

The  
**New Zealand  
Medical Journal**

Journal of the New Zealand Medical Association

Vol 130 | No 1450 | 17 February 2017

A photograph of a woman with long dark hair holding a young child in a striped shirt. They are standing in a grassy field with trees in the background, and the scene is backlit by a bright sun, creating a warm, golden glow and lens flare effects.

**Patchy advances in child health hide a systematic failure to prioritise children in public policy**

Six new studies about diabetes: what can we learn that might benefit Māori and Pacific people?

**Proposed new industry code on unhealthy food marketing to children and young people: will it make a difference?**

**Ethnic and geographic variations in the incidence of pancreatitis and post-pancreatitis diabetes mellitus in New Zealand**

**Screening for diabetes in pregnancy in a regional area with a high Māori population**

---

The  
**New Zealand  
Medical Journal**  
Publication Information

published by the New Zealand Medical Association

---

**NZMA Chairman**

Dr Stephen Child

To contribute to the *NZMJ*, first read:  
[www.nzma.org.nz/journal/contribute](http://www.nzma.org.nz/journal/contribute)

**NZMJ Editor**

Professor Frank Frizelle

**Other enquiries to:**

NZMA  
PO Box 156  
The Terrace  
Wellington 6140  
Phone: (04) 472 4741

**NZMA Communications Manager**

Sharon Cuzens

**NZMJ Production Editor**

Rory Stewart

© NZMA 2016

---

To subscribe to the *NZMJ*, email  
[julie@nzma.org.nz](mailto: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 *NZMJ* 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](http://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](mailto:julie@nzma.org.nz)

The NZMA also publishes the *NZMJ 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**

Individuals*	\$306
Institutions	\$530
Individual article	\$25

**Overseas subscription rates**

Individual	\$426
Institutions	\$571
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](mailto:julie@nzma.org.nz) for more information.

Individual articles are available for purchase by emailing [nzmj@nzma.org.nz](mailto:nzmj@nzma.org.nz)

---

EDITORIAL

**8**

Six new studies about diabetes:  
what can we learn that might  
benefit Māori and Pacific people?  
Timothy W Kenealy, Nicolette F  
Sheridan, Brandon J Orr-Walker

**12**

Patchy advances in child health  
hide a systematic failure to  
prioritise children in public policy  
Amanda J D'Souza, Louise Signal,  
Richard Edwards

ARTICLES

**16**

23 years of managing diabetic  
ketoacidosis at Auckland Hospital  
Geoffrey Braatvedt, Amelia Tekiteki,  
Holly Britton, John Wallace, Manish  
Khanolkar

**25**

Screening for diabetes in  
pregnancy in a regional area with  
a high Māori population  
Barbara Daly, Isabel Raiman, Jennifer  
Goodson

**32**

Unhealthy food marketing to New  
Zealand children and adolescents  
through the internet  
Stefanie Vandevijvere, Karuna Sagar,  
Bridget Kelly, Boyd Swinburn

**44**

Psychosocial enhancement of the  
Green Prescription for obesity  
recovery: a randomised controlled  
trial  
Doug Sellman, Ria Schroder, Daryle  
Deering, Jane Elmslie, James Foulds,  
Chris Frampton

**55**

Ethnic and geographic variations  
in the incidence of pancreatitis  
and post-pancreatitis diabetes  
mellitus in New Zealand: a  
nationwide population-based  
study  
Sayali A Pendharkar, Juby Mathew,  
Jinfeng Zhao, John A Windsor, Daniel J  
Exeter, Maxim S Petrov

**69**

Long-term survival following  
diabetic vitrectomy  
Bia Z Kim, Kuo-Luong Lee, Stephen J  
Guest, David Worsley

**78**

Prevalence of diabetic retinopathy  
at first presentation to the retinal  
screening service in the greater  
Wellington region of New Zealand  
2006–2015, and implications for  
models of retinal screening  
Lily YL Chang, Arier C Lee, Wilson Sue

**89**

Discharge outcomes of patients  
referred to specialist eye clinic  
from diabetic retinopathy  
screening in Northland (2014–15)  
Pragnya Jagadish, David Dalziel

VIEWPOINT

**94**

Proposed new industry code on  
unhealthy food marketing to  
children and young people: will it  
make a difference?  
Boyd Swinburn, Stefanie Vandevijvere  
on behalf of submitting health  
professors

CLINICAL CORRESPONDENCE

**102**

Giant cell arteritis treatment failure  
resulting from probable steroid/  
antiepileptic drug-drug interaction  
Eoin Mulroy, John Highton, Sarah Jordan

LETTERS

**105**

High incidence of medulloblastoma  
in Māori and Pacific populations in  
New Zealand

J Mark Elwood, Phyu Sin Aye

**107**

High levels of rheumatic fever and  
sore throat awareness among a  
high-risk population screened for  
rheumatic heart disease

Jason K Gurney, Angela Chong, Nicola  
Culliford-Semmens, Elizabeth Tilton,  
Nigel J Wilson, Diana Sarfati

METHUSELAH

**111**

Community-acquired pneumonia  
incidence before and after proton  
pump inhibitor prescription

100 YEARS AGO

**112**

Sir Walter Raleigh's Nostrum  
April 1917

PROCEEDINGS

**113**

Selected proceedings of the APAC  
(Asia Pacific) Forum  
2016

## 23 years of managing diabetic ketoacidosis at Auckland Hospital

Geoffrey Braatvedt, Amelia Tekiteki, Holly Britton, John Wallace, Manish Khanolkar

We aim to examine the length of stay and need for intensive care of people admitted with diabetic ketoacidosis (DKA) to a single centre between 1988 and 2011. DKA remains an important reason for admission to this hospital, but the severity of DKA at presentation has reduced over time. The need for intensive care admission and length of stay has fallen dramatically.

---

## Screening for diabetes in pregnancy in a regional area with a high Māori population

Barbara Daly, Isabel Raiman, Jennifer Goodson

Gestational diabetes is increasing globally mainly due to the obesity epidemic. The Ministry of Health recommends that all pregnant women are screened for prediabetes and type 2 diabetes in early pregnancy, and for women with blood glucose levels, to be screened for gestational diabetes to reduce maternal and infant complications during pregnancy and birth. Despite this recommendation, only 56% of Māori women compared with 76% of European women were screened for diabetes in a regional area with a high Māori population. It is important for women to know if they have prediabetes or diabetes in pregnancy so they can receive appropriate support and health care to ensure a healthy pregnancy and infant, and to make lifestyle changes to reduce their risk of getting type 2 diabetes following birth.

---

## Unhealthy food marketing to New Zealand children and adolescents through the internet

Stefanie Vandevijvere, Karuna Sagar, Bridget Kelly, Boyd Swinburn

Compared to traditional media, the internet allows food marketers to use engaging techniques to directly interact with children. A wide range of marketing techniques and features were identified on food brand websites. Food marketing on popular non-food websites among children and adolescents was however low. Additional assessment of food marketing to children through social and other digital media is recommended.

---

## Psychosocial enhancement of the Green Prescription for obesity recovery: a randomised controlled trial

Doug Sellman, Ria Schroder, Daryle Deering, Jane Elmslie, James Foulds, Chris Frampton

Obesity is arguably the number one health problem in New Zealand, although treatment options are limited. The Green Prescription is a key government-funded health promotion programme for which the most common referrals are people with overweight problems. This study found that Green Prescription alone brought about an average of less than 1kg per participant (0.7kg) at 12 months. However, when Kia Ākina was added, the weight-loss achieved was 5x greater, along with improved confidence about recovering from obesity in the future, better quality of life measures and overall satisfaction with the assistance received by participants. Kia Ākina is a small low-cost recovery network based on addiction principles, which offers ongoing support and encouragement to its participants. Further research and development of Kia Ākina to improve its weight-loss outcomes is needed before it is expanded.

---

## Ethnic and geographic variations in the incidence of pancreatitis and post-pancreatitis diabetes mellitus in New Zealand: a nationwide population-based study

Sayali A Pendharkar, Juby Mathew, Jinfeng Zhao, John A Windsor, Daniel J Exeter, Maxim S Petrov

Previous research on pancreatitis in New Zealand was under-representative of minorities and underserved populations, particularly the Māori and Pacific people. Also, a recent study by World Health Organization showed that population-based data on acute pancreatitis and chronic pancreatitis are available for every region except for Australasia and Africa. Given that Māori and Pacific people are most prone to developing chronic metabolic diseases, determining true nationwide burden of these diseases is important, as these diseases have a large impact on healthcare policies and budgets. Awareness of these diseases is crucial to help prevent and develop treatment strategies for everyone, but particularly for Māori and Pacific people, who are worst affected.

---

## Long-term survival following diabetic vitrectomy

Bia Z Kim, Kuo-Luong Lee, Stephen J Guest, David Worsley

Complications of diabetes in the eye can be vision-threatening but also life-threatening. Nearly a third of diabetic patients undergoing surgery for the ocular complications die within five years following surgery, at around 60 years of age. The poor survival rates are associated with severe systemic complications, such as kidney disease. These complications of diabetes are preventable with careful regular monitoring and appropriate treatment that is closely shared across various medical specialties.

---

## Prevalence of diabetic retinopathy at first presentation to the retinal screening service in the greater Wellington region of New Zealand 2006–2015, and implications for models of retinal screening

Lily YL Chang, Arier C Lee, Wilson Sue

Blindness due to diabetic retinopathy (DR) is the most common cause of newly reported cases of visual loss in working-age adults (20–74 years of age), which has debilitating implications to daily living, as well as the ability to remain in the workforce. The aim of the paper is to provide New Zealand-specific prevalence of DR in patients at first presentation for eye screening in the greater Wellington region, with the intent of service evaluation. This is a retrospective study using data collected from patients newly referred DR screening between 2006–2015. This study identified a large proportion (97.7%) of patients with non-sight-threatening or no DR, who can be managed in the community by primary care providers, and do not require referral to secondary care ophthalmology. In addition to early detection of DR, the optometrist-based DR screening service in the greater Wellington region is an early opportunity for patient education, and provides a feasible solution to alleviate the volume of DR screening that currently strains ophthalmology services in public hospitals.

### Discharge outcomes of patients referred to specialist eye clinic from diabetic retinopathy screening in Northland (2014–15)

Pragnya Jagadish, David Dalziel

The study was a retrospective analysis of 98 patients seen at specialist eye clinic after being referred from diabetic retinopathy screening (DRS). We found that 45% of the patients were re-enrolled back into DRS after being seen in specialist clinic and 49% stayed under specialist clinic for further treatment. Only 5.9% patients were not re-enrolled back to DRS after being seen in specialist eye clinic. Non-attendance at clinic appointments was high among the Māori population.

---

### Proposed new industry code on unhealthy food marketing to children and young people: will it make a difference?

Boyd Swinburn, Stefanie Vandevijvere on behalf of submitting health professors

The Advertising Standards Authority recently reviewed its self-regulatory codes on food marketing to children and have proposed a new single code. Over 70 health professors provided a submission to this process, and in this paper analyse the proposed new code. They find that there are no improvements or uncertain improvements over the previous codes and that children will continue to be heavily exposed to unhealthy food marketing—especially adolescents (not covered in the code), through sponsorship and package labelling (excluded from the code) and at peak TV viewing hours and through social media (unsatisfactory definitions in the code). The health professors are calling for government to take the lead and develop a stronger regulatory environment to protect children.

---

# Six new studies about diabetes: what can we learn that might benefit Māori and Pacific people?

Timothy W Kenealy, Nicolette F Sheridan, Brandon J Orr-Walker

This edition of the Journal includes five observational papers on aspects of diabetes epidemiology and service delivery in New Zealand, and one randomised controlled trial of an obesity intervention. We shall refer to these as papers on retinopathy,<sup>1</sup> vitrectomy,<sup>2</sup> pancreatitis,<sup>3</sup> gestational diabetes (GDM),<sup>4</sup> diabetic ketoacidosis (DKA)<sup>5</sup> and enhanced Green Prescription (the obesity intervention trial).<sup>6</sup> The papers collectively represent a welcome contribution to the diabetes literature. It is heartening to see audit and research into diabetes in New Zealand across a wide range of topics, disciplines and institutions.

We have chosen to consider what these papers say, and where they could lead, in respect to one of the most intractable problems with diabetes in New Zealand—the unfair burden of diabetes on Māori and Pacific peoples. This disproportionate burden has been reported for at least 30 years<sup>7</sup> and Māori diabetes has been a national priority since at least 2001.<sup>8</sup> Despite real efforts and some successes,<sup>9,10</sup> even those who have contributed enormously will agree that the net effect remains incomplete and inadequate. We consider each paper in turn, recognising that we do not necessarily address the issues of central interest to the authors.

*Retinopathy paper.*<sup>1</sup> Māori and Pacific people attending for first retinal screening have similar or higher rates of retinopathy than New Zealand European. The only potentially changeable risk factor identified was improved glucose control. Retinopathy rates were calculated after excluding 28% of people due to incomplete data and the 27% who did not attend, including 44% of Māori who did not attend. The authors

muster arguments for a community optometrist-based retinal screening. We would argue that no service is acceptable with this level of missing data and non-attendance. Both issues need attention that is likely to involve further audit and research.

*Vitrectomy paper.*<sup>2</sup> Māori have a disproportionate rate of vitrectomy, and non-New Zealand European have reduced survival after vitrectomy (hazard ratio 2.2 for mortality compared with New Zealand European). The only potentially changeable risk factor identified was an association with renal failure, itself dependent on blood pressure, glucose control and other known risk factors including ethnicity. We already know that Māori and Pacific develop diabetes up to 10 years earlier than New Zealand European, and that they progress faster to cardiovascular disease<sup>11</sup> and renal failure.<sup>12</sup> At the level of the individual patient, vitrectomy is a marker on the road to perdition and should trigger specialist diabetes review. At a population level the vitrectomy data supports a strong case for preferential allocation of people and resources, and probably new population-based and community-based strategies, to address Māori and Pacific diabetes.

*Pancreatitis paper.*<sup>3</sup> Māori have the highest reported rate of acute pancreatitis worldwide. No potentially changeable risk factors were identified in the data analysis, although uneven geographical distribution may point to regional differences in service access and delivery, and the literature cited suggested other risk factors including alcohol. Overall rates of hospital admission for pancreatitis are likely to be accurate. Distinction between acute and chronic pancreatitis, based on discharge codes,

must be less definitive, and a diagnosis of diabetes any time after pancreatitis must be a tenuous link as diabetes is much more common than pancreatitis. Nevertheless, our comments are essentially the same as for vitrectomy—hospital admission for pancreatitis or diabetes should trigger a specialist diabetes review.

*GDM paper.*<sup>4</sup> Screening rates for GDM were 76% for New Zealand European women and 56% for Māori women. No potentially changeable risk factors were identified. The obvious first step to improving Māori screening rates is to establish universal screening, rather than targeted screening, in line with current guidelines.<sup>13</sup> After that it is important to re-audit screening rates by ethnicity and actively manage any ongoing differential. This may require prioritising resources to screening Māori women.

*DKA paper.*<sup>5</sup> Māori and Pacific rates of DKA in adults have not obviously changed over time, but this condition is primarily due to type 1 diabetes where the proportions of Māori and Pacific are relatively low. It remains important to routinely cut audit and research results by ethnicity.

*Green Prescription paper.*<sup>6</sup> Most of the participants did not have diabetes (personal communication D Sellman, 2017) but mean BMI was about 40, and any intervention that can improve obesity at this level is relevant to diabetes prevention and management. The study was well-conceived and well-conducted and, given the theoretical basis of the trial, it was reasonable to combine ethnic groups. However, in trials where intervention effect might be different by ethnicity, ie most trials, we should include sufficient numbers of Māori and/or Pacific people to achieve “equal explanatory power” compared to New Zealand European.<sup>14</sup>

If the purpose of health services research is to “pursue knowledge that will inform and influence health policy, practice and service innovation”,<sup>15</sup> whose job is it to take the findings from these papers and elsewhere and work to improve outcomes for Māori and Pacific with diabetes? Firstly, all of us. “One has a moral obligation to consider helping alleviate suffering of individuals wherever one finds them, in light of and commensurate to one’s capacity to do so. In

other words, one ought to help if one can and to the extent one can.”<sup>16</sup> Secondly, we would argue that the academic community has its own specific imperative to translate their findings into real-world justice.<sup>17</sup> We find the current papers, as a group, relatively weak on implications in general, especially as they relate to Māori and Pacific diabetes.

A raft of known service strategies could help address the burden of diabetes for Māori and Pacific. We have noted the need for universal screening for GDM, for using clinical events to trigger specialist review and the need to prioritise resources to Māori and Pacific. A longer list includes using community health workers, self-management support, devolving services to Māori and Pacific providers, health services that are free to the user, systematic (universal) processes in primary and secondary care, tighter integration between primary and secondary care, and public reporting of service and outcome measures from primary and secondary care.

Each of these service strategies can be tested and refined by audit and research. We have already noted the need for routine and repeat audits, for routinely cutting audit and research data by ethnicity, and including sufficient numbers of Māori and Pacific in intervention trials. We suggest that audit and cross sectional studies have maximum value only when repeated after an attempt to improve practice, and longitudinal studies have maximum value only when they can explore the effects of service change. Research funders and commissioners could more actively mandate dissemination and implementation of existing research and new findings as they arise. The remaining requirements for the researcher consist of nothing less than excellent practice that includes: selecting a research topic based on potential to improve service or fairness; collecting data on factors that may be explanatory and changeable and consider these factors in their literature review, analysis and discussion; and actively disseminate findings to policy makers, funders and service providers.

The remaining requirements for policy makers, funders and service providers are to wisely enact the “best-available” evidence, rather than waiting for “best” evidence before changing policy or practice. They

might reflect that the status quo generally has a minimal evidence base and that optimal health service delivery is likely to be accumulated from decades of incremental, systematic research, not something tried once then dropped.<sup>18</sup>

The role of audit and research in respect to health service and equity is to look at

what we are doing to see if we can improve it. In addressing the unfair burden of diabetes on Māori and Pacific, we see excellent practice, and excellent research and evaluation, already in use sometimes in some places. Our plea is for more universal application, “doing science to reduce social inequalities in health while also doing excellent science.”<sup>16</sup>

**Competing interests:**

Nil.

**Author information:**

Timothy W Kenealy, Associate Professor of Integrated Care, Department of Medicine, University of Auckland, Auckland; Nicolette F Sheridan, Associate Dean Equity, Faculty of Medical and Health Science, Associate Professor, School of Nursing, University of Auckland, Auckland; Brandon J Orr-Walker, Endocrinologist and Clinical Head Department of Endocrinology and Diabetes, Division of Medicine, Counties Manukau District Health Board, Auckland.

**Corresponding author:**

Timothy Kenealy, Associate Professor of Integrated Care, University of Auckland South Auckland Clinical School, Middlemore Hospital, Private Bag 93311, Otahuhu, Auckland 1640.

t.kenealy@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7153>

**REFERENCES:**

1. Chang LL, Lee A, Wilson S. Prevalence of diabetic retinopathy at first presentation to the retinal screening service in the greater Wellington region of New Zealand 2006–2015, and implications for models of retinal screening. *NZ Med J* 2017; 130(1450):78–88.
2. Kim B, Lee KL, Guest S, Worsley D. Long-term survival following diabetic vitrectomy. *NZ Med J* 2017; 130(1450):69–77.
3. Pendharkar SA, Matthew J, Zhao J, Windsor JA, Exeter DJ, Petrov MS. Ethnic and Geographic Variations in the Incidence of Pancreatitis and Diabetes After Pancreatitis in New Zealand: A Nationwide Population-Based Study. *NZ Med J* 2017; 130(1450):55–68.
4. Daly B, Raiman I, Goodson J. Factors Associated with Screening for Diabetes in Pregnancy in a predominantly Māori population. *NZ Med J* 2017; 130(1450):25–31.
5. Braatvedt G, Tekiteki A, Britton H, Wallace J, Khanolkar M. 23 years of managing diabetic ketoacidosis at Auckland Hospital. *NZ Med J* 2017; 130(1450):16–24.
6. Sellman D, Schroder R, Deering D, Elmslie J, Foulds J, Frampton C. Psychosocial enhancement of the Green Prescription for Obesity Recovery: A Randomized Controlled Trial. *NZ Med J* 2017; 130(1450):44–54.
7. Issacs R, Scott D. Diabetic patient discharges from Middlemore Hospital in 1983. *N Z Med J*. 1987; 100:629–631.
8. King A. He Korowai Oranga: Maori Health Strategy. Wellington: Ministry of Health;2001.
9. Kenealy T, Eggleton K, Sheridan N, Robinson E. Systematic care to reduce ethnic disparities in diabetes care. *Diabetes Res Clin Pract*. 2010; 89:256–261.
10. Tipene-Leach DC, Coppell KJ, Abel S, Pahau HL, Ehau T, Mann JI. Ngati and healthy: translating diabetes prevention evidence into community action. *Ethn Health*. 2013; 18(4):402–414.
11. Kenealy T, Elley C, Robinson E, et al. An association between ethnicity and cardiovascular outcomes for people with type 2 diabetes in

- New Zealand. *Diabet Med.* 2008; 25:1302–1308.
12. Elley CR, Robinson T, Moyes S, et al. Derivation and Validation of a Renal Risk Score for People with Type 2 Diabetes. *Diabetes Care.* 2013; 36(10):3113–3120.
  13. Ministry of Health. Screening, Diagnosis and Management of Gestational Diabetes in New Zealand: A clinical practice guideline. Wellington: Ministry of Health; 2014.
  14. Te Rōpū Rangahau Hauora a Eru Pōmare. Mana Whakamārama - Equal Explanatory Power: Māori and non-Māori sample size in national health surveys. Wellington: Te Rōpū Rangahau Hauora a Eru Pōmare, Wellington School of Medicine and Health Sciences, University of Otago; 2002.
  15. den Hertog P, van der Wietze A, de Jong MW. Capabilities for managing service innovation: towards a conceptual framework. *Journal of Service Management.* 2010; 21(4):490–514.
  16. Venkatapuram S. Justice and Global Health Research. *Am J Bioeth.* 2016; 16(10):46–47.
  17. Kenealy T. How universities can help create a wiser world: The urgent need for an academic revolution. Commentary on Nicholas Maxwell. *J Eval Clin Pract.* 2014; 21(3):535–537.
  18. Gawande A. Testing, testing. The health-care bill has no master plan for curbing costs. Is that a bad thing? *The New Yorker Digital Edition* 2009.

# Patchy advances in child health hide a systematic failure to prioritise children in public policy

Amanda J D'Souza, Louise Signal, Richard Edwards

Recent years have seen some welcome advances in child health in New Zealand, and some hard-fought gains in public policy. These achievements should be recognised and celebrated. For example, once unbelievably low, immunisation coverage is now soaring. Children have better legal protection from assault and better access to primary care. New Zealand's child mortality rate has declined, although it is still twice that of the best-performing countries. There have also been encouraging developments relating to whānau ora and vulnerable children, including structural changes for the child protection system. A promising change is the creation of 'VOYCE - Whakarongo Mai', an independent organisation to ensure the voices of children in care are heard. Notably, the new Prime Minister Bill English appointed a Minister for Children in his Cabinet reshuffle in December, although it is unclear the extent to which this role will be focused on all children and accompanied by a dedicated government unit or Ministry with sufficient resourcing to introduce real change.

However, important questions remain such as: to what extent has overall child health improved; are the underlying social and environmental drivers of health and wellbeing being adequately addressed; and does New Zealand have a political culture and policy process that appropriately prioritises the wellbeing of children?

Childhood obesity has increased significantly in New Zealand in recent years. It is an example of a specific health issue for which effective action is urgently needed. The Childhood Obesity Plan, launched by the Health Minister in 2015, was a welcome development. Unfortunately, the plan

neglects to include many of the important evidence-based policy interventions recommended by the World Health Organization's Commission on Ending Childhood Obesity, chaired by Sir Professor Peter Gluckman, the Prime Minister's Chief Science Advisor.<sup>1</sup> That Commission called for comprehensive government-led action that encompassed the creation of healthy food environments for children, through policy interventions like a sugary drinks tax, and reduced exposure of children to unhealthy food and beverage marketing.

This week's letter from 73 health professors highlights the New Zealand Government's failure to act on this advice.<sup>2</sup> The Professors decry the failure to protect New Zealand's children from unhealthy food and other marketing. The increasing ubiquity of this marketing and complexity of the policy task is highlighted by Vandevijvere and colleagues<sup>3</sup> in their new study outlining unhealthy food marketing on the Internet and in new media, and by Chambers et al,<sup>4</sup> highlighting how sports sponsorship is used to advertise alcohol, including to children.

Unfortunately, such specific policy inadequacies are repeated by failures to effectively address key social and economic determinants of child health and wellbeing, such as child poverty and inadequate housing. These are not hypothetical concerns; New Zealand's once proud reputation of being a great country for children has been seriously eroded over the last 25 years.<sup>5</sup> Despite good evidence of its far-reaching consequences, the most recent data from the Ministry of Social Development show that 28% of New Zealand children still live in households that meet

the criteria for income poverty.<sup>6</sup> Homelessness has increased and more than half of the 41,000 homeless New Zealanders are children and youth.<sup>7</sup> Almost half of all Pacific children live in overcrowded housing.<sup>8</sup> New Zealand is yet to adequately address the enormous rise in serious infectious diseases in childhood that started in the 1990s.<sup>9</sup> Indeed, childhood hospitalisation rates for serious skin infections, bronchiolitis, bronchiectasis and dental caries increased during the 2000s.<sup>8,10</sup> Compared to other advanced economies, New Zealand has one of the highest rates of child maltreatment, the third highest rate of child obesity and the highest rate of youth suicide. Huge ethnic and socioeconomic disparities persist for all of these conditions.<sup>8,10</sup>

The causes of these problems are likely complex, however, many of New Zealand's current policy settings represent missed opportunities for disease prevention and the promotion of healthy development and child wellbeing. This suggests that protecting children's wellbeing and supporting parents and families has inadequate priority within the political and policy process. Too many children, parents and caregivers are living in stressful circumstances that are hazardous to health, and do not have sufficient access to resources, supports and health-promoting environments.<sup>11</sup> There is increasing recognition of systemic institutional failures; the maltreatment of children in the care and protection system is especially salient, however, the lack of a child-centred approach to policy formulation appears more widespread.

This month the Children's Commissioner expressed shock at the lack of consultation with children regarding amendments to the Education Act despite children being primary stakeholders.<sup>12</sup> As yet there is little indication of meaningful poverty reduction; a task hindered by a lack of a coherent plan, targets and substantive investment.<sup>5</sup> The lack of action on climate change can also be viewed as a failure to prioritise children's futures.<sup>13</sup> Not only do these suboptimal policy settings not serve children and their families well, they are associated with considerable social and economic costs to the nation.

It is apparent that children's wellbeing, and the concept of children as rights-bearers, are yet to become embedded as

'business as usual' within public law and institutional policies and practices. New Zealand could look to other countries that do better for children. For example, Sweden has a culture of prioritising children in policy-making, supported by specific laws and institutional mechanisms that promote the consideration of children's rights in policy; there is a broad-based consensus ensuring the longevity of this approach.<sup>14</sup> Some countries, such as Norway, have incorporated the UN Convention of the Rights of the Child (UNCRC) into domestic human rights legislation. New Zealand children could benefit from such an approach. However, last year the UN Committee on the Rights of the Child was concerned that 25 years on from ratifying UNCRC the New Zealand Government had yet to develop and implement a coherent national plan of action for all children.<sup>15</sup> UNCRC aims to give structure and effect to what is a moral responsibility towards children and is based on their developmental needs. Not only does it help policy-makers to make progress towards appropriate protections and provisions for children and their caregivers, it prompts thinking about children as capable citizens whose own experiences and views matter. Further, it is a framework that promotes a whole-of-government approach and is consistent with the Treaty of Waitangi in striving for non-discrimination and a collaborative and empowering partnership with communities, iwi and hapu.

Policy-making is hard; it requires balancing diverse, and often competing, interests in a resource-limited, high pressure and constantly changing environment. However, the pressing policy issues for children in New Zealand seem to be portrayed as unnecessarily challenging, and the priority accorded to children is vastly inadequate in relation to the extent of the problems, the profound impacts and costs of a poor start in life and the Government's duty of care to children under UNCRC.

Our Australian counterparts have highlighted how challenging it can be to get children's issues higher on the policy agenda.<sup>16</sup> Children do not vote and have little opportunity themselves to directly participate in the policy process. Further, Stanley and Daube<sup>17</sup> highlight the unfair power differentials involved, drawing attention to the savviness and resourcing of industries

who seek to maximise profit from products that are harmful to children's health, such as the food, tobacco and alcohol industries. In contrast, they suggest there is limited resourcing for child public health advocacy.

Despite this the lack of action is curious given the importance of the issues and the potential for societal benefits, particularly when many policy interventions already have strong public support, such as for smoke-free cars and a sugary drinks tax. For example, Child Poverty Action Group research suggests that 75% of the public view child poverty as an important problem and only 19% felt that Government action was sufficient.<sup>18</sup> Even where support is less strong, Governments can show leadership and spearhead change, and often public support will follow. For example, this occurred with the 2007 repeal of Section 59 of the Crimes Act and the resulting prohibition of physical punishment of children.<sup>19</sup>

So unfortunately, the prospects for progress in improving and safeguarding child health and addressing the huge

disparities that exist appear bleak in New Zealand unless there is a paradigm shift in the priority accorded to children in public policy and the political process. Positive policy developments have been slow and ad hoc—much more can be done to address a range of specific health issues, such as the appropriate regulation of marketing of junk food, sugary drinks and alcohol. There is a pressing need to address wider issues of child poverty, housing, climate change and other major environmental and economic drivers of child health. However, progress is likely to be slow without broader culture change within government and the public sector, supported by structures and processes that ensure that children are properly considered in all aspects of policy formulation. UNCRC provides specific and clear guidance in this regard. It is possible that by adopting a goal to be one of the best countries in the world for children's wellbeing, New Zealand could make a remarkable comeback. The question is: why is it taking us so long?

---

**Competing interests:**

Nil.

**Author information:**

Amanda J D'Souza, HRC Clinical Research Training Fellow, Health Promotion and Policy Research Unit, Department of Public Health, University of Otago, Wellington; Louise Signal, Associate Professor, Health Promotion and Policy Research Unit, Department of Public Health, University of Otago, Wellington; Richard Edwards, Professor, Health Promotion and Policy Research Unit, Department of Public Health, University of Otago, Wellington.

**Corresponding author:**

Dr Amanda J D'Souza, Health Promotion and Policy Research Unit, Department of Public Health, University of Otago, PO Box 7343, Wellington South 6242.  
amanda.dsouza@otago.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7154>

---

**REFERENCES:**

1. World Health Organization. Commission on Ending Childhood Obesity. 2016; World Health Organization: Geneva.
2. Swinburn B, Vandevijvere S. Proposed new industry code on unhealthy food marketing to children and young people: Will it make a difference? *NZMJ*. 2017;130(1449):94–101.
3. Vandevijvere S, Sagar K, Kelly B, et al. Unhealthy food marketing to New Zealand children and adolescents through the Internet. *NZMJ*. 2017;130(1449):32–43.
4. Chambers T, Signal L, Carter MA, et al. Alcohol sponsorship of a summer of sport: a frequency analysis of alcohol marketing during major sports events on New Zealand television. *NZMJ*. 2017; 130(1448):27–33.
5. Boston J. Child Poverty in New Zealand: Why it

- matters and how it can be reduced. *Educational Philosophy and Theory*. 2014; 46:962–988.
6. Simpson J, Duncanson M, Oben G, et al. *Child Poverty Monitor 2016 Technical Report*. 2016; NZ Child & Youth Epidemiology Service, University of Otago: Dunedin.
  7. Amore K. *Severe housing deprivation in Aotearoa/New Zealand: 2001-2013*. 2016; He Kainga Oranga/Housing & Health Research Programme, University of Otago: Wellington.
  8. Simpson J, Oben G, Craig E, et al. *The Determinants of Health for Children and Young People in New Zealand 2014*. 2016; New Zealand Child and Youth Epidemiology Service, University of Otago: Dunedin.
  9. O’Sullivan CE, Baker MG, Zhang J. Increasing hospitalizations for serious skin infections in New Zealand children, 1990–2007. *Epidemiology and Infection*. 2011; 139:1794–1804.
  10. Simpson J, Duncanson M, Oben G, et al. *The Health Status of Children and Young People in the Hutt Valley, Capital & Coast and Wairarapa DHBs 2015*. 2016; New Zealand Child and Youth Epidemiology Service, University of Otago: Dunedin.
  11. Shonkoff JP. Leveraging the biology of adversity to address the roots of disparities in health and development. *Proceedings of the National Academy of Sciences*. 2012; 109(Supplement 2):17302–17307.
  12. Sachdeva S. Education system revamp must stop until kids are heard - Children’s Commissioner. *Dominion Post*. 2017: Wellington.
  13. Bennett H, Jones R, Keating G, et al. Health and equity impacts of climate change in Aotearoa-New Zealand, and health gains from climate action [Special Article]. *NZMJ*. 2014; 127(1406):16–31.
  14. Scott A. Sweden’s leading role in reducing child poverty and improving children’s wellbeing, in *Northern Lights. The Positive Policy Example of Sweden, Finland, Denmark and Norway*. 2014; Monash University Publishing, Victoria:63–97.
  15. UN Committee on the Rights of the Child. *Concluding observations on the fifth periodic report of New Zealand CRC/C/NZL/CO/5*. 2016; United Nations: Geneva.
  16. Oberklaid F. Child advocacy and the Queen’s representative; an unlikely link. *Archives of Disease in Childhood*. 2003; 88(11):980.
  17. Stanley F, Daube M. Should industry care for children? Public health advocacy and law in Australia. *Public health*. 2009; 123:283–6.
  18. MMRResearch. *New Zealanders’ Attitudes to Child Poverty*. Research Report. 2014; Child Poverty Action Group: New Zealand.
  19. D’Souza AJ, Russell M, Wood B, et al. Attitudes to physical punishment of children are changing. *Archives of Disease in Childhood*. 2016; 101:690–693.

# 23 years of managing diabetic ketoacidosis at Auckland Hospital

Geoffrey Braatvedt, Amelia Tekiteki, Holly Britton, John Wallace, Manish Khanolkar

## ABSTRACT

**AIMS:** To examine the length of stay and need for intensive care of people admitted with diabetic ketoacidosis (DKA) to a single centre between 1988 and 2011.

**METHODS:** Patients aged  $\geq 15$  years admitted for the first time with DKA (plasma glucose  $\geq 10$  mmol/L and a bicarbonate concentration  $\leq 15$  mmol/L and a pH  $< 7.35$ , and raised plasma or urine ketones or anion gap) to Auckland City Hospital from 1988–2011 were identified retrospectively. The patients were divided into four cohorts (1988–1996; 1997–2001; 2002–2006; 2007–2011). Over this time period there was no significant change to the insulin infusion protocol.

**RESULTS:** There were 576 admissions with DKA in 388 people over the 23 years. The mean age of the patients and glucose concentration at presentation to hospital fell significantly over time. The admission pH and bicarbonate concentration was higher in more recent cohorts. The length of stay and need for intensive care admission fell significantly over time, but the number of patients subsequently readmitted with DKA remained high. In-hospital mortality remained low.

**CONCLUSIONS:** DKA remains an important reason for admission to this hospital, but the severity of DKA at presentation has reduced over time. The need for intensive care admission and length of stay has fallen dramatically.

Diabetic ketoacidosis (DKA) remains a life-threatening, complex metabolic disorder complicating diabetes, and is a common cause for admission to acute medical units. A previous study of 125 people admitted with DKA between 1988–1996 to Auckland Hospital,<sup>1</sup> reported that the patients had a mean length of stay (LOS) of about a week, and nearly a third were admitted to the intensive care unit. In-hospital mortality was relatively low, with 2.4% of patients dying during their index hospital admission. However, 25% of these patients had a readmission with DKA during the audit period.

The protocol used to manage DKA in this hospital has not substantially changed since that time and remains “glucose-centric” (Appendix 1). It was previously available only as a hard copy sheet but available in all ward areas. In 2000, Auckland Hospital published its own Resident Medical Officer Handbook online and in hard copy (and more recently available to staff as a phone

app) detailing the management guidelines for a large variety of medical conditions including DKA. Hospital policy encouraged the use of the DKA protocol wherever possible, and the protocols were made widely available through the hospital intranet and by hard copy in the Emergency Department and on all medical wards. The protocol has always been printed on green double-sided A4 paper, which also serves as a prescription for insulin and fluids, as well as records the insulin infusion delivered and the blood glucose results. If the patient’s hourly measured capillary glucose levels are not falling, the rate of insulin infused per hour is rapidly escalated by protocol and on a varying scale (with separate scales for patients with known severe renal impairment). For most patients, scale “B” or “C” is used initially, and for a patient with significant hyperglycaemia at presentation, the dose of insulin infused per hour is about 6–8 units—this approximates the new UK guidelines<sup>2</sup> of 0.1 unit/kg/hour for

a person of average weight. The insulin infusion continues to be infused alone until the capillary glucose falls to  $<15\text{mmol/L}$ , when insulin is continued and 10% dextrose is added at 80ml per hour (lower rates on renal protocol). Fluids containing varying concentrations of potassium are also infused by protocol according to the patient's renal function and potassium concentrations, which are measured frequently. Once the patient is eating and drinking and glucose values are stable, subcutaneous insulin is commenced and the insulin infusion is weaned to stop. The protocol recommends frequent measurement of venous bicarbonate, potassium and pH, but does not stipulate repeat measures of plasma beta-hydroxybutyrate. This glucose-centric protocol has remained in use in this hospital over the past 23 years.

In 2011, the Joint British Society Guideline for the Management of DKA was published<sup>2</sup> with a major shift away from a glucose-centric protocol to a ketone-centric one, with the recommendation to commence a weight-based fixed dose of insulin infusion until ketonaemia (measured at the bedside) is cleared. Most NHS trusts in the UK have now adopted this protocol, and a similar shift has occurred in some New Zealand Hospitals. A recent UK audit of the use of the new protocol in managing 50 people presenting with DKA<sup>3</sup> reported a median LOS of 2 and mean 3.3 days.

The aim of the current study is to describe our experience in the management of DKA over a 23-year time frame, during which the management protocol has become more widely available and adopted, but has remained glucose-centric so as to help determine if adopting the new UK ketone-centric protocol should be considered.

### Patients and methods

Auckland City Hospital serves the central Auckland population (currently approximately 400,000 people), and the Department of General Medicine has approximately 12,000 medical discharges per year.<sup>4</sup> Children  $<15$  years of age are admitted to a separate specialist children's hospital. All patients discharged from Auckland Hospital are coded using standard internationally agreed codes. Patient records have been prospectively stored in electronic format

only for many years, and as patients get admitted or readmitted, any past stored paper records are scanned to an electronic format, which enables review of patient records remotely. All laboratory results have been available electronically from the late 1990s. Every New Zealand citizen has a unique national patient identifying number, allowing for accurate searching of their health records.

Patients presenting with DKA are assessed either in the emergency department and then referred to general medicine or can be referred directly to general medicine if the referring Doctor has discussed the admission with the general medical registrar on call. The "team of the day" looks after the patients (there were a total of eight General Medicine teams consisting of House Officer, Registrar and Consultant Physician until 2000 when the number of teams was increased to 12), and the diabetes service is only involved in their inpatient care by referral. Unless the patients require intensive care, they are looked after in a general medical ward. A new hospital on the same site as the old one was opened in 2005 (1,200 beds) with a bigger emergency floor space, with an additional acute assessment area having 65 beds available for 24-hour care before transfer up to the medical wards. Both the emergency and acute assessment units are co-located on the same floor, with excellent lines of communication between the two departments. Patients admitted to the emergency department are expected to be referred to an appropriate service for ongoing inpatient care or be discharged within six hours. General medicine therefore is involved in DKA management early in the patient's admission.

Data from the previously published 1988–1996 cohort<sup>1</sup> has been compared with three subsequent five-year cohorts to 2011 (1997–2001, 2002–2006, 2007–2011). All people aged  $\geq 15$  years admitted to Auckland Hospital with DKA were identified using the hospital discharge codes. While all admissions with DKA during the time period were recorded, only the first admission of that person with DKA during the study period (the index admission) was analysed and used in comparative studies, but the number and

**Table 1:** Details of the first admission of people admitted with DKA to Auckland City Hospital over 23 years by cohort (n=388 people).

	1988–1996	1997–2001	2002–2006	2007–2011	P
Number	125	62	81	120	
Age (yr.)	42 ± 16	38 ± 18	35 ± 17	31 ± 11	<0.05
Duration known diabetes (yr.)	11.6 ± 11.4	11 ± 10	9.4 ± 11	10.6 ± 12	
Female (%)	n/a	56	52	48	
European (%)	81	56	57	64	<0.05
Māori (%)	15*	16	14	10	
Pacific (%)		18	14	7	<0.05
Other (%)	4	11	16	18	<0.05

p<0.05 group over time. \*This includes Māori and Pacific for the first cohort. Details of the cohort 1988–1996 have previously been published.<sup>1</sup> Other = Asian/Indian. Data are mean ±SD.

timing of any subsequent readmissions with DKA was recorded. We continued to use the same definition of DKA as we had in the initial study—venous glucose  $\geq 10$ mmol/L **and** arterial pH <7.35 **and** bicarbonate  $\leq 15$ mmol/L **and** raised venous/urine ketones or raised anion gap. The electronic records of each person's admission were reviewed and data extracted. One way ANOVA, t-tests and  $\chi^2$  tests were used to compare differences between the time periods. Local ethics committee approval for the study was granted.

## Results

Over the 23-year period, a total of 576 DKA admissions in 388 patients met the study criteria. For the cohort 1997–2011, 516 patients were identified from coding as having had DKA, and 263 met our inclusion criteria.

Table 1 details the demography of the index admission of each patient. It can be seen that the mean age at presentation with DKA is lower in recent times and ethnic diversity is greater.

The age of the patient, glucose and pH at admission with DKA, and LOS was no different between patients of New Zealand European origin compared with all other ethnicities (data not shown).

Table 2 details admission metabolic parameters and LOS. Patients admitted in the more recent cohorts have had lower admission blood glucose and a higher pH and bicarbonate concentration reflecting less severe ketoacidosis. The number of patients admitted to the intensive care unit (ICU) has fallen dramatically, and LOS has fallen by around 50%. The mortality rate in hospital has remained low, and in the most recent cohort was <1%. However, the readmission

**Table 2:** Metabolic parameters, length of stay (LOS) and number (%) cared for in the intensive care unit (ICU) for 388 people admitted with diabetic ketoacidosis to Auckland City Hospital.

	1988–1996	1997–2001	2002–2006	2007–2011	P
Plasma glucose (mmol/L)	43 ± 21	34 ± 15	33 ± 15	31 ± 11	<0.05
pH	7.12 ± 0.12	7.16 ± 0.18	7.15 ± 0.15	7.18 ± 0.14	<0.05
Bicarbonate (mmol/L)	n/a	10.6 ± 7.2	9.1 ± 5.2	13.4 ± 6	
LOS (days)	6.6 ± 4	5.94 ± 6.6	4.8 ± 5.0	3.4 ± 3.6	<0.05
LOS median/ range(days)		4/0–35	4/0–35	2/0–29	
ICU (%)	30.4	24	22	9	<0.05
Readmitted* with DKA (%)	25	28	23	15	
In hospital mortality (%)	2.4	1.6	1.2	0.9	<0.05

Details of the cohort 1988–1996 have been previously published.<sup>1</sup> p<0.05 group over time. Data are mean ±SD. N/A = not available. \*within five years.

**Table 3:** Length of stay (LOS) of patients admitted with severe diabetic ketoacidosis (pH of  $\leq 7.1$  and/or bicarbonate  $\leq 5.0$  mmol/l) by cohort year of admission.

	1997–2001	2002–2006	2007–2012	P
Number of patients	20	30	31	
LOS (days)	7.45 $\pm$ 8.9	5.93 $\pm$ 6.9	4.23 $\pm$ 3.7	0.21
LOS range (days)	2–35	1–35	1–18	
ICU %	45	33	23	0.16

ICU = % of patients admitted to intensive care unit. Data are mean  $\pm$ SD.

rate with DKA has remained high at between 15–28% during the audit period following the index admission.

To investigate the influence of severity of DKA on LOS and ICU admission rates, two further analyses were done. Individual data from the 1988–1996 cohort was no longer available, and thus patient details from the three subsequent cohorts were examined. Table 3 presents LOS and ICU admission data of patients with severe DKA (pH  $\leq 7.1$  and/or bicarbonate  $\leq 5$  mmol/l) by cohort and demonstrates that patients with similar DKA severity had a (non-significant) shorter LOS and fewer requirements for ICU admission over time.

Table 4 presents data on all patients for 1997–2011 divided by tertile of admission pH, and demonstrates a shorter LOS and less need for ICU admission in those with highest pH on admission.

The number of patients admitted with DKA per 100,000 population aged  $>15$  in the hospital catchment area did not change over the study period (1988–1996 8.4/100,000; 1997–2001 6.8/100,000; 2002–2006 8.1/100,000; 2007–2011 9.0/100,000).

## Discussion

This report details one hospital's experience in the management of DKA over a 23-year time frame, during which time the management protocol has remained

glucose-centric (and essentially unchanged, including no change in the hard copy) and under the care of the general medicine "team of the day". The insulin infusion protocol has however become much more readily accessible since 2000, and the size and staffing of the emergency and acute assessment floor has improved significantly since the new hospital was opened in 2005. The study has shown that in-hospital mortality has remained low, and compares highly favourably with other reports, which showed a mortality of 3.9% for admissions between 1971–1991, and 1.8% for admissions between 2000–2009.<sup>5–7</sup> The LOS has dramatically fallen from nearly a week to under four days, and the number of patients admitted to the intensive care unit has reduced two-fold. Similar trends in reduced LOS for other general medicine patient admissions have also been observed. The high readmission rate remains a concern.

Previous studies have shown that following protocols of care for complex disorders such as DKA improves outcome.<sup>8–10</sup> Over the past 40 years many different protocols and guidelines for the management of DKA have been published.<sup>11–13</sup> All agree that relatively low-dose IV insulin infusion with vigorous fluid resuscitation and close monitoring of patients is important. Recent studies from the UK<sup>3,14</sup> have however shown that standard protocols for the management

**Table 4:** Length of stay (LOS) of patients admitted with diabetic ketoacidosis between 1997–2011 by tertile of pH.

pH tertile	Number of patients	ICU (%)	LOS (days)	LOS range (days)
6.7–6.92	34	67	9.4 $\pm$ 10.6	1–35
6.93–7.15	121	20	4.2 $\pm$ 3.1	1–18
7.16–7.35	233	6.3*	3.7 $\pm$ 3.7*	1–29

ICU (%) = % of patients admitted to intensive care unit \* $p < 0.01$  across tertiles.

of DKA are in fact poorly followed. The recent UK Guidelines<sup>2</sup> have recommended that emphasis should shift away from glucose concentration-driven protocols to ketone and pH-driven considerations using frequent bedside ketone and glucose testing to inform when insulin infusions can be safely changed to subcutaneous insulin. While the protocol may have merit, we are not aware of large and robust RCT evidence that this has advantages for patient outcomes that matter—mortality, LOS and early readmission with DKA due to a failed discharge. Our own experience has shown that the widespread use of a single protocol is probably the main contributor to better outcomes over time, although the patients in more recent years appear to have presented with less severe DKA than in the earlier years, perhaps reflecting the widespread use of modern analogue insulins in the community, better education of patients and other factors. Furthermore, patient's ability to better self-monitor glucose and ketones could have led to an earlier discharge in more recent years without the need to ensure full metabolic clearance of ketones in a hospital setting.

Although the number of patients available for comparison has reduced statistical power, the LOS (and need for ICU admission) of patients with severe DKA (Table 3) has shown a progressive reduction over time suggesting that improvements in initial care, perhaps in the Emergency and Acute Assessment Departments, has led to the fall in length of stay observed in the whole cohort, as no changes to the protocol were made during that time. For the most recent three five-year cohorts, stratifying patients by tertiles of pH (Table 4) does show a reduction in LOS for those with the highest tertile of pH compared with patients admitted with the lowest pH tertile, suggesting this could be one of the reasons why the length of stay has reduced over time as patients in recent years had less severe DKA. We did not specifically analyse how well the protocol was followed over the study period, but note it has become more widely available since 2000 when it was more formally published in a handbook, and particularly in recent years with the availability of the protocol electronically, on smart phones, intranet and iPads.

The reduction in admissions to intensive care over the study period is striking. This could reflect less severe presenting acidosis in recent times, better immediate care in the emergency room and acute assessment area and an ability to manage acutely unwell patients in these well-staffed and fully monitored areas independent of whatever insulin infusion protocol is in place. Recent UK guidelines recommend HDU-ICU admission for patients meeting a number of criteria including a bicarbonate  $<5$  or pH  $<7.1$ .<sup>2</sup> The number of patients fulfilling these criteria over the three recent five-year cohorts was 31%, 34% and 22% respectively. However, the number of patients admitted to ICU in each time frame has reduced from 45% to 33% to 23% respectively (Table 3). Interestingly, a recent<sup>15</sup> large study of DKA admissions to New Zealand and Australian ICUs between 2000–2013 showed that the incidence of DKA admission has increased as a proportion of other reasons for admission to ICU, but the pH and bicarbonate has increased and glucose decreased consistent with our study, showing that patients are presenting to hospital with less severe DKA. The LOS in the ICU rose over time, but the overall LOS in hospital fell. Twenty-seven percent of patients were not on established insulin on admission to ICU, suggesting a large number of these patients had DKA in the context of type 2 diabetes. Our own study has also shown a rising prevalence of DKA in non-New Zealand Europeans, also suggesting that DKA is not an uncommon complication of type 2 diabetes. Although we did not record the presence of co-morbidities in our cohorts with DKA, the age, metabolic parameters and LOS of patients of New Zealand ethnicity was no different from those patients of other ethnicities, confirming the increasing evidence of an earlier age of presentation with type 2 diabetes in non-New Zealand Europeans.

The number of patients readmitted with DKA over the subsequent five years is of concern. Although the apparent reduction in readmission rate in the most recent cohort to 15% could reflect a real reduction in readmission rates, it could also reflect the fact that by definition this cohort has only been followed for a maximum of five years, with some patients near the close-out time point of the audit having been followed only for a

few weeks to months. Potential reasons for the high readmission rate in these cohorts have been reported in a separate study.<sup>16</sup>

Study limitations include that this was a retrospective review and based on discharge codes to identify patients. It is likely that a number of episodes of DKA were incorrectly coded, and thus the number of patients admitted with DKA is not accurate. However, the discharge codes have not changed over the years, and thus case ascertainment is likely to have been equal (good or poor) across the time periods. The number of patients admitted with DKA per 100,000 of population did not change over time, despite the known rising incidence of type 1 diabetes in New Zealand. This could suggest that case ascertainment was worse in recent cohorts, but equally that the incidence of DKA in patients with type 1 diabetes has fallen. Most patient details were available through the electronic record, which also allowed the opportunity to double audit patients with any queries identified from a first review. We also did not review whether over time the protocol was followed more rigorously or not, nor how much fluid was infused, as our main focus was to examine trends in patient outcomes over time. The LOS was recorded in “days” and not hours, and thus patients with a stay of <24 hours could have a LOS of “0” days. A recent study<sup>17</sup> from Christchurch has shown that many patients with DKA only need an overnight admission. Finally a new hospital was opened in 2005 with much better emergency room and acute assessment area integration and communication, which may well have contributed to better patient outcomes independent of what protocol was in place. We do however have a unique opportunity to investigate this further, as North Shore City Hospital in Auckland, which serves a similar number of patients with the same acuity as Auckland

Hospital, uses the same pool of medical registrars in training rotating through both hospitals and opened a new emergency department in 2010, did change its protocol of care for DKA to the UK guidelines in 2012. We are thus currently comparing outcomes in LOS, need for intensive care, rates of hypoglycaemia and time to resolution of acidosis in the cohorts admitted with DKA in 2013 to each hospital.

The ethnic origin of the people admitted with DKA has changed substantially over the study period, with many more patients of Māori, Pacific and other ethnic origin admitted in later cohorts (Table 1). Type 1 diabetes in these communities is relatively rare,<sup>18</sup> and thus many of these patients are likely to have had type 2 diabetes. The incidence of type 2 diabetes in Māori and Pacific people in Auckland is increasing, and the age at presentation is much younger than in people of New Zealand European origin.<sup>19, 20</sup>

## Conclusion

In conclusion, our retrospective cohort study has described the outcome of patients with DKA over a long period of time, and has shown that DKA remains a common reason for presentation to our medical unit, but that the in-hospital mortality is low, and the length of stay and number of patients admitted to intensive care has fallen dramatically over time. The readmission rate for DKA, however, remains high. During this time the insulin infusion protocol has not changed, and suggests therefore that the improvements shown are likely systems of care improvements independent of what protocol is used. Whether a different protocol of care such as the UK ketone-centric one, results in even better patient outcomes is the subject of another study comparing patients admitted with DKA in two neighbouring hospitals in Auckland that use different insulin infusion protocols.

**Competing interests:**

Dr Braatvedt reports affiliation with Eli Lilly and Novo nordisk outside the submitted work.

**Author information:**

Geoffrey Braatvedt, Department of Medicine, University of Auckland, Park Road, Auckland; Amelia Tekiteki, General Medicine, Auckland City Hospital, Auckland; Holly Britton, General Medicine, Auckland City Hospital, Auckland; John Wallace, General Medicine, Auckland City Hospital, Auckland; Manish Khanolkar, Diabetes Centre, Green Lane Hospital, Auckland.

**Corresponding author:**

Geoffrey Braatvedt, Department of Medicine, University of Auckland, Park Road, Auckland 1111. g.braatvedt@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7155>

**REFERENCES:**

1. Bagg W, Sathu A, Streat S, Braatvedt G. Diabetic ketoacidosis in adults at Auckland Hospital 1988-1996. *Aust NZ J Med* 1998; 28:604-608.
2. Savage MW, Dhatariya KK, Kilvert A, Rayman G, Rees JAE, Courtney CH, Hilton L, et al. for the Joint British Diabetes Societies. Joint British Diabetes Societies guidelines for the management of diabetic ketoacidosis. *Diabetic Med* 2011; 28:508-515.
3. Crasto W, Htike ZZ, Turner L, Higgins K. Management of diabetic ketoacidosis following implementation of the JBDS guidelines: Where are we and where should we go? *Br J Diabetes Vasc Dis* 2015; 15:11-16.
4. Toomath R, Szecket N, Nahill A, Denison T, Spriggs D, Lay C, et al. Medical service redesign shares the load, saving 6000 bed days and improving morale. *Int Med J* 2014; 34:785-790.
5. Basu A, Close CF, Jenkins D, et al. Persisting mortality in diabetic ketoacidosis. *Diabet Med* 1993; 10:282-4.
6. Wright J, Ruck K, Rabbitts R, Charleton M, De P, Barrett T, et al. Diabetic ketoacidosis in Birmingham, UK, 2000-2009: an evaluation of risk factors for recurrence and mortality. *Br J Diabetes Vasc Dis* 2009; 9:278-282.
7. Chen HF, Wang CY, Lee HY, See TT, Chen MH, Jiang JY, et al. Short term case fatality rate and associative factors among inpatients with diabetic ketoacidosis and hyperglycaemic hyperosmolar state: a hospital based analysis over a 15 year period. *Inter Med* 2010; 49:729-737.
8. Meade M, Ely E. Protocols to improve the care of critically ill pediatric and adult patients. *JAMA* 2002; 288:2601-2603.
9. Bull SV, Douglas IS, Foster M, Albert RK. Mandatory protocol for treating adult patients with diabetic ketoacidosis decreases intensive care unit and hospital length of stay: results of a non-randomised trial. *Crit Care Med* 2007; 35:41-46.
10. Thuzar M, Malabu UH, Tisdell B, Sangla KS. Use of a standardised diabetic ketoacidosis management protocol improved clinical outcomes. *Diabetes Research and Clinical Practice* 2014; 104:e8-e11.
11. Kitabchi AE, Umpierrez GE, Fisher JN, Murphy MB, Stentz FB. 30 years of personal experience in hyperglycemic crisis: diabetic ketoacidosis and hyperglycemic hyperosmolar state. *J Clin Endocrinol Metab* 2008; 93:1541-1552.
12. Kitabchi AE, Umpierrez GE, Miles JM, Fisher JN. Hyperglycemic crisis in adult patients with diabetes: a consensus statement from the American Diabetes Association. *Diab Care* 2009; 32:1335-1343.
13. Eledrisi MS, Alshanti S, Faiq Shah M, Brolosy B, Jaha N. Overview of the diagnosis and management of diabetic ketoacidosis. *Am J Med Sci* 2006; 331:243-251.
14. Jervis A, Champion S, Figg G, Langley J, Adams G. Prevalence of diabetes ketoacidosis rises and still no strict treatment adherence. *Current Diabetes Reviews* 2013; 9:54-61.
15. Venkatesh B, Pilcher D, Prins J, Bellomo R, Morgan TJ, Bailey M. Incidence and outcome of adults with diabetic ketoacidosis admitted to ICUs in Australia and New Zealand. *Critical Care* 2015; 19:451-463.
16. Cooper H, Tekiteki A, Khanolkar M, Braatvedt GD. Risk factors for recurrent admissions with diabetic ketoacidosis – a case-controlled observational study *Diab Med* 2016; 33:523-528.
17. Yong KW, Moore MP, Lunt H. Medically facilitated discharge of adult

- diabetic ketoacidosis admissions: precipitants and average length of stay. NZMJ 2014; 127:86–93.
18. Wu D, Kendall D, Lunt H, Willis J, Darlow B, Framp-ton C. Prevalence of type 1 diabetes in New Zealanders aged 0–24 years. NZMJ 2005; 118; no1218 ISSN 11758716.
19. Berkeley J, Lunt H. Diabetes epidemiology in New Zealand – does the whole picture differ from the sum of its parts? NZMJ 2006; 119; no1235 ISSN 11758716.
20. McGrath NM, Parker GN, Dawson P. Early presentation of type 2 diabetes mellitus in young New Zealand Māori. Diab Res Clin Pract 1999; 43:205–209.

## Appendix

### Suggested medical management DKA

1. Admit under the team of the day.
2. Consider resuscitation status.
3. Look for underlying illness, eg Sepsis, MI.
4. Nil by mouth if vomiting or reduced level of consciousness for the first 12h at least.
5. Vital signs q2h (may need q1h initially) for 8h then q4h until stable.
6. Capillary blood glucose q1h until off insulin infusion.
7. Urine output q1h initially; be concerned if less than 30 mL/h.
8. Consider intensive care referral if reduced level of consciousness, BP <90 systolic, pH <7.2 or renal impairment.
9. Contact nursing supervisor early: these patients often require one-on-one nursing for the first 8–12h.

### Investigations/management:

- Electrolytes, creatinine, FBC.
- ABG at presentation plus beta hydroxybutyrate. Thereafter venous bicarbonate is usually adequate to monitor progress.
- Culture of blood, urine and other clinically indicated sites.
- CXR.
- Nasogastric tube if vomiting (ileus is common in DKA).
- ECG.
- Troponin T if >30y and no obvious alternative precipitant or ECG abnormal.
- Repeat electrolytes at least q2h until stable especially watching for hypokalaemia.
- Use a flowchart to record fluid balance, pH or bicarbonate and electrolytes.

### Fluids and insulin:

- The first litre of hydrating solution should be sodium chloride 0.9% given as quickly as possible in the first hour and followed by 500–1000mL/h of sodium chloride 0.45% of 0.9% (depending on the state of hydration and serum sodium) during the next 2h.
- The type and rate of continued fluid replacement will depend on assessment of clinical and biochemical factors.
- If hypernatraemic (>146mmol/L) consider sodium chloride 0.45%.
- Repeat electrolytes regularly as above. K<sup>+</sup> may be required in large amounts (often >20mmol/L). Do not begin to replace until K<sup>+</sup> <5.0mmol/L and urine output >30mL/h. When K<sup>+</sup> <5.0mmol/L begin replacement at 20mmol/hour—don't wait until K<sup>+</sup> is low.
- Insulin: do not strive for rapid control as glucose will often fall significantly with rehydration alone. Commence an IV insulin infusion according to Table 1.
- Start with Scale B as it is the most commonly appropriate and should be the initial default scale.

**Table 1:**

<b>Scale A</b>	Thin small individuals, especially women: some very sensitive normal (total daily dose <30units/day); athletes; hypopituitary, hypoadrenal and hypothyroid patients; post-pancreatectomy patients (surgical or functional); some well controlled non-obese diet controlled type 2 diabetics.
<b>Scale B</b>	Individuals (type 1 or type 2) with no special circumstances; use if normal daily insulin requirements approximately 30–80units/day.
<b>Scale C</b>	Seriously ill (high fever etc.); moderate steroid doses (up to 20mg/daily); very obese or insulin resistant patients (>80units/day); uncomplicated post-infarct patients.
<b>Scale D</b>	High steroid doses (>20mg daily); very stressed individuals; complicated post-infarct patients.
<b>Scale E</b>	Major cardiac surgery (never use this scale without specialist physician/anaesthetic advice).

**Table 2:**

<b>Blood glucose</b>	<b>Scale A</b>	<b>Scale B</b>	<b>Scale C</b>	<b>Scale D</b>	<b>Scale E</b>
<5.0mmol/L	Stop/consult	0.5–1 unit/h or Scale A	2 units/h or Scale B	4 units/h or Scale C	8 units/h or Scale D
5.0–11.0mmol/L	1 unit/h	3 units/h	4 units/h	8 units/h	16 units/h
11.1–17mmol/L	3 units/h	6 units/h	8 units/h	16 units/h	32 units/h
>17mmol/L	6 units/h or Scale B	12 units/h or Scale C	16 units/h or Scale D	32 units/h or Scale E	64 units/h

- If blood glucose persistently below 5mmol/L, ie on two or more tests one hour apart, move one scale to the left and/or ask for advice.
- If blood glucose persistently high, ie on two or more tests one hour apart, check pump for correct rate and line for patency, then move one scale to the right and/or ask for advice.
- Once glucose is <15mmol/L on two consecutive tests one hour apart, introduce 10% dextrose at 80mL/hr.

## GIK Infusions

GIK is an insulin infusion run in conjunction with an infusion of 500ml of glucose 10% with 10mmol of potassium chloride. This combination is suitable for most patients (unless the patient is sodium depleted or is a renal patient).

The insulin infusion should be prescribed on the Insulin Chart. Scale B (see table above) is most commonly appropriate and should be in the initial default scale. The insulin infusion should be prepared in a 50ml syringe by mixing 50 units Actrapid® in 49.6ml sodium chloride 0.9% (to make a 1unit/ml solution). This should be administered using a syringe pump.

The glucose 10% infusion with 10mmol of potassium chloride should be prescribed on the fluid balance chart and given at a rate of 80ml/h via a volumetric pump. This regimen is designed to control diabetes. Other problems such as fluid depletion, other electrolyte imbalance etc. should be managed through separate infusions.

## Discontinuing a GIK

An insulin infusion is best discontinued 2–4 hours after a meal. The usual pre-meal dose of insulin is given, and the infusion should be continued for around a further 2 hours if Humalog® or Novorapid® is used or 4 hours if Actrapid® or Humulin R® is used. This overlap allows time for the subcutaneous insulin to reach peak concentration and prevent hyperglycaemia.

Patients with type 1 diabetes should also have their intermediate/long-acting subcutaneous insulin restarted or initiated. (Protaphane® or Humulin NPH® or Glargine). This is especially important if a rapidly acting analogue is used. The patient's usual dose of insulin should be prescribed.

Patients with DKA not previously known to have diabetes should be started initially on a twice daily dose of intermediate acting insulin (eg Protaphane® or Humulin NPH®) at a dose of 0.2–0.4 units/kg/day and specialist referral made. Short-acting insulin will be added when they are stable and ambulant.

Diabetologist/diabetic education referral (early). DKA is usually a failure of education/self-care.

# Screening for diabetes in pregnancy in a regional area with a high Māori population

Barbara Daly, Isabel Raiman, Jennifer Goodson

## ABSTRACT

**AIMS:** To identify and document factors associated with screening for diabetes in pregnancy in a regional area with a high Māori population in New Zealand.

**METHODS:** An audit was undertaken of routine hospital data collected from all 656 women who gave birth, between June and December in 2013 and 2014, in two Mid-North Island hospitals in the Bay of Plenty region.

**RESULTS:** Of the 656 woman who gave birth during these periods, only 416 (63%) were screened for diabetes in pregnancy, including 390 (60%) for gestational diabetes mellitus later in pregnancy. After controlling for age, screening was less common in Māori (56%) compared with European women (76%). After adjusting for ethnicity, women aged 35–40 years were more likely to be screened compared with women aged 25–29 years (77% versus 61%;  $p=0.02$ ). Screening was associated with longer hospital stays following birth, with screened women more likely to stay >5 days than <1 day, compared with unscreened women (84% versus 56%;  $p<0.0001$ ). Screening was significantly higher in 2014 than 2013 (68% versus 58%;  $p=0.008$ ).

**CONCLUSIONS:** Greater effort is required to increase screening, especially for Māori women who have increased risk of type 2 diabetes and gestational diabetes mellitus and of poorer outcomes.

Gestational diabetes mellitus (GDM) is increasing globally.<sup>1</sup> Its prevalence varies depending on the ethnic make-up of populations and screening criteria adopted, with 2–6% prevalence reported for pregnancies in Europe,<sup>1</sup> 7% in the US,<sup>2</sup> and up to 20% in high risk populations,<sup>3</sup> including New Zealand.<sup>4</sup> GDM is strongly associated with body mass index (BMI), with the prevalence being 13% for women whose BMI is >25–39kg/m<sup>2</sup> and 21% for those >40kg/m<sup>2</sup>.<sup>5</sup> Over 10% of all New Zealand births (61,038 in 2015)<sup>6</sup> occur at National Women's Hospital (Auckland) and in 2014, 40% of women who delivered at National Women's were overweight (BMI >25), 17% were obese, 9.8% were diagnosed with GDM, which was higher for Indian (21%), Asian (16%), Pacific (11%) and Māori (6%) women compared with European women (5%).<sup>4</sup> Another important driver of increasing GDM cases is the higher fertility rates for Pacific and Māori women, which are 2.7 and 2.5 births per woman respectively, compared with 1.9 for European and 1.7 for Asian women.<sup>7</sup>

As a consequence of the increasing prevalence and importance of GDM, the New Zealand Ministry of Health (MoH) recommends that all pregnant women have glycosylated haemoglobin (HbA<sub>1c</sub>) levels tested in early pregnancy to identify undiagnosed type 2 diabetes, and to inform the screening sequence later in pregnancy.<sup>8</sup> Diagnosis for GDM is based on the original New Zealand Society for the Study of Diabetes criteria<sup>9</sup> of a fasting glucose  $\geq 5.5$ mmol/L or two-hour oral glucose tolerance test (OGTT)  $\geq 9$ mmol/L.<sup>8</sup> If glucose is 7.8–11mmol/L for the one-hour Polycose test, women are advised to have an OGTT.<sup>8</sup>

There is a paucity of information on the prevalence of screening for diabetes in pregnancy in New Zealand.<sup>10</sup> More recently, screening in Counties Manukau, South Auckland, had increased to approximately 80% in 2011<sup>11</sup> and 85% in 2013, with a reported 6% prevalence of GDM.<sup>12</sup> In 2011, National Women's reported screening rates for GDM >90%.<sup>5</sup> However, there do not appear to be any previous published

reports on screening for diabetes in pregnancy from regional areas.

The Bay of Plenty District Health Board (DHB) serves a population of 222,235 people and has a greater proportion of older people, Māori people (25% compared with 16% nationally),<sup>13</sup> and has more people categorised as most deprived socioeconomically (24%) compared with the national average (20%).<sup>13,14</sup> The aim of this paper is to report the prevalence of screening for diabetes in pregnancy and to identify associated risk factors in a regional area with a high Māori population.

## Methods

An audit was undertaken of routinely collected hospital data from 656 women who gave birth over two six-month periods (June to December in 2013 and 2014) from two hospitals in the Eastern Bay of Plenty DHB, serving a regional area with a high Māori population (51%).

Maternal demographic and laboratory data are routinely collected on all women who attend outpatient clinics or on admission to hospital. All data are entered into the hospital-based patient management system. Each contact with the hospital generates a code indicating the type

of service provided, health professional consulted, diagnoses and procedures undertaken. All laboratory tests are conducted through public funded laboratories whose tests results are accessed electronically through the software management program 'Éclair' by Primary Health Organisations and hospitals in the Bay of Plenty region.

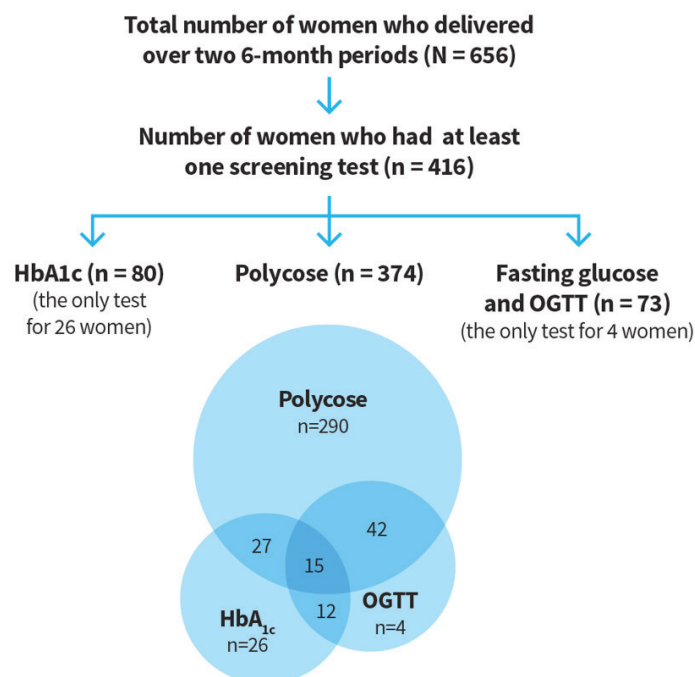
Health related and laboratory data for all women who had delivered in the two hospitals serving the Eastern Bay of Plenty region were accessed to complete this audit. Data were cross-checked for accuracy and anonymised prior to data analyses.

Standard univariate and multivariate methods were used for analysing categorical and continuous outcome data, using PROC FREQ, PROC UNIVARIATE and PROC REGRESS in SAS version 9.3 (SAS Institute, Cary, North Carolina, 2010).

## Results

The proportion of all women audited (n=656) who were screened for any type of diabetes in pregnancy was 63%. Figure 1 outlines the screening tests undertaken by 416 of the total cohort of women who delivered in two of the three hospitals in the Bay of Plenty DHB, over two six-month periods (June to December in 2013 and 2014).

**Figure 1:** Number of women who delivered in two hospitals, serving a regional population in the Bay of Plenty region who underwent screening tests for diabetes in pregnancy.



**Table 1:** Demographic maternal characteristics for women who were screened compared with those not screened for diabetes in pregnancy (n=656).

Variable and level	Total n (%) <sup>*</sup>	Screened women n=416 n (%) <sup>#</sup>	Non-screened women n=240 n (%) <sup>#</sup>	P-value
Age (years)				
< 20	95 (14)	59 (62)	36 (38)	0.07
21–24	168 (26)	99 (59)	69 (41)	
25–29	184 (28)	112 (61)	72 (39)	
30–34	130 (20)	85 (65)	45 (35)	
35–40	79 (12)	61 (77)	18 (23)	
Ethnicity				
NZ European/Other	206 (31)	157 (76)	49 (24)	<0.0001
Māori	410 (63)	231 (56)	179 (44)	
Asian	30 (5)	23 (77)	7 (23)	
Pacific	10 (1.5)	5 (50)	5 (50)	
Hospital of birth				
Secondary	618 (94)	394 (64)	224 (36)	0.40
Minor	37 (6)	21 (57)	16 (43)	
Antenatal bloods				
Yes	628 (96)	415 (66)	213 (34)	<0.0001
No	28 (4)	1 (4)	27 (96)	
Length of stay				
<1 (days)	299 (46)	166 (56)	133 (45)	<0.0001
2	131 (20)	85 (65)	46 (35)	
3–4	140 (21)	93 (66)	47 (34)	
>5	86 (13)	72 (84)	14 (16)	
Year				
2013 (June–December)	303 (46)	177 (58)	126 (42)	0.01
2014 (June–December)	353 (54)	239 (68)	114 (32)	

P-value showing significance of variation in percentages in subgroups, from the chi-square value from the Fisher test.

\* Percent by column.

# Percent by row.

Of the 416 women who were screened, only 12% had an HbA<sub>1c</sub> test for prediabetes or type 2 diabetes, 57% underwent a Polycose test between 24 and 28 weeks and 11% had a fasting glucose test and OGTT. Thirteen (3%) of the 416 women screened were diagnosed with GDM, two women had pre-existing diabetes, one each with type 1 and type 2 diabetes, and the status of nine women (4%) could not be determined. Of the 13 women diagnosed with GDM, nine (69%) were Māori, three were European and one of Indian ethnicity.

Of the 80 women who had HbA<sub>1c</sub> levels tested, 19% (n=15) had had levels  $\geq 40$ mmol/mol (including one  $\geq 50$ mmol/mol), and of those, two had pre-existing diabetes, nine had a fasting glucose and an OGTT (including two women who had a Polycose

test) and one woman had a Polycose test. The remaining three women had no fasting or glucose challenge test. Two women who underwent the Polycose test had glucose levels  $>11$ mmol/L, 14 women who had a fasting glucose test had levels  $\geq 5.5$ mmol/L and 11 women who had an OGTT had glucose levels  $\geq 9$ mmol/L.

Table 1 compares demographic characteristics of the 416 women who underwent screening for diabetes in pregnancy with the 240 women not screened who gave birth over two six-month periods in two hospitals in the Eastern Bay of Plenty region. Significantly more New Zealand European and Asian women were screened compared with Māori and Pacific women, and a higher proportion of women were screened in 2014 compared with 2013 (P=0.01).

**Table 2:** Multivariate prevalence rates (RR) for factors associated with screening for diabetes in pregnancy, adjusted for age & ethnicity as appropriate (n=656).

Variable and level	Screened N (%)		RR (95% CI) Adjusted*	P-value Adjusted*
Age (years)				
25-29	112	61	1.00	
< 20	59	62	1.15 (0.92-1.44)	0.21
21-24	99	59	1.05 (0.88-1.25)	0.59
30-34	85	65	1.04 (0.87-1.23)	0.68
35-40	61	77	1.24 (1.06-1.47)	0.02
Ethnicity				
European/Other	157	76	1.00	
Māori	231	56	0.73 (0.65-0.83)	<0.0001
Asian	23	77	1.01 (0.81-1.26)	0.91
Pacific	5	50	0.67 (0.36-1.27)	0.11
Length of stay (days)				
<1	166	56	1.00	
2	85	65	1.08 (0.92-1.28)	0.36
3-4	93	66	1.14 (0.97-1.34)	0.13
>5	72	84	1.44 (1.25-1.66)	<0.0001
Year				
2013 (June-Dec)	177	58	1.00	
2014 (June-Dec)	239	68	1.17 (1.04-1.32)	0.008

P-value showing significance of variation in percentages in subgroups, from the chi-square value from the Fisher test.  
\*Age adjusted for ethnicity; ethnicity adjusted for age; and length of stay and year adjusted for age and ethnicity.

Table 2 shows women aged 35–40 years were more likely to be screened than women 25–29 years after controlling for ethnicity, and Māori and Pacific women were less likely to undergo screening compared with European women after controlling for age, although the latter was not significant due to the small number of Pacific women (n=10). After controlling for age and ethnicity, women who were screened were more likely to remain in hospital for at least five days compared with women who had not undergone screening. The proportion of women who underwent screening significantly increased from 58% to 68% between the last six months of 2013 and 2014.

## Discussion

This report documents the prevalence of screening for diabetes in pregnancy and associated risk factors in a regional area with a high Māori population. Only 12% of women underwent an HbA1c screening test for prediabetes and type 2 diabetes in early pregnancy, with 19% of those women having elevated levels, and only 60% of

women were screened for GDM at 24 to 28 weeks. Screening rates for Māori and Pacific were unacceptably low and significantly lower than those for New Zealand European, European and Asian women. Women aged 35–40 years of age were more likely to be screened compared with women 25–29 years of age.

Women who were screened were more likely to stay in hospital for  $\geq 5$  days compared with women not screened. Although reasons are not known for the extended hospital stays, women with a pre-existing health condition or pregnancy-related complication were more likely to undergo screening, perhaps due to greater engagement with health care services.

The 60% screening prevalence for GDM for this regional area was far lower than the two recently reported for the Auckland region, being >90% for National Women's for all ethnic groups<sup>4</sup> and 85% in Counties Manukau in South Auckland.<sup>12</sup> However, despite the overall high screening rate for Counties Manukau, the prevalence was only 61% for Māori women,<sup>11</sup> and remains

the lowest compared with all other ethnic groups; 81–83% for Asian, 77% for European and 72% for Pacific women in 2011,<sup>11</sup> and only slightly higher than the 56% for Māori women in our survey.

Internationally, indigenous women (including Māori) are more likely to have undiagnosed type 2 diabetes during pregnancy compared with European women.<sup>8,15</sup> Early identification and treatment of women with borderline and type 2 diabetes and with GDM is associated with reductions in pregnancy and perinatal complications.<sup>1,9,16,17</sup> In addition, GDM is an established risk factor for progression to type 2 diabetes, which carries up to a 70% lifetime risk,<sup>18</sup> and a diagnosis of borderline GDM, is important to women.<sup>19</sup> In a study in Northland, 60% (n=110) of all women diagnosed with GDM between 1997 and 2005 were followed up for a median 2.4 year period, and of those, 32% had an abnormal fasting blood glucose test or had developed diabetes or impaired glucose tolerance.<sup>20</sup>

Despite on-going controversy about the ideal diagnostic criteria for GDM,<sup>21,22</sup> universal screening in early pregnancy for type 2 diabetes and in the second trimester for GDM is recommended.<sup>8</sup> This report shows the status of diabetes in pregnancy was not known for 30% of the women and, based on the New Zealand National Health 2008/9 survey, potentially 2.3% of Māori women aged 25–44 years could have undiagnosed diabetes and 31% have prediabetes,<sup>23</sup> and be at increased risk of GDM.<sup>8</sup>

The new MoH guideline<sup>8</sup> may help assist lead maternity carers (LMC), who have previously reported that a lack of information and clear guidelines for screening for diabetes in pregnancy is a barrier in advising and arranging for women to undergo screening.<sup>10</sup> Targeting women at risk of GDM for screening, rather than all women,<sup>10,24</sup> results in underreporting of GDM.<sup>25</sup>

Achieving universal screening for diabetes in pregnancy is a challenge, but was achieved in the Cook Islands after the introduction of a screening programme that offered screening to all eligible women.<sup>26</sup> One New Zealand study that interviewed 26 Māori women to identify barriers to screening for diabetes in pregnancy, reported that two of the five women who had not undergone screening felt additional tests

were unnecessary, as previous pregnancies had been uneventful.<sup>10</sup> In contrast, reasons given by the 21 women who underwent screening included having a positive relationship with their LMC, understanding the consequences of GDM and knowing they were at increased risk of GDM.<sup>10</sup>

Findings from this paper showed screening rates for GDM increased between 2013 and 2014. A new multidisciplinary hospital-based maternity service for women with diabetes in pregnancy may have contributed to this increase or it may be following a natural upward trend. Further initiatives and active engagement with Māori and Pacific communities are required to reduce the ethnic variation in screening and inequity in accessing health care services to achieve universal screening rates in line with National Women's<sup>5</sup> and in the Cook Islands.<sup>26</sup> The new MoH guideline provides an opportunity to encourage all DHBs to audit and report screening trends. Without these data, the impact of the new guideline and local initiatives to improve existing services to maximise screening opportunities will remain elusive.

Limitations of this audit include the limited number of variables collected, including the absence of well-known risk factors for GDM, such as BMI, to establish if they were associated with screening and their association with pregnancy and perinatal complications. No details were available for the small number of women who had a home delivery in this region. In addition, it is possible that pregnancy-related adverse outcomes were underestimated, as a small number of high risk women may have been transferred out of the area. Despite these limitations, this audit included all women who gave birth at two of the three hospitals in the Bay of Plenty region and is representative of all women undergoing screening for diabetes in pregnancy residing in a regional area with a high Māori and socioeconomically disadvantaged population.

This report highlights the poor uptake of screening for prediabetes, type 2 diabetes and GDM in pregnancy in a high risk population group. Further resources are required to increase engagement with Māori and Pacific communities to achieve universal and equitable screening rates across all ethnic groups and regions in New Zealand.

**Competing interests:**

Nil.

**Acknowledgements:**

We would like to acknowledge the Bay of Plenty District Health board for their support in undertaking this audit.

**Author information:**

Barbara Daly, Senior Lecturer in Nursing, School of Nursing, University of Auckland, Auckland; Isabel Raiman, Medical Service, Nurse Practitioner (Diabetes), Bay of Plenty, District Health Board, Tauranga; Jennifer Goodson, Clinical Study Coordinator, Medical Service, Bay of Plenty, District Health Board, Tauranga.

**Corresponding author:**

Barbara Daly, Senior Lecturer in Nursing, School of Nursing, University of Auckland, 85 Park Road, Grafton, Auckland 1023.

b.daly@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7156>

**REFERENCES:**

- Buckley BS, Harreiter J, Damm P, et al. Gestational diabetes mellitus in Europe: prevalence, current screening practice and barriers to screening. A review. *Diabet Med* 2012;29:844-54.
- American Diabetes A. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2014;37 Suppl 1:S81-90.
- Galtier F. Definition, epidemiology, risk factors. *Diabetes Metab* 2010;36:628-51.
- Auckland District Health Board. National Women's Annual Clinical Report 2014. Secondary National Women's Annual Clinical Report 2014. 2015. <http://nationalwomenshealth.adhb.govt.nz/Portals/0/Annual%20Reports/ACR%20master%20MOH%20July%202015.pdf>
- Auckland District Health Board. National Women's annual clinical report 2011. Secondary National Women's annual clinical report 2011. 2011. [http://nationalwomenshealth.adhb.govt.nz/Portals/0/Annual%20Reports/Final\\_2011\\_ACR\\_PDF\\_24\\_July\\_2012.pdf](http://nationalwomenshealth.adhb.govt.nz/Portals/0/Annual%20Reports/Final_2011_ACR_PDF_24_July_2012.pdf)
- Statistics New Zealand. Births and Deaths: Year ended December 2015. Secondary Births and Deaths: Year ended December 2015 2016. [http://www.stats.govt.nz/browse\\_for\\_stats/population/births/BirthsAndDeaths\\_HOTPYeDec15.aspx](http://www.stats.govt.nz/browse_for_stats/population/births/BirthsAndDeaths_HOTPYeDec15.aspx)
- Statistics New Zealand. Births and Deaths: Year ended December 2014. Secondary Births and Deaths: Year ended December 2014 2015. [http://www.stats.govt.nz/browse\\_for\\_stats/population/births/BirthsAndDeaths\\_HOTPYeDec14.aspx](http://www.stats.govt.nz/browse_for_stats/population/births/BirthsAndDeaths_HOTPYeDec14.aspx)
- Ministry of Health. Screening, Diagnosis and Management of Gestational Diabetes in New Zealand: A clinical practice guideline. Secondary Screening, Diagnosis and Management of Gestational Diabetes in New Zealand: A clinical practice guideline. Wellington (New Zealand): Ministry of Health; 2014.
- Simmons D, Rowan J, Reid R, et al. Screening, diagnosis and services for women with gestational diabetes mellitus (GDM) in New Zealand: a technical report from the National GDM Technical Working Party. *NZMJ* 2008;121:74-86.
- Ministry of Health. Māori health literacy research: Gestational diabetes mellitus. Secondary Māori health literacy research: Gestational diabetes mellitus. Wellington (New Zealand): Ministry of Health; 2014.
- Winnard D, Anderson P. Diabetes in pregnancy in CMDHB - report on trends over time, a 2011 snapshot and service implications. Secondary Diabetes in pregnancy in CMDHB - report on trends over time, a 2011 snapshot and service implications. 2013. <http://countiesmanukau.health.nz/assets/About-CMH/Reports-and-planning/Diabetes/2011-Diabetes-in-Pregnancy-Trends-2011-snapshot.pdf>
- Ekeroma AJ, Chandran GS, McCowan L, et al. Impact of using the International Association of Diabetes and Pregnancy Study Groups criteria in South Auckland: prevalence, interventions and outcomes. *The Australian & New Zealand journal of obstetrics & gynaecology* 2015;55:34-41.

13. Ministry of Health. Population of Bay of Plenty District Health Board. Secondary Population of Bay of Plenty District Health Board. 2014/15. <http://www.health.govt.nz/new-zealand-health-system/my-dhb/bay-plenty-dhb/population-bay-plenty-dhb>
14. Atkinson J, Salmond C, Crampton P. NZDep2013 Index of Deprivation. Secondary NZDep2013 Index of Deprivation. Wellington (New Zealand): Department of Public Health, University of Otago; 2014. <http://www.health.govt.nz/publication/nzdep2013-index-deprivation>
15. Chamberlain C, McNamara B, Williams ED, et al. Diabetes in pregnancy among indigenous women in Australia, Canada, New Zealand and the United States. *Diabetes Metab Res Rev* 2013;29:241-56.
16. Simmons D. Diabetes and obesity in pregnancy. Best practice & research. *Clinical obstetrics & gynaecology* 2011;25:25-36.
17. Rowan JA, Budden A, Ivanova V, et al. Women with an HbA of 41-49 mmol/mol (5.9-6.6%): a higher risk subgroup that may benefit from early pregnancy intervention. *Diabet Med* 2015.
18. Kim C, Newton KM, Knopp RH. Gestational diabetes and the incidence of type 2 diabetes: a systematic review. *Diabetes Care* 2002;25:1862-8.
19. Han S, Middleton PF, Bubner TK, Crowther CA. Women's views on their diagnosis and management for borderline gestational diabetes mellitus. *Journal of diabetes research* 2015;209215.
20. McGrath NM, Evans C, Holgate A. Post-partum follow-up of women with gestational diabetes mellitus from Northland, New Zealand. *Diabet Med* 2007;24:218-9.
21. Cundy T, Ackermann E, Ryan EA. Gestational diabetes: new criteria may triple the prevalence but effect on outcomes is unclear. *BMJ* 2014;348:g1567.
22. Rowan J, Allen H, Budden A, et al. New Zealand National GDM Guidelines: an alternative view of some good practice points. *The Australian & New Zealand journal of obstetrics & gynaecology* 2015;55:17-20.
23. Coppel 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. *NZMJ* 2013;126:23-42.
24. Rush E, Crook N, Simmons D. Point-of-care testing as a tool for screening for diabetes and pre-diabetes. *Diabet Med* 2008;25:1070-5.
25. Simmons D, Devers MC, Wolmarans L, Johnson E. Difficulties in the use of risk factors to screen for gestational diabetes mellitus. *Diabetes Care* 2009;32:e8.
26. Aung YY, Sowter M, Kenealy T, et al. Gestational diabetes mellitus screening, management and outcomes in the Cook Islands. *NZMJ* 2015;128:21-8.

# Unhealthy food marketing to New Zealand children and adolescents through the internet

Stefanie Vandevijvere, Karuna Sagar, Bridget Kelly, Boyd Swinburn

## ABSTRACT

**AIM:** To assess the extent and nature of unhealthy food marketing to New Zealand children and adolescents through the internet.

**METHODS:** Internet traffic data for January 2014 was purchased from AC Nielsen to identify the most popular websites (n=110) among children and adolescents aged 6–17 years. In addition, websites (n=70) of food and beverage brands most frequently marketed to children through television, sports, magazines and Facebook were included. Marketing techniques and features on those websites were analysed.

**RESULTS:** The extent of food marketing on popular non-food websites was low. A wide range of marketing techniques and features was, however, identified on food brand websites, including advercation (87%), viral marketing (64%), cookies (54%), free downloadable items (43%), promotional characters (39%), designated children's sections (19%) and advergaming (13%). Most techniques appeared more frequently on websites specifically targeting children and adolescents, than on other websites targeting the general public.

**CONCLUSION:** Compared to traditional media, the internet allows food marketers to use engaging techniques to directly interact with children. While the range of marketing techniques and features identified on food brand websites was extensive, the most popular websites among children and adolescents were non-food related, and the extent of food marketing on those websites was found to be low. Additional assessment of food marketing to children through social and other digital media is recommended.

The prevalence of childhood obesity has increased dramatically worldwide since the 1980s, and is considered as one of the most serious public health issues of the 21<sup>st</sup> century.<sup>1,2</sup> The most recent New Zealand Health Survey (2015/2016)<sup>3</sup> showed that one in three children are overweight or obese; a two percentage-point increase since 2006/2007.<sup>4</sup>

Unhealthy food marketing to children is one risk factor for childhood obesity.<sup>5,6</sup> Marketing messages reach children through a variety of media, such as television, magazines, radio, sports and increasingly the internet and new media.<sup>7</sup> Unhealthy food products, such as sugary drinks, savoury snacks, confectionary and sweetened breakfast cereals, are the products most frequently marketed to children on television.<sup>5</sup> A recent review revealed a variety

of persuasive marketing techniques used by advertisers to promote food products to children on television,<sup>8</sup> including free gifts, toys, coupons, celebrity endorsements, discounts and competitions. These forms of marketing techniques have been found to increase children's preferences for the advertised foods.<sup>8</sup> The latest New Zealand study on television food marketing in 2006 found that the majority of unhealthy food advertisements occurred during children's peak television viewing times.<sup>9</sup> In addition, some major food brands sponsor sports<sup>10,11</sup> and pay for advertising in children's magazines<sup>12</sup> in New Zealand, which may further accumulate exposure of children to certain brands.

Unlike in several other developed countries (Sweden, Norway, Ireland, UK, Chile),<sup>13</sup> there are currently no regulations in place by the New Zealand Government to reduce

exposure of children to unhealthy food marketing through any type of medium. The Advertising Standards Authority, an industry body, established the Children's Code for Advertising Food in 2010.<sup>14</sup> This Code defines the age of a child as less than 14 years, and states that '*Food advertisements should not undermine the food and nutrition policies of the Government, the Ministry of Health Food and Nutrition Guidelines nor the health and wellbeing of children*', and '*advertisements should not encourage over-consumption of any food*'.<sup>14</sup> In addition, free-to-air television in New Zealand supplements this Code by voluntary rules concerning advertising during children's programmes as outlined in the document 'Getting it right for children', last updated in 2008.<sup>15</sup> However, research in New Zealand<sup>9–12,16</sup> and internationally<sup>17</sup> has consistently shown that self-regulation of food marketing by the food industry does not lead to measurable reductions in children's exposure to unhealthy food marketing.

With the emergence of the internet and new media over the past decade, there has been a shift in some of children's screen time from television to the internet. The World Internet Project Survey reported a steady increase in internet users from 82% in 2007 to 92% in 2013 in New Zealand.<sup>18</sup> In addition, the percentage of households with an internet connection has continued to increase from 37.4% in 2001 and 60.5% in 2006 to 76.8% in 2013. In 2014 about half of the children 6–14 years old used the internet at home every day.<sup>19</sup> In 2006 it was found that around 65% of Australian children aged between 5 to 14 years were using the internet, and 79% of children aged between 5 to 8 years went online at home.<sup>20</sup>

It has been found previously in Australia that food companies attract the attention of children and extend their exposure time on the internet through interesting games, website-featured competitions, promotional characters, email greeting cards and free downloadable items.<sup>21</sup>

In addition, advergames (where the product is an essential part of the game) engages the player through entertainment and competitions,<sup>22</sup> and has been shown to increase children's preferences for advertised food products.<sup>23,24</sup> Monitoring the extent and nature of unhealthy food marketing to children through a wide range

of media is necessary to increase accountability of governments and the food industry to reduce childhood obesity.<sup>25</sup> This study specifically focuses on assessing, for the first time, the extent and nature of unhealthy food and non-alcoholic beverage promotion to New Zealand children and adolescents through the internet. Previously, other studies in New Zealand have investigated food marketing to children through television, sport sponsorship (through analysis of websites from national and regional New Zealand sporting organisations), food product packaging and magazines.<sup>9–12,16</sup>

## Methods

The study was approved by the University of Auckland Human Participants Ethics Committee.

### Sampling of websites

The most popular websites among New Zealand children and adolescents aged 6–17 years for January 2014 were selected by purchasing internet traffic data from AC Nielsen, a market research company. All websites that attracted an audience greater than 1.5% of the target population ( $\geq 10,365$  New Zealand children and adolescents 6–17 years) were included.

This list included mainly non-food websites, such as search engines, websites related to games, news, television, magazines, movies, cartoons, online shopping, banks, and encyclopaedia. One super-market food brand website was included (countdown.co.nz).

Additional food brand websites were selected based on the frequency of marketing of food brands through other media, such as television, sport sponsorship and magazines, as derived from previous New Zealand studies<sup>10,12,26</sup>, and from Social-bakers, an online social media monitoring company.<sup>27</sup> The Australian website of the selected food brands was considered for inclusion where there was no existing New Zealand website (as a few brands use one website for both Australia and New Zealand).

### Coding of websites

Two coding tools, one for the popular, non-food websites, and one for the food brand websites, were adapted from an Australian study,<sup>21</sup> and used to measure the extent and nature of unhealthy food

promotion through the internet in New Zealand. Marketing techniques and related features used on food brand websites (eg advercation, advergaming, viral marketing, promotional characters (see Table 1)) were recorded. The coding tools were pilot tested for a small sample of websites (n=4), prior to the actual data collection. The coding for these four websites by KM was checked by one of the lead researchers (SV), and no discrepancies were found. The data collection took place during May–June 2014.

A small sample of food brand websites (n=18), mainly fast food restaurant websites and websites specifically targeting children and adolescents, were re-coded by KM during the month of September 2014 to identify any changes in the frequency of food marketing techniques and features used by internet food marketers over time.

All food and beverage products marketed on websites were classified as “Everyday” (healthy), “Sometimes” or “Occasional” (unhealthy) foods, according to the New Zealand Ministry of Health Food and Beverage Classification System.<sup>28</sup> Foods were categorised into seven groups: 1) non-alcoholic beverages, 2) vegetables and fruit, 3) breads and cereals, 4) milk and milk products, 5) meat, fish, poultry and meat alternatives, 6) mixed meal dishes and 7) snack items.<sup>28</sup>

### Coding of food brand websites

Characteristics of websites assessed included the target audience (children 6–12 years, adolescents 13–17 years and general population) and a range of marketing techniques, such as brand identifiers, gaming and children’s sections, promotional characters, promotions, opportunities for extending website experience, marketing partnerships and tie-ins, brand benefit claims, nutrition and health claims, protection for children, registration and accounts and educational material.

Details on the features assessed within each marketing technique are given in Table 1. All the games on the food brand websites were played once by KM to identify the presence of food products, brand logos, spokes characters, music, sound effects and animation during the games.

Websites were classified as targeting children when they included fun, fantasy and adventure themes or games, and were classified as targeting adolescents when they included fashion, image and sexuality themes. The promotional characters were categorised into six types, adapted from Hebden et al:<sup>29</sup> cartoon/company-owned character, licensed character, amateur sportsperson, famous sportsperson, celebrity and movie tie-in. Premium offers (downloads, buy one, get one free, etc.) were also captured.<sup>29</sup> An internationally standardised system was used to categorise health-related labelling components on food products (eg health and nutrition claims).<sup>30</sup>

For specific brand or standalone websites (eg [weetbix.co.nz](http://weetbix.co.nz)), the entire site was coded. For the food brand websites which were just a page/link on the company’s website, the entire page, including the information located within two mouse clicks away from the brand page, was coded. For the food company websites, the home page and all the pages two mouse clicks away from the home page were coded.

### Coding of popular non-food websites

Both branded and non-branded food references were captured and coded. Unlike for the food brand websites, for which the unit of analysis was the website, for popular non-food websites, the unit of analysis was the food referenced. Every pictorial or written reference to food on the websites was included.

For branded food references, the type of advertisement, the size of the advertisement and the marketing techniques and features (similar as for food brand websites as explained above) were captured.

The type of advertisement was categorised into (a) direct advertisements; (b) part of editorial content (interviews, comics, stories); (c) product competition or promotion; (d) activity (games, puzzles and quizzes); (e) inclusion in a recipe; (f) association with an icon (cartoon characters, celebrities, sporting figureheads); or (g) links to other media marketing (such as food company’s internet site or their Facebook page).<sup>31</sup>

## Statistical analysis

Since the number of websites that appeared to be specifically targeting adolescents was very low ( $n=4$ ), they were analysed in combination with the websites targeting children. The data were analysed in SPSS version 20 for Windows (SPSS Inc., Chicago, USA). The frequency of marketing techniques and features used on websites targeting children and adolescents and websites targeted to the general population were compared using chi square tests.

Furthermore, frequency of marketing techniques used on a selected sample of food brand websites was compared between two different time periods, using chi square tests.

Due to multiple comparisons, the significance level was adjusted to 0.003 instead of 0.05 (Bonferroni correction). The proportion of healthy 'Everyday' foods versus unhealthy 'Occasional' foods was calculated for both food brand and popular, non-food websites.

## Results

### Food brand websites

In total, 70 food brand/company websites were included in the study, of which 40 were stand-alone websites and 30 were a page link on a food company website. The majority of the included food brands/companies ( $n=66$ ) had a New Zealand website, which was analysed. For the other four brands, the Australian website was analysed. The majority of websites were classified as 'targeted towards the general population' ( $n=46$ ), followed by 'targeting children' ( $n=20$ ), and only four websites were classified as 'targeting adolescents'.

All food brand websites displayed brand logos, 93% of websites contained different brand variants (eg size or flavours), 89% of websites included product packaging graphics, and 87% of websites included images of products in the background. Many websites contained advercation (87%), brand benefit claims for taste and quality of food products (96%) and viral marketing (such as links to Facebook and other social media pages) (64%). Overall, 33% of websites included television advertisements, and 34% of websites included competitions and/or giveaways. Rebates, such as combo meals, value packs or special

discounted items, were found on 13% of websites, predominantly on fast food restaurant websites (Table 1).

About 19% of websites contained a children's designated area, and 13% of websites contained advergames (Table 1). In total, 91 advergames were identified on food brand websites, ranging from one to 76 games per website. The games mainly included adventure games (15%), creative games (15%) and sport games (14%). The majority of advergames included features to extend the game (76%), such as leader boards—including rank, nickname, best game and total score of all games, the opportunity to post scores publicly, and play again option. Animations appeared within 60% of games, special sound effects within 59% of games and music within 41% of games. Some games (30%) created opportunities to personalise with the character/player in the game. None of the websites with advergames specified age restrictions or required parental consent to play the games.

The use of promotional characters (67% vs 24%,  $p=0.001$ ) and tie-ins (58% vs 44%,  $P>0.050$ ) was more concentrated on websites targeting children and adolescents, than websites targeting the general population. Websites targeting children and adolescents offered advergames (25% vs 7%,  $p=0.054$ ) and giveaways (29% vs 11%,  $p=0.054$ ) more frequently than websites targeting the general population. The most common giveaway item was a 'toy' with a children's product or a kid's meal. Viral marketing was more frequent on websites targeting children and adolescents (79% vs 57%,  $p=0.061$ ) than on websites targeting the general population, and was the most common feature used on websites targeting children to increase website experience and brand exposure. More than half of the websites targeted to children and adolescents (54%) offered free downloadable items, such as branded characters (eg Ronald MacDonald), logos, menus or colouring pages.

About 50% of websites targeting children offered the opportunity to register and become a member or enter in a promotion, whereas only 2% of websites targeted to the general population offered those options ( $p<0.001$ ). Moreover, only 4% of children's websites required parental consent, and

13% contained information for parents related to registrations and accounts.

A higher proportion of websites targeting children and adolescents than those targeting the general population (75% vs. 44%,  $p=0.012$ ) used cookies to save passwords, to keep track of what was purchased, to estimate number of users and to determine overall traffic patterns on the website.

In total, 74.6% food brand websites advertised food products, which were of low nutritional value, and were classified as unhealthy “Occasional” foods according to the Ministry of Health food and beverage classification system. The highest frequency of marketing was for sweet snacks (including ice cream) (23%), followed by mixed meal dishes (13%) and breakfast cereals (10%). The prevalence of marketing for “occasional” foods was found to be higher on websites targeting children and adolescents (88%) than on websites targeting the general population (57%).

There were no significant differences ( $p>0.05$ ) in the frequency of use of any of the marketing techniques on food brand websites between two different time periods. However, it has to be noted that the sample size of websites, which were re-analysed, was small.

### Popular non-food websites

Out of 110 popular non-food websites analysed, only 15 websites included branded or non-branded food references ( $n=65$  in total, of which only  $n=5$  were branded). The majority of food references were found on game websites. The food categories referenced most frequently were mixed meal dishes (38%), followed by sweet snacks (28%). The majority of foods referenced (63%) were classified as unhealthy “occasional” foods, and 12% were classified as healthy “everyday” foods. Since only five branded food references were found on non-food websites, further details on marketing techniques are not reported here.

## Discussion

This is the first study to comprehensively assess the extent and nature of food marketing to New Zealand children and adolescents through the internet, including through both food brand and popular non-food websites.

While the extent of food marketing on popular, non-food websites was low, a wide range of marketing techniques and features were identified on food brand websites. In general, marketing techniques appeared more frequently on websites specifically targeting children and adolescents, than on other websites targeting the general population.

Surprisingly, the frequency of food references on non-food websites (2014) was very low in our study, compared to Australia (2006), where about 11 food references per website were found.<sup>21</sup> Also in the US, food advertising on third-party websites was found to be much more common than in New Zealand.<sup>37</sup> One of the potential explanations might be that companies increasingly focus their marketing efforts on social and other digital media (eg Facebook), which have become much more popular since the Australian study was conducted ten years ago. A few recent Australian studies,<sup>43,44</sup> and a recently published report from the World Health Organization Europe<sup>45</sup> show that unhealthy food advertising through Facebook and other digital media is concerning, since it amplifies advertising in traditional media and is very poorly regulated and monitored.<sup>7</sup>

The dynamic and sophisticated nature of digital media enables food marketers to directly connect and engage with their target audiences in new ways, which are beyond those delivered by the traditional media, such as television.<sup>7,45</sup>

Viral marketing, for example, encourages children to send marketing messages to their friends, without understanding the blurring line between advertising and content. Furthermore, the use of ‘cookies’ (small files that track user’s online activities and collect user information to facilitate targeted marketing) allows marketer’s direct, inconspicuous insight into children’s behaviour in ways not available in the past.<sup>38</sup> Food marketers also encourage children to register and open an account, by providing benefits to members, such as access to special games, free downloadable items, etc. In addition, advergames on some food company websites engage children with branded characters and branded food items. One study in the US found a significantly higher proportion of children visitors on

branded food company websites with advergames than those without advergames.<sup>24</sup>

Similar to traditional media platforms,<sup>9,10,12</sup> internet marketing was found to predominantly promote unhealthy foods to New Zealand children and teenagers. Consequently, there is a need for regulations to protect children from unhealthy food and beverage marketing messages. Presently there are no regulations on any marketing activities in New Zealand. According to the World Health Organization, government regulations have the highest potential to decrease the extent of exposure of children to unhealthy food and beverage marketing.<sup>46</sup> In this study, the majority of the food brand websites were New Zealand specific, which is important for policy implementation within New Zealand. While a new draft self-regulatory Code for advertising to children and adolescents has recently been proposed,<sup>47</sup> this is very unlikely to have any impact to reduce unhealthy food marketing to children through digital media.

Restriction of the use of cookies as a violation of children's privacy could be introduced, such as in the US, where digital tracking of children younger than 13 years is limited.<sup>48</sup> Verifiable parental consent is needed to collect personal information from children younger than 13 years old, and tracking across platforms with geo-location or behavioural advertising is not allowed.<sup>45</sup> Verifiable parental consent should be required for membership or registration on websites, especially for those featuring advergames. Hardly any of the food brand websites investigated in this study contained regulated age restrictions and parental consent.

In addition, food companies should include privacy policy information with clear specification of age of target audience, any marketing features used on websites, such as advergames, and personal information collected from children. Viral marketing needs to be restricted to reduce the spread

and reach of unhealthy food marketing to children and adolescents. Other strategies to protect children against harmful effects of food marketing could also be used, such as mass media campaigns, including counter advertising.<sup>49</sup> In addition, companies could be much more transparent about their policies related to restricting unhealthy food marketing to children, including types of media and marketing techniques, and put those clearly up on their website.<sup>50</sup>

The strengths of the study include the selection of websites based on net ratings data for the target population and food brands most frequently marketed through a range of media in New Zealand. The large sample of websites included in the study assisted to assess the potential exposure of children and adolescents to marketing messages. Furthermore, a small sample of food websites were reanalysed at a later time to identify changes in frequency of different types of promotional activities over time.

The limitations include that actual exposure of children and adolescents to food marketing through the internet (especially through food brand websites), and the extent of food marketing through social and other digital media (eg Facebook) was not assessed.

## Conclusions

Compared to traditional media, the internet allows food marketers to use engaging techniques to target children and directly interact with them. While the range of marketing techniques and features identified on food brand websites was extensive, the most popular websites among children and adolescents were non-food related, and the extent of food marketing on those websites was found to be low.

Additional assessment of food marketing to children and adolescents through social and other digital media is crucial, since companies may have shifted their marketing efforts to those new media.

**Competing interests:**

Dr Swinburn and Dr Vandevijvere report grants from Health Research Council of New Zealand, during the conduct of the study.

**Acknowledgements:**

The authors acknowledge support from the University of Auckland Faculty Research Development Fund for carrying out this study. SV is supported by a research fellowship from the Heart Foundation of New Zealand. SV and BS designed the study; BK, SV and KS designed the tool for data collection, KS collected the data and analysed the results; SV drafted the manuscript; all authors critically revised draft versions of the manuscript and approved the final version.

**Author information:**

Stefanie Vandevijvere, Senior Research Fellow, University of Auckland, Auckland; Karuna Sagar, Honours Student, University of Auckland, Auckland; Bridget Kelly, Senior Research Fellow, University of Wollongong, Australia; Boyd Swinburn, Professor, University of Auckland, Auckland.

**Corresponding author:**

Dr Stefanie Vandevijvere, Senior Research Fellow, University of Auckland, Auckland.  
s.vandevijvere@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7157>

**REFERENCES:**

- Morgen CS, Sorensen TI. Obesity: global trends in the prevalence of overweight and obesity. *Nature reviews Endocrinology*. 2014; 10(9):513–4.
- Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet*. 2014; 384(9945):766–81.
- Ministry of Health. Annual Update of Key Results 2015/16: New Zealand Health Survey. Wellington: Ministry of Health; 2016.
- Ministry of Health. A Portrait of Health: Key results of the 2006/07 New Zealand Health Survey. Wellington: Ministry of Health; 2008.
- Cairns G, Angus K, Hastings G. The extent, nature and effects of food promotion to children: a review of the evidence to December 2008. Geneva: World Health Organization; 2009.
- Hastings G, McDermott L, Angus K, Stead M, Thomson T. The extent, nature and effects of food promotion to children: a review of the evidence. Technical Paper prepared for the World Health Organization. Geneva: World Health Organization; 2007.
- Kelly B, Vandevijvere S, Freeman B, Jenkin G. New media but same old tricks: food marketing to children in the digital age. *Current obesity reports*. 2015.
- Jenkin G, Madhvani N, Signal L, Bowers S. A systematic review of persuasive marketing techniques to promote food to children on television. *Obes Rev*. 2014; 15(4):281–93.
- Wilson N, Signal L, Nicholls S, Thomson G. Marketing fat and sugar to children on New Zealand television. *Prev Med*. 2006; 42(2):96–101.
- Carter MA, Signal L, Edwards R, Hoek J, Maher A. Food, fizzy, and football: promoting unhealthy food and beverages through sport - a New Zealand case study. *BMC Public Health*. 2013; 13:126.
- Carter MA. Is Junk Food Promoted Through Sport? : University of Otago; 2013.
- No E, Kelly B, Devi A, Swinburn B, Vandevijvere S. Food references and marketing in popular magazines for children and adolescents in New Zealand: A content analysis. *Appetite*. 2014; 83:75–81.
- World Cancer Research Fund. WCRF International Food Policy Framework for Healthy Diets: NOURISHING 2014 [12/06/2014]. Available from: [http://www.wcrf.org/policy\\_public\\_affairs/nourishing\\_framework/food\\_marketing\\_advertising](http://www.wcrf.org/policy_public_affairs/nourishing_framework/food_marketing_advertising)
- Advertising Standards Authority. Children's Code For Advertising Food 2010 [17/06/2013]. Available

- from: [http://www.asa.co.nz/code\\_children\\_food.php](http://www.asa.co.nz/code_children_food.php)
15. Think TV. Advertising on Television. Getting it right for children [01/10/2013]. Available from: [http://www.thinktv.co.nz/wp-content/uploads/Booklet\\_Mar\\_2011.pdf](http://www.thinktv.co.nz/wp-content/uploads/Booklet_Mar_2011.pdf)
  16. Devi A, Eyles H, Rayner M, Mhurchu CN, Swinburn B, Lonsdale-Cooper E, et al. Nutritional quality, labelling and promotion of breakfast cereals on the New Zealand market. *Appetite*. 2014; 81:253–60.
  17. Galbraith-Emami S, Lobstein T. The impact of initiatives to limit the advertising of food and beverage products to children: a systematic review. *Obes Rev*. 2013; 14(12):960–74.
  18. Gibson A, Miller M, Smith P, Bell AC, Crothers C. World Internet Project: Internet trends in New Zealand (2007-2013). Auckland: AUT University; 2013.
  19. Broadcasting Standards Authority. Children's Media Use Study: How our children engage with media today: Broadcasting Standards Authority; 2015. Available from: [http://bsa.govt.nz/images/assets/Research/Childrens\\_Media\\_Report\\_2015\\_FINAL\\_for\\_publishing\\_2.pdf](http://bsa.govt.nz/images/assets/Research/Childrens_Media_Report_2015_FINAL_for_publishing_2.pdf)
  20. Australian Bureau of Statistics. Children's internet access and mobile phone ownership, selected characteristics. Children's participation in culture and leisure. Australian Bureau of Statistics; 2012.
  21. Kelly B, Bochynska K, Kornman K, Chapman K. Internet food marketing on popular children's websites and food product websites in Australia. *Public Health Nutr*. 2008; 11(11):1180–7.
  22. Culp J, Bell RA, Cassady D. Characteristics of food industry web sites and “advergames” targeting children. *J Nutr Educ Behav*. 2010; 42(3):197–201.
  23. Mallinckrodt V, Mizerski D. The effects of playing an advergame on young children's perceptions, preferences and requests. *Journal of Advertising*. 2007; 36(2):87–100.
  24. Harris JL, Speers SE, Schwartz MB, Brownell K. US food company branded advergames on the Internet: children's exposure and effects on snack consumption. *Journal of Children and Media*. 2012; 6(1):51–68.
  25. Kelly B, King L, Baur L, Rayner M, Lobstein T, Monteiro C, et al. Monitoring food and non-alcoholic beverage promotions to children. *Obes Rev*. 2013; 14 Suppl 1:59–69.
  26. Jenkin G, Wilson N, Hermanson N. Identifying ‘unhealthy’ food advertising on television: a case study applying the UK Nutrient Profile model. *Public Health Nutrition*. 2009; 12(05):614–23.
  27. Socialbakers [11/11/2013]. Available from: <http://www.socialbakers.com/>
  28. Ministry of Health. Food and beverage classification system for years 1-13. Wellington: Ministry of Health; 2007.
  29. Hebden L, King L, Kelly B, Chapman K, Innes-Hughes C. A Menagerie of Promotional Characters: Promoting Food to Children through Food Packaging. *Journal of Nutrition Education and Behavior*. 2011; 43(5):349–55.
  30. Rayner M, Wood A, Lawrence M, Mhurchu CN, Albert J, Barquera S, et al. Monitoring the health-related labelling of foods and non-alcoholic beverages in retail settings. *Obes Rev*. 2013; 14 Suppl 1:70–81.
  31. Kraak V, Pelletier DL. How marketers reach young consumers: implications for nutrition education and health promotion campaigns. *Family Economics and Nutrition Review*. 1998; 11(4):31.
  32. Alvy LM, Calvert SL. Food marketing on popular children's web sites: a content analysis. *J Am Diet Assoc*. 2008; 108(4):710–3.
  33. Cheyne AD, Dorfman L, Bukofzer E, Harris JL. Marketing sugary cereals to children in the digital age: a content analysis of 17 child-targeted websites. *Journal of health communication*. 2013; 18(5):563–82.
  34. Henry AE, Story M. Food and beverage brands that market to children and adolescents on the internet: a content analysis of branded web sites. *J Nutr Educ Behav*. 2009; 41(5):353–9.
  35. Lingas EO, Dorfman L, Bukofzer E. Nutrition content of food and beverage products on Web sites popular with children. *Am J Public Health*. 2009; 99 Suppl 3:S587–92.
  36. Weber K, Story M, Harnack L. Internet food marketing strategies aimed at children and adolescents: a content analysis of food and beverage brand web sites. *J Am Diet Assoc*. 2006; 106(9):1463–6.
  37. Ustjanauskas AE, Harris JL, Schwartz MB. Food and beverage advertising on children's web sites. *Pediatr Obes*. 2014; 9(5):362–72.
  38. Moore E, Rideout V. The Online Marketing of Food to Children: Is It Just Fun and Games? *Journal of*

- Public Policy & Marketing. 2007; 26(2):202–20.
39. Kelly B, Chapman K. Food references and marketing to children in Australian magazines: a content analysis. *Health Promot Int.* 2007; 22(4):284–91.
  40. Jones S, Wiese E, Fabrianesi B. Following the links: food advertising and promotion on children's magazine websites. *Journal of Nonprofit & Public Sector Marketing.* 2008; 20(2):165–90.
  41. Brady J, Mendelson R, Farrell A, Wong S. Online marketing of food and beverages to children: a content analysis. *Canadian journal of dietetic practice and research : a publication of Dietitians of Canada = Revue canadienne de la pratique et de la recherche en dietetique : une publication des Dietetistes du Canada.* 2010; 71(4):166–71.
  42. Lee M, Choi Y, Quilliam ET, Cole RT. Playing With Food: Content Analysis of Food Advergemes. *J Consum Aff.* 2009; 43(1):129–54.
  43. Freeman B, Kelly B, Baur L, Chapman K, Chapman S, Gill T, et al. Digital Junk: Food and Beverage Marketing on Facebook. *Am J Public Health.* 2014:e1–e9.
  44. Boelsen-Robinson T, Backholer K, Peeters A. Digital marketing of unhealthy foods to Australian children and adolescents. *Health Promot Int.* 2016; 31(3):523–33.
  45. World Health Organization Europe. Tackling food marketing to children in a digital world: trans-disciplinary perspectives Geneva: World Health Organization Europe; 2016.
  46. World Health Organization. A framework for implementing the set of recommendations on the marketing of foods and non-alcoholic beverages to children. Geneva, Switzerland: World Health Organisation; 2012.
  47. Advertising Standards Authority. Report on the review of the Children's Code for Advertising Food and the Code for Advertising to Children 2016 [27/10/2016]. Available from: <http://www.asa.co.nz/wp-content/uploads/2016/10/Final-Report-Childrens-Codes-Review.pdf>
  48. Federal Trade Commission. COPPA - Children's Online Privacy Protection Act [12/06/2014]. Available from: <http://www.coppa.org/>
  49. Dietz WH. New strategies to improve food marketing to children. *Health affairs.* 2013; 32(9):1652–8.
  50. Sacks G, Mialon M, Vandevijvere S, Trevena H, Crino M, Swinburn B. Comparison of food industry policies and commitments on marketing to children and product (re) formulation in Australia, New Zealand and Fiji. *Critical Public Health.* 2014.

**Table 1:** Marketing techniques and features used on food and beverage brand and company websites.

<b>Marketing techniques</b>	All websites (n=70)	Websites targeting children + adolescents (n=24)	Websites targeting general population (n=46)
Marketing features	Number of websites (n) and percentage (%)	Number of websites (n) and percentage (%)	Number of websites (n) and percentage (%)
<b>Brand identifiers</b>	<b>70 (100%)</b>	<b>24 (100%)</b>	<b>46 (100%)</b>
Brand logo	70 (100%)	24 (100%)	46 (100%)
Different brand variants	65 (92.9%)	23 (95.8 %)	42 (91.3%)
Product packaging graphics	62 (88.6%)	21 (87.5%)	41 (89.1%)
Product as part of the background	61 (87.1%)	23 (95.8%)	38 (82.6%)
Product serving suggestions	43 (61.4%)	15 (62.5%)	28 (60.9%)
Image of person consuming the product	19 (27.1%)	6 (25.0%)	13 (28.3%)
<b>Designated children's section</b>	<b>13 (18.6%)</b>	<b>6 (25.0 %)</b>	<b>7 (15.2%)</b>
<b>Advergaming</b>	<b>9 (12.9%)</b>	<b>6 (25.0%)</b>	<b>3 (6.5%)</b>
<b>General gaming</b>	<b>3 (4.3%)</b>	<b>3 (12.5 %)*</b>	<b>0 (0.0%)</b>
<b>Promotional characters</b>	<b>27 (38.6%)</b>	<b>16 (66.7%)**</b>	<b>11 (23.9%)</b>
Cartoons, company owned	18 (25.7%)	12 (50.0%)**	6 (13.0%)
Licensed characters	7 (10.0 %)	5 (20.8 %)*	2 (4.4%)
Famous sports persons	4 (5.7%)	3 (12.5%)	1 (2.2%)
Famous celebrities	3 (4.3%)	0 (0.0%)	3 (6.5 %)
Amateur sport persons	1 (1.4%)	1 (4.1%)	0 (0.0%)
Movie tie-ins	0 (0.0%)	0 (0.0%)	0 (0.0%)
<b>Premium offers</b>	<b>11 (15.7%)</b>	<b>4 (16.7%)</b>	<b>7 (15.2%)</b>
<b>Promotions</b>	<b>49 (70%)</b>	<b>16 (66.7%)</b>	<b>33 (71.7%)</b>
Television adverts	23 (32.9%)	9 (37.5%)	14 (30.4%)
Giveaways	12 (17.1%)	7 (29.2%)	5 (10.9%)
Competitions	12 (17.1%)	4 (16.7%)	8 (17.4%)
Fundraising	11 (15.7%)	4 (16.7%)	7 (15.2%)
Vouchers	9 (12.9%)	2 (8.3%)	7 (15.2%)
Rebates	9 (12.9%)	3 (12.5%)	6 (13.0%)
Sample offers	2 (2.9%)	2 (8.3%)	0 (0.0%)
Other	12 (17.1%)	4 (16.7%)	8 (17.4%)
<b>Opportunities to extend the website experience</b>	<b>67 (95.7%)</b>	<b>23 (95.8%)</b>	<b>44 (95.7%)</b>
Expanding usage of the brand (recipes)	55 (78.6%)	17 (70.8%)	38 (82.6%)

**Table 1:** Marketing techniques and features used on food and beverage brand and company websites (Continued).

Link to other food product web site or pages	54 (77.1%)	19 (79.2%)	35 (76.0%)
Viral marketing	45 (64.3%)	19 (79.2%)	26 (56.5%)
Free download items	30 (42.9%)	13 (54.2%)	17 (37.0%)
Draws	22 (31.4%)	8 (33.3%)	14 (30.4%)
Direct messages to children	9 (12.9%)	7 (29.2%)	2 (4.4%)
Link to other non- food websites	5 (7.1%)	1 (4.2%)	4 (8.7%)
<b>Marketing partnership and tie-ins</b>	<b>34 (48.6%)</b>	<b>14 (58.3%)</b>	<b>20 (43.5%)</b>
Other brands incorporated in premiums, giveaways, competition, sponsorship	24 (34.3%)	8 (34.8%)	16 (33.3%)
Television tie-ins	13 (18.6%)	5 (20.8%)	8 (17.4%)
Cartoon character tie-ins	5 (7.1%)	3 (12.5%)	2 (4.4%)
Movie tie-ins	1 (1.4%)	1 (4.2%)	0 (0.0%)
<b>Brand benefit claims</b>	<b>67 (95.7%)</b>	<b>23 (95.8%)</b>	<b>44 (95.7%)</b>
Sensory based characteristics	51 (72.8%)	20 (83.3%)	31 (67.4%)
Convenience	43 (61.4%)	17 (70.8%)	26 (56.5%)
Suggested use	37 (52.8%)	14 (58.3%)	23 (50.0%)
New brand development	27 (38.5%)	10 (41.7%)	17 (37.0%)
Suggested users	25 (35.7%)	12 (50.0%)	13 (28.3%)
Price	17 (24.2%)	5 (20.8%)	12 (26.1%)
Emotive claims	17 (24.2%)	7 (29.2%)	10 (21.7%)
Puffery	19 (27.1%)	10 (41.7%)	9 (20.0%)
<b>Nutrition labels</b>	<b>53 (75.7%)</b>	<b>20 (83.3%)</b>	<b>33 (71.7%)</b>
Basic nutrition information	50 (71.4%)	17 (70.8%)	33 (71.7%)
Specific nutrition claims	22 (31.4%)	9 (37.5%)	13 (28.3%)
Health claims	11 (15.7%)	4 (16.7%)	7 (15.2%)
Healthy eating strategies	7 (10.0%)	3 (12.5%)	4 (8.7%)
<b>Claims</b>	<b>39 (55.7%)</b>	<b>14 (58.3%)</b>	<b>25 (54.3%)</b>
Nutrient content claim	25 (35.7%)	9 (37.5%)	16 (34.8%)
Health related ingredient claim	21 (30.0%)	8 (33.3%)	13 (28.3%)
General health claim	11 (15.7%)	3 (12.5%)	8 (17.4%)
Nutrition and other function claim	8 (11.4%)	1 (4.2%)	7 (15.2%)
Reduction of disease risk claim	5 (7.1%)	0 (0.0%)	5 (10.8%)
Nutrient comparative claim	4 (5.7%)	0 (0.0%)	4 (8.7%)
Other claims (eg organic)	4 (5.7%)	2 (8.3%)	2 (4.4%)
<b>Registration and accounts</b>	<b>13 (18.6%)</b>	<b>12 (50.0%)**</b>	<b>1 (2.2%)</b>

**Table 1:** Marketing techniques and features used on food and beverage brand and company websites (Continued).

Website memberships	13 (18.6 %)	12 (50.0 %)**	1 (2.2%)
Benefits for members	13 (18.6 %)	12 (50.0%)**	1 (2.2 %)
<b>Protection for children</b>	<b>59 (84.3%)</b>	<b>24 (100%)*</b>	<b>35 (76.1%)</b>
Legal information available	59 (84.3%)	24 (100%)*	35 (76.1%)
Use of cookies	38 (54.3%)	18 (75%)*	20 (43.5%)
Information to parents	5 (7.1 %)	3 (12.5%)	2 (4.4%)
Age blocks	4 (5.7%)	3 (12.5%)	1 (2.2%)
Parents' consent	1 (1.4%)	1 (4.2 %)	0 (0.0%)
Time restriction	0 (0.0%)	0 (0.0%)	0 (0.0%)
<b>Educational material (adverca- tion)</b>	<b>61 (87.1%)</b>	<b>22 (91.6%)</b>	<b>39 (84.7%)</b>
<i>Type of education</i>			
Details on product ingredients	43 (61.4%)	13 (54.1%)	30 (65.2%)
Historical facts	31 (44.3%)	14 (58.3 %)	17 (37.0%)
General nutrition	17 (24.3%)	3 (12.5%)	14 (30.4%)
Sports information	4 (5.7%)	1 (4.2%)	3 (6.5%)
Other	9 (12.9%)	4 (16.7%)	5 (10.9%)

\*P<0.05 (none is <0.003); \*\* p<0.003.

# Psychosocial enhancement of the Green Prescription for obesity recovery: a randomised controlled trial

Doug Sellman, Ria Schroder, Daryle Deering, Jane Elmslie, James Foulds, Chris Frampton

## ABSTRACT

**AIMS:** Kia Ākina is a low-cost obesity recovery network providing ongoing addiction-orientated psychosocial support. This study explored the impact of Kia Ākina when added to the Green Prescription, a key government-funded health promotion programme in New Zealand.

**METHODS:** A randomised controlled trial (ACTRN12613001160729) involving 108 participants recruited from primary care compared Green Prescription plus Kia Ākina (KA/GRx) with Green Prescription alone (GRx) over 12 months. The primary *a priori* outcome measure was achieving 5% loss of weight from baseline.

**RESULTS:** KA/GRx participants lost more weight overall than GRx (3.6kg vs 0.7kg,  $p=0.03$ ), while 39% of the GRx group *gained* weight compared with 21% of KA/GRx ( $p=0.04$ ). However, KA/GRx and GRx had similar proportions with weight loss of 5% or greater (20% vs 17%,  $p=0.62$ ). KA/GRx participants had greater changes in confidence about recovery ( $p=0.02$ ), and quality of life measures ( $p=0.03$ ) and greater overall satisfaction with assistance received ( $p<0.001$ ) compared with GRx participants.

**CONCLUSIONS:** Psychosocial support provided through Kia Ākina enhanced treatment outcomes for people with obesity at 12 months when added to GRx, although weight-loss outcomes were modest. Before Kia Ākina is expanded, improved weight-loss outcomes are required, which may be achieved through individualised assessment and targeted dietary assistance.

The global increase in people with obesity<sup>1</sup> is well represented in New Zealand where obesity rates have sharply increased over the past 30 years so that over 30% of the general population 15 years and above are now obese.<sup>2</sup>

While governments across the world struggle with this epidemic,<sup>3</sup> a fundamental challenge for health care systems is how to assist the thousands of people who now suffer from obesity.

Despite decades of research and formulation of weight-loss guidelines<sup>4-6</sup> long-term non-surgical treatment outcomes for obesity remain disappointing.<sup>7,8</sup> Even achieving the minimal permanent weight loss of 5% required to produce clinically significant improvements has proved too difficult for the majority.<sup>9,10</sup> Nevertheless, metabolic benefits can occur with healthy nutrition and

increased physical activity in the absence of significant weight loss.<sup>11</sup>

Primary care is a setting widely recognised as the most appropriate for the treatment of obesity and associated health conditions. However, the effectiveness of interventions by generalist health professionals and counselling by physicians is questionable.<sup>12,13</sup> Lack of training, time and infrastructural support plus cynicism about treatment effectiveness have been identified as factors undermining physicians' effectiveness.<sup>14</sup>

The Green Prescription has been demonstrated to be an inexpensive intervention for increasing activity in sedentary people<sup>15</sup> and is a key government-funded programme for assisting people with a range of chronic conditions, including obesity, to improve their health primarily through increasing physical activity.<sup>16</sup> The most common

referrals are people who are overweight. This strategy is supported by evidence that exercise alone can be effective in weight reduction,<sup>17</sup> and that, even when no weight loss occurs, exercise will improve general health as reflected in the reduction of cardiovascular disease risk factors.

Research on the role of addictive processes in obesity has been accelerating in recent years, and the use of addiction methods and therapies in assisting people with obesity is gaining traction.<sup>18,19</sup>

Kia Ākina (“be encouraged and supported”) is an evolving obesity recovery network that emerged out of a study comparing Weight Watchers and Overeaters Anonymous. The study identified the need for a group-based addiction treatment programme for people with obesity wanting to lose weight, that provided ongoing support, was financially accessible and “non-religious”.<sup>22</sup> Kia Ākina provides ongoing psychosocial support incorporating addiction and standard weight-loss strategies, encourages self-discovery and focuses on weight-loss based on a sustainable new recovery lifestyle.

Weight loss is the primary outcome measure in clinical studies of obesity.<sup>10</sup> However, at times this focus has been to the exclusion of broader personal functioning measures, including quality of life data, limiting the overall clinical and life significance of weight-loss results.<sup>20</sup>

This study aimed to investigate whether psychosocial support provided by Kia Ākina enhances the weight-loss and other outcomes from the Green Prescription programme within New Zealand primary care services.

## Methods

The design was a parallel two-group, randomised controlled trial comparing Green Prescription plus Kia Ākina (KA/GRx) with Green Prescription alone (GRx) for people with obesity (BMI >30) (kg/m<sup>2</sup>), recruited during a routine primary care consultation.

The Green Prescription was provided by the GP before the GP or practice nurse obtained formal consent for the study, undertook BMI measurement and conducted a baseline physical fitness test. Contact details of each recruited participant were

then relayed to a National Addiction Centre researcher (RS) who arranged for baseline self-report measures to be completed via an online questionnaire. Randomisation to one of the two treatment groups (KA/GRx or GRx) occurred once these on-line measures were completed.

Randomisation involved a computer-generated random allocation sequence (1:1), stratified for gender and primary care venue, independent of the study’s clinicians.

Participants in the KA/GRx arm were invited to attend an introductory Kia Ākina workshop. Both treatment groups were actively encouraged by their primary care physician and practice nurse to become involved in the Green Prescription opportunities.

The study was registered with the Australian and New Zealand Clinical Trials Registry (ANZCTR) (ID: ACTRN12613001160729). Ethics approval was given by the Southern Health and Disabilities Ethics Committee (Ref: 13/STH/151).

## Sample

One hundred and fifty-nine patients were screened during 2013/2014 in four geographically separate (North, South, West, North/West) general primary care venues in Christchurch, New Zealand. Inclusion criteria were: 23–65 years old, not currently involved in other weight loss programmes, with no current significant medical condition or undergoing medical treatment likely to significantly affect weight, or which would make weight loss or dietary restriction contraindicated. Fifty-one patients were excluded (11 did not meet inclusion criteria, 37 declined to participate and three for reasons unknown). The remaining 108 were recruited for the study, and randomised into the two groups. The percentage of men who were screened in but declined to participate (23%) was significantly higher than the percentage of men who subsequently participated in the study (9%),  $p < 0.01$ .

## Interventions

The Green Prescription primarily provides free consultations with a qualified and experienced physical activity coach who helps to support each person to discover suitable physical activity options in their community. Each participant also has

the opportunity to try a range of activities in a supported environment, discuss topics that support a healthy lifestyle, establish a plan of activity suited to meet individual need and be supported by other participants and Green Prescription staff. During the time of the study, the Green Prescription programme in Canterbury underwent a widening of scope from a primary focus on physical activity to incorporate instruction about healthy food and eating behaviour. An eating programme “Appetite for Life”,<sup>22</sup> group support and education sessions about healthy living, as well as text and email encouragement were added.

Kia Ākina is an obesity recovery network which utilises six standard addiction treatment strategies: permanent life-style change;<sup>23</sup> safe non-stigmatising venue;<sup>24</sup> motivational enhancement principles;<sup>25</sup> abstinence-based food-rules;<sup>26</sup> harm reduction<sup>27</sup> and care of long-term medical conditions;<sup>28</sup> and self-help recovery group processes. Kia Ākina primarily provides ongoing psychosocial support, but is grounded in traditional evidenced-based approaches to weight loss, involving a combination of Food/diet modification, increased physical Activity and Behavioural strategies, termed the FAB approach.<sup>4</sup>

Participants are encouraged to set personal weight loss goals, either on their own or in interaction with the network, select from a range of options to be involved in and work at their own pace. The options include face to face meetings—six monthly workshops (two hours each) and weekly facilitated group discussion meetings (one hour), with topics determined by participants—an ongoing email discussion group based on a weekly email message addressing one of five key principles (Take Control, Get Active, Eat Well, Persist, Enjoy Life),<sup>29</sup> a daily text buddy system and regular motivational text messages.

A list of 50 energy-dense, nutrient-poor foods high in fat, sugar and/or containing alcohol, has been developed,<sup>30</sup> referred to as the NEEDNT Food List (Non-Essential, Energy-Dense, Nutritionally-deficient). This list provides a starting point for participants to identify problematic foods to work on.

## Measures

The primary *a priori* outcome was a 5% reduction in weight at 12 months or

not. Additional weight measures included 12-month total weight loss, % weight loss and % excess weight loss (%EWL) using a BMI of 29 as the target weight. Secondary measures included: physical fitness using the 2-minute step in place test (Step Test)<sup>31</sup> combined with heart recovery rate at 1 minute (HRR1)<sup>32, 33</sup> the Kessler 10 Psychological Distress Scale (K10);<sup>34</sup> two Likert scales (0–10 scale) measuring the two central constructs of readiness for change<sup>25</sup>—the importance of change to the individual and confidence that change is possible; and a quality of life questionnaire, WHOQOL-BREF.<sup>35</sup>

Following initial baseline assessment, the practice nurse completed three-monthly weight (digital scales) and six-monthly physical fitness measures. The Step Test measures the number of steps a participant achieves in 2 minutes—right knee raised above a set level to achieve one step. HRR1 is calculated as the heart rate immediately at the end of the Step Test minus the heart rate one minute later. In addition to demographic, weight-loss and substance use questions at baseline, the other measures were completed on-line at baseline, six- and 12-months.

Likert scales (ranging 1–7) were administered six-monthly to check the total amount of food advice given by the GP and practice nurse. The Communication, Comfort and Rapport subscales of the Medical Interview Satisfaction Scale<sup>36</sup> measuring the quality of the therapeutic relationships were administered online six-monthly.

Finally, acceptability of adding Kia Ākina to the Green Prescription was assessed through a measure of overall satisfaction of assistance received using a 5-point Likert Scale (1. Very Satisfied–5. Very Unsatisfied).

## Statistical analysis

The primary analyses of weight changes at 12 months were undertaken on an intention to treat basis, with missing weight-loss data imputed from 12-month group means. Secondary sensitivity analyses were undertaken on those who remained in the KA/GRx or GRx groups at 12 months. T-tests and chi-square tests were undertaken to compare outcomes where appropriate, with comparisons summarised as odds ratios and effect sizes (Cohen d).

**Table 1:** Demographic, substance and weight characteristics of the sample (n=108), and by treatment groups (n=54 each).

	Total	(sd)	KA/GRx	(sd)	GRx	(sd)
Current age (years):	43.7	(10.9)	45.1	(10.9)	42.4	(10.9)
Baseline weight (kg):	111.2	(21.4)	111.6	(21.2)	110.8	(21.9)
Baseline BMI (kg/m <sup>2</sup> ):	40.9	(7.1)	41.0	(7.0)	40.8	(7.3)
Weight age 20 years (kg):	79.5	(18.7)	79.3	(18.1)	79.7	(19.4)
Age first obese (years):	26.1	(11.3)	26.1	(11.2)	26.1	(11.5)
Highest weight ever (kg):	118.0	(23.9)	120.0	(25.3)	115.9	(22.4)
Female:	91	(84%)	45	(83%)	46	(85%)
Ethnicity:						
Pākehā (NZ European)	74	(69%)	38	(70%)	36	(67%)
Māori/Pacific	13	(12%)	6	(11%)	7	(13%)
Other	21	(19%)	10	(19%)	11	(20%)
Marital Status:						
Single	28	(26%)	14	(26%)	14	(26%)
Married/defacto	71	(66%)	33	(61%)	38	(70%)
Separated/divorced	9	(8%)	7	(13%)	2	(4%)
Highest level of education:						
Secondary	37	(34%)	16	(30%)	21	(39%)
Tertiary	71	(66%)	38	(70%)	33	(61%)
Main source of income: (n=103)						
Full-time employment	51	(48%)	27	(51%)	24	(44%)
Part-time employment	19	(18%)	8	(15%)	11	(20%)
Government benefit	17	(16%)	6	(11%)	11	(20%)
Supported by partner	14	(13%)	6	(11%)	8	(15%)
Other	2	(2%)	2	(4%)	0	(0%)
Smoking status: (n=107)						
Never smoked daily	64	(60%)	32	(59%)	32	(60%)
Non-smoker	34	(32%)	15	(28%)	19	(36%)
Current smoker	9	(8%)	7	(13%)	2	(4%)
Drinking status: (n=107)						
Never	15	(14%)	7	(13%)	8	(15%)
Ex-drinker	4	(4%)	2	(4%)	2	(4%)
Less than once a week	51	(48%)	23	(43%)	28	(52%)
1–2x/week	24	(22%)	11	(21%)	13	(24%)
3–4x/week	5	(5%)	3	(6%)	2	(4%)
5+x/week	8	(7%)	7	(13%)	1	(2%)

## Power analysis

The primary outcome was a 5% reduction in weight at 12 months from baseline. It was estimated 25% of experimental participants (KA/GRx arm) would achieve this goal at 12 months based on preliminary Kia Ākina data, compared with 5% of controls (GRx) as anticipated from exercise programmes.<sup>37</sup> Using a two-tailed alpha of 0.05, there was 80% power to detect this difference between experimental (KA/GRx) and control (GRx) participants, with a sample size of 49 participants in each group.

## Results

Table 1 outlines baseline demographic, substance use and weight characteristics of the sample.

The mean number of formal previous attempts at weight loss was 2.2 (range=0–8). The four most common methods were, in order, Weight Watchers, prescribed diet pills, Jenny Craig and the Atkins Diet.

Over the course of the study 18 participants formally withdrew, nine from each treatment group, and a further 13 partic-

**Table 2:** Distribution of participants according to the two treatment groups (KA/GRx vs GRx) across six weight loss categories at 12 months follow-up, for both those followed up (n=84) and the full imputed sample (n=108).

OUTCOME	Full sample (imputed) (n=108)				Completed follow-up (n=84)			
	KA/GRx		GRx		KA/GRx		GRx	
10+% weight loss	5	(9%)	2	(4%)	5	(12%)	2	(5%)
5.0–9.9% weight loss	6	(11%)	7	(13%)	6	(15%)	7	(16%)
0.0–4.9% weight loss	32	(59%)	24	(44%)	19	(46%)	13	(30%)
0.1–4.9% weight gain	8	(15%)	18	(33%)	8	(20%)	18	(42%)
5.0–9.9% weight gain	3	(6%)	3	(6%)	3	(7%)	3	(7%)
10+% weight gain	0	(0%)	0	(0%)	0	(0%)	0	(0%)
	54		54		41		43	

Participants were not available for follow-up at 12 months, seven from KA and six from GRx. This left 77 participants from whom complete data at 12 months were obtained (71% follow-up rate). There was no significant difference between the two treatment groups (Chi square=0.21, df=1, p=0.64) in terms of the overall percentage who withdrew or were lost to follow-up.

At 12-month follow-up, home weight measurements using digital scales were obtained from an additional seven participants from whom secondary follow-up measures were not completed (three from KA/GRx, four from GRx). This resulted in 84

participants followed up for weight (75% follow-up rate).

### Weight loss

Weight change at 12 months varied considerably across the total sample from 10.5kg gained to 21.0kg lost.

Weight loss data were categorised into six clinically meaningful groups,<sup>10</sup> as seen in Table 2. 11/54 (20%) of KA/GRx had a weight loss of 5% or greater compared with 9/54 (17%) in GRx (p=0.62, OR=0.78 (0.30, 2.07)). 21/54 (39%) of the GRx group gained weight compared with 11/54 (21%) of the KA/GRx group (p=0.04, OR=2.49 (1.05, 5.88)).

**Table 3:** Comparison of weight outcome measures between participants in the KA/GRx vs GRx groups for both those followed up (n=84) and the full sample (imputed) (n=108).

	Total	(sd)	KA/GRx	(sd)	GRx	(sd)	t	p	D
Baseline weight (n=84)	112.5	(22.3)	112.3	(21.5)	112.7	(23.1)			
12-month weight	110.4	(22.5)	108.6	(20.9)	112.0	(24.0)			
Weight loss (kg)	2.1	(6.2)	3.6	(6.5)	0.7	(5.6)	2.15	0.03	0.48
% Weight loss	1.9	(5.3)	3.1	(5.5)	0.7	(4.9)	2.15	0.03	0.46
% Excess weight loss	8.7	(28.5)	14.0	(33.1)	3.7	(22.6)	1.68	0.09	0.36
Baseline weight (n=108)	111.2	(21.4)	111.6	(21.2)	110.8	(21.9)			
12-month weight	109.0	(21.5)	108.0	(20.6)	110.0	(22.6)			
Weight loss (kg)	2.2	(5.5)	3.6	(5.7)	0.7	(5.0)	2.79	0.006	0.54
% Weight loss	1.9	(4.7)	3.2	(4.8)	0.7	(4.4)	2.84	0.005	0.54
% Excess weight loss	9.3	(25.6)	14.6	(29.3)	4.1	(20.2)	2.17	0.03	0.42

**Table 4:** Comparison of secondary outcome measures between KA/GRx vs GRx participants at 12-month follow-up (n=68-81).

	Total	(sd)	KA/GRx	(sd)	GRx	(sd)	t	p	d
<b>PHYSICAL FITNESS (n=68)</b>									
Baseline steps	80.1	(20.0)	80.5	(18.7)	79.8	(21.2)			
12-month steps	94.6	(24.3)	94.2	(27.3)	94.8	(21.9)			
Change steps	14.4	(17.1)	13.7	(18.3)	15.0	(16.3)	0.31	0.76	0.08
<b>Baseline HRR1</b>									
12-month HRR1	26.8	(12.2)	26.5	(13.7)	27.1	(11.1)			
Change HRR1	31.7	(14.4)	31.2	(14.9)	32.1	(14.3)	0.09	0.93	0.02
<b>PSYCHOLOGICAL DISTRESS (n=81)</b>									
Baseline K10 score	21.9	(7.3)	20.8	(7.3)	22.8	(7.2)			
12m K10 score	18.5	(6.4)	16.8	(4.4)	20.0	(7.5)			
Change K10 score	3.3	(6.2)	4.0	(6.5)	2.7	(4.0)	0.92	0.36	0.21
<b>READINESS TO CHANGE (n=81)</b>									
Baseline importance	10.1	(1.6)	9.9	(1.7)	10.2	(1.4)			
12m importance	9.7	(1.8)	9.5	(1.8)	9.8	(1.8)			
Change importance	0.4	(1.5)	0.4	(1.5)	0.4	(1.5)	0.01	0.99	0.00
<b>Baseline confidence</b>									
12m confidence	7.0	(2.5)	7.0	(2.6)	6.9	(2.6)			
Change confidence	7.0	(2.6)	7.8	(2.8)	6.3	(2.6)	2.41	0.02	0.56
<b>QUALITY OF LIFE (n=81)</b>									
Baseline physical health	51.2	(9.9)	51.3	(10.8)	51.1	(9.1)			
12m physical health	66.4	(17.3)	66.1	(18.6)	66.6	(16.3)			
Change physical health	15.2	(15.8)	14.8	(16.5)	15.5	(15.4)	0.19	0.85	0.04
<b>Baseline psychological</b>									
12m psychological	49.9	(12.9)	49.3	(12.1)	50.3	(13.6)			
Change psychological	58.3	(19.1)	61.0	(17.7)	56.0	(20.2)	1.86	0.07	0.41
<b>Baseline relationships</b>									
12m relationships	57.7	(20.7)	60.3	(22.8)	55.4	(18.5)			
Change relationships	62.0	(19.7)	65.1	(17.5)	59.3	(21.3)	0.26	0.80	0.05
<b>Baseline environment</b>									
12m environment	65.6	(14.6)	66.5	(16.2)	64.8	(13.2)			
Change environment	68.6	(14.0)	72.9	(13.4)	64.8	(13.5)	2.44	0.02	0.55
<b>Baseline percep QOL</b>									
12m percep QOL	66.6	(25.8)	60.8	(29.8)	71.5	(20.8)			
Change percep QOL	71.9	(20.8)	72.3	(21.1)	71.5	(20.8)	2.28	0.03	0.51
<b>Baseline percep Health</b>									
12m percep Health	30.2	(22.3)	30.3	(25.4)	30.2	(19.3)			
Change percep Health	43.8	(24.9)	47.4	(23.8)	40.7	(25.6)	1.08	0.28	0.24
<b>OVERALL SATISFACTION OF ASSISTANCE RECEIVED (n=79)</b>									
	2.4	(1.1)	1.8	(1.0)	2.9	(1.0)	4.96	<0.001	1.10

KA/GRx participants lost more weight than GRx participants (3.6kg vs 0.7kg,  $p=0.006$ ,  $n=108$ ), which translated into significantly greater % weight loss and %EWL (Table 3).

### Secondary measures

There were no significant differences between KA/GRx and GRx participants in the change to 12 months for the two physical fitness measures or psychological distress measure, although these improved from baseline across the whole sample (Table 4).

There was no difference between KA/GRx and GRx participants in terms of the change to 12 months in perceived importance of recovery, but there was a significant difference between the two groups in the change in confidence for recovery. KA/GRx participants increased in confidence over the 12 months (7.0 to 7.8), whereas GRx participants' confidence decreased (6.9 to 6.3) ( $p=0.02$ ,  $d=0.56$ ).

The WHOQOL-BREF measures quality of life (QOL) on six domains: Physical Health, Psychological Health, Relationships, Environment, Overall Perception of QOL and Overall Perception of Health, each scored as a percentage. There were significant quality of life improvements for KA/GRx participants compared with GRx for Environment ( $p=0.02$ ,  $d=0.55$ ) and Overall Perception of QOL ( $p=0.03$ ,  $d=0.51$ ), and some indication of a differential advantage to KA/GRx in terms of Psychological Health ( $p=0.07$ ,  $d=0.41$ ). There were significant quality of life improvements for the whole sample for each of the six domains, with medium to very large effect sizes detected for Physical Health ( $p<0.001$ ,  $d=1.08$ ), Psychological Health ( $p<0.001$ ,  $d=0.52$ ) and Perception of Health ( $p<0.001$ ,  $d=0.58$ ).

Participants ( $n=79$ ) reported high overall satisfaction with the assistance received over the 12 months. The total mean score on the 5-point Likert scale was 2.4. KA/GRx participants scored significantly more satisfaction than GRx participants (1.8 vs 2.9,  $p<0.001$ ,  $d=1.10$ ). Eighty-nine percent of KA/GRx participants said they were either satisfied or very satisfied compared with 28% of GRx participants ( $p<0.001$ ,  $OR=20.7$  (6.0, 71.0)).

### Threats to validity

There were no differences between KA/GRx and GRx participants in terms of the

measures of quality of therapeutic relationships with GP and practice nurse at six months and 12 months. However, at six months, GRx participants reported their GP talking with them more about the types of food to eat ( $p=0.047$ ,  $d=0.47$ ) and how much to eat ( $p=0.063$ ,  $d=0.43$ ). Following this finding, GP and practice nurse colleagues at the four primary care venues were reminded about the importance of clinical equipoise with study participants. The 12-month check revealed no significant differences in these two questions ( $p=0.67$ ,  $d=0.09$  and  $p=0.28$ ,  $d=0.24$  respectively) or any others.

## Discussion

This randomised controlled trial aimed to investigate the impact of adding Kia Ākina, a novel addiction-orientated weight-loss programme providing ongoing psychosocial support to the Green Prescription. The overall 25% drop-out rate at 12-months compared well with other obesity studies.<sup>38,39</sup>

KA/GRx participants lost more weight overall (3.6kg) than GRx (0.7kg). However, the two groups were not differentiated according to the primary *a priori* outcome measure, 5% loss of weight from baseline. The GRx group, with its widened scope involving eating advice as well as exercise coaching, was over three times more effective than anticipated (17% cf 5% estimated), whereas the KA/GRx group was a little less effective than was predicted (20% cf 25% estimated) in terms of 5% weight-loss from baseline.

The 12-month weight loss of 3.6kg in KA/GRx participants compares well with the 3.0kg weight loss found in an independent study of Weight Watchers, the most prominent commercial weight-loss programme in New Zealand.<sup>40</sup>

One in five of the KA/GRx group and one in six of the GRx group achieved a 5% weight loss in this 12-month study. This contrasts favourably with the estimated natural history in the US population of one in eight and one in seven morbidly obese men and women who achieve this annually.<sup>41</sup>

The odds of *gaining* weight in the KA/GRx group were two and a half times less than in the GRx group, suggesting the addition of Kia Ākina may be most helpful at reducing

an ongoing progression of obesity at the harder-to-treat end of the obesity spectrum.

The further advantages for KA/GRx in increasing confidence about recovery and enhancing aspects of quality-of-life suggest the additional content of Kia Ākina has some impact on improving participants' life functioning and providing hope for future recovery from obesity. These are elements consistent with a contemporary addiction treatment orientation; ie concern with broader life functioning than simply whether there has been a cessation of addictive behaviour.<sup>42</sup>

Five of the six strategies underpinning Kia Ākina—permanent lifestyle change, safe non-stigmatising venue, motivational enhancement, long-term harm reduction and recovery group process—are standard addiction treatment strategies, but not unique to addiction treatment. The one unique strategy, abstinence rules for NEEDNT food,<sup>30</sup> is different to the traditional moderation approach in obesity treatment,<sup>43,44</sup> although is unlikely to be a single factor underlying Kia Ākina's apparent effectiveness.

Strengths of this study were its real-life setting, procedures involving routine primary care consultations, ensuring the main outcome measures (weight and fitness) were obtained during routine care and the use of a comprehensive outcome measurement package.<sup>21</sup>

An important limitation was the relatively short follow-up period of 12-months, which precludes any definitive conclusion about Kia Ākina being an effective weight-loss addition to the Green Prescription in obesity. Kia Ākina is continuing to run as a recovery network, emphasising the importance of ongoing recovery work while enjoying life now, and providing the necessary support for maintenance of weight-loss,<sup>45</sup> making positive longer-term outcomes a possibility. A further limitation was the relative lack of men in the study brought about in part by significantly more men declining to participate following being screened in. While

there is little evidence that effective weight loss strategies are different between men and women,<sup>46</sup> there is a growing literature on specific needs for men in weight-loss programmes.<sup>47</sup> Further research is needed to determine whether Kia Ākina is inherently less appealing to men than women.

The positive results found for KA/GRx participants indicate that Kia Ākina has promise as an addition to the Green Prescription, especially when more psychosocial support is required, although weight-loss outcomes at 12-months were modest. Nevertheless, the high satisfaction rating by KA/GRx participants compared with GRx participants indicates the Kia Ākina programme is providing participants with something important and of value. However, before expanding the programme there are three challenges:

1. To better understand what it is that is valued by participants. For instance, is it the existence of a safe place among peers to feel accepted and supported, or is it the provision of hope that longer-term recovery is possible?
2. To undertake longer follow-up of participants in order to investigate whether positive outcomes are obtained over time; and
3. To explore enhancement of Kia Ākina in order to improve the weight-loss outcomes while maintaining participants' high satisfaction with the programme. These developments could include clinical assessment, tailoring approaches to the type of obesity and providing individualised dietary assistance. These additions would move Kia Ākina away from being a low-cost recovery network towards functioning more as a specialist obesity clinic. However, these two orientations could feasibly be combined in a service utilising the best dietary approaches available while continuing a peer-based recovery network.

**Competing interests:**

Dr Sellman reports non-financial support from Radiant Health Ltd outside the submitted work; Dr Elmslie reports grants from HRC, grants from T-Gear Trust, Nelson, New Zealand, during the conduct of the study.

**Acknowledgements:**

We acknowledge our primary care partners for this study: Dr Margaret Metherell, Dr Clive Hunter, Dr Brett Mann and Ms Helen Johnson. We feel indebted to 26 original participants, who we began interacting with five years ago, and the 108 participants in this current study, from whom we have learnt much. Finally, we acknowledge the funding from the Health Research Council of New Zealand for the study (Grant Number: 13/541), and additional funding support from the T-GEAR Charitable Trust to assist with further development of Kia Ākina.

**Author information:**

Doug Sellman, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch; Ria Schroder, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch; Daryle Deering, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch; Jane Elmslie, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch; James Foulds, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch; Chris Frampton, National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch.

**Corresponding author:**

Prof Doug Sellman, C/- National Addiction Centre, Department of Psychological Medicine, University of Otago, Christchurch, PO Box 4345.  
doug.sellman@otago.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7158>

**REFERENCES:**

- World Health Organization. Obesity and overweight. Fact Sheet #311, updated January 2015. <http://www.who.int/mediacentre/factsheets/fs311/en/> (accessed 12/3/2015).
- Ministry of Health. New Zealand Health Survey. Annual update of key findings 2012/2013. Wellington: Ministry of Health, 2013.
- James WPT. WHO recognition of the global obesity epidemic. *Int J Obes (Lond)*. 2008 Dec; 32 Suppl 7:S120-126. doi: 10.1038/ijo.2008.247.
- Ministry of Health, Clinical Trials Research Unit. Clinical guidelines for weight management in New Zealand adults. Wellington: Ministry of Health, 2009.
- National Health and Medical Research Council (NHMRC). Clinical practice guidelines for the management of overweight and obesity in adults, adolescents and children in Australia. Melbourne: National Health and Medical Research Council, 2013.
- National Institute for Health and Care Excellence (NICE). Managing Overweight and Obesity in Adults: Lifestyle Weight Management Services, 2014. <http://www.nice.org.uk/guidance/ph53> (accessed 25/9/2015).
- Fabricatore AN, Wadden TA. Obesity. *Annu Rev Clin Psychol*. 2006; 2:357-77.
- Laddu D, Dow C, Hingle M, et al. A review of evidence-based strategies to treat obesity in adults. *Nutr Clin Pract*. 2011 Oct; 26(5):512-25. doi: 10.1177/0884533611418335.
- Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. 2002 7; 346(6):393-403.
- Douketis JD, Macie C, Thabane L, Williamson DF. Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. *Int J Obes (Lond)*. 2005; 29(10):1153-67.
- Huang T, Tobias D, Hruby A, Rifai N, Tworoger S, Hu F. An Increase in Dietary Quality Is Associated with Favorable Plasma Biomarkers of the Brain-Adipose Axis in Apparently Healthy US Women. *Journal of Nutrition* 2016; 146:1101-8.
- Tsai AG, Wadden TA. Treatment of obesity in primary

- care practice in the United States: A systematic review. *Gen Intern Med.* 2009; 24(9): 1073–1079. doi: 10.1007/s11606-009-1042-5. Epub 2009 Jun 27.
13. Hartmann-Boyce J, Johns DJ, Jebb SA, et al. Behavioural weight management programmes for adults assessed by trials conducted in everyday contexts: systematic review and meta-analysis. *Obes Rev.* 2014; 15(11): 920-932. doi: 10.1111/obr.12220. Epub 2014 Aug 11.
  14. Forman-Hoffman V, Little A, Wahls T. Barriers to obesity management: a pilot study of primary care clinicians. *BMC Fam Pract.* 2006; Jun 6; 7:35.
  15. Elley R, Kerse N, Arroll B, et al. Cost-effectiveness of physical activity counselling in general practice. *N Z Med J.* 2004; 117(1207) [http://www.nzma.org.nz/\\_data/assets/pdf\\_file/0011/17948/Vol-117-No-1207-17-December-2004.pdf](http://www.nzma.org.nz/_data/assets/pdf_file/0011/17948/Vol-117-No-1207-17-December-2004.pdf)
  16. Ministry of Health. Green Prescription, 2014, <http://www.health.govt.nz/our-work/preventative-health-wellness/physical-activity/green-prescriptions> (accessed 6/9/2015).
  17. Shaw KA, Gennat HC, O'Rourke P, Del Mar C. Exercise for overweight or obesity. *Cochrane Database of Syst Rev.* 2006 Oct 18; (4): CD003817 DOI:10.1002/14651858.CD003817.pub3.
  18. Brownell KD, Gold MS (eds). *Food and Addiction: A Comprehensive Handbook.* Oxford University Press, New York, 2012.
  19. Barry D, Clarke M, Petry N. Obesity and its relationship to addictions: Is overeating a form of addictive behavior? *Am J Addict.* 2009; 18(6):439–451. doi: 10.3109/10550490903205579.
  20. Schroder RN, Stevenson R, Sellman JD, et al. Abstinence vs. moderation in obesity treatment: client perceptions of what works. A commissioned paper for the National Heart Foundation of New Zealand, 2011.
  21. Avenell A. Systematic review of the long-term effects and economic consequences of treatment for obesity and implications for health improvement. *Health Technol Assess.* 2004; 8(21):1–182.
  22. Cutler L, King B, McCarthy N, et al. Appetite for life: an evaluation of a primary care lifestyle programme. *J Prim Health Care.* 2010; 2(4):281–287.
  23. Vaillant GE. What can long-term follow-up teach us about relapse and prevention of relapse in addiction? *British J Addict.* 1988; 83(10):1147–1157.
  24. Thomas SL, Hyde J, Karunaratne A, et al. Being fat in today's world: A qualitative study of the lived experiences of people with obesity in Australia. *Health Expect.* 2008; 11(4): 321-330. doi: 10.1111/j.1369-7625.2008.00490.x. Epub 2008 Jul 28.
  25. Rollnick S, Allison J. Motivational interviewing. Chapter 7 in N Heather & T Stockwell (eds), *The Handbook of Treatment and Prevention of Alcohol Problems*, John Wiley & Sons Ltd: England, 2004.
  26. Koob GF, Volkow ND. Neurocircuitry of addiction. *Neuropsychopharmacology.* 2010; 35(1): 217-238. doi: 10.1038/npp.2009.110.
  27. Cheung YW. Substance abuse and developments in harm reduction. *CMAJ.* 2000; 162(12):1697–1700.
  28. Barr VJ, Robinson S, Marinlink B, et al. The expanded chronic care model: An integration of concepts and strategies from population health promotion and the chronic care model. *Hosp Q.* 2003; 7(1):73–82.
  29. Sellman JD. *Real weight loss: A practical guide to changing your lifestyle and achieving long-term weight loss.* Nelson, New Zealand: Craig Potton Publishing, 2008.
  30. Elmslie JL, Sellman JD, Schroder RN, Carter FA. The NEEDNT Food List: non essential, energy-dense, nutritionally-deficient foods. *N Z Med J.* 2012; 125(1350):84–92.
  31. Rikli R, Jones J. Development and validation of a functional fitness test for community-residing older adults. *J Aging Phys Act.* 1999; 7: 129–161.
  32. Prado DM, Silva AG, Trombetta IC, et al. Exercise training associated with diet improves heart rate recovery and cardiac autonomic nervous system activity in obese children. *Int J Sports Med.* 2010; 31(12): 860–865. doi: 10.1055/s-0030-1267158. Epub 2010 Nov 11.
  33. Guazzi M, Myers J, Peberdy MA, et al. Cardiopulmonary exercise testing variables reflect the degree of diastolic dysfunction in patients with heart failure – normal ejection fraction. *J Cardio-pulm Rehabil Prev.* 2010; 30(3): 165–172. doi: 10.1097/HCR.0b013e3181d0c1ad.
  34. Kessler RC, Barker PR, Colpe LJ, et al. Screening for serious mental illness in the general population. *Arch Gen Psychiatry.* 2003; 60(2):184–9.
  35. WHOQOL Group. Development of the World Health Organization WHOQOL-BREF Quality of

- Life Assessment. *Psychol Med.* 1998; 28(3):551–558.
36. Meakin R, Weinman J. The Medical Interview Satisfaction Scale (MISS-21) adapted for British general practice. *Fam Pract.* 2002; 19(3):257–263.
  37. Johns DJ, Hartmann-Boyce J, Jebb SA, Aveyard P. Diet or exercise interventions vs combined behavioural weight management programs: a systematic review and meta-analysis of direct comparisons. *J Acad Nutr Diet.* 2014; 114(10):1557–1568. doi: 10.1016/j.jand.2014.07.005.
  38. Mutsaerts MAQ, Kuchenbecker WKH, Mol BW, et al. Dropout is a problem in lifestyle intervention programs for overweight and obese infertile women: a systematic review. *Hum Reprod.* 2013; 28(4):979–986. doi: 10.1093/humrep/det026. Epub 2013 Feb 20.
  39. Hadziabdic MO, Mucalo I, Hrabac P, et al. Factors predictive of drop-out and weight loss in weight management of obese patients. *J Hum Nutr Diet.* 2015; 28 Suppl 2: 24–32. doi: 10.1111/jhn.12270. Epub 2014 Sep 13.
  40. Jebb SA, Ahern AL, Olson AD, et al. Primary care referral to a commercial provider for weight loss treatment versus standard care: A randomised controlled trial. *Lancet.* 2011; 378(9801):1485–92.
  41. Fildes A, Charlton J, Rudisill C, et al. Probability of an obese person attaining normal body weight. Cohort study using electronic health records. *Am J Public Health.* 2015; 105(9): e54–9. doi: 10.2105/AJPH.2015.302773. Epub 2015 Jul 16.
  42. Miller PG, Miller WR. What should we be aiming for in the treatment of addiction. *Addiction.* 2009; 104(5): 685–686. doi: 10.1111/j.1360-0443.2008.02514.x.
  43. McMillan-Price J, Brand-Miller J. Dietary approaches to overweight and obesity. *Clin Dermatol.* 2004; 22(4):310–314.
  44. Lindstrom J, Peltonen M, Tuomilehto J. Lifestyle strategies for weight control: experience from the Finnish Diabetes Prevention Study. *Proc Nutr Soc.* 2005; 64(1):81–8.
  45. Soleymani T, Daniel S, Garvey WT. Weight maintenance: challenges, tools and strategies for primary care physicians. *Obes Rev.* 2016; 17(1):81–93. doi: 10.1111/obr.12322. Epub 2015 Oct 21.
  46. Williams RL, Wood LG, Collins CE, Callister R. Effectiveness of weight loss programmes – is there a difference between men and women: A systematic review. *Obes Rev.* 2015; 16:171–186.
  47. Sabinsky MS, Toft U, Raben A, Holm L. Overweight men's motivations and perceived barriers towards weight loss. *Eur J Clin Nutr.* 2007; 61:526–531.

# Ethnic and geographic variations in the incidence of pancreatitis and post-pancreatitis diabetes mellitus in New Zealand: a nationwide population-based study

Sayali A Pendharkar, Juby Mathew, Jinfeng Zhao, John A Windsor, Daniel J Exeter, Maxim S Petrov

## ABSTRACT

**AIM:** To determine the incidence of acute pancreatitis (AP), chronic pancreatitis (CP), and post-pancreatitis diabetes mellitus (DP) in New Zealand, and the effect of ethnic and geographic variations.

**METHODS:** Data were collected from all district health boards in New Zealand by the Ministry of Health (Manatū Hauora). Diagnosis of AP, CP and DP was determined by the International Classification of Diseases-10 codes. Incidence rates per 100,000 population per year were calculated using incident AP, CP and DP cases as the numerator, and the adult resident population of New Zealand as the denominator. Poisson distribution was used to estimate 95% confidence intervals. The district health board domicile codes and corresponding incidence rates were used to map geographical variations for AP, CP and DP.

**RESULTS:** On average, 2,072 new cases of AP, CP and DP were diagnosed in New Zealand every year. The crude incidence of AP was 58.42 [57.55, 59.30], CP - 3.97 [3.74, 4.20], and DP - 7.95 [7.62, 8.27] per 100,000 population per year. Māori had the highest incidence of AP (95.21 [91.74, 98.68] per 100,000 population per year), CP (6.27 [5.37, 7.16] per 100,000 population per year), and DP (18.23 [16.71, 19.76] per 100,000 population per year). Incidence of AP and DP was at least 1.8 and 2.6 times higher in Māori than New Zealand Europeans in every age group, and incidence of DP was at least 1.9 times higher in Pacific people than New Zealand Europeans in every age group. Auckland/Northland had the highest incidence of AP (135.25 [134.82, 135.68] per 100,000 population), and CP (9.03 [8.60, 9.46] per 100,000 population), while Lakes/Waikato had the highest incidence of DP (20.64 [20.21, 21.07] per 100,000 population) in New Zealand.

**CONCLUSIONS:** New Zealanders have a very high incidence rate of AP, with Māori having the highest reported incidence of AP worldwide. There is a significant geographic variation in incidence of pancreatic diseases, with the Upper North Island having the highest incidence rates of AP, CP and DP in the country. Future high-quality studies are required to understand the mechanisms of pancreatitis and DP in order to develop preventive and therapeutic strategies that would benefit New Zealanders in general and Māori in particular.

Inflammatory diseases of the pancreas are common worldwide and pose a substantial burden on healthcare systems.<sup>1,2</sup> The global incidence of acute pancreatitis (AP) is 33.7 per 100,000 population per year, whereas the global incidence of chronic pancreatitis (CP) is 9.6 per 100,000 population per year.<sup>3</sup> In the USA alone, pancreatitis results in more than 800,000 visits to

hospitals and costs more than \$2.6 billion.<sup>4</sup> Further, while burden of type 1 and type 2 diabetes mellitus has long been acknowledged worldwide and in New Zealand,<sup>5,6</sup> post-pancreatitis diabetes mellitus (DP) has only gained attention recently.<sup>7-13</sup> In particular, a nationwide population-based study from Taiwan showed that patients after AP have a 2.5 times higher risk of developing

newly diagnosed diabetes mellitus than individuals in the general population.<sup>14</sup> The risk of diabetes appears to be independent of the degree of mechanical destruction of the pancreas,<sup>15,16</sup> suggesting that patients with pancreatitis are at increased risk of developing DP regardless of pancreatitis severity.

Although research on pancreatitis has been conducted in New Zealand and Australia for years, it was limited to single-centre studies only.<sup>17–20</sup> These studies typically had a small sample size, and were prone to selection and participation bias, including underrepresentation of minorities and underserved populations, particularly the Māori. Further, the recent comprehensive systematic review and meta-analysis on the global burden of diseases of the pancreas showed that large population-based data on incidence of acute and chronic pancreatitis are available for every World Health Organization (WHO) region except for Australasia and Africa.<sup>3</sup> Given that Māori and Pacific people are at an increased risk of developing chronic metabolic diseases,<sup>21–23</sup> population-based studies, particularly nationwide studies, are essential for determining the true incidence of diseases, effect of covariates and pattern of healthcare utilisation, as they have large sample size and lack of selection and participation bias.

The aim of this population-based study was to determine the incidence of AP, CP and DP in New Zealand using national-level health dataset, as well as investigate the effect of ethnicity and geographical location.

## Methods

### Data source

Data for this study were obtained from and prepared by New Zealand's Ministry of Health Analytical Services Database (National Health Board, Ministry of Health, New Zealand). The dataset included information on patients' age, sex, ethnicity, tenth revision International Classification of Diseases (ICD-10) codes and the hospital admitted to. All patients were anonymised by the Analytical Services for the purpose of this study. In line with the Ministry of Health guidelines, ethical review was not required for this study. No contact was made with the study population.

### Study population

All New Zealand residents have a unique alpha-numeric code, the National Health Index, assigned at the very first contact with the health-care system. The population for this study ( $n=2,597,217$ ) constituted registered patients admitted to a public hospital across New Zealand from 1<sup>st</sup> January 2006 to 31<sup>st</sup> October 2015 with the diagnosis of AP (ICD-10 K85.0, ICD-10 K85.1, ICD-10 K85.2, ICD-10 K85.3, ICD-10 K85.8, ICD-10 K85.9), CP (ICD-10 K86.0, ICD-10 K86.1) or DP (ICD-10 E-1364, ICD-10 E1365, and non-diabetic patients who had new diabetes mellitus ICD-10 codes after the date of AP or CP).<sup>24</sup> All patients who had a prior principal or secondary diagnosis of acute or chronic pancreatitis or diabetes back to 1 January 2005 were excluded. Further, patients who had an initial principal or secondary diagnosis of CP, followed by a later diagnosis of AP, were excluded from those designated as having AP. In case of multiple admissions for a patient, data from only the very first admission were used, with duplicate entries excluded.

In this study, people less than 20 years of age ( $n=1117$ ) were excluded from analyses, as non-adults according to the WHO guidelines.<sup>25</sup> Age for all remaining patients was standardised according to the WHO standardised age groups.<sup>25</sup>

Ethnic groups were categorised in line with Statistics New Zealand Census (2013)<sup>26</sup> and prioritised in the following order: New Zealand European, Māori, Asian, Pacific people and other. The "other" category comprised of the following ethnic groups: Middle Eastern, African, Latin American, North American Indian, Mauritian, South African and those who did not identify with any ethnic group.

New Zealand district health boards (DHBs) were aggregated into seven broad regions from North to South according to the geographic location. The regions contained sufficient number of cases of AP, CP and DP to meaningfully map the geographical variations in crude incidence of AP, CP and DP per 100,000 population, and by ethnicity. Maps for incidence rates were created at the DHB region level using the Jenks classification, which groups similar values in each class and maximises the differences between

classes.<sup>27</sup> All maps were created using the ArcGIS 10.3.1 for Desktop (ESRI 2015. ArcGIS Desktop: Version 10.3.1.4959 Redlands, CA: Environmental Systems Research Institute).

### Statistical analyses

Discrete variables were presented as counts. Percentage incidence and incidence rate per 100,000 population per year were determined using incident AP, CP, and DP cases as the numerator and the adult resident population of New Zealand at the start of the study period (2006), reported by Statistics New Zealand, as the denominator. These were calculated by ethnicity, geographical variation, age and sex. Poisson distribution was used to estimate 95% confidence intervals (CI). All analyses were conducted using SPSS for Windows Version 23 (SPSS Inc., Chicago, IL, USA).

## Results

There were a total of 20,198 incident cases of AP, CP and DP. The annual incidence of AP was 58.42 [57.55, 59.30] per 100,000 population per year, CP - 3.97 [3.74, 4.20] per 100,000 population per year, and DP - 7.95 [7.62, 8.27] per 100,000 population per year. On average 1,721 people were newly diagnosed with AP, 117 with CP and 234 people with DP every year. Median (interquartile range) duration between an incident event of pancreatitis and DP was 25 (6–44) months.

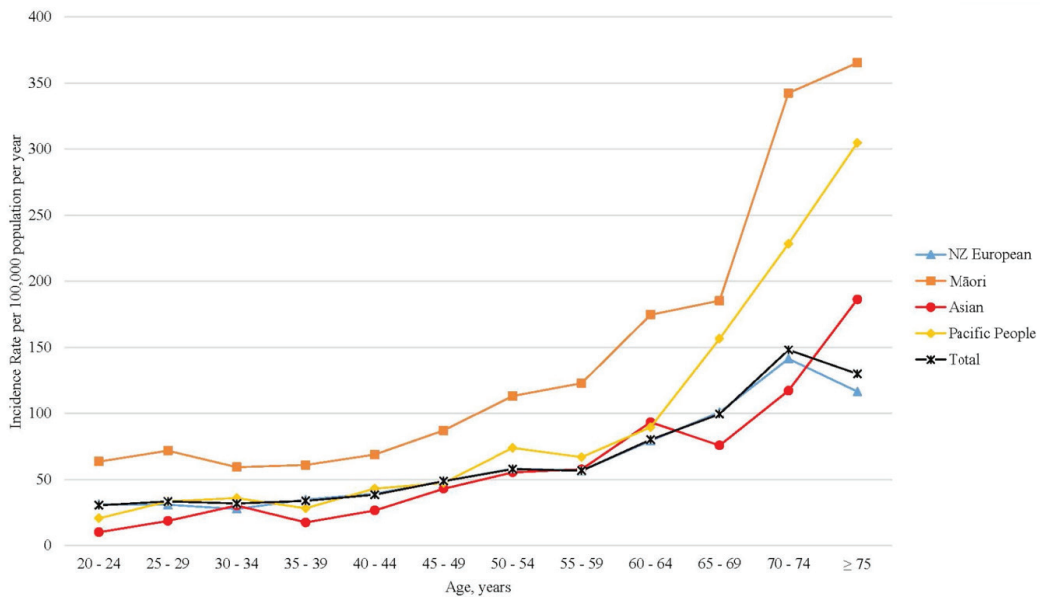
### Acute pancreatitis

The study cohort included 16,753 incident cases of AP, of whom 8,063 (47.65%) were men. The incidence rate of AP in the different ethnic groups is shown in Table 1.

**Table 1:** Incidence of acute pancreatitis, chronic pancreatitis and post-pancreatitis diabetes mellitus by ethnicity.

	Incident cases	Population	Crude incidence, %	Incidence rate per 100,000 population per year [95% CI]
<b>Acute pancreatitis</b>				
NZ European	10,063	1,699,938	0.59	60.22 [59.05, 61.39]
Māori	2,872	306,873	0.94	95.21 [91.74, 98.68]
Asian	855	245,523	0.35	35.43 [33.06, 37.80]
Pacific people	736	137,931	0.53	54.28 [50.37, 58.19]
Other	2,227	206,952	1.08	109.47 [104.95, 113.99]
<b>Chronic pancreatitis</b>				
NZ European	729	1,699,938	0.04	4.36 [4.05, 4.68]
Māori	189	306,873	0.06	6.27 [5.37, 7.16]
Asian	66	245,523	0.03	2.73 [2.07, 3.39]
Pacific people	26	137,931	0.02	1.92 [1.18, 2.65]
Other	133	206,952	0.06	6.54 [5.43, 7.65]
<b>Post-pancreatitis diabetes mellitus</b>				
NZ European	1,096	1,699,938	0.06	6.56 [6.17, 6.95]
Māori	550	306,873	0.18	18.23 [16.71, 19.76]
Asian	198	245,523	0.08	8.20 [7.06, 9.35]
Pacific people	231	137,931	0.17	17.04 [14.84, 19.23]
Other	227	206,952	0.11	11.16 [9.71, 12.61]

**Figure 1:** Distribution of population incidence of acute pancreatitis per 100,000 per year by age and ethnicity.



**Table 2:** Incidence ratios of acute pancreatitis, chronic pancreatitis and post-pancreatitis diabetes mellitus in Māori and Pacific People compared to New Zealand Europeans.

Demographic factors	Māori			Pacific People		
	AP	CP	DP	AP	CP	DP
<b>Age, years</b>						
20-24	2.04	1.61	N/E	0.66	0.22	N/E
25-29	2.33	1.13	13.84	1.08	0.25	4.40
30-34	2.15	1.26	6.93	1.30	0.50	5.48
35-39	1.75	1.01	6.22	0.81	0.39	3.19
40-44	1.76	1.74	2.74	1.10	0.47	4.37
45-49	1.79	1.54	2.61	0.97	0.55	1.90
50-54	1.95	1.69	2.63	1.27	0.43	6.34
55-59	2.17	2.46	3.23	1.18	0.30	5.87
60-64	2.21	2.22	8.19	1.13	1.07	5.57
65-69	1.84	1.60	3.91	1.55	1.03	4.28
70-74	2.42	2.39	5.93	1.62	0.76	3.60
≥75	3.14	3.66	6.70	2.62	1.28	6.86
<b>Sex</b>						
Men	1.83	1.65	3.36	1.02	0.74	2.74
Women	2.08	1.94	3.59	1.14	0.24	3.75

**Abbreviations:** AP, acute pancreatitis; CP, chronic pancreatitis; DP, post-pancreatitis diabetes mellitus; N/E, not estimable.

**Footnote:** The incidence ratios for AP, CP and DP were calculated by dividing the incidence rate of Māori and Pacific People by the incidence rate of Europeans for each pancreatic disease.

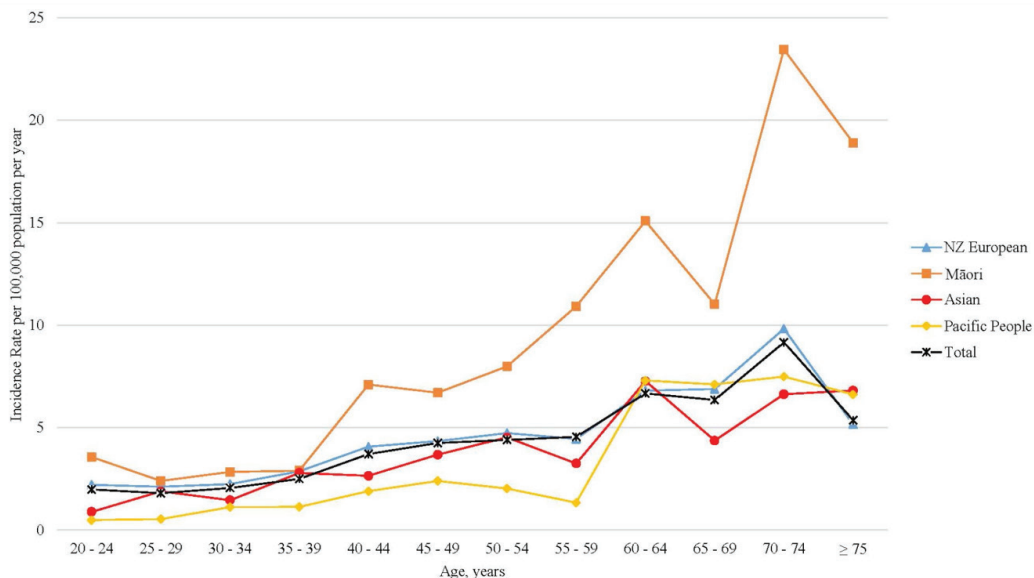
Māori had a significantly higher overall incidence of AP at 95.21 [91.74, 98.68] per 100,000 population per year, than both New Zealand European at 60.22 [59.05, 61.39] per 100,000 population per year, and New Zealanders altogether at 57.84 [56.97, 58.72] per 100,000 population per year ( $p < 0.05$ ) (Table 1). Although the overall incidence of AP was observed to increase with increasing age in all ethnic groups (Figure 1), Māori were more likely to develop AP than New Zealand Europeans in every age group, ranging from 1.75 times higher in the 35–39 group to 3.14 times higher in the  $\geq 75$  group (Table 2). Pacific people had overall incidence of AP at 54.28 [50.37, 58.19], which was not significantly different from both New Zealand Europeans and all New Zealanders. However, in those aged  $\geq 50$  years, Pacific people were consistently more likely to develop AP than New Zealand Europeans, ranging from 1.13 times in the 60–64 group to 2.62 in the  $\geq 75$  group (Table 2).

## Chronic pancreatitis

The study cohort included 1,143 incident cases of CP, of whom 671 (58.30%) were men. The incidence rate of CP in the different ethnic groups is shown in Table 1.

Māori had a significantly higher incidence rate of CP at 6.27 [5.37, 7.16] per 100,000 population per year, than both New Zealand Europeans and all New Zealanders at 4.36 [4.05, 4.68] per 100,000 population per year and 3.95 [3.72, 4.18] per 100,000 population per year, respectively ( $p < 0.05$ ). Although no significant overall change in incidence of CP with increasing age was observed in any of the ethnic groups (Figure 2), Māori were more likely to develop CP than New Zealand Europeans among people older than 40, ranging from 1.5 times higher in the 45–49 group to 3.66 times higher in the  $\geq 75$  group (Table 2). Pacific people had overall incidence of CP at 1.92 [1.18, 2.65], which was significantly lower than both New Zealand Europeans and all New Zealanders.

**Figure 2:** Distribution of population incidence of chronic pancreatitis per 100,000 per year by age and ethnicity.



### Post-pancreatitis diabetes mellitus

The study cohort included 2,302 incident cases of DP, of whom 1,462 (63.51%) were men. The incidence rate of DP in the different ethnic groups is shown in Table 1.

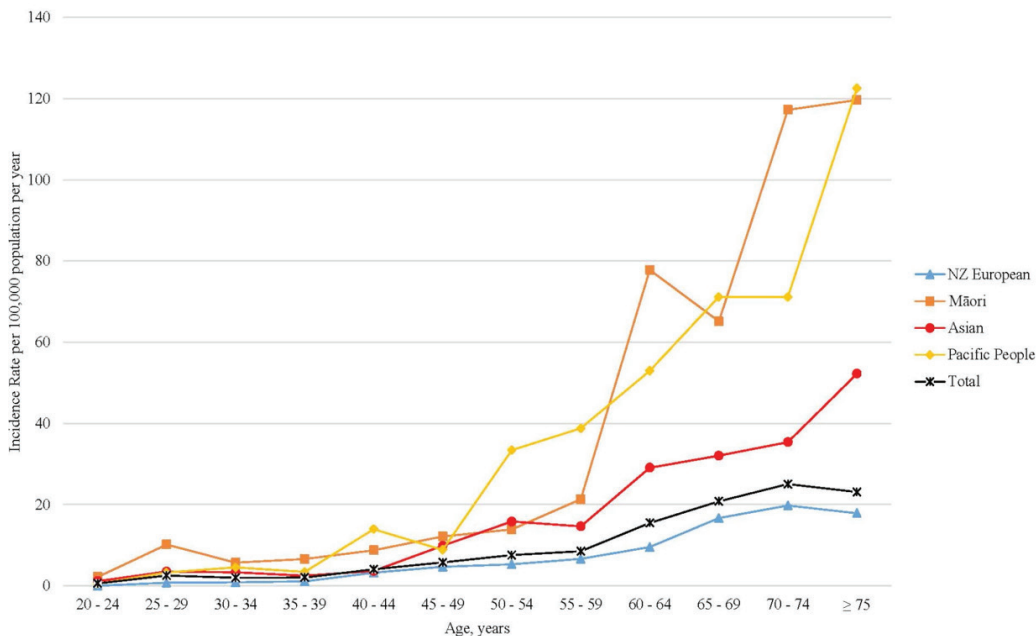
Both Māori and Pacific people had a significantly higher incidence of DP at 18.23 [16.71, 19.76] per 100,000 population per year, and 17.04 [14.84, 19.23] per 100,000 population per year, respectively, than New Zealand Europeans at 6.56 [6.17, 6.95] per 100,000 population per year ( $p < 0.05$ ). Increase in incidence of DP was significantly associated with increasing age in New Zealand Europeans, Māori and Pacific people (Figure 3). Māori were more likely to develop DP than New Zealand Europeans in every age group, ranging from 3.23 times higher in the 55–59 group to 13.84 times higher in the 25–29 group (Table 2). Pacific people were also more likely to develop DP than New Zealand Europeans in every age group, ranging from 1.90 times in the 45–49 group to 6.86 in the  $\geq 75$  group (Table 2).

### Geographical variation in acute pancreatitis, chronic pancreatitis and post-pancreatitis diabetes mellitus

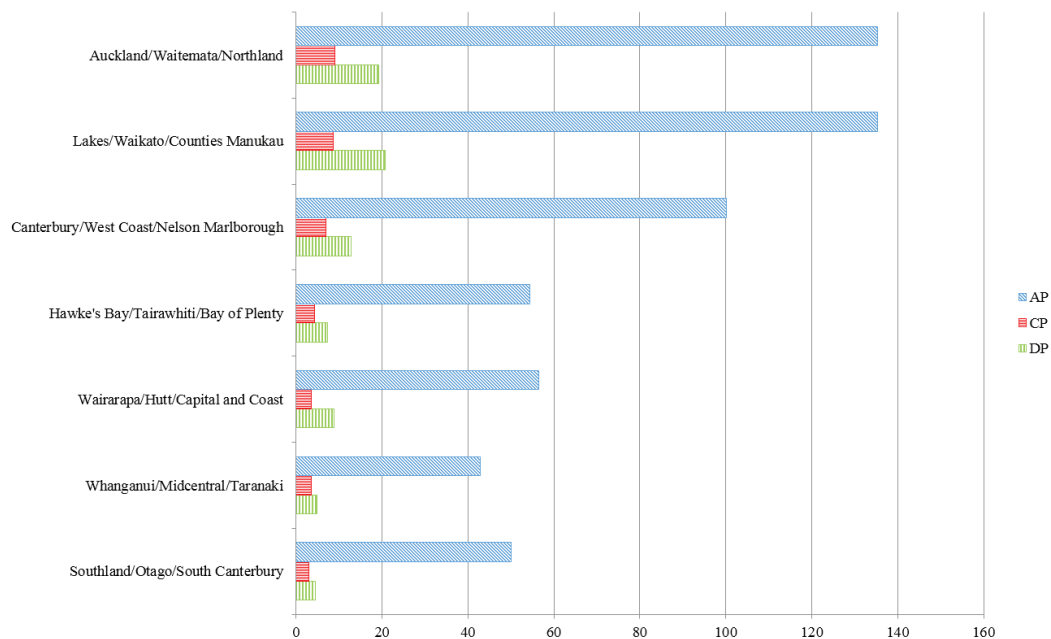
The Auckland/Waitemata/Northland region had the overall highest incidence of AP (135.25 [134.82, 135.68] per 100,000 population) and CP (9.03 [8.60, 9.46] per 100,000 population), while the Lakes/Waikato/Counties Manukau region had the overall highest incidence of DP (20.64 [20.21, 21.07] per 100,000 population) in New Zealand. By contrast, the Whanganui/Midcentral/Taranaki region had the overall lowest incidence of AP (42.80 [42.77, 42.88] per 100,000 population), while the Southland/Otago/South Canterbury region had the overall lowest incidence of CP (2.99 [2.91, 3.07] per 100,000 population) and DP (4.51 [4.44, 4.59] per 100,000 population) (Figures 4 and 5).

New Zealand Europeans in the Canterbury/West Coast/Nelson Marlborough region had the highest incidence of AP and CP (646.91 [646.06, 647.29], and 46.13 [45.30,

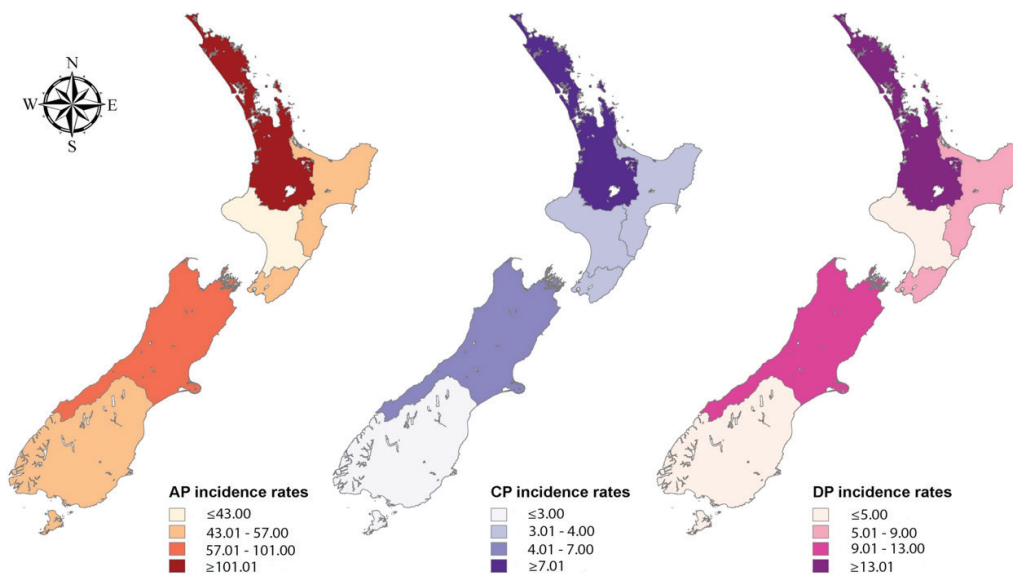
**Figure 3:** Distribution of population incidence of post-pancreatitis diabetes mellitus per 100,000 per year by age and ethnicity.



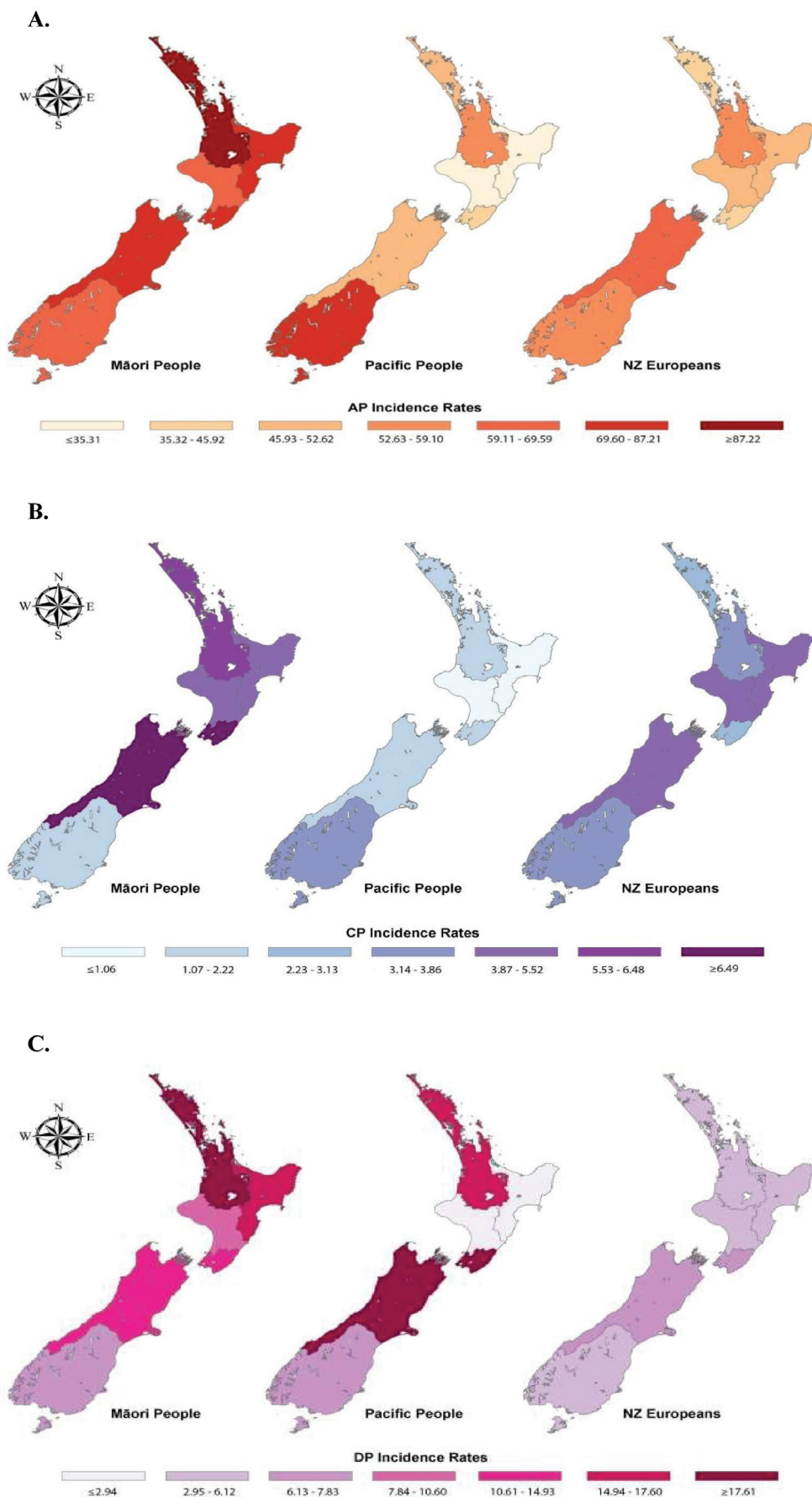
**Figure 4:** Geographic variation in incidence of pancreatitis and post-pancreatitis diabetes mellitus in New Zealand.



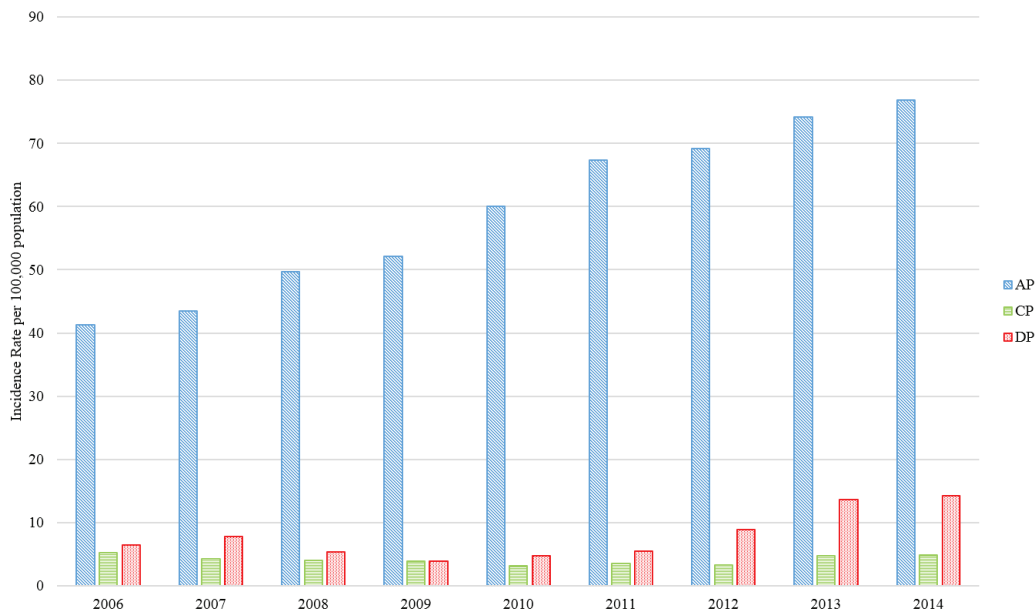
**Figure 5:** Incidence of acute pancreatitis, chronic pancreatitis, and post-pancreatitis diabetes mellitus, per 100,000 population by geographic regions.



**Figure 6:** Incidence of acute pancreatitis (A), chronic pancreatitis (B), and post-pancreatitis diabetes mellitus (C), per 100,000 population by geographic regions and ethnicity.



**Figure 7:** Distribution of population incidence of pancreatitis and post-pancreatitis diabetes mellitus per 100,000.



46.56] per 100,000 population, respectively) (Figures 6A and 6B), while those in the Wairarapa/Hutt/Capital and Coast region had the highest incidence of DP (68.40 [67.56, 68.82] per 100,000 population) (Figure 6C). Māori in the Lakes/Waikato/Counties Manukau region had the highest incidence of AP and DP (1102.29 [1101.92, 1102.36] and 234.03 [233.86, 234.20] per 100,000 population, respectively) (Figures 6A and 6C), while those in the Auckland/Waitemata/Northland region had the highest incidence of CP (64.83 [63.76, 65.90] per 100,000 population) (Figure 6B). Pacific people, from the Lakes/Waikato/Counties Manukau region, had the highest incidence of AP, CP and DP (591.04 [590.96, 591.30], 22.23 [21.24, 23.22], and 176.01 [175.65, 176.37] per 100,000 population, respectively) (Figure 6).

### Trends in incidence of acute pancreatitis, chronic pancreatitis and post-pancreatitis diabetes mellitus

Incidence of AP increased significantly ( $p < 0.05$ ) from 2006 (41.27 [41.04, 41.51] per 100,000 population per year) to 2014 (76.81 [76.49, 77.13] per 100,000 population per year). The incidence of CP did not change significantly from 2006 to 2014 (5.26 [5.18, 5.35] per 100,000 population per year to 4.92 [4.84, 5.00] per 100,000 population per year

in 2014). Post-pancreatitis diabetes mellitus significantly increased from 2006 (6.48 [6.39, 6.58] per 100,000 population per year) to 2014 (14.19 [14.05, 14.33] per 100,000 population per year) (Figure 7).

## Discussion

A recent comprehensive systematic review and meta-regression showed that the global crude incidence of AP is 33.74 [23.33, 48.81] per 100,000 per year and CP - 9.62 [7.86, 11.78] per 100,000 per year.<sup>3</sup> Findings from our study show, for the first time, that New Zealand sits at the higher end of the global spectrum with an incidence of 58.42 [57.55, 59.30] per 100,000 per year for AP, at par with North America, which has an incidence of 58.20 [56.90, 59.50] per 100,000 per year.<sup>3,28</sup> By contrast, the crude incidence of CP in New Zealand sits at the lower end of the global spectrum and is 3.97 [3.74, 4.20] per 100,000 population per year compared to the higher incidence rate in Europe of 10.82 [8.12, 14.41] per 100,000 per year. This study is also first in the literature to determine the incidence of DP in a population-based dataset. The crude incidence rate of DP in New Zealand is 7.95 [7.62, 8.27] per 100,000 population per year.

In New Zealand, the annual incidence of AP is 1.7 times higher in New Zealand than in the world in general and two times

higher than in Europe in particular.<sup>3</sup> This is indicative of the immense AP-related costs and associated burden likely posed on New Zealand's healthcare system. While data on cost of treating AP is not available in New Zealand, the direct costs of hospitalisation for AP in the USA exceeded \$2.6 billion in 2003.<sup>29</sup> In addition, older patients as well as patients in urban and teaching hospitals had higher costs per hospitalisation, based primarily on longer length of hospitalisation. Further, a steady increase in the incidence of and hospitalisations due to AP worldwide has been reported over the past 3–4 decades.<sup>29–34</sup> A similar trend was observed in New Zealand, with findings from our study showing that the incidence of AP has nearly doubled since 2006, and it is likely attributed to increased prevalence of metabolic disorders.<sup>9,35</sup> An interaction between defective glucose and lipid metabolism and abdominal adiposity is thought to increase the risk of developing AP. Evidence suggests that hyperglycaemia, hypertriglyceridaemia and abdominal or general adiposity significantly increase the risk of developing AP.<sup>9</sup> Nonetheless, evidence on impact of obesity on AP is variable and difficult to determine.

Diabetes continues to increase and is a significant national and global concern.<sup>6</sup> In the US, it is the seventh leading cause of mortality, and poses a considerable socioeconomic burden.<sup>6</sup> Accumulating evidence shows that diabetes after either AP or CP presents a unique pathophysiology, and is not an uncommon clinical entity.<sup>7,8,16</sup> Findings from our recent meta-analysis show that nearly 40% of patients after just one episode of AP develop new pre-diabetes or diabetes,<sup>10,16</sup> while nearly 80% of DP is attributed to CP.<sup>36</sup> However, diabetes in general is often undiagnosed with type 2 diabetes undiagnosed in 30–50% of New Zealand's population.<sup>5</sup> In light of the evidence that DP is often under- and mis-diagnosed as type 2 diabetes,<sup>8</sup> it is reasonable to suggest that at least 30–50% of DP is undiagnosed in New Zealand. Findings from our study show that DP is an increasingly significant problem in New Zealand, with incidence rate having more than doubled over the last decade, and it requires clinical attention, correct diagnosis, and appropriate preventive and therapeutic measures.

Incidence of AP, CP and DP differs in various ethnic groups. Findings from our study show that Māori are not only 1.6 times, 1.5 times and 2.8 times more likely to develop AP, CP and DP, respectively, than New Zealand Europeans, but also have the highest reported incidence of AP in the world (95.21 [91.74, 98.68] per 100,000 population per year).<sup>3</sup> A recent systematic review and meta-analysis of high-quality population-based studies on risk factors for pancreatic diseases<sup>2</sup> showed that tobacco use, high BMI and heavy alcohol consumption are the three most perilous risk factors for developing pancreatitis. Compared with non-smokers, smokers are 1.7 times more likely to develop a pancreatic disease. Current smokers are at a 20% higher risk of developing a pancreatic disease compared with ex-smokers.<sup>2</sup> Tobacco use is a leading risk factor for detrimental health worldwide. New Zealand is characterised by marked discrepancy in smoking prevalence between Māori and non-Māori population (33% versus 14% of New Zealand Europeans).<sup>38</sup> The Māori population has over twice as high all-cause mortality rates as the non-Māori population, with the non-Māori population living on average 7.3 years longer than the Māori population.<sup>38</sup> Compared with alcohol non-consumers, alcohol consumers are at a 1.12 times higher risk of developing disease of the exocrine pancreas. Heavy drinkers ( $\geq 4$  drinks per day) have a 40% increase in risk of developing a pancreatic disease compared to non-heavy alcohol users.<sup>2</sup> Alcohol consumption is another major cause of mortality, disease and injury globally. Internationally, alcohol consumption alone causes 2.7 million deaths annually.<sup>39</sup> In New Zealand, 5.4% of deaths in people aged less than 80 years are attributed to alcohol, with Māori having 2.5 times higher death rate attributable to alcohol than non-Māori.<sup>39</sup> In light of this evidence, it is not surprising that Māori have the highest incidence of pancreatitis and DP in the country. High quality studies are now needed to investigate factors protective against pancreatitis and DP and develop preventive strategies, particularly among Māori.

This study also brings to the forefront the geographic variation and confirmation of significant difference in the incidence

of pancreatic diseases between the north and the south of the country. Northern New Zealand is 2.7 times, 3 times and 4.75 more likely to develop AP, CP and DP, respectively, than southern New Zealand. The pattern persisted when stratified by ethnicity, with Māori in north of the country being 4.78 times, 3.01 times and 5.45 times more likely to develop AP, CP and DP, respectively, than those in the south of the country. Pacific people mirror the pattern, with those residing in north being 8.38 times, 13.67 times and 7.3 times more likely to develop AP, CP and DP, respectively, than those residing in the south of New Zealand. The New Zealand deprivation index<sup>40</sup> illustrates the difference between the north and the south of the country: while most of the North Island has a deprivation index of five to ten, with ten being most deprived, the South Island has a deprivation index of one to five, with one being least deprived. Deprivation indices are based on household income, access to healthcare, unemployment, housing and other factors. Facilities that may compromise health, such as alcohol outlets and advertising of unhealthy products, are preferentially located in most deprived areas often due to more affordable price barriers and utility costs.<sup>41</sup> Findings from our study bring to the forefront the ethnic disparities across New Zealand in terms of the diseases of the pancreas. That Māori and Pacific people often reside in the most deprived areas, while New Zealand Europeans populate the least deprived areas, may help explain the stark inequality in the incidence rates of pancreatitis and DP between Māori and Pacific people and New Zealand Europeans. This marked geographical variance necessitates further research into socioeconomic factors such as availability of resources and access to healthcare, and potentially the need to develop area-based prevention strategies.<sup>42</sup>

This population-based study has several limitations. First, only public hospital records nationwide were used to determine the incidence rates of pancreatitis and DP.

It is possible that cases diagnosed in the community and private hospitals were not transferred to public hospitals and were thus not entered into the used database,<sup>43</sup> resulting in underestimation of incidence rates. In future, population-based studies should strive to combine data from both public and private records to provide more accurate estimates of incidence rates. Second, definitions of AP, CP and DP were determined solely from the ICD-10 codes rather than from clinical, laboratory or pathological evidence.<sup>44-46</sup> Third, the “other” ethnic group was heterogeneous. No further information was available to tease out each individual ethnicity constituting the “other” group and explore the high incidence rates of diseases of the pancreas observed in this group. Further, the study focused a priori on the ethnicities that are highly prevalent in New Zealand. Last, the healthcare costs associated with AP, CP and DP could not be investigated, as this data were not readily available and are government funded. In future studies, information on costs associated with pancreatitis and DP should be ascertained to get a better insight into the socioeconomic burden these disease states pose on New Zealand’s healthcare system.

In conclusion, this nationwide population-based study shows that New Zealanders in general have one of the highest incidence rates of AP in the world, with Māori having the highest reported incidence of AP worldwide. It has also estimated the incidence of DP, which is a growing problem nationally and internationally, and showed that DP affects Māori and Pacific people disproportionately. Further, geographical location appears to be an important factor of inequity in incidence of pancreatitis and DP throughout the country. High quality research is needed in New Zealand to better understand the drivers of AP, CP and DP, and relationships between these three diseases to develop preventive and treatment strategies that are needed for everyone, but particularly for Māori who are worst affected by these diseases.

**Competing interests:**

Nil.

**Acknowledgements:**

The authors would like to thank Ying Huang (Section of Epidemiology and Biostatistics, University of Auckland), for her help with some of the statistical analyses.

This study was part of the Clinical and epidemiological investigations in Metabolism, nutrition, and pancreatic diseases (COSMOS) program. COSMOS is supported in part by the Health Research Council of New Zealand (grant 15/035 to Dr. Petrov), which played no role in the study design; collection, analysis, or interpretation of data; or writing of the manuscript.

**Author information:**

Sayali A Pendharkar, Department of Surgery, University of Auckland, Auckland; Juby Mathew, Department of Surgery, University of Auckland, Auckland; Jinfeng Zhao, Section of Epidemiology and Biostatistics, University of Auckland, Auckland; John A Windsor, Department of Surgery, University of Auckland, Auckland; Daniel J Exeter, Section of Epidemiology and Biostatistics, University of Auckland, Auckland; Maxim S Petrov, Department of Surgery, University of Auckland, Auckland.

**Corresponding author:**

Dr Max Petrov, Department of Surgery, University of Auckland, Room 12.085A, Level 12, Auckland City Hospital, Private Bag 92019, Victoria Street West, Auckland 1142.  
max.petrov@gmail.com

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7159>

**REFERENCES:**

1. Sankaran SJ, Xiao AY, Wu LM, et al. Frequency of progression from acute to chronic pancreatitis and risk factors: a meta-analysis. *Gastroenterology*. 2015; 149:1490–1500.
2. Alsamirai A, Das SLM, Windsor JA, Petrov MS. Factors that affect risk for pancreatic disease in the general population: a systematic review and meta-analysis of prospective cohort studies. *Clin Gastroenterol Hepatol*. 2014; 12:1635–1644.
3. Xiao AY, Tan MLY, Wu LM, et al. Global incidence and mortality of pancreatic diseases: a systematic review, meta-analysis, and meta-regression of population-based cohort studies. *Lancet Gastroenterol Hepatol*. 2016; 1:45–55.
4. Peery AF, Crockett SD, Barritt AS, et al. Burden of Gastrointestinal, Liver, and Pancreatic Diseases in the United States. *Gastroenterology*. 2015; 149:1731–1741.
5. 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; 126:23–42.
6. NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. *Lancet*. 2016; 387:1513–30.
7. Cui Y, Andersen DK. Pancreatogenic diabetes: special considerations for management. *Pancreatol*. 2011; 11:279–94.
8. Pendharkar SA, Mathew J, Petrov MS. Age- and sex-specific prevalence of diabetes associated with diseases of the exocrine pancreas: a population-based study. *Dig Liver Dis*. 2017; [Epub ahead of print].
9. Petrov MS. Abdominal fat: a key player in metabolic acute pancreatitis. *Am J Gastroenterol*. 2013; 108:140–2.
10. Pendharkar SA, Asrani VM, Xiao AY, et al. Relationship between pancreatic hormones and glucose metabolism: a cross-sectional study in patients after acute pancreatitis. *Am J Physiol Gastrointest Liver Physiol*. 2016; 311:G50–58.
11. Kennedy JIC, Askelund KJ, Premkumar R, et al. Leptin is associated with persistence of hyperglycemia in acute pancreatitis: a prospective clinical study. *Medicine (Baltimore)*. 2016; 95:e2382.
12. Goodger RL, Asrani VM, Windsor JA, Petrov MS. Impact of metabolic comorbidities on outcomes of patients with acute pancreatitis: a scoping review. *Panminerva Med*. 2016; 58:86–93.

13. Das SLM, Kennedy JIC, Murphy R, et al. Relationship between the exocrine and endocrine pancreas after acute pancreatitis. *World J Gastroenterol.* 2014; 20:17196–205.
14. Shen H, Yang C, Chang Y, et al. Risk of diabetes mellitus after first-attack acute pancreatitis: a national population-based study. *Am J Gastroenterol.* 2015; 110:1698–706.
15. Lee YK, Huang MY, Hsu CY, Su YC. Bidirectional relationship between diabetes and acute pancreatitis: a population-based cohort study in Taiwan. *Medicine (Baltimore).* 2016; 95:e2448.
16. Das SLM, Singh PP, Phillips ARJ, et al. Newly diagnosed diabetes mellitus after acute pancreatitis: a systematic review and meta-analysis. *Gut.* 2014; 63:818–31.
17. Anderson K, Brown LA, Daniel P, Connor SJ. Alanine transaminase rather than abdominal ultrasound alone is an important investigation to justify cholecystectomy in patients presenting with acute pancreatitis. *HPB (Oxford).* 2010; 12:342–7.
18. Nesvaderani M, Eslick GD, Cox MR. Acute pancreatitis: update on management. *Med J Aust.* 2015; 202:420–3.
19. Petrov MS, McIlroy K, Grayson L, et al. Early nasogastric tube feeding versus nil per os in mild to moderate acute pancreatitis: a randomized controlled trial. *Clin Nutr.* 2013; 32:697–703.
20. McKenzie SJL, Premkumar R, Askelund KJ, et al. The effect of enteral nutrition on adipokines in patients with acute pancreatitis. *J Nutr Sci.* 2015; 4:e33.
21. Jeffreys M, Wright C, Mannetje A, et al. Ethnic differences in cause specific mortality among hospitalised patients with diabetes: a linkage study in New Zealand. *J Epidemiol Community Health.* 2005; 59:961–6.
22. Jeffreys M, Stevanovic V, Tobias M, et al. Ethnic inequalities in cancer survival in New Zealand: linkage study. *Am J Public Health.* 2005; 95:834–7.
23. Wang TKM, Ramanathan T, Stewart R, et al. Māori have worse outcomes after coronary artery bypass grafting than Europeans in New Zealand. *N Z Med J.* 2013; 126:12–22.
24. Wu JM, Ho TW, Kuo TC, et al. Glycemic Change After Pancreaticoduodenectomy: a population-based study. *Medicine (Baltimore).* 2015; 94:e1109.
25. Ahmad O, Boschi-Pinto C, Lopez A, et al. Age-standardization of rates: a new WHO standard. <http://www.who.int/healthinfo/paper31.pdf>. 2001.
26. 2013 Census - Major ethnic groups in New Zealand. *Statistics NZ.* <http://www.stats.govt.nz/Census/2013-census/profile-and-summary-reports/infographic-culture-identity.aspx>. 2013.
27. Jenks GF. Generalization in statistical mapping. *Ann Ass Am Geogr.* 1963; 53:15–26.
28. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology.* 2013; 144:1252–61.
29. Fagenholz PJ, Fernandez-del Castillo C, Harris NS, et al. Direct medical costs of acute pancreatitis hospitalizations in the United States. *Pancreas.* 2007; 35:302–7.
30. Floyd A, Pedersen L, Nielsen GL, et al. Secular trends in incidence and 30-day case fatality of acute pancreatitis in North Jutland County, Denmark: a register-based study from 1981–2000. *Scand J Gastroenterol.* 2002; 37:1461–5.
31. Eland IA, Sturkenboom MJ, Wilson JH, Stricker BH. Incidence and mortality of acute pancreatitis between 1985 and 1995. *Scand J Gastroenterol.* 2000; 35:1110–6.
32. Tinto A, Lloyd DAJ, Kang JY, et al. Acute and chronic pancreatitis—diseases on the rise: a study of hospital admissions in England 1989/90–1999/2000. *Aliment Pharmacol Ther.* 2002; 16:2097–105.
33. Lindkvist B, Appelros S, Manjer J, Borgström A. Trends in incidence of acute pancreatitis in a Swedish population: is there really an increase? *Clin Gastroenterol Hepatol.* 2004; 2:831–7.
34. Spanier BWM, Dijkgraaf MGW, Bruno MJ. Trends and forecasts of hospital admissions for acute and chronic pancreatitis in the Netherlands. *Eur J Gastroenterol Hepatol.* 2008; 20:653–8.
35. Premkumar R, Phillips ARJ, Petrov MS, Windsor JA. The clinical relevance of obesity in acute pancreatitis: targeted systematic reviews. *Pancreatology.* 2015; 15:25–33.
36. Lin YK, Johnston PC, Arce K, Hatipoglu BA. Chronic Pancreatitis and Diabetes Mellitus. *Curr Treat Options Gastroenterol.* 2015; 13:319–31.
37. Yadav D, Hawes RH, Brand RE, et al. Alcohol consumption, cigarette smoking, and the risk of recurrent acute and chronic pancreatitis. *Arch Intern Med.* 2009; 169:1035–45.
38. Glover M, Kira A, Cowie N, et al. Health conse-

- quences of tobacco use for Māori—cessation essential for reducing inequalities in health. *N Z Med J.* 2013 2; 126:60–73.
39. MacLennan B, Kypri K, Connor J, et al. New Zealand's new alcohol laws: protocol for a mixed-methods evaluation. *BMC Public Health.* 2016; 16:1–11.
40. Atkinson J, Salmond C, Crampton P. NZDep2013 index of deprivation. Wellington: Department of Public Health, University of Otago; 2014.
41. Stevenson A, Pearce J, Blakely T, et al. Neighbourhoods and health: a review of the New Zealand literature. *N Z Geographer.* 2009; 65:211–21.
42. Warin B, Exeter D, Zhao J, et al. Geography matters: the prevalence of diabetes in the Auckland region by age, gender and ethnicity. *N Z Med J.* 2016;129: 25–38.
43. Hazra N, Gulliford M. Evaluating pancreatitis in primary care: a population-based cohort study. *Br J Gen Pract.* 2014; 64:e295–301.
44. Gillies NA, Pendharkar SA, Asrani VM, Mathew J, Windsor JA, Petrov MS. Interleukin-6 is associated with chronic hyperglycemia and insulin resistance in patients after acute pancreatitis. *Pancreatology.* 2016; 16:748–55.
45. Singh RG, Pendharkar SA, Gillies NA, Miranda-Soberanis V, Plank LD, Petrov MS. Associations between circulating levels of adipocytokines and abdominal adiposity in patients after acute pancreatitis. *Clin Exp Med* 2017; [Epub ahead of print].
46. Pendharkar SA, Singh RG, Petrov MS. Cross-talk between innate cytokines and the pancreatic polypeptide family in acute pancreatitis. *Cytokine* 2017; 90:161–8.

# Long-term survival following diabetic vitrectomy

Bia Z Kim, Kuo-Luong Lee, Stephen J Guest, David Worsley

## ABSTRACT

**AIM:** To update long-term survival data on patients with proliferative diabetic retinopathy undergoing vitrectomy and to identify associated risk factors.

**METHODS:** Retrospective clinical record review at a single New Zealand tertiary referral centre. A total of 182 eyes that underwent a vitrectomy for a diabetic vitreous haemorrhage and/or tractional retinal detachment between March 2000 and December 2010 were included. Kaplan-Meier survival curves and Cox-regression analyses were performed for survival rates and associated risk factors.

**RESULTS:** The mean age of patients was 55 years (range 22 to 85) at time of surgery. The three-year survival rate following diabetic vitrectomy was 83.5%, and the five-year survival rate (N=154) was 70.1%. Increasing age, dialysis and high serum creatinine were associated with poorer survival on multivariate Cox regression analyses (hazard ratio of 1.035, 4.216 and 1.930 respectively with p-values of 0.018, <0.001 and 0.046).

**CONCLUSION:** Survival rates after diabetic vitrectomy remain relatively poor but comparable to earlier New Zealand and international reports. However, there remain significant differences between ethnic groups within New Zealand that need to be addressed in addition to renal disease, which appears to be a major risk factor for poor survival. Overall, the contemporary survival outcomes observed in this study may influence decision making by patients and clinicians as well as encourage a review of current healthcare resource allocation in diabetes care.

Proliferative diabetic retinopathy (PDR) affects 7% of diabetic patients globally (approximately 17 million people), and half of these patients will be blind if untreated.<sup>1,2</sup> However, timely evaluation and treatment can reduce blindness by more than 90%, provide sustained improvement in visual function, improve quality and/or length of life and be highly cost-effective.<sup>2-6</sup> Treatment often involves a vitrectomy for non-clearing vitreous haemorrhage and/or tractional retinal detachment involving or threatening the macula.<sup>7</sup>

Patients with PDR not only suffer from a serious ocular disease but may also have important systemic comorbidities. This is reflected in studies reporting five-year survival following diabetic vitrectomy in predominantly Caucasian populations of between 68–86%, significantly lower than those without PDR (66–99%).<sup>8-10</sup> These studies date back two or more decades; survival rates are anticipated to be higher

following advances in management of diabetes in the 21<sup>st</sup> century.

The purpose of this study was to determine contemporary long-term survival rates and associated prognostic factors in patients undergoing diabetic vitrectomy. Survival rates are crucial for decision-making in the clinical setting and for planning healthcare resource allocation. A comparison with earlier studies may also highlight advances and shortcomings in diabetes and more specifically diabetic retinopathy management.

## Methods

A retrospective review of clinical records was performed of all patients that underwent vitrectomy for diabetic vitreous haemorrhage and/or tractional retinal detachment. All surgeries were performed between March 2000 and December 2010 at Waikato Public Hospital by two vitreo-retinal surgeons. Morbidity data and

**Table 1:** Ethnic distribution of study population (N=182 patients).

Ethnicity	Percentage of study population	Percentage of Waikato district population†
Māori	49.5%	20.7%
NZ European	40.1%	73.4%
Pasifika	5.5%	3.6%
Other	3.3%	
Not identified	1.6%	

†2013 New Zealand Census.<sup>11</sup>

survival rates were taken from the time of the initial vitrectomy surgery. Institutional approval was granted from Waikato DHB.

Clinical records were reviewed and data collected on patient demographics, medical comorbidities, serum creatinine, serum HbA1c, medications, pre-operative ocular characteristics and visual acuity. Date of patient death was confirmed from the national health record database.

Statistical analyses were performed using SPSS version 22 (Statistical Package for the Social Sciences GmbH Software, Munich, Germany). P values <0.05 were considered statistically significant. Kaplan-Meier survival curves were analysed for the group. Univariate Cox-regression analyses were performed for all pre-operative variables to identify risk factors for mortality. Significant variables were included in the multivariate analysis.

**Table 2:** Medical and medication history as self-reported at the pre-operative assessment or documented in clinical records prior to vitrectomy (N=182 patients).

Medical status	Number of cases (%)	Missing data (N)
Hypertension	155 (85.2%)	3
Dyslipidaemia	106 (58.2%)	6
Neuropathy	46 (25.3%)	7
Ischaemic heart disease	42 (23.1%)	4
Cerebrovascular disease	14 (7.7%)	4
HbA1c >55 mmol/mol‡§	69 (37.9%)	89
High serum creatinine†§	91 (50%)	34
Antiplatelet and/or anticoagulant	88 (48.4%)	7
Insulin	134 (73.6%)	1
Aspirin	77 (42.3%)	7
Statin	91 (50.0%)	10
Dialysis	27 (14.8%)	3
Smoking status		20
• current smoker	47 (25.8%)	
• ex-smoker	43 (23.6%)	
• non-smoker	72 (39.6%)	

†Normal creatinine in adult males 60–105 µmol/L, adult females 45–90 µmol/L.

‡Microvascular complication risk increases markedly when HbA1c above 55 mmol/mol.<sup>12</sup>

§Result of most recent blood test, taken within three months prior to vitrectomy.

**Table 3:** Indications for vitrectomy and pre-operative eye characteristics (N=182 eyes).

Pre-operative eye status	Number of cases (%)
Vitreous haemorrhage	143 (78.6)
Retinal detachment	94 (51.6)
Both vitreous haemorrhage and retinal detachment	55 (30.2)
Phakic	139 (76.4)
Neovascular glaucoma	3 (1.6)
Previous anti-VEGF treatment†	21 (11.5)

†Anti-VEGF = anti-vascular endothelial growth factor.

## Results

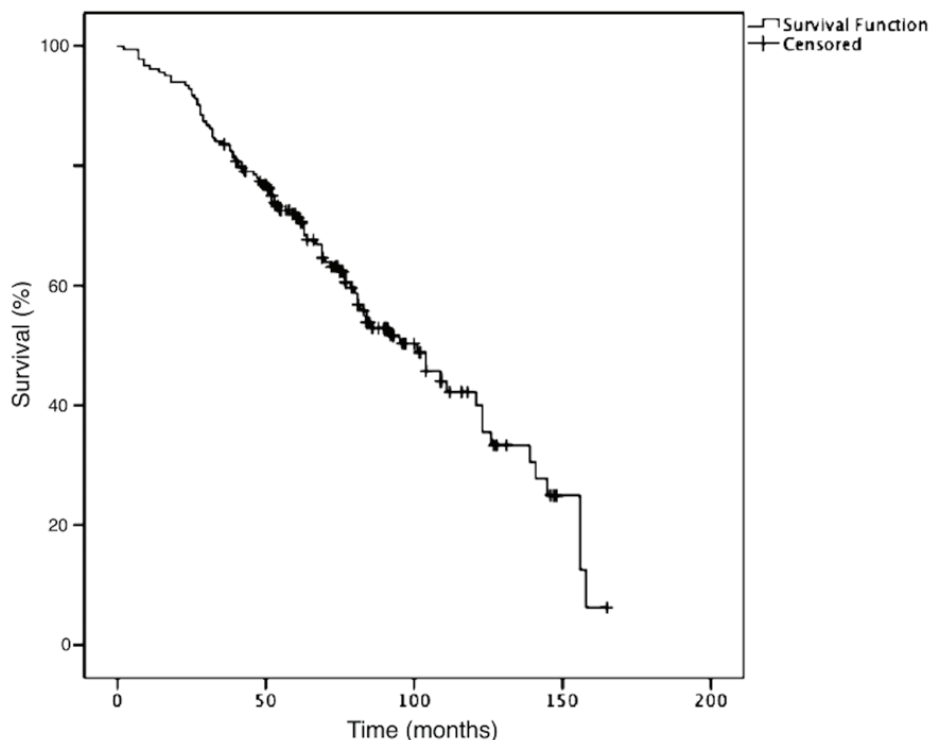
A total of 182 eyes were included in this study. The mean age was 55 years  $\pm$  12 SD (range 22 to 85), 53.8% were male, ethnicity was 49.5% Māori and 40.1% New Zealand (NZ) European (Table 1). Non-Europeans were younger at presentation than NZ Europeans (mean age 52  $\pm$  11 SD vs 59  $\pm$  13 SD,  $P < 0.001$ ) and with a shorter duration of diabetes than NZ Europeans (14 vs 22 years,  $p < 0.001$ ).

Type 1 diabetes mellitus accounted for 16.5%, and type 2 accounted for 83.5% of cases. Mean time from diagnosis of diabetes to time of surgery was 18 years  $\pm$  10 (range 0–59). Table 2 displays the medical and medication history of all patients.

Table 3 shows the indications for surgery and pre-operative eye status. Pre-operative best-corrected visual acuity (BCVA) in the operated eye was 1.23 logMAR (~6/120 Snellen equivalent). Post-operative BCVA was 0.471 logMAR (~6/18 Snellen equivalent).

### Use of anti-vascular endothelial growth factor (anti-VEGF) therapy in PDR

Anti-VEGF agents were introduced in 2007 at this institution. They were used alone or in conjunction with retinal photocoagulation and/or vitrectomy for treatment of PDR. Following its introduction, the proportion of vitrectomy cases for vitreous haemorrhage reduced from 86.4% to 68.4% ( $p = 0.006$ ).

**Figure 1:** Kaplan-Meier survival curve for patients undergoing vitrectomy for proliferative diabetic retinopathy (N=182).

**Table 4:** Univariate Cox proportional hazard model for baseline characteristics associated with mortality in patients undergoing diabetic vitrectomy (N=182).

Variables	P value	Hazard ratio	95% CI for hazard ratio
Age	*0.004	1.027	1.009–1.047
Male	0.377	0.829	0.548–1.256
Ethnicity Non-NZ European vs NZ European	0.149 *0.013	1.785	1.132–2.815
Right eye	0.836	1.045	0.688–1.589
Vitreous haemorrhage	0.799	1.075	0.615–1.880
Retinal detachment	0.565	0.885	0.583–1.343
Neovascular glaucoma	0.967	0.959	0.133–6.931
Pseudophakia	*0.004	1.915	1.225–2.995
Pre-operative visual acuity	0.459	1.115	0.837–1.485
Post-operative visual acuity	0.154	1.314	0.903–1.912
Pre-operative anti-VEGF therapy†	0.504	0.731	0.292–1.833
Type 1 diabetes mellitus	*0.007	0.319	0.139–0.732
Years since diagnosis of diabetes	0.553	1.006	0.986–1.027
HbA1c‡	0.432	0.884	0.650–1.202
Insulin	0.281	0.772	0.481–1.237
Serum creatinine‡	*<0.001	1.003	1.001–1.004
Dyslipidaemia	*0.044	1.585	1.012–2.483
Dialysis	*<0.001	4.446	2.662–7.426
Neuropathy	*0.017	1.778	1.110–2.848
Hypertension	*0.047	2.195	1.010–4.771
Ischaemic heart disease	0.107	1.466	0.920–2.337
Cerebrovascular disease	0.136	1.662	0.853–3.241
Smoking status	0.714		
• Current smoker	0.696	1.123	0.628–2.009
• Ex-smoker	0.416	1.244	0.736–2.103
Antiplatelet/anticoagulant	*0.022	1.648	1.073–2.529
Aspirin	0.061	1.502	0.981–2.298
Warfarin	0.955	1.023	0.470–2.228
Statin	0.807	1.056	0.683–1.631

†VEGF = vascular endothelial growth factor.

‡Result of most recent blood test, taken within three months prior to vitrectomy.

\*Statistically significant at p&lt;0.05 level.

**Table 5:** Multivariate Cox proportional hazard model for baseline patient characteristics affecting mortality in undergoing diabetic vitrectomy (N=182).

Variables	P value	Hazard ratio	95% CI for hazard ratio
Age	*0.007	1.040	1.011–1.071
Non-NZ European vs NZ European	*0.024	2.157	1.106–4.207
Pseudophakia	0.943	1.020	0.595–1.749
Type 1 diabetes mellitus	0.327	0.588	0.204–1.699
Serum creatinine†	*0.032	2.026	1.063–3.865
Dyslipidaemia	0.646	1.145	0.643–2.036
Dialysis	*<0.001	4.155	2.188–7.887
Neuropathy	0.079	1.621	0.946–2.778
Hypertension	0.969	0.983	0.411–2.348
Antiplatelet/anticoagulant	0.199	1.417	0.833–2.409

†Result of most recent blood test, taken within three months prior to vitrectomy.

\*Statistically significant at  $p < 0.05$  level.

Concurrent intra-vitreous anti-VEGF treatment was given in 23 cases at time of vitrectomy.

### Mortality following vitrectomy and associated risk factors

Mean follow-up after vitrectomy was 97 months  $\pm$  4 SE (95% CI 88–105, range 2–165) (Figure 1). Ninety patients (49.5%) were deceased as of 31<sup>st</sup> December 2013. Mean time to death was 60 months  $\pm$  4 SE (95% CI 52–68, range 2–158). Following vitrectomy, the three-year survival rate was 83.5% (152/182), and the five-year survival rate was 70.1% (108/154).

Variables that were significantly associated with mortality on univariate regression analysis were age, non-European ethnicity, pseudophakia, antiplatelet or anticoagulant therapy, dyslipidaemia, dialysis, serum creatinine, neuropathy, hypertension, type 1 diabetes mellitus (Table 4).

Variables that were significantly associated with mortality on stepwise multivariate regression analysis were age, dialysis, and serum creatinine (Table 5).

## Discussion

Patients with PDR are expected and observed to have much lower survival rates than those without proliferative retinopathy or diabetes, due to severe micro- and macro-vascular complications.<sup>8,9,13,14</sup> In the

current study, the survival rate following diabetic vitrectomy was 83.5% at three years, reducing to 70.1% at five years. These rates are comparable to previous reports (68–95.8% at five years), suggesting no great change has occurred over the last decade or more.<sup>8,9,13,15–17</sup>

Diabetes care is costly, and diabetic retinopathy care is a considerable part of this expense.<sup>18,19</sup> The costs of admissions for ophthalmic complications at Canterbury DHB, where diabetes was the primary diagnosis, amounted to \$579,462 in the 2005/06 financial year, accounting for 18% of total admissions for diabetes.<sup>20</sup> Indirect costs, such as those associated with vision loss, are estimated to be three-fold greater than such direct costs.<sup>21,22</sup> It is important to continually evaluate the prognosis and survival of these patients in order to guide healthcare management and clinical advisories in the future utilisation of healthcare resources and setting of treatment priorities. The relatively poor survival rates post-vitrectomy need to be considered in the mix with direct and indirect costs both to individual patients and within the wider health economic field.

Despite the high costs associated with diabetic retinopathy, early vitrectomy for diabetic vitreous haemorrhage is associated with a significant gain of 0.41 quality-adjusted life years (QALY), at a relatively low cost per QALY gain (<US\$10,000).<sup>4,5</sup>

Understanding the survival rates and the related prognostic factors may influence clinical decisions around unilateral versus bilateral surgery, complex surgery with poor visual prognosis or high risk of ocular morbidity.

There are reports of exceptional five-year survival rates, as in Japan (95.8%), which are close to that of type 1 diabetics without retinopathy (99%).<sup>10,23</sup> This Japanese cohort had a markedly lower mean age (43 years), possibly a shorter duration of diabetes (12.4 ± 7.7 years) and smaller proportion of pre-operative systemic microvascular complications than described in other studies, probably accounting for the high survival rates.<sup>23,24</sup> In the current study, half of the patients were Māori, whose life expectancy is, in general, shorter than non-Māori by 7.1 years. Furthermore, with diabetes, the standardised mortality rate for Māori is 62.5 per 100,000 versus 11 per 100,000 for non-Māori.<sup>25,26</sup> Thus we would expect a relatively lower survival rate in the current study.

In a New Zealand study by Vote et al, where the cohort was 35% Māori and 26% Pasifika (compared to the current study with 49.5% Māori and 5.5% Pasifika), the mean time to death was 4.3 years following primary vitrectomy for diabetic retinopathy.<sup>15</sup> There has been a marginal increase to 5.0 years in the current study, although the overall five-year mortality rate has not changed from around 70% in 1992–1996.<sup>15</sup> Between the study by Vote et al and the current study, the mean age at time of surgery has increased from 52 to 55, and the mean time between diagnosis of diabetes and vitrectomy has increased from 16 to 18 years.<sup>15,24</sup> This prolonged interval before requiring a vitrectomy may reflect improvements in overall diabetes management over the decade in New Zealand delaying the need for vitrectomy. Although, any effect of better diabetic control on post-vitrectomy survival rates in the later New Zealand cohort may be being offset by the greater age at which vitrectomy is being performed, and thus underestimated.

In the current multivariate Cox regression model, age at time of surgery, dialysis and serum creatinine were statistically significant factors related to reduced survival following diabetic vitrectomy. Matsumoto et al reported an association between age, nephropathy

and neuropathy with lower survival.<sup>23</sup> In the current study, neuropathy was only significant at the univariate level and not in the multivariate model. Similarly, Gollamudi et al showed age, duration of diabetes and nephropathy had an effect on survival in patients undergoing diabetic vitrectomy in the US.<sup>8</sup> Markers of nephropathy vary between studies making comparisons difficult. The incidence of diabetes-related end-stage renal disease (ESRD)—the most common cause of ESRD in New Zealand—has doubled between 1992 and 2003 in New Zealand.<sup>27</sup> The higher rate of dialysis in the current study as compared with the earlier New Zealand data (14% vs 11%) may reflect this change.<sup>15</sup> The similar survival rates, despite worsening nephropathy, may suggest that the management of ESRD in particular has improved.

Māori have an earlier onset of type 2 diabetes mellitus than NZ Europeans (by 8–10 years) and a high failure-to-attend-screening rate (32.3% versus 18.7% overall).<sup>28</sup> Thus patients in the current study may be diagnosed late and actually have a longer duration of diabetes before vitrectomy than observed. This may partly account for the lower survival rate in the current study compared to the UK study by Banerjee et al (70% vs 86% at five years), where a longer duration of diabetes was associated with poorer survival.<sup>15,24</sup> Although there was no significant association between duration of diabetes and survival in the current study, there is a significant interval before vitrectomy, where diabetic screening and monitoring could be further encouraged, especially for Māori. This may take the form of transport subsidies, integrated specialty services for diabetes to reduce the number of hospital visits and promote a multidisciplinary approach to treatment. Community visits by respective healthcare professionals may also improve awareness of disease and community support for patients. Although pre-operative visual acuity remains around 6/120 (1.23 logMAR in the current study versus 1.38 logMAR a decade earlier in New Zealand), the mean post-operative visual acuity was better [0.471 logMAR (~6/18) versus 1.19 logMAR (~6/90)].<sup>15</sup> This improvement may be due to better surgical instrumentation and techniques, more sub-specialty trained

vitreoretinal surgeons, improved access to care, less severe disease at time of surgery and more eyes with combined or earlier non-surgical interventions (pan-retinal photocoagulation, long-acting steroids such as triamcinolone and/or anti-VEGF therapy). More than 10% of the current study population had prior anti-VEGF therapy, and 13% had anti-VEGF at time of their diabetic vitrectomy. The significant reduction in vitreous haemorrhage cases since 2007 (when anti-VEGF treatment was introduced for PDR at this institution) may be at least in part due to these new agents. Although the survival rates have not significantly changed over the years, the better visual outcomes following diabetic vitrectomy may result in improved quality of life.

The proportion of vitrectomy patients on insulin has increased from 67% in 1992–1996 to 73.6% despite a reduction in the proportion of type 1 diabetes from 23.7% to 16.5% during the same period.<sup>15</sup> This may reflect an increase in appropriate medical treatment of diabetes. Alternatively, the increased use of insulin therapy may reflect a greater disease severity, in which case non-surgical treatments for diabetic retinopathy would appear to be delaying the need for vitrectomy. Furthermore, despite delaying vitrectomy both in terms of diabetes duration and patient age, the proportion of retinal detachments as an indication for diabetic vitrectomy has decreased from 70% in 1992–1996 to 52%,

suggesting that patients have less severe ocular disease or present earlier.<sup>15</sup> Again, this may be secondary to improvements in screening, medical and laser treatments and access to treatment.

Limitations of this study include its retrospective nature and incomplete data collection. Measures of serum creatinine and HbA1c were within three months of the surgery and were not all taken at pre-operative assessment.

In summary, this study provides updated survival data of patients undergoing diabetic vitrectomy. The relatively poor survival rate associated with the need for diabetic vitrectomy is an important consideration for both individual patient care and for wider health resource allocation. Since the 1990s to 2000s, the mean age of diabetic vitrectomy patients and their duration of diabetes have increased. Furthermore, post-operative visual acuity results have improved. These changes may reflect improvements in eye screening, management of diabetes and retinopathy and improved access to care. The long-term survival of patients undergoing diabetic vitrectomy appears to be associated with age and severe nephropathy. Ongoing efforts are needed to improve the renal care of these patients, especially in non-European populations. Overall, vitrectomy is considered a highly successful and cost-effective intervention, and ongoing updates of survival data are recommended for its utility analysis.

**Competing interests:**

Nil.

**Acknowledgements:**

The authors would like to thank Mrs Julie Loughnan (Administrator, Waikato DHB, New Zealand) and Dr Rick Cutfield, Diabetologists at Waitemata DHB, New Zealand for their support in this study.

**Author information:**

Bia Z Kim, Non-Training Registrar, Department of Ophthalmology, Waikato District Health Board, Hamilton; Kuo-Luong Lee, Vitreo-retinal Fellow, Department of Ophthalmology, Waikato District Health Board, Hamilton; Stephen J Guest, Consultant Ophthalmologist, Department of Ophthalmology, Waikato District Health Board, Hamilton; David Worsley, Consultant Ophthalmologist, Department of Ophthalmology, Waikato District Health Board, Hamilton.

**Corresponding author:**

Dr Bia Z Kim, Department of Ophthalmology, Faculty of Medical and Health Sciences, Private Bag 92019, University of Auckland, Auckland.

bia.kim@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7160>

**REFERENCES:**

1. Yau JW, Rogers SL, Kawasaki R, et al. Global prevalence and major risk factors of diabetic retinopathy. *Diabetes Care*. 2012; 35:556–64.
2. Ferris FL. Results of 20 years of research on the treatment of diabetic retinopathy. *Prev Med*. 1994; 23:740–2.
3. Brown MM, Brown GC, Brown HC, et al. The comparative effectiveness and cost-effectiveness of vitreoretinal interventions. *Curr Opin Ophthalmol*. 2008; 19:202–7.
4. Brown MM, Brown GC, Lieske HB, Lieske PA. Preference-based comparative effectiveness and cost-effectiveness: a review and relevance of value-based medicine for vitreo-retinal interventions. *Curr Opin Ophthalmol*. 2012; 23:163–74.
5. Sharma S, Hollands H, Brown GC, et al. The cost-effectiveness of early vitrectomy for the treatment of vitreous hemorrhage in diabetic retinopathy. *Curr Opin Ophthalmol*. 2001; 12:230–4.
6. The Diabetic Retinopathy Vitrectomy Research Group. Early vitrectomy for severe vitreous hemorrhage in diabetic retinopathy. Four-year results of a randomized trial: Diabetic Retinopathy Vitrectomy Study Report 5. *Arch Ophthalmol*. 1990; 108:958–64.
7. Sharma T, Fong A, Lai TY, et al. Surgical treatment for diabetic vitreoretinal diseases: a review. *Clin Experiment Ophthalmol*. 2016; 44:340–54.
8. Gollamudi SR, Smiddy WE, Schachat AP, et al. Long-term survival rate after vitreous surgery for complications of diabetic retinopathy. *Ophthalmology*. 1991; 98:18–22.
9. Helbig H, Kellner U, Bornfeld N, Foerster MH. Life expectancy of diabetic patients undergoing vitreous surgery. *Br J Ophthalmol*. 1996; 80:640–3.
10. Klein R, Moss SE, Klein BEK, DeMets DL. Relation of ocular and systemic factors to survival in diabetes. *Arch Int Med*. 1989; 149:266–72.
11. Statistics New Zealand. 2013 Census: Statistics New Zealand; 2015. Available from: <http://www.stats.govt.nz/Census/2013-census.aspx>. Accessed April 15, 2016.
12. New Zealand Guidelines Group. New Zealand Primary Care Handbook 2012. 3rd ed. Wellington: New Zealand Guidelines Group; 2012.
13. Davis MD, Hiller R, Magli YL, et al. Prognosis for life in patients with diabetes: relation to severity of retinopathy. *Trans Am Ophthalmol Soc*. 1979; 77:144–70.
14. Lux A, Ostri C, Dyrberg E, et al. Survival rates after diabetic vitrectomy compared with standard

- diabetes and general populations. *Acta Ophthalmol.* 2012;90:e650–2.
15. Vote BJ, Gamble GD, Polkinghorne PJ. Auckland Proliferative Diabetic Vitrectomy Fellow Eye Study. *Clin Experiment Ophthalmol.* 2004; 32:397–403.
  16. Summanen P, Karhunen U, Laatikainen L. Characteristics and survival of diabetic patients undergoing vitreous surgery. *Acta Ophthalmol (Copenh).* 1987; 65:197–202.
  17. Uchio E, Inamura M, Ohno S, et al. Survival rate after vitreous surgery in patients with diabetic retinopathy. *Ophthalmologica.* 1993; 206:83–8.
  18. Palmer AJ, Weiss C, Sendi PP, et al. The cost-effectiveness of different management strategies for type I diabetes: a Swiss perspective. *Diabetologia.* 2000; 43:13–26.
  19. Caro JJ, Ward AJ, O'Brien JA. Lifetime costs of complications resulting from type 2 diabetes in the U.S. *Diabetes Care.* 2002; 25:476–81.
  20. Sheerin I. Hospital expenditure on treating complications of diabetes and the potential for deferring complications in Canterbury, New Zealand. *N Z Med J.* 2009; 122:22–9.
  21. Taylor HR, Keeffe JE, Mitchell P. Clear insight: the economic impact and cost of vision loss in Australia. Melbourne: Eye Research Australia, 2004.
  22. Meads C, Hyde C. What is the cost of blindness? *Br J Ophthalmol.* 2003; 87:1201–4.
  23. Matsumoto T, Uchio E, Gotoh K, et al. [Survival rate of patients with diabetic retinopathy after vitreous surgery]. *Nippon Ganka Gakkai Zasshi.* 1994; 98:989–93.
  24. Banerjee PJ, Moya R, Bunce C, et al. Long-term survival rates of patients undergoing vitrectomy for proliferative diabetic retinopathy. *Ophthalmic Epidemiol.* 2016; 23:94–8.
  25. Statistics New Zealand. New Zealand period life tables: 2012–14. Wellington: Statistics New Zealand; 2015. Available from: [http://www.stats.govt.nz/browse\\_for\\_stats/health/life\\_expectancy/NZLifeTables\\_HOTP12-14.aspx](http://www.stats.govt.nz/browse_for_stats/health/life_expectancy/NZLifeTables_HOTP12-14.aspx). Accessed April 16, 2016.
  26. Bramley D, Hebert P, Jackson R, Chassin M. Indigenous disparities in disease-specific mortality, a cross-country comparison: New Zealand, Australia, Canada, and the United States. *N Z Med J.* 2004; 117:U1215.
  27. Joshy G, Simmons D. Epidemiology of diabetes in New Zealand: revisit to a changing landscape. *N Z Med J.* 2006; 119:U1999.
  28. Reda E, Dunn P, Straker C, et al. Screening for diabetic retinopathy using the mobile retinal camera: the Waikato experience. *N Z Med J.* 2003; 116:U562.

# Prevalence of diabetic retinopathy at first presentation to the retinal screening service in the greater Wellington region of New Zealand 2006–2015, and implications for models of retinal screening

Lily YL Chang, Arier C Lee, Wilson Sue

## ABSTRACT

**AIM:** To describe the prevalence of diabetic retinopathy (DR) in patients at first presentation for diabetic retinal screening in the greater Wellington region with the intent of service evaluation.

**METHODS:** This is a retrospective study using data collected from patients newly referred for diabetic retinal screening between 2006–2015 (prevalence analysis, n=12667). The prevalence of DR was calculated by gender, ethnicity, age, type of diabetes and glycaemic control (HbA1c). Chi-square test and multiple logistic regression was used for data analysis.

**RESULTS:** The prevalence of any DR was 22.5% (n=2852) (non-sight-threatening (NST-DR) n=2562, 20.2%, sight-threatening (ST-DR) n=290, 2.3%). Type 1 diabetes and poor HbA1c control were strongly associated with any degree of DR. Old-age (>65 years), and Asian and Pacific Island (PI) ethnicity had moderately greater odds compared with European. Male gender had marginally increased odds for any DR.

**CONCLUSION:** This study identified a large proportion (97.7%) of patients (no DR n=9815, 77.5%, NST-DR n=2562, 20.2%) who can be managed in the community by appropriately supported primary care providers, and do not require referral to secondary care ophthalmology. In addition to early detection of ST-DR (2.3%), retinal screening is an early opportunity for education of patients with no DR or NST-DR.

The prevalence of diabetes by district health board (DHB) area in New Zealand is estimated to range from 4.17% to 8.35%.<sup>1</sup> The Ministry of Health estimated 260,458 diagnosed cases of New Zealanders with diabetes as at December 2015,<sup>2</sup> and projects an average annual growth rate of new diabetes diagnoses of 5% according to Diabetes Surveillance: population-based estimates and projections for New Zealand.<sup>3</sup>

Blindness due to diabetic retinopathy (DR) is the most common cause of newly reported cases of visual loss in working-age adults

(20–74 years of age),<sup>4,5</sup> which has debilitating implications to daily living, as well as the ability to remain in the workforce. In New Zealand, approximately 20–25% of people with diabetes have DR, with an estimated 12% requiring care from ophthalmology.<sup>6,7</sup>

The benefits of retinal screening in reducing the burden to society of sight-threatening (ST) diabetic eye disease and incidental non-diabetic eye disease are long established.<sup>8</sup> However, there is ongoing discussion surrounding how to best carry out screening, and particularly, the point of

referral from primary care (collaborative teams including technicians, nurses, general practitioners (GPs) and optometrists) to secondary care (ophthalmology).<sup>9</sup>

The three DHBs that make up the greater Wellington region are Wairarapa DHB (Masterton District, Carterton District, South Wairarapa District), Hutt Valley DHB (Upper Hutt City, Lower Hutt City), and Capital & Coast DHB (Kapiti Coast District, Porirua City, Wellington City). The greater Wellington region was estimated by New Zealand Ministry of Health to have 24,499 diagnosed cases of diabetes as at December 2015.<sup>2</sup> Retinal screening was a component of the national Diabetes Get Checked Programme introduced in June 2000 that provides a free annual review for all patients with diabetes. However, prior to 2002, screening was only available from a single site at Wellington Hospital, and was limited to approximately 400 screens per annum. This meant it was not possible for regional GPs to refer all the population diagnosed with diabetes to the public health system for retinal screening.

The solution in the greater Wellington region was initiated by Wellington Independent Practitioner's Association to make use of a single visit to contracted community optometrists for screening and primary care management of DR. Since its implementation, the service has been continued by Compass Health Primary Health Organisation (PHO) (for Capital & Coast and Wairarapa DHBs) and Te Awakairangi Health Network and Ropata Medical PHO (for Hutt Valley DHB). Diagnosis and management rather than detect and refer is the clinical competence standard for New Zealand optometrist registration, and diabetic retinal screening and monitoring has been part of the optometry scope of practice since the introduction of the Health Practitioners Competence Assurance Act 2003.<sup>10,11</sup>

Retinal screening in the greater Wellington region is funded by the three DHBs, and is free to eligible patients enrolled with their local GP. Screening and reporting is completed within 90 days of referral. The greater Wellington region currently has nine community optometry sites, 24 optometrists, of whom 23 have therapeutic pharmaceutical agent (TPA) endorsement, 11 retinal

cameras and five optical coherence tomographers (OCT). Designated ophthalmologists provide support to contracted optometrists by reviewing retinal images and overseeing peer review events. Approximately 11,000 screens were performed in 2015—3.2% were referred to ophthalmology secondary care at Wellington Hospital Eye Clinic for diabetic eye disease, and 96.8% remained in the community with primary care optometry.<sup>12</sup> This model of care resonates with many aspects of the New Zealand health strategy 2016,<sup>13</sup> as the overall goal is to provide more accessible, timely and quality health care to New Zealanders.

The aim of this study is to provide New Zealand-specific epidemiology data on DR in the greater Wellington region, and to assess the effectiveness of this model in health care delivery. Coverage and did-not-attend (DNA) rates will be used to evaluate completeness and equity of care between population groups.

## Methods

### Research design

This is a retrospective evaluation study. Anonymised patient data from 1<sup>st</sup> January 2006 to 31<sup>st</sup> December 2015 was obtained from Compass Health PHO. A scope of review was submitted to The Health and Disability Ethics Committees (HDEC), and this study is exempt from full ethics review. An out-of-scope letter was issued by HDEC in February 2016. The letter stated this study is consistent with an audit and related activity to evaluate how the optometrist screening delivery model may support regional service design.

### Data inclusion criteria

Eligible patient were those diagnosed with diabetes and referred by their GPs or nurses for diabetic retinal screening with a contracted community optometrist for the first time. Referrals are posted, faxed or emailed by GPs or practice nurses to the community optometrist. The GP practices use in-house systems to check that referral has been made and outcome screening report is received from the optometrist. GP practices checks off retinal screening as a component that must be completed for the DHB-funded "Get Checked" diabetic health review (replaced in 2012 by Diabetes Care

Improvement Package), which is also a PHO performance Programme indicator.<sup>14</sup> Patient data was included for analysis if all reporting fields described in the screening report were complete. Patients with null values in the age, gender, HbA1c, type of diabetes or DR grading fields were excluded for analysis. DR grading of a patient was defined based on the most advanced DR grading of the two eyes.

### Screening process

The screening procedure was in accordance with the standards for retinal screening methods in the National Diabetes Retinal Screening Grading System and Referral Guidance,<sup>12</sup> which included measurement of the patient's habitual and pinhole visual acuities, retinal photography, grading the severity of DR,<sup>12</sup> immediate discussion of results and patient education by the screening optometrist, and scheduling for rescreening/monitoring or referral. Habitual and pinhole visual acuities were functional measures of the patient's vision and estimation of best corrected vision respectively. Reduced visual acuity may be indicative of cataract, diabetic macula oedema or other non-diabetes-related eye diseases. Mydriatics were indicated when ocular media opacity and/or pupil miosis inhibit the view of the retina. Clinical examination with binocular indirect ophthalmoscopy or slit lamp biomicroscopy was performed when photos were inadequate or views outside of the photographable area were required, eg patients diagnosed with type 1 diabetes for longer than 15 years are at higher risk for chronic hypoxia-induced neovascularisation in the retinal periphery.<sup>12</sup> The use of dilated retinal photography and clinical examination by trained clinicians have been described as sensitive screening and monitoring tests.<sup>15</sup> In cases of suspected macula oedema, OCT was used to aid the diagnosis, and informed screening optometrists of referral urgency for ophthalmology care. The screening report was sent to the referrer and Compass Health, or a referral was sent to the hospital as required.

### Screening report

The report included patient details such as ethnicity, age, latest HbA1c, type of diabetes, year of diagnosis for diabetes, co-existing health problems and medications. Screening

details in the report specified whether mydriatics was used, whether digital retinal photography or clinical examination was conducted, VA, grading of DR and any relevant notes from the screening optometrist. If ophthalmology referral was actioned, the referral reasons and ophthalmology clinic were specified on the report.

### Study parameters

1. Prevalence of non-sight-threatening DR (NST-DR) \* at first presentation from a cohort of patients with type 1 or type 2 diabetes.
2. Prevalence of sight-threatening DR (ST-DR) \*\* at first presentation from a cohort of patients with type 1 or type 2 diabetes.
3. Odds ratio of DR (adjusted by age, gender, and ethnicity and disease characteristics (diabetes type and glycaemic control as indicated by HbA1c) using logistic regression).
4. The DNA rate to diabetic screening at first presentation, and for all referrals (including first referrals and returning patients).
5. Coverage rate calculated as the number of screenings done by optometrists at the last quarter in 2015 divided by the number of enrolled diabetic patients in the region.

\* NST-DR in the Wellington model is defined as disease grading R1-R3 (minimal-moderate) for retinopathy, and M1-M3 (minimal-moderate) for maculopathy.

\*\* ST-DR in the Wellington model is defined as disease grading R4 (severe non-proliferative DR) or R5 (proliferative DR) for retinopathy, and M4 (moderate) or M5 (severe) for maculopathy.

Statistical analysis was performed by Chi-square test and multiple logistic regression, using SAS® software version 9.4 (SAS Institute Inc., Cary, NC, USA).

The definitions of NST-DR and ST-DR used in this study are consistent with the New Zealand Diabetic Retinal Screening, Grading, Monitoring and Referral Guidance 2016.<sup>12</sup> ST-DR is DR where additional medical management and/or surgical treatment may be required to prevent permanent functional vision loss.<sup>12</sup> In the Wellington model this is the clinical referral point from

primary care (community optometrist) to secondary care (ophthalmology clinic in hospital setting). Such clinical referral point evolved over the years through frequent communication, peer review and support from ophthalmologists.<sup>7</sup>

## Results

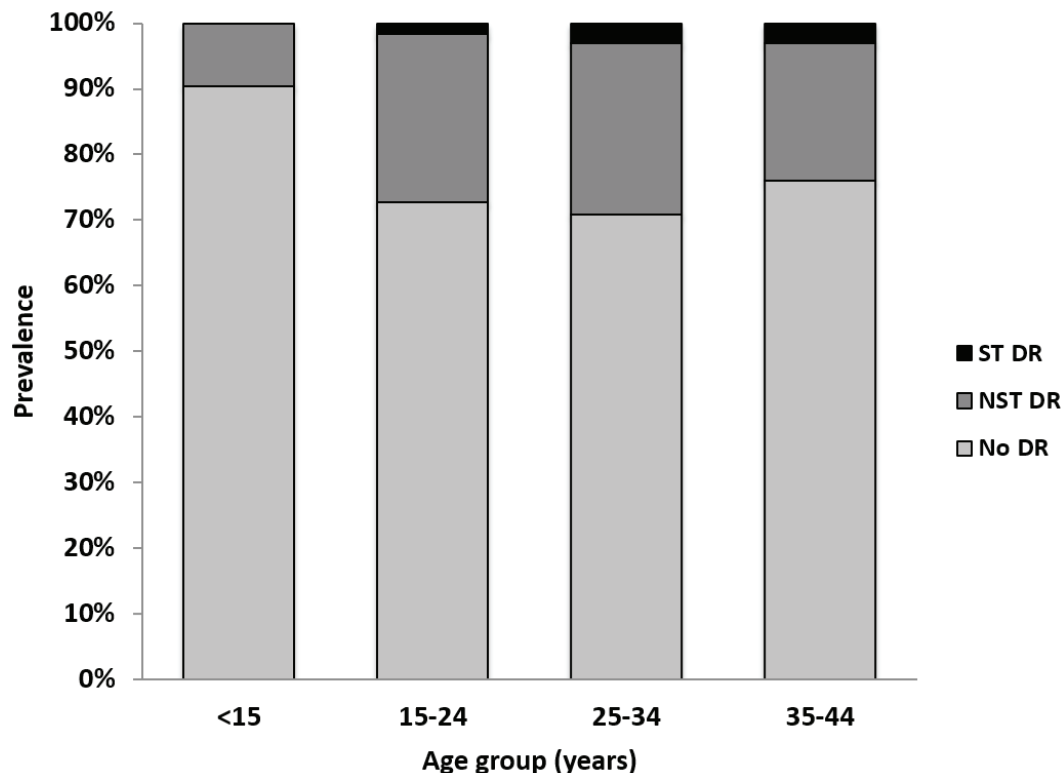
Seventeen thousand five hundred and eight patients attended their first diabetic retinal screening from 2006–2015, and those with incomplete data for age, gender, HbA1c, type of diabetes or DR grading (n=4,841) were excluded for DR prevalence analysis. This led to a total of 12,667 patients being included in DR prevalence calculation. The overall prevalence of any form of DR is 22.5% (n=2,852), with 20.2% (n=2,562)

being NST-DR (R1, 2, 3, and M1, 2, 3), and 2.3% (n=290) being ST-DR (R4, R5, M4, M5). Table 1 shows patient demographics and prevalence of DR by gender, ethnicity and age. Fifty-five percent of the study cohort were male (n=6,968), and 45% were female (n=5,699). Most of first-time retinal screening patients were European (n=7,165, 56.6%). The majority of patients fell into the middle-aged category (45–64 years old, n=6,156, 48.6% of cohort), followed by the old-age category (>65 years, n=3,876, 30.6% of cohort). Univariate analysis by chi-square test showed a statistically significant difference in the prevalence of DR when compared by gender (DF = 2,  $p < 0.001$ ), ethnicity (DF=8,  $p < 0.001$ ) and age groups (DF=4,  $p < 0.001$ ).

**Table 1:** Prevalence of DR by gender, ethnicity and age groups.

						Chi-square test	
	Total (n)	Total %	No DR	NST-DR	ST-DR	Degree of freedom (DF)	p-value
<b>Total</b>							
	12,667	100.0%	9,815 (77.5%)	2,562 (20.2%)	290 (2.3%)		
<b>Gender</b>							
Male	6,968	55.0%	5,307 (76.1%)	1,482 (21.3%)	179 (2.6%)	2	0.0002
Female	5,699	45.0%	4,508 (79.1%)	1,080 (19.0%)	111 (1.9%)		
<b>Ethnicity</b>							
European	7,165	56.6%	5,619 (78.4%)	1,418 (19.8%)	128 (1.8%)	8	<0.0001
Māori	1,641	13.0%	1,294 (78.9%)	307 (18.7%)	40 (2.4%)		
PI	1,335	10.5%	994 (74.4%)	296 (22.2%)	45 (3.4%)		
Asian	1,486	11.7%	1,106 (74.4%)	340 (22.9%)	40 (2.7%)		
Other/ Unknown	1,040	8.2%	802 (77.1%)	201 (19.3%)	37 (3.6%)		
<b>Age</b>							
Young (≤44)	2,635	20.8%	1,981 (75.2%)	583 (22.1%)	71 (2.7%)	4	0.0003
Mid (45–64)	6,156	48.6%	4,851 (78.8%)	1,158 (18.8%)	147 (2.4%)		
Old (≥65)	3,876	30.6%	2,983 (77.0%)	821 (21.2%)	72 (1.9%)		

Figure 1: Prevalence of DR in the young-age category ( $\leq 44$  years) in 10-year age bands.



It is interesting to note that although the young-age category ( $\leq 44$  years,  $n=2,635$ , 20.8%) had the smallest proportion of the entire cohort, DR was the most prevalent for both the NST-DR and ST-DR categories (Table 1). Following this observation, all patients  $\leq 44$  year old were further stratified into 10-year age bands. In the  $<15$  years old age group, 9.6% of the patients had NST-DR, and there was no ST-DR. In the 15–24 year-old age group, there was a marked  $>2.5x$  increase in the prevalence of NST-DR (25.7%) (Figure 1). As for ST-DR, the most significant increase in prevalence was from none in the  $<15$  group to 1.6% in the 15–24 year-old group, which then nearly doubled to 3.0% in the 25–34 year-old age group.

Table 2 shows prevalence of DR in different diabetes disease types and levels of HbA1c control. 7.2% ( $n=918$ ) of the patients in this cohort had type 1 diabetes, and 92.8% ( $n=11749$ ) had type 2 diabetes. There was significantly greater prevalence for both NST-DR and ST-DR in type 1 diabetes ( $DF=2$ ,  $p<0.0001$ ) using univariate analysis by chi-square test. HbA1c provided information on the level of glycaemic control, and patients were grouped into

three categories, (1) HbA1c  $\leq 64$  mmol/mol (good control), (2) HbA1c = 65–75 mmol/mol (moderate control), and (3) HbA1c  $>75$  mmol/mol (poor control), with reference to The Diabetic Retinal Screening, Grading, Monitoring and Referral Guidance.<sup>12</sup> Poor HbA1c control associated with a significantly higher prevalence of both NST-DR and ST-DR, using chi-square test ( $DF=4$ ,  $p<0.0001$ ).

The association of DR with gender, ethnicity, age group, diabetes type and HbA1c control was assessed using logistic regression (Table 3). Having type 1 diabetes and poor HbA1c control is strongly associated with any degree of DR in this patient cohort. The odds of people with type 1 diabetes having any DR is 3.17 (95% CI 2.70–3.72) times the odds of people with type 2 diabetes, and the odds of people with poor HbA1c control having any DR is 2.22 (95% CI 1.98–2.49) times the odds of people with good control. The old-age category ( $OR = 1.72$ , 95% CI 1.50–1.98), Asian ethnicity ( $OR = 1.51$ , 95% CI 1.32–1.73), and PI ethnicity ( $OR = 1.33$ , 95% CI 1.15–1.54) also have moderately greater odds when compared to the reference levels of young-age and European ethnicity respectively, for having any degree

**Table 2:** Prevalence of DR in different diabetes disease types and glycaemic control categories.

						Chi-square test	
	Total (n)	Total %	No DR	NST-DR	ST-DR	Degree of freedom (DF)	p-value
<b>Diabetes type</b>							
Type 1	918	7.2%	530 (57.7%)	330 (36.0%)	58 (6.3%)	2	<0.0001
Type 2	11,749	92.8%	9,285 (79.0%)	2,232 (19.0%)	232 (2.0%)		
<b>Glycaemic control (HbA1c in mmol/mol)</b>							
Good (≤64)	9,419	74.3%	7,606 (80.8%)	1,679 (17.8%)	134 (1.4%)	4	<0.0001
Moderate (65–75)	1,353	10.7%	962 (71.1%)	352 (26.0%)	39 (2.9%)		
Poor (>75)	1,895	15.0%	1247 (65.8%)	531 (28.0%)	117 (6.2%)		

of DR. Male patients have approximately 20% increase in the odds of having any DR (OR=1.21, 95% CI 1.11–1.32) compared to female patients.

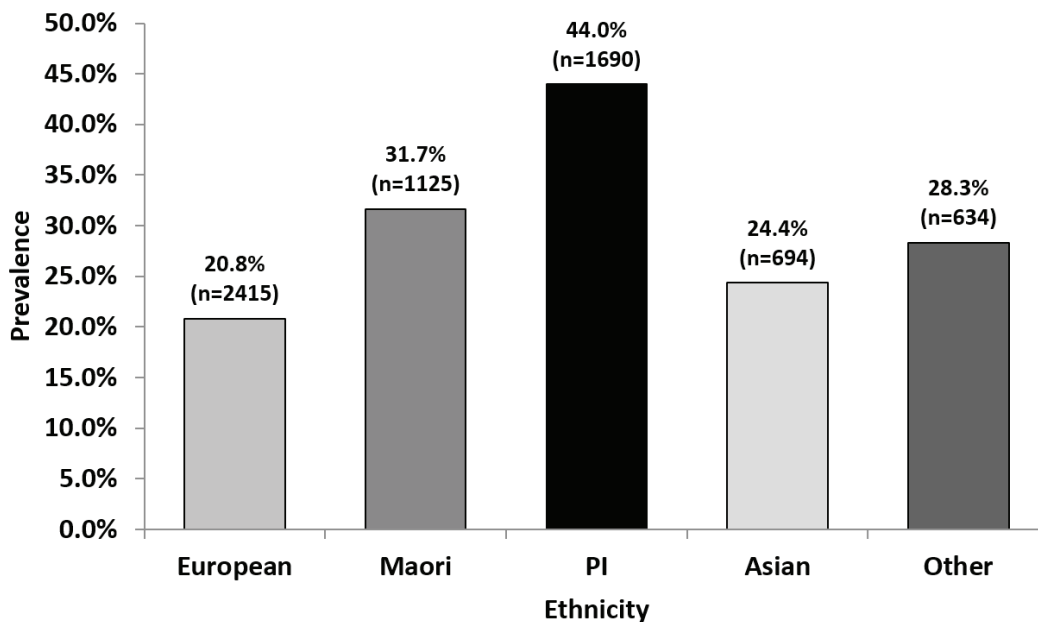
The mean DNA rate (DNA n=6,558, divided by the total number of referrals n=24,066)

of first-time referrals between 2006–2015 was 27.3% (Figure 2). Patients identified with PI ethnicity had the highest DNA rate (DNA n=1,690, attendance n=2,153, 44.0%), followed by Māori ethnicity (DNA n=1,125, attendance n=2,427, 31.7%). The mean DNA rate of all referrals between 2006–2015

**Table 3:** Odds ratio estimates for any DR, adjusted by age, gender, ethnicity, diabetes type and HbA1c control using multiple logistic regression.

Categories	Effect	Point estimate	95% Wald confidence limits	
<b>Age category</b>	Middle-age vs young	1.29	1.14	1.45
	Old-age vs young	1.72	1.50	1.98
<b>Gender</b>	Male vs female	1.21	1.11	1.32
<b>Ethnicity</b>	Māori vs European	1.06	0.92	1.21
	PI vs European	1.33	1.15	1.54
	Asian vs European	1.51	1.32	1.73
	Other/unknown vs European	1.14	0.97	1.34
<b>Diabetes Type</b>	Type 1 vs 2	3.17	2.70	3.72
<b>HbA1c Control</b>	Moderate control vs good control	1.64	1.44	1.88
	Poor control vs good control	2.22	1.98	2.49

Figure 2: DNA rate of first-time referrals between 2006–2015.



(including first presentation and returning patients) was 12.9% (DNA n=13,191, attendance n=89,017).

The estimated mean coverage rate (Q4 2015) of diabetic retinal screening for the three DHBs in the greater Wellington region is 88.9% (numerator is the number of patients diagnosed with diabetes and enrolled in a CCH, Wairarapa and Hutt Valley GP practice who have received a valid retinal screening in the last two years to the end of the reporting quarter on 31<sup>st</sup> December 2015 (n=17,237), and the denominator is the number of patients diagnosed with diabetes and enrolled in a CCH, Wairarapa or Hutt Valley GP practice on the 31<sup>st</sup> December 2015 (n=19,397)—data provided by Compass Health PHO and Hutt Valley DHB and Te Awakairangi Health Network).

## Discussion

We report in this study that 77.5% of those referred for the first time for diabetic retinal screening between 2006–2015 in the Wellington region had no DR, which was comparable with that reported in Waikato (78.0%),<sup>16</sup> and lower than Northland (81%).<sup>6</sup> ST-DR was 2.3% in this study for the Wellington region, which was lower than Waikato (3.1%), and higher than Northland (1.8%). However, it should be noted that the definition of ST-DR is different by region, as the Wellington model regarded grade 4–5 retinopathy/maculopathy as sight-threatening, while the Waikato and Northland

study considered grade 3–5 retinopathy/maculopathy as sight-threatening.<sup>6,12,16</sup> With an appropriate primary care workforce and supporting systems in place, the Wellington model referral threshold for ST-DR is safe and an efficient use of limited public hospital secondary care resources.<sup>7</sup>

The findings from this study also identified the characteristics associated with having any degree of DR. Those with type 1 diabetes had the greatest odds, followed by poor and moderate HbA1c control, when compared within their respective variable group. Old-age and Asian ethnicity were also found to have moderately increased odds. The reasons to increased risks for DR may be due to a combination of genetic and environmental factors such as lifestyle and increased co-morbidities, especially in old-age.<sup>17</sup> Our finding was in agreement with evidence from the literature that the prevalence of DR and the prevalence of blindness due to DR is greater in type 1 than type 2 diabetes.<sup>4,18</sup> It is also well established in the literature that the mean HbA1c was the dominant predictor for DR, and that a 10% reduction in HbA1c is associated with 43–45% lower risk in DR progression.<sup>19</sup> Similarly, an increased prevalence of DR in old age (>65 years of age) and Asian ethnicity have been described in the literature.<sup>20,21</sup> Patients with the aforementioned demographic and disease characteristics require the most attention from primary care providers to optimise referral and attendance to screening appointments.

It is also interesting to note that the data presented in Figure 1 showed no ST-DR, 9.6% NST-DR in the <15-years age group, while 90.4% had no DR. The prevalence of NST-DR then increased dramatically in the 15–24-years age group to 25.7%. This data suggests that it may be beneficial to screen those <15 years of age, and by educating these young patients and their parents at an early stage, this dramatic increase in the prevalence of DR may be preventable. The American Diabetes Association recommends annual screening for young-onset diabetes patients beginning 3–5 years after diagnosis of diabetes once the patient is 10 years or older.<sup>22</sup> This reiterated the value of diabetic retinal screening in the adolescent population, which provides us the opportunity for patient education, with the end-goal of minimising DR progression.

The coverage rate (Q4 2015) of diabetic retinal screening for the three DHBs in the greater Wellington region is 88.9%. This compared with 77% in Northland, New Zealand,<sup>6</sup> and 78.7% in the national DR service in Scotland, UK.<sup>23</sup> The high screening rate in the greater Wellington region compares with the study by Frederikson and Jacobs (2008), in which retinal screening was accessed by 92% of diabetic patients. Such sustained high coverage rate is likely due to the differences in the patient journey offered in Wellington compared with Northland and national retinal screening service in Scotland, UK. There are close links between GPs, community optometrists, lead ophthalmologist and Compass Health in Wellington, with a common goal of developing and maintaining accessible retinal screening service. Convenient appointment times and locations are also available in Wellington. Long-established community optometry sites provide continuity of care and make use of existing optometry practice infrastructure. A large amount of time and effort is put into the process between GP referral and retinal screening attendance. There are up to five contact interactions (optometrist to patient up to three times, and GP/nurse to patient up to two times) used as opportunities to find out why DNA is happening, and then provide a solution for that patient (standard operating procedures provided by contracted community optometrists and Compass Health PHO).

Upon referral from GP, the optometrist (receptionist) phones the patient directly to book an appointment; a reminder (text or phone message) is given the day before the appointment. At the first and second DNA occurrence, the optometrist (receptionist) repeats the booking process. At third DNA the patient is returned to the referring GP/nurse, who then contacts patient, and if appropriate, another referral to the optometrist for retinal screening to start the process again. In the Wellington model, patients have the opportunity to view the photographs, have it explained by a clinician, be advised of the outcome, and the opportunity to ask questions during the appointment, rather than having to wait up to four weeks for the results.<sup>24</sup> A retinal screening patient in Northland or Scotland may require multiple appointments to achieve the same outcome as one appointment in Wellington, with a clinical optometrist working within their scope of practice. Northland model is primarily technician-based in mobile clinics, with grading of retinopathy undertaken offsite after the appointment. National retinal screening in Scotland, UK operates with computer-generated referral letters and communications to patients, GPs, optometrists, ophthalmologists and diabetologists,<sup>25</sup> and a specialist software for DR photography. Grading of retinal photographs undergo three levels of grading—Level 1 uses auto-grading by a computer programme that detects microaneurysms, Level 2 graders are optometrists or nurse practitioners and Level 3 graders are medical retina trained ophthalmologists.<sup>24</sup>

The mean DNA rate was 27% for first time referrals, compared to 12.9% in all referrals (this study) and 7% in all referrals reported by Frederikson and Jacobs.<sup>26</sup> This suggests that new referrals may be more likely to miss their screening appointments than those who have had screening services, and warrants further investigation. This indicates the importance of promoting diabetic retinal screening services, especially to newly diagnosed diabetic patients. Māori and PI ethnicity had the highest DNA rate, and therefore, were likely to be under-represented in this study cohort. This is consistent with findings from analysis of the national diabetes annual review that Māori and ethnic minorities

are often under-represented.<sup>27</sup> Such barrier to health care may be attributed to lack of motivation, social, family and transport difficulties.<sup>27</sup> We propose that liaison with GPs and community health care providers may improve patients' understanding of long-term consequences of diabetes, such as preventable blindness due to DR. This could in turn raise patient awareness and the value of diabetic retinal screening, despite the barriers. The main benefit of retinal screening for most participants at first presentation is the opportunity for face-to-face education about their health condition. Diabetes self-management education programmes have been shown to assist behavioural changes,<sup>28</sup> which are significant in limiting the long-term health consequences and economic impact of diabetes. A technician-only retinal screening programme where grading might be performed offsite and after the patient has gone is a lost opportunity for concurrent patient education. In the Wellington model, patients are shown the photos of their own eyes at the time of the screening appointment, and have the opportunity for immediate discussion with an optometrist who can answer their questions about their health condition.

Importantly, this study suggests 97.7% (those with no DR and NST-DR) of first presentation population can be managed in the community by primary care. This is possible because the scopes of practice for health practitioner have changed since the introduction of Health Practitioners Competence Assurance Act 2003,<sup>11</sup> and because of the increased availability of digital imaging technology such as retinal photography and OCT. There are 472 optometrists with

TPA endorsement of 704 optometrists in practice nationwide, and 71% of optometrists in practice are under the age of 50 (data provided by New Zealand Optometrists and Dispensing Opticians Board). There is one ophthalmology clinical nurse specialist (data provided by Nursing Council of New Zealand), of 145 nurse practitioners in practice,<sup>29</sup> and 45% of registered nurses in practice are aged 50 or over.<sup>13</sup> As the New Zealand health workforce is ageing, the challenge is to continually invest time and funds in training new health professionals. The Wellington model makes use of an existing trained primary care ophthalmic workforce and existing fully equipped community optometry sites supported by local general practice and hospital ophthalmology. Thus, the current New Zealand optometric workforce provides a feasible solution to alleviate the volume of diabetic retinal screening that currently strains ophthalmology services in public hospitals.<sup>30</sup>

## Conclusion

The prevalence of ST-DR is low in those referred for diabetic retinal screening for the first time. This reflects a large proportion of patients at first presentation having no DR or NST-DR, who do not require ophthalmology care and are safe to remain in community screening. The optometrist-based diabetic retinal screening service in the greater Wellington region has a high coverage rate and comparable attendance rate with other models, and is well accepted by local clinicians and patients. Patient education and optimisation of attendance, especially those with greater risk profiles will benefit patient outcome.

**Competing interests:**

Lily YL Chang was awarded the New Zealand Association of Optometrists (NZAO) postdoctoral scholarship for manuscript preparation (funded by NZAO and Ministry of Health (MOH)).

Arier C Lee's service of statistical analysis was funded by the NZAO and MOH.

**Acknowledgements:**

We would like to acknowledge Dr Keith Maslin (consultant ophthalmologist) and Dr Lesley Frederikson (national director for NZAO) for their support and advice during manuscript preparation, the New Zealand Optometrists and Dispensing Opticians Board and Nursing Council of New Zealand for providing additional information on New Zealand optometrists and nurses. We are also grateful for Chris Gellen (Compass Health PHO), for assisting with preparation of raw data, calculation of coverage rate and DNA rate, and acknowledge assistance of Chris Kerr and Liz Dutton (Compass Health PHO), Jodi Caughley (Hutt Valley Health), and Diane Taylor (Te Awakairangi Health Network).

**Author information:**

Lily YL Chang, School of Optometry & Vision Science, University of Auckland, Auckland;  
Arier C Lee, Section of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland; Wilson Sue, Bentley & Sue Optometrists, Upper Hutt Wellington.

**Corresponding author:**

Lily YL Chang, School of Optometry & Vision Science, University of Auckland, Grafton, Auckland 1023.

[lily.chang@auckland.ac.nz](mailto:lily.chang@auckland.ac.nz)

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7161>

**REFERENCES:**

- Coppell KJ, et al. Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: findings from the 2008/09 Adult Nutrition Survey. *The New Zealand Medical Journal* (Online), 2013. 126(1370).
- Ministry of Health. Virtual Diabetes Register 2016. Available from: <http://www.health.govt.nz/our-work/diseases-and-conditions/diabetes/about-diabetes/virtual-diabetes-register-vdr>. (Accessed October 2016).
- Ministry of Health. 2007. Diabetes Surveillance: Population-based estimates and projections for New Zealand, 2001–2011: Public Health Intelligence Occasional Bulletin No. 46. Wellington: Ministry of Health.
- Fong DS, et al. Retinopathy in diabetes. *Diabetes care*, 2004. 27(suppl 1): p. s84-s87.
- Klein R, et al. The Wisconsin Epidemiologic Study of Diabetic Retinopathy XXII: the twenty-five-year progression of retinopathy in persons with type 1 diabetes. *Ophthalmology*, 2008. 115(11):p. 1859–1868.
- Papali'i-Curtin AT, Dalziel DM. Prevalence of diabetic retinopathy and maculopathy in Northland, New Zealand: 2011–2012. *The New Zealand Medical Journal* (Online), 2013. 126(1383).
- Avery N, Chan K, Maslin K. Progression of diabetic maculopathy in patients on the Wellington Diabetic Screening Programme initially graded M3. *The New Zealand Medical Journal* (Online), 2013. 126(1372).
- James M, et al. Cost effectiveness analysis of screening for sight threatening diabetic eye disease. *Bmj*, 2000. 320(7250): p. 1627–1631.
- Squirrell DM, Talbot JF. Screening for diabetic retinopathy. *Journal of the Royal Society of Medicine*, 2003. 96(6): p. 273–276.
- Optometrist and Dispensing Opticians Board. Standards of Clinical Competence. 2010: Wellington, New Zealand. p. 1–11.
- New Zealand Government. Health Practitioners Competence Assurance Act 2003. 2003. p. 1–148.
- Ministry of Health. 2016. Diabetic Retinal Screening, Grading, Monitoring and

- Referral Guidance. Wellington: Ministry of Health.
13. Ministry of Health. 2016. New Zealand Health Strategy: Future direction. Wellington: Ministry of Health.
  14. New Zealand Best Practice Advocacy Centre. (2010) Screening for diabetic retinopathy in primary care, 2010. Available from: <http://www.bpac.org.nz/BPJ/2010/August/retinopathy.aspx> (Accessed December 2016).
  15. Hutchinson A, et al. Effectiveness of screening and monitoring tests for diabetic retinopathy—a systematic review. *Diabetic medicine*, 2000. 17(7): p. 495–506.
  16. Reda E, et al. Screening for diabetic retinopathy using the mobile retinal camera: the Waikato experience. *The New Zealand Medical Journal* (Online), 2003. 116(1180).
  17. Bourdel-Marchasson I, et al. Key priorities in managing glucose control in older people with diabetes. *JNHA-The Journal of Nutrition, Health and Aging*, 2009. 13(8): p. 685–691.
  18. Eppens MC, et al. Prevalence of diabetes complications in adolescents with type 2 compared with type 1 diabetes. *Diabetes care*, 2006. 29(6): p. 1300–1306.
  19. Diabetes Control and Complications Trial, The relationship of glycemic exposure (HbA1c) to the risk of development and progression of retinopathy in the diabetes control and complications trial. *Diabetes*, 1995. 44(8): p. 968–983.
  20. Kato S, et al. Retinopathy in older patients with diabetes mellitus. *Diabetes research and clinical practice*, 2002. 58(3): p. 187–192.
  21. Raymond NT, et al. Higher prevalence of retinopathy in diabetic patients of South Asian ethnicity compared with White Europeans in the Community A cross-sectional study. *Diabetes care*, 2009. 32(3): p. 410–415.
  22. Lueder GT, J Silverstein. Screening for retinopathy in the pediatric patient with type 1 diabetes mellitus. *Pediatrics*, 2005. 116(1): p. 270–273.
  23. National Health Service, Scotland. Scottish Diabetic Retinopathy Screening Collaborative Annual Report 2014. 2014: Edinburgh, Scotland p. 1–49.
  24. Zachariah S, Wykes W, Yorston D. The Scottish Diabetic Retinopathy Screening programme. *Community Eye Health*, 2015. 28(92): p. s22.
  25. Diabetic Retinopathy Screening Implementation Group, Scottish Government. Diabetic retinopathy screening services in Scotland: recommendations for implementation, 2003. Available from: <http://www.gov.scot/Publications/2003/07/17638/23077> (Accessed December 2016).
  26. Frederikson LG, Jacobs RJ. Diabetes eye screening in the Wellington region of New Zealand: characteristics of the enrolled population (2002–2005). *The New Zealand Medical Journal* (Online), 2008. 121(1270).
  27. Porter T, Le Lièvre C, Lawrenson R. Why don't patients with diagnosed diabetes attend a free 'Get Checked' annual review? *Journal of primary health care*, 2009. 1(3): p. 222–225.
  28. New Zealand Guidelines Group. 2012. Effective health behaviour change in long-term conditions: A review of New Zealand and international evidence. Wellington: Ministry of Health. / New Zealand Guidelines Group. 2011. RapidE: Chronic Care: Health Literacy Interventions – A brief summary. Wellington: Ministry of Health.
  29. Nursing Council of New Zealand., Annual Report 2015. 2015: Wellington, New Zealand p. 1–66.
  30. McDonald EM, Ram FS. Seeing into the future: ophthalmologists and specialist nurses working together. *The New Zealand medical journal*, 2016. 129(1438): p. 12.

# Discharge outcomes of patients referred to specialist eye clinic from diabetic retinopathy screening in Northland (2014–15)

Pragnya Jagadish, David Dalziel

## ABSTRACT

**AIMS:** To determine the discharge outcomes of patients seen in specialist eye clinic after referral from diabetic retinopathy screening (DRS).

**METHODS:** Retrospective analysis of outcomes of a sample of 98 patients referred from DRS to specialist eye clinic.

**RESULTS:** A sample of 98 patients were analysed following referral by DRS to specialist eye clinic from 16/4/14 to 16/4/15. Age at screening ranged from 13–88 years, with the main ethnic groups being Māori (57.1%), European (39.79%) and Indian (3.06%). A majority of the patients were referred to specialist eye clinic for diabetic retinopathy (60%) or cataracts (35%). After being seen in specialist eye clinic, 45% of the patients were enrolled back into DRS and 49.1% stayed under care of ophthalmology service for further treatment, and a further 5.9% were discharged to care of GP or optometrist without re-enrolment back to DRS. Of those referred back to DRS, 30% were re-enrolled after further imaging with optical coherence tomography (OCT), and 24% of patients were referred back to DRS due to non-attendance. Non-attendance at clinic appointments was high among the Māori population.

**CONCLUSION:** Our study identified that 94% of patients referred to specialist eye clinic were either referred back to DRS or kept under care with only five patients not re-enrolled back into DRS. Despite good service delivery, Northland remains a high-risk population for diabetes, where non-attendance at clinic appointments remained an issue with the Māori patient population. In addition, a significant proportion of patients were re-referred back to DRS after OCT, and a consideration is to include OCT in the screening pathway.

Diabetes affects more than 200,000 New Zealanders, and is one of the fastest growing health epidemics in the country.<sup>1</sup> Diabetic retinopathy (DR) is a serious complication of diabetes, and is the leading cause of preventable blindness and vision impairment in New Zealand.

The aim of retinal screening programs is to detect diabetic retinopathy so that timely interventions can be made. Screening is undertaken by the district health boards (DHBs), and referrals are made to specialist eye clinic/s in accordance with the National Diabetes Retinal Screening Grading System and Referral Guidelines.<sup>2,7</sup>

In 2014, there were a total of 257,776 patients diagnosed with diabetes in New Zealand, of which 11,000 patients were from the Northland district.<sup>1</sup> Northland consists of 3.6% of the New Zealand population, and has a total of 151,692 people residing in an area which spans 13,789 km.<sup>3</sup> The main ethnic groups are European (75.7), Māori (32.4%), Pacific islander (3.2%) and Asian (2.8%).<sup>3</sup>

Northland also has a high level of deprivation with large inequalities in health care outcomes. Approximately 30% of people with diabetes have DR, with 10% presenting with sight-threatening retinopathy.<sup>2</sup> In Northland, the rate of avoidable hospitalisation from

diabetic complications is nearly twice the national average, and the mortality rates for diabetes-related conditions is up to eight times higher for the Māori population.<sup>4</sup> In 2006, a strategy called STAND (Successfully Taking Action for Northland Diabetes) was undertaken by the Northland DHB with the aim to prevent diabetes through advocacy of lifestyle changes, early detection and intervention in order to reduce the risks of diabetic complications.<sup>4</sup>

The DRS in Northland was launched in 1994 and includes 22 screening sites. It comprises a mobile retinal screening van, which is attended by a qualified medical photographer.<sup>5</sup> Mobile retinal screening takes place in a range of settings—hospital outpatient clinics, medical centres and community centres, and covers areas from Te Kao, south of Cape Reinga to Mangawhai near Northland's southern border. Patients from DRS were referred to specialist eye clinic in Whangarei in accordance with the National Diabetes Retinal Screening Grading System and Referral Guidelines 2006.<sup>2</sup>

This study aims to examine the outcomes of a sample of 98 patients who were referred from DRS in Northland and seen in specialist eye clinic between April 2014–April 2015. It also aims to examine whether patients are re-enrolled back into DRS after being reviewed in the specialist eye clinic.

## Methods

This study was a retrospective audit of a sample of 98 patients referred from DRS to specialist eye clinic at Whangarei hospital over a 12-month period. Since this was a retrospective audit, we did not require ethics approval.

**Research design**—The study accessed data from the *Optimise* database, which stores the diabetic retinal screening photographs. This database recorded demographics, disease data and digital retinal photographs. Screening data of 98 patients consecutively referred to eye clinic was acquired from April 2014 to April 2015, and retrospectively assessed.

**Screening centres**—Screening took place in 22 clinics, with a mobile screening van attending outpatient clinics, GP surgeries and community centres in Northland.<sup>5</sup> After

pre-assessment and mydriatic administration, a Zeiss ProNM camera was used to take three digital photographs, each with a 45 degree field of view, in accordance with National Guidelines.<sup>2</sup>

**Grading criteria** was applied to patients in accordance with the National Diabetes Retinal Screening grading system.<sup>2</sup> All patients with M3 R4 or other significant pathology, including cataracts, were referred to specialist eye clinic. The primary grader was a qualified medical photographer, and the secondary grader was the designated ophthalmologist at Whangarei Base Hospital. Each of the retinal photographs were screened into low, medium and high urgency (marked green, orange and red respectively) by the primary grader, and referred to the secondary grader.

Low urgency (marked green) included mostly patients who required OCT assessment; medium urgency (marked orange) included patients with significant maculopathy or impaired fundal view from cataracts; and high urgency (marked red) included patients with evidence of proliferative DR or vitreous haemorrhage. The secondary grader assessed the referrals from the primary grader, and patient appointments were made according to National Guideline Screening Intervals. Patients referred with cataracts did not meet requirements for referral reporting, and hence screening was unable to be completed.

To address the issue of concordance between graders, 10% of the primary grader's images were reviewed by the secondary grader in a random fashion, and a high concordance rate was reported.<sup>5</sup>

## Results

### Patient demographics

**Diagnosis on referral**—A majority of the patients (60.7%) were referred for Diabetic retinopathy (non-proliferative and proliferative with complications eg vitreous haemorrhage), followed by (35.7%) for cataract or complication from cataract (eg YAG capsulotomy). There were singular cases of exotropia (1.1%), intracranial hypertension (1.1%) and keratoconus (1.1%) referred by DRS to eye clinic.

**Table 1:**

Parameter	Study population n=98
Gender, n (%)	
Male	45, (46%)
Female	52, (54%)
Ethnicity	
Māori	56, ( 57.1%)
Caucasian	39, (39.79%)
Indian	3, (3.06%)
Age (mean, range)	Mean 61, (13–88)

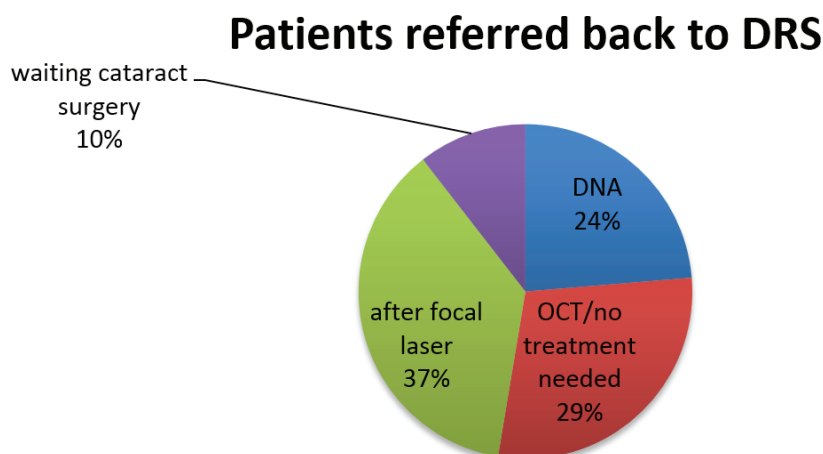
**Outcomes of patients referred to specialist eye clinic**—Around 45% of the patients were enrolled back into DRS, and 49.1% stayed under care of ophthalmology service for further treatment. A small proportion (5.9%) were discharged to care of GP or optometrist.

**Reasons why patients were referred back to DRS**—Around 37% of patients were referred back to DRS after retinal laser, 29% had further imaging, eg optical coherence tomography (OCT), and returned to DRS since no active treatment was necessary, and a further 10% were referred back while awaiting cataract surgery. Around 24% of patients were referred back to DRS due to non-attendance (DNA). Ninety percent of DNA patients were of Māori ethnicity. All DNA patients had a recall notice sent.

**Outcomes of patients not re-enrolled into DRS**—Five patients were not re-enrolled back into DRS from specialist eye clinic. Four of five were discharged to GP, out of these, three were discharged following non-attendance after more than two recall appointments. One patient was discharged to GP to optimise HbA1C pre-cataract surgery, and one was referred to optometrist post-cataract surgery.

## Discussion

The Northland population remains a high-risk population for diabetes, with 11,000 patients diagnosed with the condition over the last year.<sup>1</sup> Diabetic retinopathy is an avoidable complication of diabetes, and can be reliably detected by regular retinal screening. It is known that between

**Figure 1:** Reasons why patients were referred back to DRS.

6% and 39% of people with type 2 diabetes have retinopathy at diagnosis, with 4% to 8% having sight-threatening disease.<sup>4</sup> In the next 20 years, it is projected that the prevalence of diabetes in New Zealand will, if left unchecked, increase by 90% in Māori, 109% in Pacific peoples and 39% in Europeans.<sup>1</sup> Prevalence of retinopathy is higher in Northland (20% total across all ethnicities) than other district health boards (Waikato 9–10%, Lower Hutt 11–12%).<sup>4</sup> Northland also has a higher proportion of Māori (7.7 percent of New Zealand's Māori population), with 43,527 people residing in Northland Region.<sup>4</sup> The STAND (Successfully Taking Action for Northland Diabetes) was a program taken up in 2006 by the Northland DHB, which aims to prevent diabetes through lifestyle advocacy, address inequalities in healthcare and reduce the impact on quality-of-life from diabetic complications.<sup>4</sup>

This study aims to examine the outcomes of patients referred to specialist eye clinic from DRS. Of a total of 98 patients who were referred to DRS, 46% were male and 54% female. A majority of the population referred were of Māori ethnicity, (57.1%) despite comprising only 30% of the total Northland population. Of the patients seen in the eye clinic, around 49.1% stayed under care of ophthalmology service for further treatment and 45% of the patients were enrolled back into DRS after assessment. Around 30% were re-enrolled after optical coherence tomography (OCT) and 24% of patients were referred back to DRS due to non-attendance. There were only five patients not referred back to DRS, and three of these were discharged to GP after multiple non-attendances. Interestingly, around 90% of the patients who did not attend were of Māori ethnicity, which remains a challenge in addressing the health inequalities in the region.

One of the goals of the STAND strategy is to improve the health among Māori populations and continually identify and address barriers to people accessing programmes and services in Northland. A majority of the referrals to specialists were of Māori origin (57.1%), which correlates with existing data that Māori are three times as likely to have type 2 diabetes as non-Māori, and are more likely to develop complications.<sup>4</sup> However, the rate of non-attendance is high among the

Māori population, which resulted in three patients discharged to care of GP rather than DRS. The STAND strategy investigated the barriers to access in Māori populations for attendance at diabetic retinal screening through patient satisfaction survey and addressed issues with measures, such as flexibility in changing appointments, new referral forms and having a telephone confirmation of attendance prior to appointment.<sup>4</sup> To address the non-attendance rates at specialist eye clinic from DRS referral, further research is being undertaken through patient surveys and focus groups on what can be done to improve the experience of referral and attendance for patients.

In those referred back to DRS, around 30% were re-enrolled after imaging of the fundus and retina with OCT. The OCT is the gold standard for evaluating the layered structure of the retina, and is useful in determining macular oedema or intraretinal fluid. Despite investment in new retinal camera, which affords good views without pupil dilation, some of the referrals to the eye clinic were due to poor views from camera and requirement of further imaging. The Ophthalmic Photographic Diabetic Review (OPDR) in the UK comprehensively combines digital photography and OCT for patients who require more frequent reviews than the usual annual screening service.<sup>6</sup> In centres such as the Birmingham Diabetes Centre, the OCT has already been incorporated into the DRS service. The use of OCT helps monitoring for the high-risk patient population, eg pregnant diabetic women, patients with early maculopathy virtually and avoids the burden of specialist appointments in hospitals.<sup>7</sup> Similarly in New Zealand, the Wellington region has trialled incorporating optometry services into DRS pathway for patients. It targets for moderate non-proliferative DR and quiescent (previously treated) proliferative DR. This involves monitoring by optometrist with OCT in either the primary or secondary health care setting.<sup>11</sup> Although the cost of having an OCT in DRS needs to be accounted for, it is a worthy measure to consider, given that 30% of patients were referred back after OCT in the specialist eye clinic.

The study supports the efficiency of the current process of referrals and re-referrals

back to DRS. Out of 84 patients, only five patients were not re-enrolled back to DRS. Of these, three were due to non-attendance, and were discharged to care of GP. One patient was discharged to GP to optimise blood sugar levels prior to cataract surgery, and one was discharged to the optometrist post-cataract operation. Although it is likely all five patients would be referred back to DRS from the GP or optometrist, it flags the need to improve the identification of patients who are referrals from DRS. Currently, the referral letters from DRS are not linked into *Concerto*, which is a program used for letter dictations, and hence there is no easy identification of patients as being from DRS. Suggestions to improve identification include alerts on *Concerto*, which flags that the patient is from DRS, and creation of links between applications, eg *Optimise* and *Concerto*, in a way that data from DRS is easily accessible to clinicians, GPs and optometrists who are involved in patient care.

Recently, the National Diabetes Retinal Screening grading system and Referral Guidelines were updated in March 2016.<sup>7</sup>

The new guidelines involve measures to monitor DR by revising the screening interval to three-yearly for those without clinical modifiers, and involves monitoring by optometrist with an ophthalmologist who oversees the region's program. However, in Northland, we have not introduced the increased recall periods due to high incidence of retinopathy in the high-risk population with a significant DNA rate. Improvement of the service should involve collaboration with GPs and optometrists in order to streamline a process of referral and communication for patients seen in the specialist eye clinic.

In conclusion, Northland remains a high-risk population for diabetes, and measures to identify and prevent complications from diabetes that have been initiated with the STAND strategy. Our study identified that a majority of patients referred to eye clinic were referred back to DRS after being seen at specialist eye clinic. A large proportion of our referrals from DRS were of Māori ethnicity and non-attendance at clinic appointments remained an issue in this patient population.

---

#### Competing interests:

Nil.

#### Author information:

Pragnya Jagadish, Whangarei Hospital, Whangarei; David Dalziel, Ophthalmology Department, Whangarei Hospital, Whangarei.

#### Corresponding author:

Dr Pragnya Jagadish, Whangarei Hospital, Maunu Rd, Maunu, Whangarei 0110.  
pjag141@gmail.com

#### URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7162>

---

#### REFERENCES:

1. Ministry of Health. Estimated diagnosed cases of diabetes by DHB as at December 2014. Wellington: Ministry of Health 2015.
2. Ministry of Health. National Diabetes Retinal Screening, Grading System and Referral Guidelines 2006. Wellington: Ministry of Health 2006.
3. Statistics New Zealand. 2013 QuickStats about a place: Northland Region [Internet]. Wellington: Statistics New Zealand; 2013 [cited 15 August 2016]. Available from: [http://www.stats.govt.nz/Census/2013-census/profile-and-summary-reports/quickstats-about-a-place.aspx?request\\_value=13068&tab-name](http://www.stats.govt.nz/Census/2013-census/profile-and-summary-reports/quickstats-about-a-place.aspx?request_value=13068&tab-name)
4. Northland District Health Board (NDHB). The Northland Diabetes Strategy Implementation plan 2006 - STAND. Northland: NDHB, 2006.
5. Papali'i-Curtin A, Dalziel D. Prevalence of diabetic retinopathy and maculopathy in Northland, New Zealand: 2011-2012. *N Z Med J.* 2013; 126:20-8.
6. Dodson PM, Quant L, Leigh R, Wharton H. Ophthalmic Photographic Diabetic Review (OPDR): a virtual clinic approach for management of referable diabetic maculopathy. Poster 2015.
7. Ministry of Health. National Diabetes Retinal Screening, Grading System and Referral Guidelines 2016. Wellington: Ministry of Health 2016.

# Proposed new industry code on unhealthy food marketing to children and young people: will it make a difference?

Boyd Swinburn, Stefanie Vandevijvere on behalf of submitting health professors\*

## ABSTRACT

Reducing the exposure of children and young people to the marketing of unhealthy foods is a core strategy for reducing the high overweight and obesity prevalence in this population. The Advertising Standards Authority (ASA) has recently reviewed its self-regulatory codes and proposed a revised single code on advertising to children. This article evaluates the proposed code against eight criteria for an effective code, which were included in a submission to the ASA review process from over 70 New Zealand health professors. The evaluation found that the proposed code largely represents no change or uncertain change from the existing codes, and cannot be expected to provide substantial protection for children and young people from the marketing of unhealthy foods. Government regulations will be needed to achieve this important outcome.

## Viewpoint

New Zealand has an unacceptably high prevalence of childhood obesity,<sup>1</sup> and the Minister of Health, Dr Jonathan Coleman, has made it one of his priorities to reduce this rate through the government's childhood obesity plan.<sup>2</sup> Achieving this goal will require a significant reduction in the marketing of unhealthy foods and beverages to children and young people.

The WHO Commission on Ending Childhood Obesity, which is co-chaired by the Prime Minister's Chief Science Advisor, Professor Sir Peter Gluckman, had its report<sup>3</sup> endorsed by all Member States, including New Zealand, at the World Health Assembly in May 2016. This report supports a strong regulatory approach to reduce unhealthy food marketing to children, as do New Zealand public health experts.<sup>4,5</sup>

In 2015, the Advertising Standards Authority (ASA), the industry body responsible for the self-regulatory codes on advertising, pre-empted the launch of the

government's 22-point plan<sup>2</sup> by announcing a review of the two ASA codes which relate to advertising to children. In September 2016, the ASA's independent Review Panel submitted its report to the ASA Board, recommending including the amended code provisions to be included into a new single, combined ASA code.<sup>6</sup> Dr Coleman commented that the revised ASA code would make significant improvements to protect children and young people from exposure to the marketing of unhealthy food products.<sup>7</sup> This analysis critically assesses whether the revised ASA code will achieve that outcome.

The ASA Review Panel received 91 submissions, including 52 from public health and nutrition organisations that called for substantially strengthened codes, and 15 from the food and beverage industry sector, which largely opposed stronger codes.<sup>8</sup> For example, the Food and Grocery Council called for a lowering of the age to which the ASA code would apply from 14 years to 12 years.<sup>9</sup> Six weeks after the release of the Review Panel report, ASA asked submitters

to comment once again on two issues: the nutrient profiling system to use (see point 6 below) and the definition of targeting children (see point 7 below).

Although there is no evidence that industry-controlled, voluntary codes are effective in reducing marketing to children,<sup>10</sup> public health groups actively participated in the ASA review in the hope of strong outcomes. A group of over 70 health professors provided a submission with eight outcomes (in bold below) that they would use to assess the strength of the revised code.<sup>11</sup> How well does the proposed new code meet these outcomes?

1. **The UN Convention on the Rights of the Child (UNCROC) is expressed throughout the code.** As in the existing ASA codes, UNCROC is recognised in the introduction of the proposed combined code. However, the rules of the proposed code do not take a rights-based approach, which would give primacy to children's interests. Instead, they are a compromise between protecting the interests of children and protecting the interests of the private sector, which advertises unhealthy foods to children. Assessment: no change, outcome not achieved.
2. **The definition of children aligns with UNCROC (under 18 years).** Despite widespread agreement in the submissions from public-interest groups that a child should be defined as under 18 years, as per UNCROC, the review recommended no change from the current age of 14 years. For children aged 14–18 years, the review suggested that 'a special duty of care be taken', but did not further define what such a duty would entail. As a result, none of the new provisions in the proposed ASA code apply to adolescents 14–18 years. Assessment: no change, outcome not achieved.
3. **The objectives of the code are to reduce the exposure and power of unhealthy food marketing to children.** While the purpose of the ASA review stated that 'the best interests of children are a primary consideration', the code remains focused on the appropriateness of single advertisements rather than the overall marketing to which children are exposed. The revised code, thus, fails to address the crux of the public health concern. Although the code mentions restrictions on the use of celebrities and licensed characters popular among children, it is unclear how these restrictions will be operationalised, as commercial sponsorship and advertising on the packaging have been excluded from the proposed code. Assessment: small changes, outcome not achieved.
4. **Recommendations are made to government to significantly invest in achieving reductions in marketing to children.** Many submissions to the ASA review called for government involvement in co-regulatory arrangements because of the failure of self-regulatory approaches in New Zealand and internationally to significantly reduce unhealthy food marketing to children. The Review Panel ruled that it was out of scope for it to make any recommendations to government. Instead, they used soft language to suggest that 'advertisers be encouraged to discuss' unresolved issues such as sponsorship, advertising on packages and an appropriate nutrient profiling system with government. Despite a large number of submissions calling for independent monitoring, the Review Panel recommended that the ASA be responsible for monitoring the code. Assessment: no change, outcome is not achieved.
5. **The code covers all forms of marketing.** As in the existing ASA codes, the term advertising is defined very broadly in the proposed code, but sponsorship and advertising on product packaging are exempted. These are major forms of marketing unhealthy foods to children. The protection from the forms of advertising which are covered within the code is highly dependent on the definition of targeting children (see point 7 below). Assessment: no change, outcome is not achieved.

- 6. The code defines unhealthy foods using a robust nutrient profiling (NP) system (WHO NP system recommended).** WHO is the world's most important standard-setting organisation for health. The WHO Europe NP system has been specifically designed for marketing to children, and was the most highly recommended NP system by public health submitters.<sup>12,13</sup> The Review Panel received specific testimony from world experts who recommended the WHO Europe NP system and provided the Panel with comprehensive comparisons of various NP systems using New Zealand data.<sup>12</sup> The Panel said it could not decide on this point and recommended the Ministry of Health food and beverage classification system<sup>14</sup> be used in the interim. The Ministry's NP system has three levels (everyday, sometimes and occasional foods), and was designed for food sold in schools so would require modification to ensure suitability for this purpose. By contrast, the WHO NP system was designed specifically to limit marketing of unhealthy food to children, and has two levels (acceptable or not). **Assessment:** improvement since previous code had included no nutrient profiling system, outcome partially achieved, but pending the further round of consultations.
- 7. The code clearly defines 'marketing to children' (Quebec model recommended).** The Review Panel recommended that 'targeting' means the advertisement has 'principal appeal to children and/or young people' with the nature of the product, theme, language and images 'taken into account'. The operationalisation of these definitions is unclear. The Panel also recommended that an advertisement is 'deemed to be targeting children or young people if they are likely to comprise 25% or more of the likely audience' (although children aged 5–14 years comprise only about 20% of the total population). This figure of 25% appears to have been chosen arbitrarily. The Review Panel had not evaluated any children audience data for the various media when it made this quantitative recommendation, and it is unclear how this rule will be operationalised. Following further inquiries on these issues to the ASA by the authors, the ASA has requested further submissions on this point. In addition, because adult viewership is in the denominator, having more adults in the audience will reduce the *percentage* of children watching but not the *total number* of children watching. The highest number of children exposed to unhealthy food advertising is during evening television (figure), which is also when the frequency of unhealthy food advertisements peaks. Indeed, in peak viewing times, 6–7pm, more than 120,000 5–13 year olds are exposed to over 15 unhealthy food advertisements an hour, creating over 2 million 'impacts' (ad impressions x viewers) per hour. Using children's viewership is a more logical and robust way to define marketing to children (see markers A and B in the figure) than including adult viewership in the metric. In Quebec, the Consumer Protection Act defines advertising as targeting children based on a) the nature and intended purpose of the goods advertised; b) the manner of presenting such advertisement; c) the time and place it is shown.<sup>15</sup> The Office of Consumer Protection has decades of experience in implementing these definitions, which are now tightly defined through multiple case judgments. The table addresses the third of these criteria for various media as they might apply in New Zealand. **Assessment:** great uncertainty in the operationalisation of the proposed definition, outcome uncertain.
- 8. The code covers marketing in child and youth settings.** The proposed ASA code states that 'Settings where children gather must be free from all forms of occasional food and beverage product advertisements'. This is an improvement in wording

**Table 1:** Suggested criteria, by media type, for determining if the timing and placement of marketing of unhealthy foods is likely to result in significant exposure to children.

Medium	Suggested criteria for excluding marketing of unhealthy foods	Comments
Television	<ol style="list-style-type: none"> <li>1. During specified children’s programming hours</li> <li>2. When a certain percentage (eg 10% or 15% see figure) of the childhood population is viewing (all channels combined).</li> </ol>	The current restrictions on marketing unhealthy foods during specified children’s programs should remain (mainly children watching but in low numbers and almost free from unhealthy food advertising). The highest children’s viewership is in the evening (mainly adults watching but also high numbers of children), and this is when the most numbers of ads for unhealthy foods are broadcast (Figure 1).
Internet	Food and beverage company websites should not have sections which market to children.	Food company websites often feature engaging techniques targeting children in branded marketing games and activities (18). Food advertising on non-food websites popular with children is very low, and is therefore not currently important.
Social media	Since marketing through social media is highly tailored to the individual, a total ban on marketing unhealthy foods to children below 14 years of age is feasible. The monitoring of compliance will, however, be more challenging than for the other media.	Children spend significant time on social media (YouTube, SnapChat, Facebook etc), and WHO has just called for urgent action on this matter in a new report. <sup>19</sup>
Magazines	Magazines which specifically cater to children and young people should not include marketing of unhealthy foods to children (including in advertorials).	Magazines specifically targeted to children and adolescents (such as Dolly, Crème and Girl-friend) contain a higher proportion of unhealthy food advertising compared to the women’s magazines which were also popular among 10–17 year olds. <sup>20</sup>
Outdoor advertising	A zone of 500m around a school is designated as free from advertising for unhealthy foods.	Children’s settings need to be expanded to cover the near school zone. <sup>21</sup>
Children’s settings	Children’s settings including early childhood settings, schools and junior sports should be free from marketing of unhealthy foods, including through sponsorships.	Disallowing marketing of unhealthy foods in children’s settings is covered in the revised ASA code, but it needs to be expanded to include sponsorship, sports settings and proximal school zones.
Super-markets and other retailers	Marketing for unhealthy foods should not specifically target children (eg in check-out aisles).	The retail sector has pledged to adhere to the proposed new code, <sup>22</sup> but how it applies in supermarkets has not been specified.

Other important criteria of the nature of the product and the manner of its presentation are not considered here.

over existing codes. However, more than 95% of schools report that they are free of food advertising (unpublished data). Sponsorship activities are the main form of marketing in children's settings, yet are exempted under the proposed code. Thus, Ronald McDonald teaching road safety to six-year-olds is not considered by the proposed code to be marketing to children. In addition, the full extent of settings included is not clear. The actual impact of this improved wording is likely to be small.

Assessment: improvement in wording, outcome uncertain.

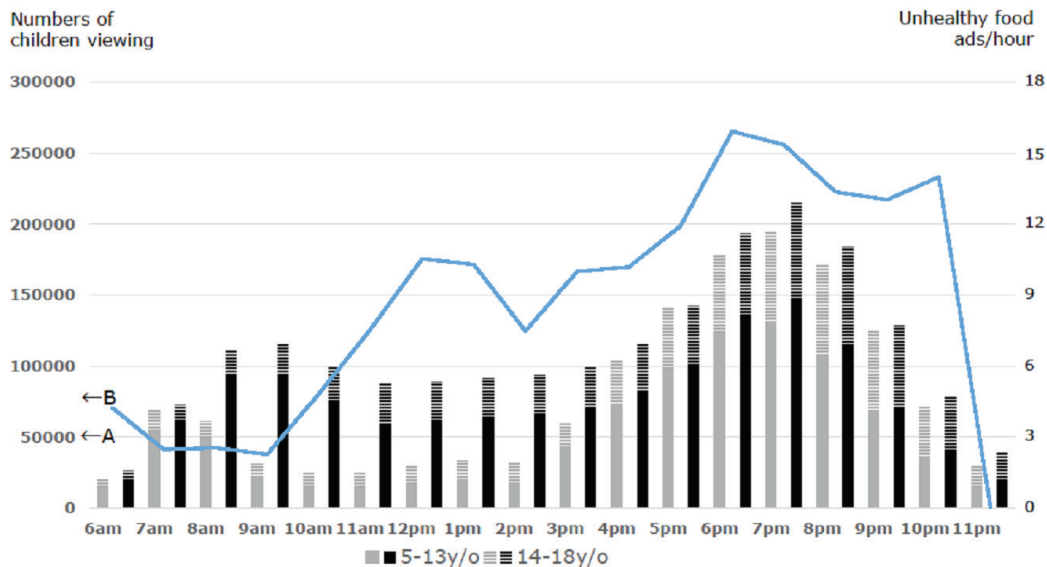
From our assessment, the revised ASA code cannot be expected to have a significant effect on restricting unhealthy food marketing to children and young people, and there is no indication that independent monitoring will be implemented to assess the code's effects. The code reflects problems endemic to self-regulation where

commercial interests conflict with public interests,<sup>10,16</sup> and falls far below current international best practice.<sup>17</sup>

In summary, we recognise the ASA's initiative in instigating a review of its codes, but we consider that, while the proposed code appears to be a small step in the right direction, it does not provide adequate protection of children and young people's interests. A further potentially serious downside is that the revised ASA code will be given as a reason by the Government for not implementing the regulations that would effectively reduce children and young people's exposure to marketing of unhealthy foods.

In our view, the Review Panel has missed a major opportunity to introduce meaningful changes that would help to reduce childhood obesity. Government regulation is urgently needed to create a policy framework which privileges children's health and well-being above commercial interests.

**Figure 1:** Numbers of children aged 5–13 years (solid bars) and 14–18 years (striped bars) in weekdays (grey bars) and weekend days (black bars) watching television (all channels combined).



A and B mark 10% and 15% respectively of the total children aged 5–13 years in New Zealand. The line represents the number of unhealthy advertisements per hour (weighted average of weekdays and weekends) as defined by WHO criteria.<sup>13</sup>

**Competing interests:**

Nil.

**Author information:**

Boyd Swinburn, Epidemiology & Biostatistics, University of Auckland, Auckland; Stefanie Vandevijvere, Morrin Road, Auckland.

\*Alistair Woodward, School of Population Health, University of Auckland; Andrew Hornblow, School of Medicine, University of Otago, Christchurch; Ann Richardson, Wayne Francis Cancer Epidemiology Research Group, University of Canterbury; Barbara Burlingame, School of Public Health, Massey University, Wellington; Barry Borman, Centre for Public Health Research, Massey University, Wellington; Barry Taylor, School of Medicine, University of Otago, Dunedin; Bernhard Breier, School of Food and Nutrition, Massey University, Albany; Bruce Arroll, School of Population Health, University of Auckland; Bernadette Drummond, Department of Oral Sciences, University of Otago, Dunedin; Cameron Grant, Department of Paediatrics, University of Auckland; Chris Bullen, National Institute for Health Innovation, University of Auckland; Clare Wall, School of Medical Sciences, University of Auckland; Cliona Ni Mhurchu, National Institute for Health Innovation, University of Auckland; David Cameron-Smith, Liggins Institute, The University of Auckland; David Menkes, Waikato Clinical Campus, University of Auckland; David Murdoch, Department of Pathology, University of Otago, Christchurch; Dee Mangin, Department of General Practice, University of Otago, Christchurch; Diana Lennon, School of Medicine, University of Auckland; Diana Sarfati, Department of Public Health, University of Otago, Wellington; Doug Sellman, Department of Psychological Medicine, University of Otago, Christchurch; Elaine Rush, School of Sport and Recreation, Auckland University of Technology; Faafetai Sopoaga, Department of Preventive and Social Medicine, University of Otago, Dunedin; George Thomson, Department of Public Health, University of Otago, Wellington; Gerry Devlin, Department of Medicine, University of Auckland, Waikato; Gillian Abel, Department of Population Health, University of Otago, Christchurch; Harvey White, School of Medicine, University of Auckland; Jane Coad, Massey Institute of Food Science and Technology, Massey University, Palmerston North; Janet Hoek, Department of Marketing, University of Otago, Dunedin; Jennie Connor, Department of Preventive and Social Medicine, University of Otago, Dunedin; Jeremy Krebs, Edgar Diabetes and Obesity Research Centre, University of Otago, Wellington; Jeroen Douwes, Centre for Public Health Research, Massey University, Wellington; Jim Mann, Edgar Diabetes and Obesity Research Centre, University of Otago, Dunedin; John McCall, Department of Surgical Sciences, University of Otago, Dunedin; John Broughton, Department of Oral Diagnostic and Surgical Sciences, University of Otago, Dunedin; John D Potter, Centre for Public Health Research, Massey University, Wellington; Les Toop, Department of General Practice, University of Otago, Dunedin; Lesley McCowan, School of Medicine, University of Auckland; Louise Signal, Department of Public Health, University of Otago, Wellington; Lutz Beckert, Department of Medicine, University of Otago, Christchurch; Mark Elwood, School of Population Health, University of Auckland; Marlena Kruger, School of Food and Nutrition, Massey University, Palmerston North; Mauro Farella, Department of Oral Sciences, University of Otago, Dunedin; Michael Baker, Department of Public Health, University of Otago, Wellington; Michael Keall, Department of Public Health, University of Otago, Wellington; Murray Skeaff, Department of Human Nutrition, University of Otago, Dunedin; Murray Thomson, Sir John Walsh Research Institute, University of Otago, Dunedin; Nick Wilson, Department of Public Health, University of Otago, Wellington; Nicholas Chandler, Department of Oral Rehabilitation, University of Otago, Dunedin; Papaarangi Reid, Department of Māori Health, University of Auckland; Patricia Priest, School of Medicine, University of Otago, Dunedin; Paul Brunton, Department of Oral Rehabilitation University of Otago, Dunedin; Peter Crampton, Division of Health Sciences, University of Otago, Dunedin; Peter Davis, COMPASS Research Centre, University of Auckland; Philip Gendall, Department of Marketing, University of Otago, Dunedin; Philippa Howden-Chapman, Department of Public Health, University of Otago, Wellington; Rachael Taylor, Edgar Diabetes and Obesity Research Centre, University of Otago, Dunedin;

Richard Edwards, Department of Public Health, University of Otago, Wellington; Robert Beaglehole, School of Population Health, University of Auckland; Robert Doughty, School of Medicine, University of Auckland; Robert Scragg, School of Population Health, University of Auckland; Robin Gauld, Otago Business School, University of Otago, Dunedin; Robert McGee, Department of Preventive and Social Medicine, University of Otago, Dunedin; Rod Jackson, School of Population Health, University of Auckland; Roger Hughes, School of Public Health, Massey University, Wellington; Roger Mulder, Department of Psychological Medicine, University of Otago, Christchurch; Ruth Bonita, School of Population Health, University of Auckland; Rozanne Kruger, School of Food and Nutrition, Massey University, Albany; Sally Casswell, SHORE and Whariki Research Centre, Massey University, Auckland; Sarah Derrett, Department of Preventive and Social Medicine, University of Otago, Dunedin; Shanthi Ameratunga, School of Population Health, University of Auckland; Simon Denny, School of Population Health, University of Auckland; Simon Hales, Department of Public Health, University of Otago, Wellington; Sue Pullon, Department of Primary Health Care and General Practice, University of Otago, Wellington; Susan Wells, School of Population Health, University of Auckland; Tim Cundy, School of Medicine, University of Auckland; Tony Blakely, Department of Public Health, University of Otago, Wellington.

**Corresponding author:**

Boyd Swinburn, Epidemiology & Biostatistics, University of Auckland, 261 Morrin Road, Auckland 1072.

boyd.swinburn@auckland.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7163>

**REFERENCES:**

1. Ministry of Health. Annual Update of Key Results 2014/15: New Zealand Healthy Survey. Wellington: Ministry of Health; 2015.
2. Ministry of Health. Childhood obesity plan Wellington: Ministry of Health; 2016. Available from: <http://www.health.govt.nz/our-work/diseases-and-conditions/obesity/childhood-obesity-plan>
3. World Health Organisation. Report of the Commission on Ending Childhood Obesity. Geneva: World Health Organisation; 2016.
4. Vandevijvere S, Swinburn B. Time to move beyond industry self-regulation of food marketing in New Zealand. *N Z Med J.* 2015; 128(1423):93–4.
5. Vandevijvere S, Dominick C, Devi A, Swinburn B. The healthy food environment policy index: findings of an expert panel in New Zealand. *Bull World Health Organ.* 2015; 93(5):294–302.
6. Advertising Standards Authority. Report on the review of the Children's Code for Advertising Food and the Code for Advertising to Children, 2016. Available from: <http://www.asa.co.nz/wp-content/uploads/2016/10/Final-Report-Childrens-Codes-Review.pdf>
7. Johnston M. New restrictions proposed on advertising food to children, Health Minister says. *NZ Herald.* 2016, 20 October 2016. Available from: [http://www.nzherald.co.nz/health/news/article.cfm?c\\_id=204&objectid=11732810](http://www.nzherald.co.nz/health/news/article.cfm?c_id=204&objectid=11732810)
8. Advertising Standards Authority. Submissions Received for the Review of the Children's Code for Advertising Food and the Code for Advertising to Children; 2016. Available from: <http://www.asa.co.nz/submissions-childrens-codes-reviews/>
9. New Zealand Food and Grocery Council. Comments from the NZFGC on the Consultation on the Review of the Code for Advertising to Children and the Children's Code for Advertising Food; 2016. Available from: <http://www.asa.co.nz/wp-content/uploads/2016/10/82.-NZ-Food-and-Grocery-Council.pdf>
10. Galbraith-Emami S, Lobstein T. The impact of initiatives to limit the advertising of food and beverage products to children: a systematic review. *Obes Rev.* 2013; 14(12):960–74.
11. Advertising Standards Authority. Submission to the Consultation on the review of the Code for Advertising to Children and the Children's Code for Advertising Food.

- From a group of 73 health professors. 2016. Available from: <http://www.asa.co.nz/wp-content/uploads/2016/10/86.-Professor-Boyd-Swinburn-on-behalf-of-73-Health-Professors.pdf>
12. Ni Mhurchu C, Mackenzie T, Vandevijvere S. Protecting New Zealand children from exposure to the marketing of unhealthy foods and drinks: a comparison of three nutrient profiling systems to classify foods. *N Z Med J.* 2016; 129(1441):41–53.
  13. World Health Organisation. WHO Regional Office for Europe nutrient profile model Copenhagen: WHO Regional Office Europe; 2015. Available from: [http://www.euro.who.int/\\_\\_data/assets/pdf\\_file/0005/270716/Nutrient-Profile-Model.pdf?ua=1](http://www.euro.who.int/__data/assets/pdf_file/0005/270716/Nutrient-Profile-Model.pdf?ua=1)
  14. Ministry of Health. Food and beverage classification system for years 1-13.; 2007.
  15. Quebec Government. Advertising directed at children under 13 Years of Age: Guide to the Application of Sections 248 and 249, Consumer Protection Act. In: Consommateur OdIPD, editor. Quebec 2012.
  16. Lumley J, Martin J, Antonopoulos N. Exposing the Charade: The failure to protect children from unhealthy food advertising. Melbourne: Obesity Policy Coalition; 2012.
  17. World Cancer Research Fund. WCRF International Food Policy Framework for Healthy Diets: NOURISHING 2014. Available from: [http://www.wcrf.org/policy\\_public\\_affairs/nourishing\\_framework/food\\_marketing\\_advertising](http://www.wcrf.org/policy_public_affairs/nourishing_framework/food_marketing_advertising)
  18. Vandevijvere S, Sagar K, Kelly B, Swinburn B. Unhealthy food marketing to children through the Internet in New Zealand. *N Z Med J.* 2016; 130(1450):32–43.
  19. World Health Organization Europe. Tackling food marketing to children in a digital world: trans-disciplinary perspectives Geneva: World Health Organization Europe; 2016. Available from: <http://www.euro.who.int/en/health-topics/disease-prevention/nutrition/publications/2016/tackling-food-marketing-to-children-in-a-digital-world-trans-disciplinary-perspectives-2016>
  20. No E, Kelly B, Devi A, Swinburn B, Vandevijvere S. Food references and marketing in popular magazines for children and adolescents in New Zealand: A content analysis. *Appetite.* 2014; 83:75–81.
  21. Vandevijvere S, Sushil Z, Exeter DJ, Swinburn B. Obesogenic Retail Food Environments Around New Zealand Schools: A National Study. *Am J Prev Med.* 2016 51(3):e57–66.
  22. Countdown. Countdown Health and Nutrition Targets 2016. Available from: [http://s3-ap-south-east-2.amazonaws.com/countdownnz/Countdown+Health+and+Nutrition+Targets\\_2016\\_FINAL.pdf](http://s3-ap-south-east-2.amazonaws.com/countdownnz/Countdown+Health+and+Nutrition+Targets_2016_FINAL.pdf)

# Giant cell arteritis treatment failure resulting from probable steroid/antiepileptic drug-drug interaction

Eoin Mulroy, John Highton, Sarah Jordan

**D**rug-drug interactions are an increasing problem in clinical practice, and may result in treatment failures. We describe an 83-year-old man with giant cell arteritis who suffered steroid treatment failure as a result of steroid metabolism induction from concurrent antiepileptic therapy.

## Case report

An 83-year-old man with previous cerebral abscess (surgically resected 55 years prior) and postoperative seizure disorder presented with headache, scalp tenderness

and reduced left eye visual acuity. His medications were: phenytoin 400mg daily, cilazapril 1.5mg daily, omeprazole 40mg daily, melatonin 3mg nightly, bendrofluzide 2.5mg daily and primidone 500mg daily.

Examination revealed visual acuity of 4/5 in the right eye, and hand movements only in the left eye. Fundoscopy showed left sided ischaemic optic neuropathy. Physical examination was otherwise normal. Bloods are shown in Table 1. Left temporal artery biopsy was diagnostic of giant cell arteritis.

**Table 1:** Full blood count, serum chemistries, liver function and inflammatory markers on presentation.

		Reference ranges
Haemoglobin	131g/L	130–175g/L
Platelets	304x10 <sup>9</sup> /L	150–400x10 <sup>9</sup> /L
Neutrophils	4.8x10 <sup>9</sup> /L	1.9–7.5 x10 <sup>9</sup> /L
Monocytes	0.6x10 <sup>9</sup> /L	0.2–1 x10 <sup>9</sup> /L
Erythrocyte Sedimentation rate	49mm/h	
C-reactive protein	86mg/L	<5mg/L
Na	128mmo/L	135–145mmol/L
K	4.1mmol/L	3.5–5.2mmol/L
Urea	4.9mmol/L	3.2–7.7mmol/L
Creatinine	72µmol/L	50–110µmol/L
Albumin	39g/L	35–50g/L
Bilirubin	5mmol/L	2–20mmol/L
Alkaline Phosphatase	103U/L	30–150U/L
Alanine Transaminase	22U/L	10–50U/L
Gamma Glutamyl Transferase	111U/L	10–50U/L

Sight-compromising disease prompted treatment with intravenous methylprednisolone 1g daily for three days. This decreased c-reactive protein (CRP) from 86mg/L to 15mg/L. Oral prednisone 60mg daily was then started, but CRP rose continuously for four days. Re-treatment with three days of intravenous methylprednisolone 500mg daily once again caused a CRP fall for three consecutive days. Upon transition to oral prednisone, this time 80mg daily, CRP rose again. A single intramuscular injection of triamcinolone acetonide 80mg, prednisone 60mg twice daily and 500mg of intravenous cyclophosphamide were prescribed, and CRP declined to normal over four days. He received one further dose of 500mg intravenous cyclophosphamide, and has successfully weaned prednisone to 15mg daily over seven months without symptomatic or biochemical disease recurrence. Though headaches and scalp tenderness resolved within four days, vision did not improve. Primidone was stopped, but phenytoin therapy continues.

His response to potent intravenous and intramuscular steroids and high dose oral steroids, but lack of response to standard treatment doses of oral prednisone, can be explained by CYP 3A4 induction by phenytoin and primidone increasing steroid metabolism.

## Discussion

The ageing global population brings with it an increased prevalence of polypharmacy.<sup>1</sup> This increases the potential for drug-drug interactions (DDIs). Our case of giant cell arteritis was unresponsive to conventional doses of oral glucocorticoids because of steroid metabolism induction from concomitant antiepileptic therapy. Clinicians must be vigilant to this interaction, especially in the elderly population.

As far back as the 1960s, the importance of DDIs on steroid metabolism were

recognised.<sup>2</sup> To understand these DDIs, a review of steroid metabolism is necessary. Prednisone is a synthetic corticosteroid, which is metabolically inactive until converted to prednisolone by hepatic 11-beta hydroxysteroid dehydrogenase.<sup>3</sup> Prednisolone, the active compound, is metabolised by CYP 3A4, which is prone to interaction with CYP inducers and inhibitors, the main culprits on the inducer list being antiepileptics (especially phenytoin, carbamazepine and phenobarbitone) and rifampicin.<sup>3,4</sup>

DDIs involving steroids are likely under-recognised. Following a flurry of publications on the subject in the late 1970s and 1980s, the topic of steroid metabolism induction appears to have fallen out of favour in the medical literature. In 1971, the plasma half-life of endogenous cortisol was noted to be reduced by co-administration of phenytoin.<sup>2</sup> In 1972, a decrease in respiratory function was noted in a group of asthmatics exposed to phenytoin, presumably due to increased steroid metabolism.<sup>5</sup> In 1976, nine steroid-dependent patients with rheumatoid arthritis were administered the enzyme inducer phenobarbitone, and showed a significant deterioration in disease control due to increased steroid metabolism.<sup>6</sup> Similar interactions were later observed in neurosurgical patients on dexamethasone and those with steroid-dependent pemphigus.<sup>7-9</sup>

The increasing prevalence of polypharmacy mandates careful consideration of DDIs when prescribing steroids. Close attention should be paid to current use of CYP-inducing medications such as those mentioned above. Elevated gamma-glutamyltransferase levels (as in our patient) may be a clue to enzyme induction, and steroid doses should be adjusted accordingly.<sup>10</sup> Doubling of usual steroid doses may be a good starting point in cases where titration over weeks is not appropriate, eg sight-compromising GCA.<sup>11</sup> Failing to do so may result in treatment failure.

**Competing interests:**

Nil.

**Author information:**

Eoin Mulroy, Department of Medicine, Dunedin Public Hospital, Dunedin; John Highton, Department of Medicine, Dunedin School of Medicine, Dunedin; Sarah Jordan, Department of Rheumatology, Dunedin Hospital, Dunedin.

**Corresponding author:**

Eoin Mulroy, Department of Medicine, Dunedin Public Hospital, Great King Street, Dunedin 9016.

eoinmulroy@gmail.com

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7164>

**REFERENCES:**

1. Nishtala PS, Salahudeen MS. Temporal Trends in Polypharmacy and Hyperpolypharmacy in Older New Zealanders over a 9-Year Period: 2005–2013. *Gerontology*. 2015; 61(3):195–202.
2. Choi Y, Thrasher K, Werk EE Jr, et al. Effect of diphenylhydantoin on cortisol kinetics in humans. *J Pharmacol Exp Ther*. 1971 Jan; 176(1):27–34.
3. Pickup ME. Clinical pharmacokinetics of prednisone and prednisolone. *Clin Pharmacokinet*. 1979 Mar-Apr; 4(2):111–28.
4. Wrighton SA, Brian WR, Sari MA, et al. Studies on the expression and metabolic capabilities of human liver cytochrome P450III<sub>A5</sub> (HLp3). *Mol Pharmacol* 1990; 38:207–213.
5. Brooks SM, Werk EE, Ackerman SJ, et al. Adverse effects of phenobarbital on corticosteroid metabolism in patients with bronchial asthma. *N Engl J Med* 1972; 286:1125–1128.
6. Brooks PM, Buchanan WW, Grove M, et al. Effects of enzyme induction on metabolism of prednisolone. Clinical and laboratory study. *Ann Rheum Dis* 1976; 35:339–343.
7. Chalk JB, Ridgeway K, Brophy T, et al. Phenytoin impairs the bioavailability of dexamethasone in neurological and neurosurgical patients. *J Neurol Neurosurg Psychiatry* 1984; 47:1087–1090.
8. McLelland J, Jack W. Phenytoin/dexamethasone interaction: A clinical problem. *Lancet*. 1978 May 20; 1(8073):1096–7.
9. Sehgal VN, Srivastava G. Corticosteroid-unresponsive pemphigus vulgaris following antiepileptic therapy. *Int J Dermatol*. 1988 May; 27(4):258.
10. Ennulat D, Walker D, Clemo F, et al. Effects of hepatic drug-metabolizing enzyme induction on clinical pathology parameters in animals and man. *Toxicol Pathol*. 2010 Aug; 38(5):810–28.
11. Petereit LB, Meikle AW. Effectiveness of prednisolone during phenytoin therapy. *Clin Pharmacol Ther*. 1977 Dec; 22(6):912–6.

# High incidence of medulloblastoma in Māori and Pacific populations in New Zealand

J Mark Elwood, Phyu Sin Aye

## ABSTRACT

In New Zealand from 1995–2010, the incidence of medulloblastoma at ages 1–19 years was significantly higher in Māori (relative risk 2.0) and in Pacific peoples (RR 2.1) than in New Zealand Europeans.

**W**e report here on the incidence of medulloblastoma in ethnic groups in New Zealand, based on data from the New Zealand national cancer registry. This legally mandated registry has had complete national data since at least 1995. Ethnic background was considered as Māori, Pacific peoples or New Zealand European, excluding Asian and Middle Eastern/Latin American/African groups.

During the period 1995–2010 at ages 0–19 years, the annual incidence of medulloblastoma in Māori was 9.1 per million population (39 cases), and in Pacific populations was 9.7 per million (19 cases). These

rates are significantly higher than in the New Zealand European population (4.5 per million, 60 cases), with relative risks of 2.0 (95% confidence limits 1.3 to 3.2) for Māori, and 2.1 (95% confidence limits 1.1 to 4.2) for Pacific groups, adjusted for age, year and gender (see Table 1). During this period 22% of the population aged 0–19 years was Māori, and 10% Pacific.

In each ethnic group, the incidence rates were higher in males than in females, but the ethnic differences were greater in females (see Table 1). In New Zealand Europeans, male rates were more than twice female rates, while in Pasifika male and

**Table 1:** Incidence of medulloblastoma, New Zealand, 1995-2010.

	NZ European	Māori	Pacific peoples
Total cases, age 0-19	60	39	19
Standardised rate per million per year*	4.54	9.11	9.73
RR and 95% CL	1 (referent)	2.0 (1.3 to 3.2)	2.1 (1.1 to 4.2)
Males, std rate	6.6	11.4	10.1
RR		1.7 (1.0 to 3.1)	1.5 (0.7 to 3.4)
Number of cases	44	25	10
Females, std rate	2.5	6.8	9.3
RR		2.7 (1.1 to 6.6)	3.8 (1.1 to 12.6)
Number of cases	16	14	9
Male to female ratio	2.8: 1	1.8:1	1.1:1

\* Rates directly standardised for age, year, gender, to the WHO world standard population.  
RR = relative risk CL = confidence limits.

female rates were almost equal; although these comparisons are based on small numbers. Overall, incidence was higher at ages 5–9 than 0–4 or 10–14 years, but the age patterns were similar in each ethnic group. There was no significant change in incidence over time, overall or in any ethnic group.

An excess of medulloblastoma in Māori has been previously shown in cancer registry data from 1948–1988, but cancer registration was incomplete at that time.<sup>1</sup> An excess in Māori was also noted in an Auckland hospital series in 1995–2004.<sup>2</sup>

An increased incidence of medulloblastoma in non-European ethnic groups has not been shown elsewhere, although the international literature is limited. In the US the incidence of medulloblastoma was lower in black and in Hispanic groups than in the white population, and it was slightly and non-significantly lower in the ‘Asian/

Pacific Islander’ group.<sup>3</sup> The rates in New Zealand Māori and Pasifika are higher than any reported in ethnic groups in the US. The overall rate in this US report was 5.2 per million, based on ages 0–14, 2007–11 and standardised to the US population; the equivalent New Zealand rate was the same, based on 52 cases.

We have no explanation for this empiric, but statistically robust, finding. Further clinical and genetic characterisation of these tumours may be informative. We are not aware of any data on subtypes or other factors related to ethnicity in New Zealand patients. The lower male:female ratio in Māori and Pasifika could be relevant, as among the four major subtypes of medulloblastoma, the wingless (WNT) subtype shows a female preponderance, and the sonic hedgehog (SHH) subtype shows about equal sex distribution, while other main groups show male excesses.<sup>4</sup>

---

#### Competing interests:

Nil.

#### Author information:

J Mark Elwood, Department of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland; Phyu Sin Aye, Department of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland.

#### Corresponding author:

Dr J Mark Elwood, Department of Epidemiology and Biostatistics, School of Population Health, University of Auckland, Auckland.  
mark.elwood@auckland.ac.nz

#### URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7165>

---

#### REFERENCES:

1. Preston-Martin S, Lewis S, Winkelmann R, Borman B, Auld J, Pearce N. Descriptive epidemiology of primary cancer of the brain, cranial nerves, and cranial meninges in New Zealand, 1948-88. *Cancer Causes Control* 1993; 4:529–538.
2. Monteith SJ, Heppner PA, Woodfield MJ, Law AJ. Paediatric central nervous system tumours in a New Zealand population: a 10-year experience of epidemiology, management strategies and outcomes. *J Clin Neurosci* 2006; 13(7):722–729.
3. Ostrom QT, de Blank PM, Kruchko C, et al. Alex’s Lemonade Stand Foundation Infant and childhood primary brain and central nervous system tumors diagnosed in the United States in 2007–2011. *Neuro Oncol* 2015;16 Suppl 10:x1–x36. doi: 10.1093/neuonc/nou327. x1–x36.
4. Massimino M, Biassoni V, Gandola L, Garre ML, Gatta G, Giangaspero F, et al. Childhood medulloblastoma. *Crit Rev Oncol Hematol* 2016; 105:35-51. doi: 10.1016/j.critrevonc.2016.05.012. Epub@2016 Jun 15.:35–51.

# High levels of rheumatic fever and sore throat awareness among a high-risk population screened for rheumatic heart disease

Jason K Gurney, Angela Chong, Nicola Culliford-Semmens, Elizabeth Tilton, Nigel J Wilson, Diana Sarfati

**E**radication of rheumatic fever has become a New Zealand health priority in recent years, with the Government investing \$65 million across a series of interventions aimed at drastically reducing rheumatic fever incidence by 2017.<sup>1</sup> It is possible to prevent rheumatic fever occurrence via the timely treatment of Group-A Streptococcal (GAS) infection with penicillin antibiotics.<sup>2</sup> Given that rheumatic fever is primarily a disease of childhood,<sup>3,4</sup> the decision to seek care generally rests with someone else—invariably the sick child's parent or caregiver—who will make this decision based on their own experiences and knowledge.

Sore throat awareness, then, is a crucial element of rheumatic fever prevention—part of which is an understanding of the potential consequences of sore throats, and what can be done to prevent them.<sup>5</sup> Little is understood about awareness in these respects among those who belong to the highest-risk population.

We interviewed the parents/caregivers of those children who were diagnosed with either definite, probable or possible/borderline RHD during the recent school-based rheumatic heart disease (RHD) echocardiographic screening programmes, conducted in multiple district health boards between 2007–2012 ('case respondents', n=91; age 12.2 years [SD 1.4], 64% of those invited to participate). We also interviewed the parents/caregivers of at least two DHB-matched 'controls' whose scan showed no cardiac abnormality ('control respondents'; n=185, age 11.9 years [SD 1.3], 51% of those invited to participate). Matching solely

on study region allowed an approximate match of cases to controls by geographic region, age, socio-economic status and time since the screening event—and, to a lesser extent, ethnicity.

The majority of the screened children were Māori ('abnormal' cases: 52%; 'normal' controls: 56%) or Pacific (cases: 48%; controls: 34%), with a minority being non-Māori/non-Pacific (cases: 5%; controls: 17%). The vast majority of both case (87%) and control (89%) respondents were parents of the screened child. The interview (79% telephone, 21% in-person) included a series of questions pertaining to rheumatic fever and sore throat awareness, and was conducted by trained interviewers using Computer-Assisted Personal Interview (CAPI) technology. The results are shown in Table 1.

Almost all respondents had heard of rheumatic fever, with no difference observed between case (92%) and control respondents (95%; p=0.458). When asked to select from a list regarding what causes rheumatic fever, the majority of respondents identified 'sore throats', with more case respondents (78%) than control respondents (65%) doing so (p=0.033).

High levels of sore throat awareness were observed regarding the best thing to do if a child has a sore throat—with the majority of both case respondents (89%) and control respondents (83%) saying that they should see a doctor or nurse straight away. The majority of respondents correctly identified that it was possible to catch a sore throat

**Table 1:** Respondent awareness with respect to sore throats and the rheumatic fever.

	Abnormal Result		Normal Result		P
	n	%	n	%	
<b>Rheumatic fever</b>					
Ever heard of rheumatic fever	84	92%	175	95%	0.458
<i>What do you think rheumatic fever is caused by?<sup>1,2</sup></i>					
Sore throats	71	78%	121	65%	0.033
Cold weather	14	15%	28	15%	0.957
Joint infections	13	14%	26	14%	0.959
Don't know	10	11%	33	18%	0.141
<i>Which part of the body can rheumatic fever affect?<sup>1</sup></i>					0.01
Kidneys	2	2%	2	1%	
Heart	82	98%	152	87%	
Liver	0	0%	2	1%	
Don't know	0	0%	19	11%	
<b>Sore throats</b>					
<i>If a child has a sore throat, what is the best thing to do?</i>					0.335
Wait a few days and see if it gets better	7	8%	16	9%	
See the nurse or doctor straight away	81	89%	152	82%	
Get something like throat lozenges for it	2	2%	13	7%	
Don't know	1	1%	4	2%	
<i>Can you catch a sore throat from someone else?</i>					0.29
Yes	59	65%	138	75%	
No	21	23%	26	14%	
Only if you are 'run down'	3	3%	6	3%	
Don't know	8	9%	15	8%	
<i>Have you ever heard of 'strep throat'?</i>	60	66%	142	77%	0.057
<i>What is the best treatment for 'strep throat'?<sup>3</sup></i>					0.334
Antibiotics	56	93%	122	86%	
Rest and wait for it to go away	2	3%	4	3%	
Other	0	0%	2	1%	
Don't Know	2	3%	14	10%	

<sup>1</sup>Multiple responses were allowed. <sup>2</sup>Among those who had heard of rheumatic fever. <sup>3</sup>Among those who had heard of 'strep throat'.

from someone else; however, nearly a third (32%) of case respondents and nearly a quarter (22%) of control respondents either said 'no' or 'don't know'. This observation is similar to that observed among New Zealand school-aged children who were asked the same question, in which less than half (49%) knew that sore throats were catching.<sup>6</sup>

The vast majority of both case (93%) and control (86%) respondents understood that a 'strep throat' needed to be treated with antibiotics—a substantially higher proportion than that observed recently in the US, where 62% of Medicaid-insured adult respondents had this understanding.<sup>7</sup>

Overall, these observations are heartening: they represent a generally high level of rheumatic fever and sore throat awareness among a population at high risk<sup>3,4</sup> of rheumatic fever—or, more accurately, a population at high risk of caring for a child with rheumatic fever. While the majority of respondents demonstrated a high level of awareness with respect to the contagious nature of sore throats, the fact that a substantial minority did not have this under-

standing suggests an opportune target for further public health education.

The New Zealand Ministry of Health has, in recent years, funded a rheumatic fever prevention programme (RFPP),<sup>8</sup> which includes a substantial rheumatic fever and sore throat awareness component—with this programme being initiated after the current cohort received screening for rheumatic heart disease, but before the respondents were interviewed. Based on international comparison<sup>7</sup> and recent evidence in the New Zealand context of a temporal improvement in awareness among this high-risk population,<sup>9</sup> we might conclude that the generally high rheumatic fever and sore throat awareness that we observed most likely reflects the current RFPP and its extensive publicity campaign, rather than education at the time of the RHD screening, which took place 3–8 years earlier. However, we note that it is also feasible that participation in the screening event itself affected participants' knowledge about rheumatic fever, which would affect the applicability of our results to high-risk populations more generally.

---

**Competing interests:**

Nil.

**Acknowledgements:**

The authors would like to acknowledge the participants and their families for taking part in this study. We would also like to acknowledge the key regional investigators from the original screening studies: Dr Rachel Webb, Dr Adrian Trenholme (South Auckland), Dr Geoff Cramp (Gisborne and Ruatoria), Dr John Malcolm (Bay of Plenty and Kawerau), Dr Roger Tuck and Dr Jonathan Jarman (Kaitaia) and Dr Nikki Blair (Porirua). This study was funded by the Health Research Council of New Zealand, the Ministry of Health, Te Puni Kōkiri, Cure Kids and the Heart Foundation (HRC ref #: 13/965).

**Author information:**

Jason K Gurney, Senior Research Fellow, Department of Public Health, University of Otago, Wellington; Angela Chong, Project Manager/Researcher, CBG Health Research Ltd, Auckland; Nicola Culliford-Semmens, Research Fellow, Starship Children's Hospital, Auckland; Elizabeth Tilton, Researcher, Starship Children's Hospital, Auckland; Nigel J Wilson, Paediatric Cardiologist, Green Lane Paediatric and Congenital Cardiology, Starship Children's Hospital, Auckland, and University of Auckland, Auckland; Diana Sarfati, Epidemiologist, Department of Public Health, University of Otago, Wellington.

**Corresponding author:**

Jason K Gurney, Senior Research Fellow, Department of Public Health, University of Otago, 23A Mein St, Newtown, Wellington.  
jason.gurney@otago.ac.nz

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7166>

---

## REFERENCES:

1. Raising awareness of rheumatic fever [press release]. 1st May 2016.
2. Heart Foundation of New Zealand. New Zealand Guidelines for Rheumatic Fever: Diagnosis, Management and Secondary Prevention of Acute Rheumatic Fever and Rheumatic Heart Disease: 2014 Update. 2014.
3. Jaine R, Baker M, Venugopal K. Epidemiology of acute rheumatic fever in New Zealand 1996–2005. *Journal of Paediatrics and Child Health*. 2008; 44(10):564–71.
4. Milne RJ, Lennon DR, Stewart JM, Vander Hoorn S, Scuffham PA. Incidence of acute rheumatic fever in New Zealand children and youth. *Journal of Paediatrics and Child Health*. 2012; 48(8):685–91.
5. New Zealand Guidelines Group. RapidE: Rheumatic Fever: A systematic review of the literature on health literacy, overcrowding and rheumatic fever. Wellington, New Zealand: 2011.
6. Harré N, Thomas D, Brown K, Raza F, Lennon D. Communicating information about sore throats and rheumatic fever to South Auckland high-school students. *New Zealand Medical Journal*. 2000; 113(1111):215–7.
7. Vaz LE, Kleinman KP, Lakoma MD, Dutta-Linn MM, Nahill C, Hellinger J, et al. Prevalence of parental misconceptions about antibiotic use. *Pediatrics*. 2015; 136(2):221–31.
8. Ministry of Health. Progress on the Better Public Services rheumatic fever target Wellington, New Zealand 2015 [Available from: <http://www.health.govt.nz/about-ministry/what-we-do/strategic-direction/better-public-services/progress-better-public-services-rheumatic-fever-target>].
9. Anderson P, King J, Moss M, Light P, McKee T, Farrell E, et al. Nurse-led school-based clinics for rheumatic fever prevention and skin infection management: Evaluation of mana kidz programme in counties Manukau. *New Zealand Medical Journal*. 2016; 129(1428):36–45.

## Community-acquired pneumonia incidence before and after proton pump inhibitor prescription

Is the use of proton pump inhibitors (PPIs) associated with an increased risk of pneumonia? Several observational studies and meta-analyses have suggested that this is so since an initial report in 2004; hence this study.

Information was derived from a primary care database in the UK. Adult patients newly treated with a PPI were selected and matched individually, according to age, sex and year of prescription, with patients who had not been prescribed PPI. The risk of pneumonia was 1.67 times higher for patients exposed to PPI. However, it was noted that in the PPI cohort the relative risk of pneumonia was 1.19 in the 30 days after prescription, but was higher in the 30 days before prescription (1.92).

The results confirm the known crude association between PPI prescriptions and an increased rate of community-acquired pneumonia.

However, the analyses show that this increased risk predates PPI prescription, and is therefore unlikely to be caused by PPIs.

*BMJ* 2016; 355:i5813

---

## Impact of providing patients with copies of their medical correspondence

In this report from Melbourne it is noted that in Australia, patients do not routinely get copies of the consultation letters and procedure reports sent to their general practitioners.

A randomised trial was designed to see whether such copies would lead to improved understanding, satisfaction or anxiety. The researchers noted no reduction in anxiety levels ( $p=0.52$ ), no increase in understanding ( $p=0.73$ ) or any increase in satisfaction ( $p=0.33$ ) in participants receiving correspondence. However, 97% wished to receive correspondence in the future and 94% in the correspondence group reported it helped them understand their medical condition.

It is recommended that patients be offered the choice of receiving copies of their clinic correspondence and procedure reports.

*Internal Medicine Journal* 2017; 47, 68–75

---

## Low-molecular-weight heparin and recurrent placenta-mediated pregnancy complications

Placenta-mediated pregnancy complications include pre-eclampsia, late pregnancy loss, placental abruption and birth of a small-for-gestational-age (SGA) neonate. These complications are leading causes of maternal, fetal and neonatal morbidity and mortality in high-income countries. In a previous study these researchers concluded that low-molecular weight heparin reduced the risk of these complications. However, there was heterogeneity in their results, so they decided to perform an individual patient data meta-analysis.

They analysed data from eight trials involving over 900 women, half of whom were treated with low-molecular-weight heparin and half who were not. Overall there was no significant difference in outcomes in the heparin or no heparin cohorts.

The researchers concluded that low-molecular-weight heparin does not seem to reduce the risk of recurrent placenta-mediated pregnancy complications in at-risk women.

*Lancet* 2016; 388:2629–41

---

### URL:

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7167>

---

# Sir Walter Raleigh's Nostrum

April 1917

In 1603 Sir Walter Raleigh, under sentence of death, was brought from trial at Winchester to the Tower of London. The sentence, however, was not executed until fifteen years later, and contrary to custom, Raleigh was not held a close prisoner in a dungeon, but was allowed a certain amount of liberty and recreation.

Scientific visitors came to the Tower to see not only Sir Walter, but also the "Wizard-Earl" of Northumberland, and Raleigh received these visitors (among whom were Bacon, rare Ben Jonson, and Heriot), in a lath and plaster shed in the garden of the Lieutenant of the Tower. In this shed, known as "the Garden House," Raleigh prepared his famous cordial, to which he gave the name of the "Balsam of Guiana." This cordial was supposed to cure nearly every ill known to mankind, and contained twenty ingredients, including "pearl, musk, hartshorn, bezoar stone, mint, gentian, mace, red rose, aloes, saffras, spirits of wine, and vipers' hearts."

Sir Walter, before his imprisonment, had enjoyed a royal patent to make wines, which brought him a large income, but his patent was transferred to Lord Howard of Effingham. "The bread and food taken from me and my children," said Lady Raleigh to King James, "will never augment my Lord of Effingham's table, though it famish us."

Raleigh's generation, and the next, firmly believed in the efficacy of the balsam

of Guiana, and it was drunk by Anne of Denmark, and Charles I. and II. Of Prince Henry it is recorded "after swallowing not more than two drops, he died." In the Garden House, the "obscure parts of learning" such as astronomy, anatomy, and theology, were discussed in the reek of drugs and chemicals, and an unfriendly critic, Sir Thomas Wilson, who, strange to say, looked upon Sir Walter as an atheist, declared that the shed contained "all the spirits in the world except the Spirit of God." The Prince said that no man but his father "would keep such a bird in such a cage." However, the balsam was the means of Raleigh's undoing, for one day one of his patients, the Countess of Beaumont, came to the Garden House for her favourite medicine, and brought with her as companion Captain Whitelocke, who was engaged in a conspiracy, and James I. affected to believe that Raleigh was also in the plot, and poor Sir Walter was closely confined, so that soon his left side became "numb," his fingers "curled," and his tongue "hardened." His doctor, Peter Turner, after many entreaties, was given permission to remove the palsied man again to the Garden House, where he lived until his release. Then came his journey to South America, his return and imprisonment, and final execution. His last joke was not inappropriate to one who had dabbled in medicine—"Can'st thou give me any plaster to set on a man's head when it is off?"

---

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7168>

---

# Selected proceedings of the APAC (Asia Pacific) Forum

2016

## Increasing the engagement of doctors in training in quality improvement

Vinters C,<sup>1</sup> Oates K<sup>2</sup>

<sup>1</sup>Program Leader, Clinical Practice Improvement Training Program, Clinical Excellence Commission, Sydney, Australia; <sup>2</sup>Director, Undergraduate Quality and Safety Education, Clinical Excellence Commission, Sydney, Australia

### Background and aims

In 2012, the New South Wales (NSW) Clinical Excellence Commission (CEC) and the Royal Australasian College of Physicians (RACP) collaborated to provide RACP Advanced Trainees (AT) with the tools to undertake a quality improvement project to improve patient outcomes using Clinical Practice Improvement (CPI) methodology.

### Methods

RACP ATs with experience in self-directed learning, leading and influencing others and working with a broad range of clinicians and managers were eligible to participate.

Participants undertook a two-day workshop on how to engage a team, lead a project, test effectiveness of interventions using Plan, Do, Study, Act cycles,<sup>1</sup> measure results and adopt practices, which increased the likelihood of sustaining their projects.<sup>2</sup>

Each participant undertook a practical improvement project, for example:

- reducing mislabelled specimens leaving an emergency department
- creating a multidisciplinary approach to reducing hypoglycaemic episodes on an endocrine ward

- establishing an acute paediatric review clinic to reduce avoidable admissions.

Participants involved patients/carers on their teams, either as active team members or by using patient stories as the impetus for change.

Each participant had an RACP-appointed supervisor and participated in a mid-point review at six months. Completed projects were presented in a final review at 12 months. Participants completed a quantitative and qualitative questionnaire at the end of the programme to provide feedback about its impact. In addition, participants completed the Attitudes to Patient Safety Questionnaire<sup>3</sup> (APSQ) before and after the workshop.

The programme has been refined since 2012. A webinar was added to increase AT supervisors' knowledge of quality improvement, and supervisors were invited to the mid-point review as well as the final graduation.

### Results

Sixty-five ATs have undertaken the programme, with 34 completing all components.

Six participants have published their work<sup>4,5</sup> and five have presented at national and international conferences. Other participants have undertaken other projects using the same methodology.

Benefits from projects include:

- 72% reduction in mislabelled specimens in a paediatric emergency department
- reduction from 23 hypoglycaemic episodes in diabetic patients on an endocrine ward to

three episodes during the same time period post-interventions

- 74% fall in avoidable paediatric pneumonia admissions.

Eighty-eight percent of participants said the programme had met or exceeded their expectations.

Eight participants provided qualitative responses. The key theme identified was the benefit of having greater involvement with the wider clinical team.

Twenty-three participants completed the APSQ. There was a slight increase in patient safety awareness.

### Conclusions

Educating junior doctors to undertake quality improvement is worthwhile. This model of collaboration could be taken up by other learned colleges in NSW and nationally.

### Declaration of conflict of interest

The lead author is the programme facilitator and main support for the participants undertaking the programme. The second author is a fellow of the RACP and presents at the workshop.

### References

1. Langley GL MR, Nolan KM, Nolan TW, Norman CL, Provost LP. The improvement guide: a practical approach to enhancing organizational performance (2nd ed). San Francisco: Jossey-Bass; 2009.
2. Taylor JRA, Mazur J. Sustainability model. NHS Improving Quality; 2014. Adapted from Maher L, Gustafson DH, Evans A. Sustainability model and guide. NHS Institute for Innovation and Improvement; 2009. Available from: [http://www.nhs.uk/media/2710414/2015\\_02\\_19\\_sustainability\\_model\\_print\\_-\\_version\\_4.pdf](http://www.nhs.uk/media/2710414/2015_02_19_sustainability_model_print_-_version_4.pdf)
3. Carruthers S, Lawton R, Sandars J, Howe A, Perry M. Attitudes to patient safety among medical students and

- tutors: developing a reliable and valid measure. *MedTeach*. 2009; 31(8):e370–e376.
4. Davis T, Nogański B. Alterations to calling criteria for Between the Flags (an early warning system). *BMJ Qual Improv Report*. 2015; 4:1 u206561.w2638 doi:10.1136/bmjquality.u206561.w2638
  5. Oates K, Vinters C, Cass-Verco J, Fletcher M, Kaur N, Mherakumombe M, Tang A. A pilot programme of clinical practice improvement for future consultant doctors. *Clin Teach*. 2016 May 3. doi: 10.1111/tct.12531

**Meeting radiation therapy patient information needs through collaborative production of videos incorporating novel 3D visualisation software**

Willis D,<sup>1</sup> Williams K,<sup>1</sup> Blencowe J,<sup>1</sup> Ind M,<sup>1</sup> Bourne M<sup>1</sup>

<sup>1</sup>North West Cancer Centre, Tamworth, Australia

**Background and aims**

Misconceptions about the radiation therapy process can compound patient anxiety before treatment. Explaining the rationale for some aspects of the process is challenging and patients in the North West Cancer Centre (NWCC) had indicated specific unmet information needs. We produced patient information videos that aimed to address those needs, incorporating footage from a three-dimensional (3D) visualisation software system to explain complex concepts.

**Methods**

A literature review and consultation with 10 patients who had recently commenced treatment informed the video content. We acquired footage of real-life treatment processes, with staff and patient consent. We used PEARL™ 3D radiation therapy visualisation software to address information needs that could not be addressed with real-life footage. NWCC staff with experience in multi-media production produced the videos. Fifteen further patients and the Radiation Oncology multi-disciplinary team (radiation oncologists, radiation therapists, specialist

nursing, medical physicists and social workers) conducted iterative reviews to determine if videos were understandable and complete. Approved videos were shown to all new patients during pre-treatment education sessions. Patients were also given a copy for viewing at home. We used a paper-based, ethics-approved patient survey to assess how well the video addressed information needs. Patients received the survey at their first treatment appointment and returned it anonymously.

**Results**

We produced five videos of 3–5 minutes' duration. Over 600 patients viewed the videos during pre-treatment education sessions. Two hundred and twelve surveys were distributed and 61 were returned (29% response rate). Survey respondents reported that the videos improved their understanding (85%) and reduced anxiety (50%). Fifty-three percent of respondents viewed videos at home, and 47% used them to explain treatment to others. Respondents commended 3D visualisation software for explaining the rationale for machine movements (81%) and the need for a preparatory CT scan (72%). In response to open ended questions, no respondents (0%) reported unmet informational needs.

The videos have been adopted by the Central Coast Cancer Centre and Crown Princess Mary Cancer Centre. The general process video has been included in the Royal Australian and New Zealand College of Radiologists' 'Targeting Cancer' campaign.

**Conclusions**

The videos met the information needs of survey respondents. The 3D visualisation software explained complex concepts clearly and succinctly. Patients valued being able to view the videos again at home and use them to explain treatment to loved ones.

**Declaration of conflict of interest**  
None.

**Patient and Whānau Centred Care Standards: elevating quality care through ward leadership**

Jenny Parr,<sup>1</sup> Jeanette Bell<sup>2</sup>

<sup>1</sup>Associate Director of Nursing, Waitemata District Health Board, Auckland; <sup>2</sup>Project Manager, Institute of Improvement and Innovation

**Background and aims**

In 2014, feedback from patients, families (whānau), staff and regular auditing indicated variability in the delivery of basic aspects of care at Waitemata District Health Board. The Patient and Whānau Centred Care Standards (PWCCS) Programme was established to develop an evidence-based, organisation-wide, care evaluation and improvement framework to promote the delivery of safe, consistent, high-quality patient care.

**Methods**

The PWCCS framework was developed using a literature review on patient-centred care and fundamentals of care, interviews with 20 senior leaders, thematic analysis of 981 items of patient/family feedback from complaints, compliments and patient surveys and a stocktake of related audits and improvement projects. A baseline audit of 19 wards and interviews with 108 patients indicated the fundamentals of care and were used to identify gaps and priorities. A multi-disciplinary steering group with independent chair and cultural and consumer representation oversaw the staged development of nine PWCCS standards over 12 months:

1. Communication
2. Clinical monitoring and management
3. Care environment
4. Comfort and pain management
5. Respect, privacy and dignity
6. Nutrition and hydration
7. Safety and prevention
8. Personal care
9. Self-care

Ward-level performance has been evaluated using an independent peer review process every six months since June 2015. Ward reviews include five patient interviews (40 items), five staff interviews (three items), ward observation, a ward management interview with each charge nurse manager and review of the previous quarter's KPI results. Language and cultural support enables non-English-speaking patients and family to participate. Participants score items using a Likert scale from yes (2) to no (0). Results are reported for each ward as percentages by question and standard, and are compared with the organisation (overall) average.

Two reviews have been undertaken; 27 wards in June 2015 and 31 wards in December 2015. In total, 302 patients were interviewed. All wards selected key areas for improvement based on their initial results. These included developing a welcome pack, a call bell campaign; "No pass zone", and local focus on providing personal care.

**Results**

Comparing the results using an independent t-test of the 27 wards, which participated in both reviews, there was a significant difference in the overall organisational results between June 2015 (M=.81, SD=.08) and December 2015 (M=.85, SD=.076);  $t(52)=2.10$ ,  $p=.041$ . In addition 74% of wards improved their overall result. There was significant improvement in five of the nine standards and 21 of the patient questions.

**Conclusion**

The PWCCS programme provides a comprehensive overview of quality at ward level that has not previously been visible and ensures the district health board provides consistent high-quality care in all our wards and patient care areas.

**Declaration of conflict of interest**  
None.

**The Respiratory Chronic and Complex Care Program—home air is fresher**

Jesina Chigavazira,<sup>1</sup> Nicole Morrison<sup>2</sup>

<sup>1</sup>CNC Respiratory, Bankstown Hospital, Sydney; <sup>2</sup>NUM Respiratory, Bankstown Hospital, Sydney

**Background and aims**

Historically, presentations for all respiratory conditions increase over winter.<sup>1,2</sup> Evidence from interventions such as the Respiratory Chronic Care Program (RCCP) suggests that many hospital presentations and admissions could be avoided with patient support at home.<sup>3,4</sup>

A 2015 review of re-admissions of patients at Bankstown Hospital with chronic respiratory diseases, mainly chronic obstructive pulmonary disease (COPD), showed that there were 235 patients/40% more re-admissions in winter (March to August) compared to summer. RCCP was proposed as the most appropriate strategy to prevent re-presentations, re-admissions, reduce length of stay and promote self-management, including reduction in smoking over winter.

While RCCP is well established across New South Wales local health districts, Bankstown Hospital did not have a well-defined programme, due to lack of financial resources. Through Nursing and Midwifery Office (NaMo) funding the pilot project was started.

**Methods**

Thirty-five respiratory patients with a history of three or more re-admissions per year were enrolled in the pilot study and followed up after discharge for six months, from March to August 2015. Input from a multidisciplinary team during the patient journey was used to recruit participants.

Patients were supported with home visits, telephone coaching and education. The majority were contacted by phone within 72 hours of enrolment; 212 phone calls were made and 105 homes visited.

Quantitative data on length of stay, number of re-admissions, presentations avoided, number of smokers who quit and patients who could self-manage their disease process was collected.

**Results**

Thirty-five patients were enrolled for the pilot programme and followed up. Of these, 30 (86%) had COPD.

- For 32/35 patients (92.6%), admissions via the Emergency Department for March to August decreased to zero.
- 18/21 (86%) smokers quit.
- There was a 2.3% decrease in episodes of care for all patients at Bankstown with the Diagnosis Related Group (DRG) for COPD in 2015 compared to 2014.
- The average length of stay for all COPD patients with complications decreased by 1.61 days, and for COPD patients without complications by 0.51 days.
- Use of overnight adult surge beds reduced from 1,149 in winter 2014 to 87 for the same period in 2015, a 92.4% reduction.

**Conclusion**

Emergency re-presentations and hospital re-admissions reduced after implementation of the programme. Therefore, provision of a respiratory outreach service that includes early discharge, follow-up education, telephone support, on-going home visits and access to outpatient clinics is associated with reduced re-admissions of chronic respiratory patients, especially COPD.

**Declaration of conflict of interest**  
None.

**References**

1. Adams S, Smith P, Allan P, Anzuetto A, Pugh J, Cornell J. Systematic review of the chronic care model in chronic obstructive pulmonary disease prevention and management. *Arch Intern Med.* 2007 Mar 26; 167(6):551–61.
2. Stone R, Lowe D, Buckingham R, Pursey N, Potter J, Roberts

CM. What happens to COPD patients before an admission with exacerbation? *Prim Health Res Dev.* 2012 Oct; 13(4):395–402. doi: 10.1017/S1463423612000187

3. Lawlor M, Kealy S, Agnew M, Korn B, Quinn J, Cassidy C, et al. Early discharge care with ongoing follow-up support may reduce hospital readmissions in COPD. *Int J Chron Obstruct Pulmon Dis.* 2009;4:55–60.
4. Koekkoek D, Bayley KB, Brown A, Rustvold DL. Hospitalists assess the causes of early hospital readmissions. *J Hosp Med.* 2011 Sep; 6(7):383–8. doi: 10.1002/jhm.909

### Telehealth: integrating care in pain management—‘Let imagination be the limit’

Martinovich J,<sup>1</sup> Johnson J<sup>2</sup>

<sup>1</sup>Telehealth Implementation Project Officer, NSW Agency for Clinical Innovation, Chatswood, NSW, Australia; <sup>2</sup>Network Manager Pain, NSW Agency for Clinical Innovation, Chatswood, NSW, Australia

#### Background and aims

In 2014, the New South Wales (NSW) Agency for Clinical Innovation (ACI) Pain Management Network recommended adoption of a Telehealth model of care to address gaps in access to chronic pain services for patients in geographically isolated local health districts. ACI undertook a pilot project with the Children’s Hospital Westmead in Sydney and Orange Hospital in rural NSW to assess the viability of Telehealth for enabling metropolitan-based specialists to deliver remote pain management services to rural consumers.

#### Methods

ACI established a partnership with Healthdirect Australia (HDA) to deliver secure, internet-based video-call technology directly to patients.

First, ACI delivered three introductory training sessions to the two pilot sites. The first introduced the technology, model of care, process and billing differences. The second was a mock consultation and the third a supported live session. A Chronic Pain Telehealth Toolkit was also developed to help clinicians plan and deliver the telehealth consultations.

Then, 32 consultation sessions were held, either one-on-one in the patient’s home or at the patient’s GP/private practice clinic with a local clinician present. Thirty-two patients and eight local clinicians (GPs, private physiotherapists and general paediatricians) were surveyed to understand their satisfaction and experiences with the telehealth service. Surveys used Likert scales and free text responses. Semi-structured interviews with five specialist pain clinicians and one administration officer were also conducted. Interview responses were thematically analysed and themes were verified by interviewees. Finally, clinicians recorded the patient’s location and we mapped the distance to the specialist pain clinic to ascertain the travel time saved.

#### Results

Fourteen patients completed the survey (43.75% response rate). All respondents (100%) strongly agreed that the session was convenient. Eighty-eight percent agreed or strongly agreed that the session was as good as face-to-face. One hundred percent were happy to continue using the telehealth service. A key theme in patients’ free text responses was reduced travel-related pain. Total patient travel distance saved was 9,180 kilometres.

All eight local clinicians surveyed reported that the sessions were convenient; that telehealth consultations helped them to support patients with pain management and that they were happy to continue using the telehealth service.

Key themes from interviews with specialist pain clinicians were perceived benefits of better access and increased networking with local healthcare professionals. All interviewees reported they were comfortable using the technology and would continue to do so after the pilot.

#### Conclusions

The Telehealth model of care is an effective way to deliver specialist pain management services to patients in rural NSW.

**Declaration of conflict of interest**  
None.

### The efficacy of the Victorian Stroke Telemedicine Program: preliminary results from 10 regional hospitals

Kathleen L Bagot,<sup>1,2</sup> Michelle Vu,<sup>1</sup> Helen M Dewey,<sup>2,3</sup> Geoffrey A Donnan,<sup>1</sup> Peter Hand,<sup>4</sup> Sharan Ermel,<sup>5</sup> Lauren Arthurs,<sup>6</sup> Jo Cottrell,<sup>7</sup> Maria Fox,<sup>8</sup> Justin Rabl,<sup>9</sup> Lisa Peters,<sup>10</sup> Casey Hair,<sup>11</sup> Anne van Berkel,<sup>12</sup> Emma Marino,<sup>13</sup> Mick O’Shea,<sup>14</sup> Janette McCabe,<sup>15</sup> Catherina De Muelenaere,<sup>16</sup>  
\*Dominique A Cadilhac,<sup>1,2</sup>

\*Christopher F Bladin,<sup>1,2</sup> on behalf of the VST investigators

\*Joint senior authors

<sup>1</sup>The Florey Institute of Neuroscience and Mental Health, Heidelberg, Victoria, Australia; <sup>2</sup>Monash University, Clayton, Victoria, Australia; <sup>3</sup>Eastern Health, Box Hill, Victoria, Australia; <sup>4</sup>Royal Melbourne Hospital, Parkville, Victoria, Australia; <sup>5</sup>Bendigo Health, Bendigo, Victoria, Australia; <sup>6</sup>Echuca Regional Health, Echuca, Victoria, Australia; <sup>7</sup>Mildura Base Hospital, Mildura, Victoria, Australia; <sup>8</sup>Swan Hill District Health, Swan Hill, Victoria, Australia; <sup>9</sup>Goulburn Valley Health, Shepparton, Victoria, Australia; <sup>10</sup>Northwest Health Wangaratta, Wangaratta, Victoria, Australia; <sup>11</sup>Ballarat Regional Hospital, Ballarat, Victoria, Australia; <sup>12</sup>Central Gippsland Health Service, Sale, Victoria, Australia; <sup>13</sup>Latrobe Regional Hospital, Traralgon, Victoria, Australia; <sup>14</sup>Albury Wodonga Hospital, Albury, NSW, Australia; <sup>15</sup>Wimmera Base Hospital, Horsham, VIC, Australia; <sup>16</sup>Bairnsdale Regional Health Service, Bairnsdale VIC, Australia

#### Background and aims

Despite several countries having well-established acute stroke telemedicine services, there has been limited availability within Australia.<sup>1</sup> Acute telemedicine programmes can increase regional hospitals’ access to time-critical delivery of intravenous stroke thrombolysis (tissue plasminogen activator; tPA)<sup>2</sup> and support rapid access to new treatments,<sup>3</sup> such as endovascular clot retrieval (ECR). The aims of the Victorian Stroke Telemedicine (VST) Program are to enhance acute stroke clinical processes of care within 16 regional hospitals.

#### Methods

VST has a multi-phase implementation process with 10

Victorian regional hospitals currently accessing the 24/7 on-call roster of metropolitan-based VST neurologists (n=12). A 12 month pre-post implementation historical-control cohort design is used. Eligibility criteria are patients ≥18 years presenting to Emergency Departments (ED) within 4.5 hours of suspected acute stroke symptom onset. Demographics, diagnosis, time of ED arrival, assessment and treatment delivery times are captured.

**Results**

Data between February 2013 and January 2016 include 1,202 patients that presented to EDs with suspected stroke during VST. Of these, 205 (median age 77 years; 49% male) had a confirmed stroke within 4.5 hours of onset (17%). Prior to VST, 1,465 patients presented with suspected stroke symptoms. Of these, 224 (median age 75 years; 55% male,) had a confirmed stroke within 4.5 hours of onset (of those presenting to ED with suspected stroke symptoms, 15% pre VST and 17% during VST had a confirmed stroke with 4.5 hours). To date, there have been 533 VST consultations and some regional hospitals are now able to provide tPA to eligible patients because of the support of VST. Faster door-to-first medical contact (p=.01) and faster door-to-needle times (p=.02) have been found (median reduction = 23 minutes) since VST was implemented. Overall, 26% of patients with ischaemic stroke presenting under 4.5 hours to EDs have received tPA compared to 16% among similar sized Australian hospitals. Further, 32 patients have been recommended and transferred for ECR therapy.

**Discussion**

The VST Program is Australia's largest stroke telemedicine service and is improving the clinical process timelines including delivering tPA faster to eligible patients in regional Victoria. VST has been leveraged to rapidly translate new evidence into practice, such as access to ECR. Without VST, these treatment options were largely unavailable to eligible patients presenting to regional hospitals.

**Declaration of conflict of interest**  
None.

**References**

1. Cadilhac DA, Vu M, Bladin C. Experience with scaling up the Victorian Stroke Telemedicine programme. *J Telmed Telecare*. 2014 Oct; 20(7):413–8.
2. Bladin CF, Cadilhac DA. Effect of telestroke on emergent stroke care and stroke outcomes. *Stroke: a Journal of Cerebral Circulation*. 2014; 45:1876–80.
3. Bagot KL, Cadilhac DA, Hand PJ, Vu M, Bladin CF. Telemedicine can aid access to optimal acute stroke care (correspondence). *Lancet*. 2016; 388:757–58.

**Pursuing health and walking the talk: a Māori advancement framework for New Zealand's Health Quality & Safety Commission**

Maria Poynter,<sup>1</sup> Kiri Rikihana,<sup>1</sup> Kayleen Katene,<sup>2</sup> Janice Wilson,<sup>1</sup> Tuwhakairiora Williams<sup>1</sup>

<sup>1</sup>Health Quality & Safety Commission, New Zealand;

<sup>2</sup>Formerly Health Quality & Safety Commission, New Zealand

**Background and aims**

Equity is an established part of quality health care, and this is reflected in the New Zealand 'Triple Aim'.<sup>1</sup> Yet we know from decades of reporting that populations do not enjoy equal health.<sup>2-4</sup> The aim of this work was for the Health Quality & Safety Commission to increase its commitment to, understanding of and planning for Māori health equity.

**Methods**

We reviewed five external Māori advancement strategies (health and non-health sector) and key Māori health equity documents. We surveyed (48 baseline, 36 repeat responses at two years) board members, staff members and Māori advisory group members about the current and desired role of the Commission in Māori health equity. Two staff members (Māori and non-Māori) led a process to develop a Māori Advancement Framework: Te Whai Oranga. An implementation plan drives key actions, with monthly reporting to senior management.

**Results**

The framework of Te Whai Oranga identifies four areas of focus: Mahi ngātahi (Partnerships), Matauranga (Knowledge), Rangatiratanga (Leadership), Whai hua (Strategic Priorities). It enables

internal stakeholders—board members, Māori advisors, senior leaders and other staff—to identify their responsibilities for each area.

Te Whai Oranga was informed by both the review of external Māori advancement frameworks and the internal stakeholder survey. In the first survey, thematic analysis identified knowledge about cultural protocols and Māori health as a key area for improvement, which allowed us to tailor a staff capability-building programme, including two board/staff training days at marae (ceremonial and cultural meeting houses), increased use of Māori language and protocol in Commission outputs, and improving relationships with key Māori health stakeholders. Our repeat survey suggests that staff have moved from not understanding why a Māori world-view is important, to wanting to further increase their knowledge.

**Discussion**

Te Whai Oranga translates as 'the pursuit of health'; we recognise that we are near the start of this journey. The Commission's commitment to equity as a component of quality must be real and long-term. Wider uptake of equity as a key concern for the health sector will be our medium-term driver, with equity of health outcomes for Māori our ultimate vision.

Key enablers have been a slow but attainable pace of change, governance and management support, and using survey data to design capability-building sessions. A key challenge has been determining how to ensure that activities like this framework have an impact on areas most in need; quality improvement approaches, like the health system, favour the majority.

**Declaration of conflict of interest**  
None.

**References**

1. Health Quality & Safety Commission. The New Zealand Triple Aim for quality improvement [cited 2016 6 September]. Available from: [www.hqsc.govt.nz/about-the-commission](http://www.hqsc.govt.nz/about-the-commission)
2. Davis P, Lay-Yee R, Dyall L, Briant R, Sporle A, Brunt D, et al. Quality of hospital care for Māori patients in New Zealand: retrospective cross-sectional assessment. *Lancet*. 2006 Jun 10; 367(9526):1920–5.

3. Robson B, Purdie G, Cormack D. Unequal impact II: Māori and non-Māori cancer statistics by deprivation and rural-urban status, 2002–2006. Wellington: Ministry of Health; 2010.
4. Ministry of Health, University of Otago. Decades of disparity III: Ethnic and socio-economic inequalities in mortality, New Zealand 1981–1999. Wellington: Ministry of Health; 2006.

### Relationship-based care: measuring the impact of patient-provider relationships on health outcomes

Katherine Gottlieb,<sup>1</sup> Michelle Tierney<sup>2</sup>

<sