior and emerging researchers and gather data and tissue samples. We encourage all researchers to engage in the work of 'future proofing' health research to ensure that responsiveness to Māori is achieved.

Competing interests:

Dr Paine is a previous Science Assessing Committee member for the Health Research Council of New Zealand, a co-opted member of the Māori Health Committee for the Health Research Council of New Zealand, and is currently involved in research projects that are funded by the Health Research Council of New Zealand and by the Ministry of Health.

Acknowledgements:

We would like to acknowledge our colleagues at the Tōmaiora Research Group and Te Kupenga Hauora Māori for their support. Our thanks to Dr Donna Cormack for her advice on an early draft of this paper.

Author information:

Papaarangi Reid, Tumuaki and Head of Department, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Sarah-Jane Paine, Senior Research Fellow, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Elana Curtis, Senior Lecturer, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Rhys Jones, Senior Lecturer, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Anneka Anderson, Lecturer, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Esther Willing, Lecturer, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland; Matire Harwood, Senior Lecturer, Te Kupenga Hauora Māori, Faculty of Medical and Health Sciences, University of Auckland, Auckland.

Corresponding author:

Sarah-Jane Paine, Te Kupenga Hauora Māori, Faculty of Medicine and Health Sciences, University of Auckland, Private Bag 92019, Auckland 1142.

sj.paine@auckland.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7414



REFERENCES:

- Ministry of Health. (2016)
 New Zealand Health
 Strategy: Future Direction.
 Wellington: Ministry
 of Health. Wellington:
 Ministry of Health.
- 2. Health Research Council of New Zealand. (2010)
 Guidelines for researchers on health research involving Māori (version 2).
 Auckland: Health Research Council of New Zealand.
- 3. Ministry of Business
 Innovation and Employment and the Ministry of
 Health. (2017) New Zealand
 Health Research Strategy
 2017–2027. Wellington,
 New Zealand: Ministry
 of Business, Innovation
 and Employment and
 Ministry of Health.
- Hudson M, Southey K, Uerata L, Beaton A, Milne M, Russell K, Smith B, Wilcox P, Toki V, Cheung M. (2016) Key informant views on biobanking and genomic research with Maori. N Z Med J. 129(1447):29–42.
- 5. National Ethics Advisory Committee. (2016)
 Strengthening Māori
 Research Ethics through
 NEAC's Ethical Guidelines
 for Health and Disability
 Research. Wellington:
 Ministry of Health.
- 6. Gifford H, Boulton
 A. (2007) Conducting
 excellent research with
 indigenous communities:
 balancing commitment to
 community and career.
 AlterNative. 3(2):24–45.
- 7. National Ethics Advisory
 Committee. (2012) Āhuatanga ū ki te tika me te pono
 mō te Rangahau Māori:
 Māori Research Ethics:
 An overview. Wellington:
 Ministry of Health.
- 8. Sporle A, Koea J. (2004)
 Māori responsiveness
 in health and medical
 research: clarifying the
 roles of the researcher and
 the institution (part 2). N
 Z Med J. 117(1199):U998.

- 9. Sporle A, Koea J. (2004) Māori responsiveness in health and medical research: key issues for researchers (part 1). N Z Med J. 117(1199):U997.
- 10. Simmonds S. (2015) A
 Framework for Māori
 Review of Research in
 District Health Boards.
 Wellington New Zealand:
 Auckland & Waitemata
 District Health Boards
 and Capital and Coast
 District Health Board.
- 11. Curtis E, Reid P. (2013)
 Indigenous Health
 Workforce Development:
 challenges and successes of the Vision 20:20
 programme. ANZ Journal
 of Surgery. 83(1–2):49–54.
- 12. Smith LT.(2012)
 Decolonizing Methodologies: Research and Indigenous Peoples. 2nd ed. London: Zed Books.
- 13. Curtis E. (2016) Indigenous positioning in health research: The importance of Kaupapa Māori theory-informed practice.

 AlterNative. 12(4):396–410.
- 14. Te Kupenga Hauora Māori. (2015) Tōmaiora Māori Health Research Group - Guidelines Auckland: Faculty of Medical and Health Sciences, University of Auckland.
- **15.** Krieger N. (2001) A glossary for social epidemiology. Journal of Epidemiology and Community Health. 55(10):693–700.
- 16. Robson B, Reid P. (2001)
 Ethnicity matters:
 Review of the Measurement of Ethnicity in
 Official Statistics Māori
 perspectives paper for
 consultation. Wellington:
 Statistics New Zealand.
- 17. Ministry of Health. (2004) Ethnicity Data Protocols for the Health and Disability Sector. Wellington: Ministry of Health.

- 18. Putaiora Writing Group. (2010) Te Ara Tika - Guidelines for Maori research ethics: a framework for researchers and ethics committee members.
- 19. Statistics New Zealand. (2013) 2013 Census QuickStats about Māori. Wellington, New Zealand.
- 20. Jones R, Crengle S, McCreanor T. (2006) How Tikanga guides and protects the research process: insights from the Hauora Tāne project. Social Policy Journal of NZ. 29:60–77.
- 21. Harwood MLN. Understanding and improving stroke recovery for Māori and their Whānau: University of Otago. 2012.
- 22. Brewer KM. (2016) The complexities of designing therapy for Māori living with stroke-related communication disorders.

 N Z Med J. 129(1435):75–82.
- 23. Te Rōpū Rangahau Hauora a Eru Pōmare. (2002) Mana Whakamārama Equal Explanatory Power: Māori and non-Māori sample size in national health surveys. Wellington: Ministry of Health.
- 24. Paine SJ, Priston M, Signal LT, Sweeney B, Muller D. (2013) Developing new approaches for the recruitment and retention of indigenous participants in longitudinal research: lessons from E Moe, Māmā: Maternal Sleep and Health in Aotearoa/New Zealand MAI Journal. 2(2):121–132.
- 25. Valencia RR.(1997) The evolution of deficit thinking: Educational thought and practice.
 London: Falmer Press.
- 26. Williams DR, Mohammed SA. (2013) Racism and Health I: Pathways and Scientific Evidence. Am Behav Sci; 57(8).



- 27. Tapera R, Harwood M, Anderson A. (2016) A qualitative Kaupapa Māori approach to understanding infant and young child feeding practices of Māori and Pacific grandparents in Auckland, New Zealand. Public Health Nutr:1–9.
- 28. Curtis E, Harwood M,
 Riddell T, Robson B, Harris
 R, Mills C, Reid P. (2010)
 Access and Society as
 Determinants of Ischaemic Heart Disease in
- Indigenous Populations. Heart, Lung and Circulation. 19(5–6):316–324.
- 29. Ministry of Health. (2007)
 Guidelines on the Use of
 Human Tissue for Future
 Unspecified Research
 Purposes: Submissions
 summary. Wellington:
 Ministry of Health.
- **30.** Kukutai K, Taylor J, editors. Indigenous data sovereignty: toward an agenda Acton, ACT: ANU Press; 2016.
- 31. Royal Commission on Genetic Modification. (2001) Report of the Royal Commission on Genetic Modification. Wellington: GM Commission.
- 32. Cram F. (2002) Māori and science: three case studies. Auckland, New Zealand: International Research Institute For Māori And Indigenous Education, University of Auckland.



Overwhelming support for smokefree cars that are carrying children—is the Government listening?

Richard Jaine, Richard Edwards, Jude Ball, Dalice Sim, George Thomson, R Beaglehole

here is convincing evidence that children exposed to secondhand smoke (SHS) are at increased risk of respiratory tract infections, asthma exacerbations, sudden unexplained death in infancy (SUDI) and bacterial meningitis.1 SHS exposure in children causes a disease burden that is entirely avoidable. As a society we fail our children when we do not take evidence-based action to avoid SHS exposure. We also fail our responsibilities under the United Nations Convention on the Rights of the Child (UNCROC) (which New Zealand ratified in 1993). Under article 24 of the UNCROC, children have a right to health.2 As part of the plan for action, countries are required to "develop legislation...to prevent the exposure of children to harmful environmental contaminants in the air". Introducing legislation to ban smoking in cars carrying children would therefore be consistent with our responsibilities under the UNCROC.

The Health Select Committee recently recommended that the Government "introduce legislation, or other measures, to ban smoking in cars carrying children under the age of 18 years". The Government responded by recognising the importance of protecting children from harm to health due to SHS exposure but decided to disregard the Health Select Committee's recommendation, largely on the grounds stating "present initiatives are sufficient to deter smoking in cars carrying children".4

We have previously presented evidence that there is substantial exposure of children to SHS in cars and that this has changed little over recent years.⁵ Another consideration in deciding whether to implement a legislative policy is whether there is public support for legislation. In the context of

policy that affects children, Article 12 of the UNCROC states that children should "be provided the opportunity to be heard". The opportunity to be heard can be achieved at different levels. At the lowest level this includes "children are listened to", and the minimum level required to meet obligations under the UNCROC is that "children's views are taken into account".

Therefore, we ask: are we meeting our UNCROC obligations to listen to our children, and taking their views into account in deciding whether to legislate for smokefree cars that are carrying children? What support is there for the legislation among adults?

Listening to our children

Two recent nationally representative surveys have asked young people's opinions of smokefree cars (Table 1).^{7,8}

We analysed data from the 2014 ASH (Action on Smoking and Health) Year 10 Snapshot Survey of over 25,000 New Zealand 14–15 year olds. This asked respondents whether they agreed or disagreed that "smoking in cars should be banned when children are in them". Support for this statement was 87%, with only 4% disagreeing. The 2012 Youth Insights Survey for the same age group had very similar findings: 88% agreed with the statement and only 4% disagreed.8

It is therefore clear that support among young people for smokefree cars is very high. This type of evidence begins to fulfil New Zealand's responsibilities under the UNCROC Article 12 by ensuring children are listened to, but unless legislation is introduced it is difficult to argue that their views are being taken into account.



Listening to our adults

There is evidence dating back to 1997 that there is overwhelming support among New Zealand adults (94%) for banning smoking in cars with children in them.⁹ Even smokers demonstrated majority support (87%) for this proposal. This has been confirmed in subsequent findings from the Health Promotion Agency's Health and Lifestyle surveys and among smokers in the New Zealand ITC project and New Zealand Smoking Monitor (Table 1).¹⁰⁻¹³

New data from the 2016 Health and Lifestyles Survey of a nationally representative sample of more than 3,800 adults demonstrates overwhelming support, with 94% of all adults supporting a ban on smoking in cars with children in them; and 89% support among adult smokers (Health and Lifestyles Survey 2016 preliminary data, personal communication, Greg Martin).

Conclusion

New Zealand is failing its obligations under the UNCROC Article 24 to protect children from harm due to exposure to SHS by not acting to introduce smokefree cars. Our evidence also clearly demonstrates strong support among young people for banning smoking in cars carrying children. Hence, although the Government could be construed as listening to our children, its failure to act on smokefree cars suggests it is not taking children's views into account, and is not meeting its obligations under UNCROC Article 12. In addition, the Government does not appear to be taking the views of adults into account either, given the sustained evidence of overwhelming public support for smokefree cars legislation. We believe it is time that the Government truly responded to the views of children and the public and addressed its duties under UNCROC by introducing legislation to make cars carrying children smokefree.

Table 1: Youth and adult support for making cars carrying children smokefree, for various New Zealand surveys, and by smoking status.

Youth support (14–15 year olds)					
Study and year	Statement	Youth support (total)	Youth support (smokers only)		
Youth Insights Survey 2012 ⁸	Smoking in cars should be banned when children are in them	88%	63%		
ASH Year 10 Snapshot Survey 2014	Smoking in cars should be banned when children are in them	87%	-		
Adult support		1			
Study and year	Statement	Adult support (total)	Adult support (smokers only)		
al-Delaimy et al 1999 ⁹	Places which should be smokefree when there are children around: private cars	94%	87%		
Wilson et al 2009 ¹⁰	Do you think smoking should be allowed in cars with preschool children in them*	-	96%*		
Health and Lifestyles survey 2008 ¹¹	Smoking should not be allowed in cars with children under the age of 14 in them	91%	82%		
Health and Lifestyles survey 2012 ¹²	Smoking in cars should be banned where children are in them	93%	-		
New Zealand Smoking Monitor survey 2014 ¹³	Smoking in cars should be banned while children are in them	-	92%		
Health and Lifestyles survey 2016, preliminary data (Greg Martin, personal communication)	Smoking in cars should be banned when children aged under 18 years old are in them	94%	89%		

^{*}This refers to percent of respondents *disagreeing* with the statement, that is, agreeing that smoking should **not** be allowed in cars with preschool children in them.



Competing interests:

Nil.

Acknowledgements:

Greg Martin from Health Promotion Agency for providing the latest Health and Lifestyles survey data.

Author information:

Richard Jaine, Senior Lecturer, Department of Public Health, University of Otago, Wellington; Richard Edwards, Professor, Department of Public Health, University of Otago, Wellington; Jude Ball, Research Fellow, Department of Public Health, University of Otago, Wellington; Dalice Sim, Senior Research Fellow/Biostatistician, Dean's Department, University of Otago, Wellington; George Thomson, Research Associate Professor, Department of Public Health, University of Otago, Wellington; Robert Beaglehole, Professor, University of Auckland, Auckland.

Corresponding author:

Richard Jaine, Senior Lecturer, Department of Public Health, University of Otago, Wellington.

richard.jaine@otago.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7415

REFERENCES:

- 1. Royal College of Physicians. Passive smoking and children. A report by the Tobacco Advisory Group. London: Royal College of Physicians, 2010.
- Hodgkin R, Newell P.
 Implementation handbook for the convention on the rights of the child. 3rd edn. Geneva: UNICEF, 2007.
- 3. New Zealand Parliament.
 Petition 2014/27 of Bridget
 Rowse on behalf of Patu
 Puauahi Smokefree
 Northland: Report of
 the Health Committee.
 Wellington: New Zealand
 Parliament, 2016.
- 4. New Zealand Parliament.
 Government response
 to report of the Health
 Committee on Petition
 2014/27 of Bridget Rowse
 on behalf of Patu Puauahi
 Smokefree Northland.
 Wellington: New Zealand
 Parliament, 2017.
- Edwards R, Sim D, Ball J, et al. Surveys shows exposure to smoking in cars among Year 10 children is not decreasing: time for the Government to act. N Z Med J. 2017; 130;56–8.

- 6. Shier H, Train PÁ. Pathways to participation: openings, opportunities and obligations: a new model for enhancing children's participation in decision-making, in line with Article 12.1 of the United Nations convention on the rights of the child. Child Soc 2001; 15:107–17.
- Action on Smoking and Health. 2014 ASH Year 10 Snapshot Survey: Information and methodology. Auckland: Action on Smoking and Health; 2014.
- 3. Health Protection Agency. Young people's opinion on extending smoking bans to cars and outdoor places where young people go. In Fact. Health Protection Agency, Wellington, 2013. http://www.hpa.org.nz/sites/default/files/2012%20 YIS%20Young%20 Peoples%20opinion%20 on%20extending%20 smoking%20bans%20 FA%20%282%29.pdf. Accessed 9 June 2017
- al-Delaimy W, Luo D, Woodward A, Howden-Chapman
 P. Smoking hygiene:

- a study of attitudes to passive smoking. N Z Med J. 1999; 112:33–6.
- 10. Wilson N, Blakely T, Edwards R, et al. Support by New Zealand smokers for new types of smokefree areas: national survey data. N Z Med J. 2009; 122(1303):80–9.
- 11. Trappitt R, Li J, Tu D.
 Acceptability of smoking
 around other people –
 Health and Lifestyles
 Survey 2008 [In Fact].
 Wellington: Health
 Sponsorship Council,
 2011. www.hsc.org.nz/
 researchpublications.html.
 Accessed 9 June 2017
- 12. Li J, Newcombe R.
 Acceptability of extended smokefree areas and smokefree cars. [In Fact].
 Wellington: Health Promotion Agency Research and Evaluation Unit, 2013.
- 13. Li J, Nelson S, Newcombe R, Walton D. Smoking in cars: knowledge, behaviours and support for smokefree cars legislation among New Zealand smokers and recent quitters. N Z Med J. 2016; 129:46–58.



The battle for better nutrition: the role of the escalating fruit and vegetable prices

Isaac Amoah, Carolyn Cairncross, Elaine Rush

The cost of fruits and vegetables is rising rapidly when compared to a loaf of bread or a box of Weetbix in New Zealand. The monthly and annual food price index reported by Statistics New Zealand revealed substantial increases in the prices of fruits and vegetables. However, wages for families in the poorest third of households have remained constant since 1982.1 In May 2017, the prices of fruits and vegetables rose by 8.2% compared with the previous month (an increase of 6.6% after seasonal adjustment) while restaurant meals and ready-toeat food prices rose by just 0.3%. In terms of annual change, fruit and vegetable prices increased by 14.0% in May 2017 compared with May 2016.1

There have been reports in national, international and social media regarding the high cost of fruit and vegetable prices in the past month. For example, expat Kiwis living in Australia took to social media—Facebook to vent their frustration with the atypical New Zealand fruit and vegetable prices by comparing the cost to those purchased in supermarkets in Australia. Broccoli was reported to cost \$A0.87 in Australia compared to \$NZ3.69 in New Zealand, while cucumber and cabbage were almost 50% cheaper in Australia than in New Zealand.2 New Zealand Food & Grocery Council spokesman Brent Webling² stated that this rise in fruit and vegetable prices was partly attributed to the economic growth of New Zealand's "markets in Asia and the East". Not only does the increase in prices of fruits and vegetables have unintended negative consequences on the socio-economic status

of the citizenry, it also has serious implications for food security and nutrition of New Zealand people. One marker of malnutrition is the prevalence of obesity and its comorbidities such as type 2 diabetes. In children and adults, the prevalence of obesity is increasing, particularly in those living in more deprived areas.³ Subsequently, costs in healthcare are also rising.⁴

Household food security implies that adequate and nutritionally appropriate food should be accessible at all times to ensure the provision of energy and nutrients required to maintain an active and healthy lifestyle. Bridging the health gap therefore requires a careful attention paid to the environment of food availability, choice and cost⁵ since consumers with limited financial resources often choose energy-dense diets high in refined grains, added sugars and fats as a way to save money and provide sufficient food to feed their families.⁶

Fruits and vegetables play a crucial role across the life course for maintaining optimal health. They should constitute half of the volume of the ideal food plate. It is known that New Zealanders should be eating more fruits and vegetables.7 New Zealand produces enough whole foods to feed 40 million people but for many New Zealanders and especially children, the supportive environment is not provided, ie, sufficient money and physical access to fruit and vegetables. The "5+ A Day" initiative promoted in New Zealand to enhance the consumption of five or more portions of fruits and vegetables a day8 is a way of promoting health. On a practical



level, meeting the 5+ a day for a family of six people implies they need to purchase and consume 21kg of fruits and vegetables per week. The rising cost of fruits and vegetables makes this recommendation even harder to achieve, particularly for those on limited incomes.

With the general election only a few days away (at time of writing), it is now time for health professionals to add their voice for the need for a fairer society and revisit the essential cornerstones of a healthy diet and life, including an adequate and varied intake of fruits and vegetables every day.

Competing interests:

Nil

Author information:

Isaac Amoah, PhD student, School of Sport and Recreation, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland;
Carolyn Cairncross, PhD, School of Sport and Recreation, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland;
Elaine Rush, PhD, School of Sport and Recreation, Faculty of Health and Environmental

Sciences, Auckland University of Technology, Auckland. Corresponding author:

Elaine Rush, PhD, School of Sport and Recreation, Faculty of Health and Environmental Sciences, Auckland University of Technology, Auckland 1010.

elaine.rush@aut.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7416

REFERENCES:

- http://www.stats.govt. nz/browse_for_stats/ people_and_communities/Households/ household-economic-survey-info-releases.aspx Accessed on 31/10/2017.
- http://www.nzherald. co.nz/business/news/ article.cfm?c_id=3&objectid=11882574 Accessed on 16/07/2017.
- http://www.health.govt. nz/nz-health-statistics/ health-statistics-and-datasets/obesity-data-and stats Accessed on 20/09/2017.

- Ettinger S. Nutritional Pathophysiology of Obesity and Its Comorbidities: A Case-study Approach. Academic Press; 2016.
- 5. Rush E, Puniani N, Snowling N, Paterson J. Food security, selection, and healthy eating in a Pacific Community in Auckland New Zealand. Asia Pac J Clin Nutr. 2007; 16(3):448–54.
- 6. Darmon N, Briend A, Drewnowski A. Energy-dense diets are associated with lower

- diet costs: a community study of French adults. Public Health Nutr. 2004; 7(1):21–7.
- 7. Ministry of Health, Annual Update of Key Results 2014/2015: New Zealand Health Survey. Wellington: Ministry of Health, 2014.
- 8. Rekhy R, McConchie R. Promoting consumption of fruit and vegetables for better health. Have campaigns delivered on the goals? Appetite. 2014: 79:113–23.



New Zealand's legal action against IQOS postponed, consultation with Big Tobacco follows

Marta Rychert

Tith an ambitious goal of eradicating tobacco use by 2025,¹ New Zealand is often considered a leader in tobacco control policy.² The 'tobacco endgame' policy and tightening controls on the industry have wide public support.³-5 While a recent study concluded new measures are needed to achieve the goal of eradicating smoking by 2025,⁶ a society without cigarettes is now becoming a very real possibility.³-9

With the eventual phasing out of cigarettes a looming prospect, the tobacco industry in New Zealand, as elsewhere,10,11 is increasingly turning its attention to alternative nicotine and new tobacco products, including electronic nicotine delivery systems (ENDS) and 'heat-not-burn' (HNB) tobacco products. However, the legal status of these products, and the applicability of laws protecting the public from passive smoke, is uncertain.12,13 For example, e-cigarettes (ie, devices that heat liquid propylene glycol to create an inhalable aerosol with nicotine¹⁴) have been available in New Zealand since around 2007, despite nicotine being legally classified as a medicine.15 In March 2017, the Government announced that nicotine-containing e-cigarettes will be regulated as consumer products.¹⁶ On the other hand, other emerging products, including HNB tobacco are currently prohibited, as the Smoke-free Environments Act 1990 (s 29(2)) bans the importation and sale of tobacco products for oral use other than smoking.17

Despite the ban on new forms of tobacco, Philip Morris International launched its HNB tobacco product IQOS (*I-Quit-Ordi-nary-Smoking*) on the New Zealand market in December 2016.18 The IQOS device heats tobacco sticks (called Heets) at 350°C, lower than the combustion point of traditional cigarettes. The product is available in Japan, Canada and many European countries, and marketed as a "lower risk" alternative to cigarettes.19 Independent research on health risks from HNB products compared to traditional cigarettes has not confirmed this claim.13 While some studies indicate that HNB products may be less harmful than traditional cigarettes, 20,21 others established serious health risks linked to HNB use, including exposure to similar levels of many cancer-causing chemicals present in traditional cigarettes. 12,22,23 Recent establishment of the Foundation for a Smoke-free World (with nearly one billion USD funding commitment from Philip Morris International)24 illustrates industry efforts to promote alternatives to traditional cigarettes on a global scale.25

In May 2017, the New Zealand Ministry of Health sued Philip Morris for importing and marketing IQOS on the grounds that it was prohibited under s 29(2) Smoke-free Environments Act 1990.26 The court hearing, initially scheduled in June 2017, was rescheduled for September.²⁷ In the meantime, three meetings between Ministry of Health officials and industry representatives (British American Tobacco, Imperial Tobacco and Philip Morris) were held in guick succession (30 May-2 June) to "discuss regulation of new tobacco and nicotine-delivery products".28 Subsequently, in August 2017, the government announced their plan to establish a pre-market approval system for smokeless tobacco products such as IQOS.29



The process through which the proposal to regulate HNB tobacco products emerged is alarming. Unlike the earlier proposal to regulate nicotine e-cigarettes, it has not been subject to similar public consultation. Also, the delay in enforcing legislation which currently prohibits sale of HNB products undermines the legitimacy of existing tobacco control laws. Finally, New Zealand is a Party to the Framework Convention on Tobacco Control and under art. 5.3 is legally obliged to protect tobacco control policy

from commercial and other vested interests of the industry. While regulation of new nicotine and tobacco products *may* indeed support achievement of the 'endgame' goal (through offering *potentially* safer alternatives to smoking), major uncertainties remain as to the health risks and normalisation effects. Given that industry survival depends on these technologies, greater transparency is needed in the process of designing regulations for the new products.

Competing interests:

Nil.

Author information:

Marta Rychert, Researcher, SHORE & Whariki Research Centre, College of Health, Massey University; Lecturer, School of Public Health and Psychosocial Studies, Auckland University of Technology, Auckland.

Corresponding author:

Marta Rychert, Researcher, SHORE & Whariki Research Centre, College of Health, Massey University, Lecturer, School of Public Health and Psychosocial Studies, Auckland University of Technology, Auckland.

m.rychert@massey.ac.nz; marta.rychert@aut.ac.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7417

REFERENCES:

- 1. New Zealand Government. Government Response to the Report of the Māori Affairs Committee on its Inquiry into the tobacco industry in Aotearoa and the consequences of tobacco use for Māori. 2011. Available from: http://www.parliament.nz/resource/en-NZ/49DBHOH_PAP21175_1/9f015010d-386fe11050cddfbb-468c2a3f5b0cb89 (accessed 20 September 2017).
- 2. Studlar DT. Tobacco Control Policy Instruments in a Shrinking World: How Much Policy Learning? International Journal of Public Administration 2006; 29:367–96.
- Edwards R, Wilson N, Peace J, et al. Support for a tobac-

- co endgame and increased regulation of the tobacco industry among New Zealand smokers: results from a National Survey. Tobacco Control 2012.
- Hoek J, Gendall P, Maubach N, et al. Strong public support for plain packaging of tobacco products. Australian and New Zealand Journal of Public Health 2012; 36:405–7.
- 5. Jaine R, Healey B, Edwards R, et al. How adolescents view the tobacco endgame and tobacco control measures: trends and associations in support among 14–15 year olds. Tobacco Control 2014.
- 6. van der Deen FS, Wilson N, Cleghorn CL, et al. Impact of five tobacco

- endgame strategies on future smoking prevalence, population health and health system costs: two modelling studies to inform the tobacco endgame. Tobacco Control 2017.
- 7. Thomson G, Edwards R, Wilson N, et al. What are the elements of the tobacco endgame? Tobacco Control 2012; 21:293–5.
- 8. Malone RE. The Race to a Tobacco Endgame. Tobacco Control 2016; 25:607–8.
- Beaglehole R, Bonita R, Yach D, et al. A tobacco-free world: a call to action to phase out the sale of tobacco products by 2040. The Lancet 385:1011–8.
- **10.** Gilmore AB. Understanding the vector in order to plan effective tobacco control



- policies: an analysis of contemporary tobacco industry materials. Tobacco Control 2012; 21:119–26.
- 11. Aguinaga Bialous S, Peeters S. A brief overview of the tobacco industry in the last 20 years. Tobacco Control 2012; 21:92–4.
- 12. Auer R, Concha-Lozano N, Jacot-Sadowski I, et al. Heat-not-burn tobacco cigarettes: Smoke by any other name. JAMA Internal Medicine 2017; 177:1050–2.
- **13.** Caputi TL. Industry watch: heat-not-burn tobacco products are about to reach their boiling point. Tobacco Control 2017; 26:609–10.
- 14. Kennedy RD, Awopegba A, De León E, et al. Global approaches to regulating electronic cigarettes. Tobacco Control 2016.
- 15. Laugesen M. Nicotine and toxicant yield ratings of electronic cigarette brands in New Zealand. New Zealand Medical Journal 2015; 128.
- 16. MOH. E-cigarettes to be regulated as consumer products (29 March 2017). 2017. Available at: http://www.health.govt. nz/our-work/preventative-health-wellness/tobacco-control/e-cigarettes Archived by WebCite at: http://www.webcitation.org/6swXcMUct (Accessed 10 May 2017).
- 17. MOH. 'Heat not burn' tobacco products. 2017. Available at: http://www. health.govt.nz/our-work/ preventative-health-wellness/tobacco-control/ smokeless-tobacco-and-nicotine-delivery-products/ heat-not-burn-tobacco-products Archived by WebCite: http://www.webcitation.org/6sv3a32M0 (Accessed 10 May 2017).
- **18.** New Zealand Herald. New high-tech tobacco

- product Iqos is illegal (2 February 2017). 2017. Available at: http://www. nzherald.co.nz/business/ news/article.cfm?c_id=3&objectid=11793233 (Accessed 14 April 2017).
- PMI. Tobacco Meets Technology. no date. Available at: http://www.pmi.com/smoke-free-products/iqos-our-tobacco-heating-system Archived by WebCite at: http://www.webcitation.org/6swdh1LfB (Accessed 10 September 2017).
- 20. Lüdicke F, Baker G,
 Magnette J, et al. Reduced
 Exposure to Harmful and
 Potentially Harmful Smoke
 Constituents With the
 Tobacco Heating System
 2.1. Nicotine & Tobacco
 Research 2017; 19:168–75.
- 21. Titz B, Boué S, Phillips B, et al. Effects of Cigarette Smoke, Cessation, and Switching to Two Heat-Not-Burn Tobacco Products on Lung Lipid Metabolism in C57BL/6 and Apoe(-/-) Mice—An Integrative Systems Toxicology Analysis. Toxicological Sciences 2016; 149:441–57.
- 22. O'Connell G, Wilkinson P, Burseg KM, et al. Heated Tobacco Products Create Side-Stream Emissions: Implications for Regulation. Journal of Environmental Analytical Chemistry 2015; 2:2380–91.
- 23. Forster M, Liu C, Duke MG, et al. An experimental method to study emissions from heated tobacco between 100–200°C. Chemistry Central Journal 2015; 9:20.
- 24. Foundation for a Smokefree World (launched 13 September 2017). 2017. Available at: http:// www.smoke-freeworld. org/about-us (Accessed 20 September 2017).

- **25.** Yach D. Foundation for a smoke-free world. The Lancet 390:1807–10.
- 26. Elder V. Ministry takes
 Philip Morris to court
 (19 May 2017). Otago
 Daily Times [serial on the
 Internet]. 2017. Available
 from: http://www.odt.
 co.nz/news/national/
 ministry-takes-philip-morris-court Archived by
 WebCite at: http://www.
 webcitation.org/6ss99oz6I
 (accessed 24 May 2017).
- 27. Philip Morris 'tobacco sticks' court prosecution postponed. (2 June 2017) 2017. Available from: http://www.stuff.co.nz/business/industries/93268568/Philip-Morris-tobacco-sticks-court-prosecution-postponed Archived by WebCite at: http://www.webcitation.org/6ss9TUi7I (accessed 10 June 2017).
- 28. Ministry of Health. Meetings with tobacco industry representatives. 2017.

 Available from: http://www.health.govt.nz/our-work/preventative-health-wellness/tobacco-control/who-framework-convention-tobacco-control/meetings-tobacco-industry-representatives

 Archived by WebCite at: http://www.webcitation.org/6ssCnTH6l (accessed 10 June 2017).
- 29. MOH. Government to establish pre-market approval system for smokeless tobacco and nicotine-delivery products (other than e-cigarettes) (2 August 2017). 2017. Available at: http://www. health.govt.nz/our-work/ preventative-health-wellness/tobacco-control/ smokeless-tobacco-and-nicotine-delivery-products Archived by WebCite at: http://www.webcitation. org/6swZsnKwA (Accessed 20 August 2017).



Antifungal susceptibility results of vaginal yeast isolates from New Zealand women, 2001–2015

Arthur Morris, Wendy McKinney, Karen Rogers, Sally Roberts, Joshua Freeman

'easts, mostly *Candida* species, can cause troublesome vulvovaginal infection. Approximately 75% of women have at least one episode of vaginal candidiasis in their lifetime. Some women experience recurrent disease, mainly in their childbearing years.¹ Culture is recommended in cases of suspected vaginal candidiasis as symptoms are non-specific. Antifungal susceptibility testing is often requested/performed on genital isolates, especially in recurrent disease. We reviewed the disk susceptibility results for six antifungal agents against genital yeast isolates recovered over the 15-year period between 2001 and 2015. Our aim was to record current susceptibility to guide empirical treatment and to enable monitoring of antifungal susceptibility over time.

Only the first isolate of a species was included for each woman. Testing followed CLSI methods using Neo-Sensitab tablets (Rosco Diagnostica, Denmark).^{2–5} The six antifungal agents tested were: clotrimazole, CLO; fluconazole, FLC; itraconazole, ITC;

ketoconazole, KTC; miconazole, MIC; and nystatin, NYS. Tablet potency and interpretive criteria are presented in Table 1.^{3,5} The clinical history and treatment details of the women are unknown. Forty women had sequential isolates and we compared their isolates' susceptibility results to see if antifungal susceptibility had changed over time.

Six hundred and fifty-six women had isolates tested; 26 had mixed infection, all with two species. Six hundred and eighty-two initial isolates were analysed. The most frequent isolates were C. albicans 55%, C. glabrata complex 24% and C. parapsilosis complex 10% (Table 2). Saccharomyces cerevisiae made up 3% of isolates. More than 90% of isolates were susceptible to all agents tested (with the exception of MIC) (Table 2). Only two isolates were resistant to nystatin (a C.albicans and a C. parapsilosis complex isolate); both susceptible to several azole agents. C. glabrata complex and Pichia kudriavzeii (previously C. krusei) were the least susceptible groups (Table 2).

Table 1: Antifungal disk potency and interpretive criteria.

			Interpretive zone criteria (mm)			
Tablet	Antifungal	Potency (ug)	Susceptible (S)	S-Dose Dependent ³ / Intermediate ⁵ (SDD/I)	Resistant (R)	
CLO	clotrimazole	10	≥20	12-195	≤11	
FLC	fluconazole	25	≥19	15–18³	≤14	
ITC	itraconazole	10	≥19	15-18 ³	≤14	
KET	ketoconazole	15	≥19	15-18 ⁵	≤14	
МІС	miconazole	10	≥20	12-195	≤11	
NYS	nystatin	50	≥15	10-145	No zone	



Table 2: Susceptibility of vaginal yeast isolates to six antifungal agents.

Species/complex (Previous name)	number, % Susceptible	CLO	FLC	ITC	KET	МІС	NYS
Candida albicans	n	368	355	356	353	369	372
	%S	98	96	96	99.4	90	99.7
C. glabrata complex	n	162	44	43	44	162	164
	%S	80	70	74	80	91	100
C. parapsilosis complex	n	69	62	63	64	70	70
	%S	100	95	98	100	36	98.6
Clavispora (Candida) lusitanae	n	13	9	9	8	13	14
	%S	100	89	89	100	85	100
Pichia kudriavzeii (C. krusei)	n	16	5	5	5	16	16
	%S	100	0	100	60	6	100
Saccaromyces cerevisiae	n	21	16	16	16	21	21
	%S	100	94	94	100	100	100
Other species	n	24	16	16	16	24	16
	%S	100	94	69	100	50	100
Total	n	673	508	508	506	675	681
	%S	94	92	93	97	81	99.7

The susceptibility of azole non-susceptible isolates to other antifungals is shown in Table 3. Non-susceptibility to all azole agents was uncommon (Table 3). Forty women (6%) had sequential isolates of the same species; 29 had two sequential isolates, eight, two and one women had three, four and five sequential isolates respectively. Of the 40 sets of sequential isolates, 29 had identical susceptibility results, eight had a

single antifungal result change from either S to SDD/I (n=4) or SDD/I to S (n=4) indicating biological variation within the limits of the test method, ie, 37 (93%) had no change in susceptibility between isolates. Two women had disc results suggesting increased resistance: one had three agents change from S or I to R, the isolate was still susceptible to three antifungals; the second had three agents change from S to SDD/I and one

Table 3: Susceptibility of non-susceptible isolates to other antifungal agents.

		Susceptible/number tested (% susceptible)						
Non-suscepti- ble* to	n	CLO	FLC	ITC	KET	MIC	NYS	Pan-azole non-susceptible*
Clotrimazole	42	-	(3/19) 16	(4/19) 21	(10/19) 53	(19/45) 45	(41/41) 100	2,5%
Fluconazole	40	(23/34) 68	-	(14/40) 35	(27/40) 68	(13/40) 33	(40/44) 100	1,3%
Itraconazole	34	(19/34) 56	(8/34) 53	-	(25/40) 73	(10/34) 29	(34/34) 100	2,6%
Ketoconazole	13	(4/13) 31	(0/13) 0	(4/13) 31	-	(7/13) 54	(13/13) 100	3,23%
Miconazole	126	(100/124) 81	(65/92) 70	(69/93) 74	(88/94) 94	-	(125/126) 99.2	2,1%
Nystatin	2	(2/2) 100	(1/1) 100	(1/1) 100	(1/1) 100	(1/2) 50	-	0

^{*}Includes Susceptible-Dose Dependent, Intermediate and Resistant isolates.



agent, ITR, move from S to R. This woman's isolate was still susceptible to NYS. One woman had three or more agents change from R to S, suggesting strain replacement with a more susceptible strain. Overall only two patients (5%) had decreased susceptibility results in sequential isolates.

Apart from miconazole, ≥92% of isolates were susceptible to antifungal agents commonly recommended and used for vaginal candidiasis.^{6,7} It was uncommon for an isolate non-susceptible to one azole agent to be non-susceptible to all azole agents (Table 3). Azole resistance was uncommon, even in repeat isolates from the same woman, supporting the observations of others.⁸⁻¹⁰ Sequential isolates from women, who probably had been treated and re-presented with symptoms, were unlikely

to have developed resistance to antifungal agents (5%). Resistance to antifungal agents remains uncommon in genital isolates in New Zealand. These susceptibility results will allow monitoring of antifungal susceptibility over time. Molecular testing for azole resistance mechanisms and to confirm strain identity would be useful additional information to give insight into the results observed in the low proportion of women with sequential isolates with apparent changes in susceptibility.

We conclude that failure of a stat dose or short course treatment does not indicate antifungal resistance and those women with recurrent vulvovaginal yeast infection can be reasonably treated empirically with a previously used agent while identification and susceptibility results are awaited.

Competing interests:

Nil.

Author information:

Arthur J Morris, Clinical Microbiologist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland; Wendy P McKinney, Medical Laboratory Scientist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland; Karen Rogers, Medical Laboratory Scientist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland; Sally A Roberts, Clinical Microbiologist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland; Joshua T Freeman, Clinical Microbiologist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland.

Corresponding author:

Arthur J Morris, Clinical Microbiologist, New Zealand Mycology Reference Laboratory, LabPlus, Auckland City Hospital, Auckland.

arthurm@adhb.govt.nz

URL

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7418

REFERENCES:

- Marchaim D, Lemanek L, Bheemreddy S, Kaye KS, Sobel JD. Fluconazole-resistant Candida albicans vulvovaginitis. Obstet Gynecol 2012; 120:1407–14.
- Clinical and Laboratory Standards Institute.
 Method for antifungal disk
- diffusion susceptibility testing of yeasts; Approved Guideline – Second Edition. CLSI document M44-A2. CLSI, Wayne, PA, USA, 2009.
- Clinical and Laboratory Standards Institute. Zone diameter interpretive standards, corresponding

minimal inhibitory concentration(MIC) interpretive breakpoints, and quality control limits for antifungal disk diffusion susceptibility testing of yeasts; third informational supplement. CLSI document M44-S3. CLSI, Wayne, PA, USA, 2011.



- Rosco Diagnostica. Neo-Sensitabs user's guide. Rosco Diagnostica, Taastrup, Denmark. 2013.
- Rosco Diagnostica. Suceptibility testing of yeasts 2011. Rosco Diagnostica, Taastrup, Denmark. 2011.
- 6. Pappas PG, Kauffman CA, Andes DR, et al. Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. Clin
- Infect Dis 2016; 62:e1-e50. doi:10.1093/cid/civ933
- 7. Reid M. Treatment of sexually transmitted and other genital infections.
 BPAC Best practice
 Journal 2009; 20:24–9.
- 8. Sobel JD, Wiesenfeld HC, Martens M, et al. Maintenance fluconazole therapy for recurrent vulvovaginal candidiasis. N Engl J Med 2004; 351:876–83.
- 9. Richter SS, Galask RP, Messer SA, et al. Antifungal

- susceptibilities of Candida species causing vulvovaginitis and epidemiology of recurrent cases. J Clin Microbiol 2005: 43:2155–62.
- 10. Sobel JD, Zervos M, Reed BD, et al. Fluconazole susceptibility of vaginal isolates obtained from women with complicated Candida vaginitis: clinical implications. Antimicrob Agents Chemother 2003; 47:34–8.



Response to Dr Caleb Armstrong: proposed Waikato medical school

Ross Lawrenson, Derek Wright, Ayla Thomas

r Armstrong¹ raises a number of points about the Waikato proposal for a third medical school, which we would like to respond to.

Firstly, the Waikato proposal has of course taken in to account the increased intake of medical students since 2002. However, these increased numbers do not overcome the long-standing shortfall in training numbers, which has resulted in the dependence on international medical graduates, and the attendant problems that Dr Armstrong has so ably described.

The reality is that we should be increasing the number of medical students taken in to medicine by 15 each year in order to keep up with the demand for doctors due to population growth, the increasing demands of an ageing population and the reduced working hours. So by 2025 we should be planning to accommodate another 100 medical students in to the New Zealand health system.

We are not proposing a zero sum game moving student numbers from one school to another. Rather we have proposed a model of training that will complement the existing programs and will help build training capacity within the system.

There is no doubt that the pressures on Waikato District Health Board in providing services to an increasing and high needs population has led to some less than ideal demands on our SMOs who also have training roles.

However, Waikato Hospital has been successfully providing clinical training for Auckland medical students for more than 20 years. It also has more than 350 junior staff, of whom 40% have been imported due to the lack of availability of New Zealand graduates.

The lack of proper investment in undergraduate training in our region means that the DHB is under resourced to provide the infrastructure required to be a tertiary training centre—when compared with Auckland, Dunedin, Christchurch and Wellington.

The Waikato proposal is for a four year graduate entry program—so that the bulk of the funding for the program actually will be spent on clinical training and will be supporting the clinical services to provide a better training environment—it certainly does not result in the University gaining "tens of millions per year" claimed by Dr Armstrong.

Waikato DHB has already taken up the suggestion of developing appropriate community placements to help encourage postgraduate trainees to consider general practice.

Our proposal also puts considerable additional resource into the development of community learning centres. We would argue that part of the pressure on the health services in our region is the 13% shortfall in doctors per head of population in our region, and our over reliance on international medical graduates.

As a DHB we have outlined a strategy to improve as a centre of excellence in education, training and research so we are better placed to meet the health needs of the population we serve. The medical school bid is an essential part of the strategy to becoming a centre of excellence for both postgraduate as well as undergraduate training.



Competing interests:

Nil.

Author information:

Ross Lawrenson, Waikato Clinical Campus, The University of Auckland, Hamilton; Derek Wright, Executive, Waikato DHB, Hamilton; Ayla Thomas, Media and Comms, Waikato DHB, Hamilton.

Corresponding author:

Derek Wright, Executive, Waikato DHB, Hamilton. derek.wright@waikatodhb.health.nz

URL:

http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1465-10-november-2017/7419

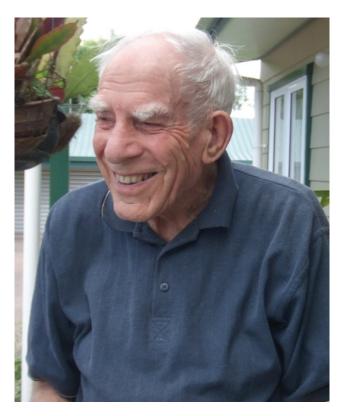
REFERENCES:

1. Armstrong C. Waikato District Health Board: a trustworthy custodian of New Zealand's future doctors? N Z Med J 2017;130(1464):75–76.



John Samuel Hopkirk

18 November 1922-24 September 2017



MB ChB (Otago 1945), FRCS (London 1951), FRACS, 1059

ohn was born in Kelburn to Cyril Spottiswoode Moy Hopkirk and Dorothy Kate (nee Saunders).

John's early life was coloured by his father's absence for veterinary science studies at Melbourne. Money was tight. At four he needed removal of an hydatid cyst from his lung. He was therefore educated at home for a year and this gave him a head start at Upper Hutt Primary School where he went directly into Standard One. His secondary schooling was at Wellington College and, at the age of seventeen, he entered Knox College to study medicine at Otago. He comments on student life:

"The lifestyle in Knox College in 1940 onwards was fundamentally one of persistent hard work. There was a war on, and we were reminded that we were a privileged group to be allowed to study at such a time. Many of our friends were in the forces, some were overseas and some died in the services. We

were reminded that if we failed to perform academically, we too would not continue to have the privilege of study. Also, because of the war, we were a young group of students: I had just turned 17 when I started. Knox was a college with strong traditions, closely allied to the Presbyterian Church by which it was governed, the master being a Presbyterian clergyman of high repute, as it was also the training school for Presbyterian ministers. Alcohol was not permitted, women only to a limited degree. Recreation was therefore different from the activities now deemed normal among students."

His final year and two house surgeon years were based in Wellington Hospital but with secondment to other places including the mining town of Denniston.

He comments on these years:

"It was a wise student who made friends with the ward sisters, and heeded their advice about patient management."



John became Surgical Registrar at Memorial Hospital, Hastings in 1948. He comments: "It was taken for granted that I would do all duties, including medicine and anaesthetics, while I wanted only surgery, so some negotiating took place. I finished up doing mostly surgery, but my anaesthetic expertise was used whenever a relaxant and intubation was required. I gave the first anaesthetic using curare in Hastings."

In Dunedin he had met Zephne Lepper, Home Science student on 4 June 1942 but was not engaged until 1947, and married on 5 April, 1948. Robin was born 24 August 1949 shortly before they embarked on MV Port Jackson for London where his parents were based at that time.

He worked in several places before obtaining his Primary in July 1950. Getting surgical experience was difficult but at Grantham he had the work to enable him to satisfy the FRCS examiners in November 1951.

He then worked at New End Hospital, Hampstead and was there for the last great smog in March 1952. "It was a blessing for London really, for the health results were so bad that measures were immediately taken to forbid the use of coal fires in Central London, and these were fully implemented."

Diane was born on 5 January 1953 and the future of the children's education was a major factor in their return to New Zealand on MV Port Hobart on 25 June that year. John immediately took up a position as Senior Surgical Registrar and Tutor at Wellington Hospital. The contacts he made proved invaluable but it became clear that the influx of returned military surgeons made staying in Wellington impractical.

He looked at provincial positions throughout the North Island before joining a Te Kuiti practice and becoming Surgeon Superintendent of the Hospital in July 1954. In 1955 their twin daughters, Philipa and Rosemary were born. Concern for family education was again a factor in the decision to return to Hastings for a two session/week appointment. General practice was necessary for survival and the first years were hard until he became established. His work was truly general, including orthopaedics, ENT, paediatric, O&G and vascular surgery. As more specialists arrived in Hastings he was able to reduce

his non-general surgical work but he always delighted in letting the author know when he was doing a gynaecological procedure.

These wide skills were necessitated by the distance from Auckland or Wellington and difficult transport. With time the vascular surgery became his sub-speciality:

"My work was my life, and the family revolved around it. Whether this was right or wrong for them, it was so. I was a young, enthusiastic surgeon, but the nature of the practice ensured that for many years I also had to conduct a type of general practice, to obtain surgical cases and to make a living."

In 1959 he obtained his Australasian Fellowship after sitting a Viva.

John's patients still remember him first for his sense of humour. His colleagues remember him for his ready availability, wisdom and skill. Sharing rooms with him was a delight and the post-clinic chats were always rewarding. Assisting him returning a newborn baby's heart to the chest from the abdomen was a privilege. That baby has since had her own family. His wideranging skills impressed the Sir James Wattie visiting professor in 1973, Professor HC Grillo (Harvard Medical School Chief of Thoracic surgery) who commented on John's skill and versatility.

He was active in local medical affairs, being President of the Hawke's Bay Division of the NZMA in 1973.

He continued to provide a sterling service and delayed his retirement by a year to 18 November, 1988 to enable his replacement John Flieschl to complete his vascular training.

John's love of surgery and the share market crash saw him working as locum from Balclutha to Kaitaia until he was forced to retire after a car accident on the way to Ashburton on 30 August 1992. His unique experience led to the following:

"Surgery in New Zealand has become more specialised and the population base to support scattered specialists is not there. Thus hospitals have to combine into regional institutions. The corollary is improved transport, not at present supplied from the health budget, so that the more indigent members of the population can no longer travel to get the treatment they require previously available in their community."



After full retirement, John did Extramural papers at Massey, usually getting an A pass, played bridge and joined Probus. He had found golf incompatible with his work and kept physically active in the garden.

Zephne's health deteriorated and despite John's own difficulties he remained her principal caregiver. They moved to Tauranga in 2014 and Zephne died in 2016. John steadily declined and died peacefully the next year. They were survived by their four children, 15 grandchildren and 17 great grandchildren.

This obituary was largely derived from John's memoirs, kindly made available by his family and with the assistance of Dr Stewart Drysdale, FRNZCGP.

Author information:

David Davidson, Retired Obstetrician and Gynaecologist, Hawke's Bay DHB, FRANZCOG. **URL:**



Lithium use in pregnancy and the risk of cardiac malformations

There has been concern that exposure to lithium early in pregnancy may be associated with a marked increase in the risk of Ebstein's anomaly and other congenital cardiac abnormalities.

As the previous data are conflicting, this cohort study was performed. The study group included over 1.3 million pregnancies. The researchers examined the risk of cardiac malformations among infants exposed to lithium during the first trimester as compared with unexposed infants and, in secondary analyses, with infants exposed to another commonly used mood stabiliser, lamotrigine.

Maternal use of lithium during the first trimester was associated with an increased risk of cardiac malformations, including Ebstein's anomaly as compared with the unexposed infants. Results were similar when compared to the lamotrigine exposed infants. However, the magnitude of this effect was smaller than had been previously postulated.

N Engl J Med 2017; 376:2245-54

Adverse events associated with statin therapy?

These researchers speculate that adverse events (AEs), particularly muscle pain and weakness, to statins have been overemphasised.

To elucidate this matter, they have reviewed data from two large trials. In a blinded randomised trial treatment when atorvastatin was compared with a placebo, there was no difference in the incidence of muscle-related AEs. However, in a non-blinded non-randomised trial of atorvastatin versus placebo the incidence of muscle-related AEs was significantly higher in those taking the statin.

These analyses illustrate the so-called nocebo effect, with an excess rate of muscle-related AE reports only when patients and their doctors were aware that statin therapy was being used and not when its use was blinded. These results will help assure both physicians and patients that most AEs associated with statins are not causally related to use of the drug and should help counter the adverse effect on public health of exaggerated claims about statin-related side effects.

Lancet 2017; 389:2473-81

Prenatal antidepressant use and risk of attention-deficit/hyperactivity disorder in offspring

Does prenatal use of antidepressants increase the risk of attention-deficit/hyperactivity disorder (ADHD) in offspring?

This population-based cohort study involved nearly 200,000 children. Three percent of them had a diagnosis of ADHD. Children whose mothers used antidepressants in the prenatal period were compared with those of mothers who did not. Children whose mothers used antidepressants before their pregnancy were also compared with non-users.

The incidence of ADHD was increased in those whose mothers used prenatal antidepressants (hazard ratio 1.39). Similar results were found in the children of those treated with antidepressants before pregnancy.

BMJ 2017; 357:j2350

URL:



Ambroise Paré, Army Surgeon

December 1917



In the dawn of history the birthplace of surgery was the battlefield. The son of Aesculapius, Machaon, he who dressed the wounds of Menelaus, was put under the special care of Nestor, for the Greeks considered a doctor to be worth in battle many soldiers.

In the Renaissance period Paré became, if not the father of modern surgery, the greatest of military surgeons. As a barber's apprentice he left the provinces for Paris in 1529, and was employed as a dresser at the Hotel Dieu. He began practice in a modest room near the favourite duelling-ground, and after eight years' residence in Paris became an Army surgeon, and served in the wars for nearly twenty years, first of all in the campaign of Francis I. In Italy. His experience in the field, and the use he made of it, gave him a great practice and a great name in Paris.

Let us consider first what manner of man he was. Neither birth nor education were his, but he had the advantage of freedom from the deadening influence of the University of Paris of that time. He wrote a great deal and his books are full of interesting cases, good stories of the camp and of the city, shrewd opinions, and contain much in the style of Pepys and Boswell. Paré, the Huegenot, is in striking contrast to his grand patients of the Court, the mad line of Kings, the Duke of Guise, Catherine Medici, the marguises, the courtiers, and the rest. He knew Vesalius and Mary Stuart and found a kindred spirit in Admiral Coligny. Piety permeated his life and found expression in his writings and in the oft-repeated phrase, "Je le pansay et Dieu le guarit"—I dressed him and God cured him. On the night of the Massacre of St. Bartholomew the King himself locked Paré in a room in the Louvre,



swearing that it was not reasonable that a man who was worth a world of men should be murdered. One of the last and greatest scenes in the life of this noble man reveals him, burdened with the weight of eighty years, tramping the hot and noisome streets of Paris, relieving the sick and poor when the city was besieged and withstanding the fury of the League and the power of the relentless Archbishop of Lyon. The dead lay unburied in the streets and the anguish and hunger of the citizens had reached the limit of human endurance. It was Master Ambroise Paré who met the Archbishop face to face in the street and called upon him to have mercy on the citizens, as he himself hoped for divine mercy. The Archbishop said afterwards that Paré had waked him up and made him think of many things, "On Thursday, December the twentieth, died Master Ambroise Paré, the King's surgeon, eighty years old; a learned man and the chief of all surgeons; who, even against the times, all his life talked and spoke openly for peace and for the people: which made him as much beloved by the good as he was opposed and hated by the wicked."

But it is Paré's work and invention as an Army doctor that appeals to us now in the days of the Great War. For some centuries in mediaeval times the surgeon had no honour, and the art was relegated to barbers, bathkeepers, sow-gelders, and mountebanks. In Prussia up to the time of Frederick the Great it was one of the duties of the Army surgeon to shave the combatant officers. But Paré had great honour. His ready sympathy endeared him to his comrades, and we read that one night he entered Metz incognito, but, being recognised, he was carried through the city by the soldiers in triumph. His reputation as an Army surgeon was built upon the sure foundation of efficiency, and his skill was a powerful aid to the Army. He seemed to exemplify the teaching of Locke, the philosopher-physician of a somewhat later period, that ideas are not innate, but that knowledge proceeds from investigation through the bodily senses. No detail was too small for his attention, and this of necessity, seeing that he had no trained nurses to depend upon. Indeed, Charles IX., dying of consumption, and the ghosts of the Massacre ever before his eyes, had no nurse save an old woman who had nursed him when he was a child, and she composed herself to sleep, as history informs us, when she was dying.

At the beginning of his career in the Army Paré's great discovery was due mainly, as he informs us, to chance. At Suse, near Mont Cenis, in 1537,

"The enemy within the castle, seeing our men come on them with great fury, did all that they could to defend themselves, and killed and wounded many of our soldiers with pikes, arquebuses, and stones: whereby the surgeons had all their work cut out for them. Now, I was at this time a freshwater soldier; I had not yet seen gunshot wounds at the first dressing. I had read in 'John de Vigo,' book one, 'Of Wounds in General,' chapter eight, that wounds made by firearms partake of venenosity, by reason of the gunpowder; and for their cure he bids you cauterise them with oil of elders, scalding hot, mixed with a little treacle. And to make no mistake, before I would use the said oil, knowing that it was to bring great pain to the patient, I asked first, before I applied it, what other surgeons used for a first dressing; which was to put the said oil, boiling, well into the wounds, with tents and setons: wherefore I took courage to do as they did. At last my oil ran short, and I was compelled, instead of it, to apply a digestive made of yolks of eggs, oil of roses and turpentine. In the night I could not sleep in quiet, fearing some default in the not cauterising, lest I should find those to whom I had not applied the said oil dead from the poison of their wounds; which made me rise very early to visit them: where, beyond my expectation, I found that they to whom I had applied my digestive had suffered but little pain, and their wounds without inflammation or swelling, having rested fairly well that night. The others, to whom the boiling oil was applied, I found feverish, with great pain, and swelling round the edges of their wounds. Then I resolved never more to burn thus cruelly poor men with gunshot wounds... See how I learned to treat gunshot wounds: not out of books."

There was a surgeon at Turin, famed above all the rest for his treatment of gunshot wounds, to whom Paré paid court for two years before he would disclose his recipe, and then only after many gifts. "Then I was joyful," writes Paré, "and my heart made glad that I had learned his remedy, which was like that which I had obtained my chance."

His second great discovery, or rather rediscovery, for it was at one time in the



practice of the ancients, was the use of ligature instead of red-hot irons to stop the bleeding of an amputation. "In 1552," he writes, "it pleased God to teach me, without I had ever seen it done in any case, no, nor read of it." In both of these discoveries he showed the fallacy of the orthodox practice based upon the pseudo Hippocratic aphorism that diseases not curable by iron are curable by fire.

What else do we owe to Paré? He invented many surgical instruments, popularised the use of a truss in cases of hernia, omitted castration as a routine part of the operation of herniotomy, advised massage, introduced artificial eyes made of gold or silver, and in 1536 performed the first excision of the elbow joint. He pointed to syphilis as the cause of aneurysm, and Howard Kelly gives to Paré the credit of assigning to flies a

great part in the transmission of infectious diseases. By ligaturing the large arteries Paré was enabled to perform many amputations, and his treatment of wounds lessened the mortality of war, well named by Pirogoff, after his experience in the Crimea, a "traumatic epidemic."

The great advance made in military surgery in the present day would not have been possible had it not been for the work of Simpson, Lister, Hunter, and Ambroise Paré of an army that has been dust and rust for three centuries. Some may say, Lister we know, and Hunter we know, but who is Paré? Let this short sketch whet the appetite of the reader for further knowledge of one of the most notable and most lovable pioneers in our art, who, fortunately, has revealed not only his thoughts but also his personality in his writings.

URL:



Leptospirosis in three workers on a dairy farm with unvaccinated cattle

Jacqueline Benschop, Julie Collins-Emerson, Allie Maskill, Patrick O'Connor, Margaret Tunbridge, Yuni Yupiana, Jenny Weston

Published: 22 September 2017 (Vol 130 No 1462)

In the first published version of this manuscript, two corrections for the above article were sent in post-publication:

- To add to the footnote of the table to attribute the work performed at ESR.
- To replace "hardjo-bovis" with "hardjo" throughout the text.

This was resolved online and in the PDF on 7 November 2017.

The state of quality improvement and patient safety teaching in health professional education in New Zealand

Gillian Robb, Susan Wells, Iwona Stolarek, Gillian Bohm

Published: 27 October 2017 (Vol 130 No 1464)

In the first published version of this manuscript, the order of the authors of the above article was incorrect. The correct order is: Gillian Robb, Susan Wells, Iwona Stolarek, Gillian Bohm.

This was resolved online and in the PDF on 7 November 2017.

URL:

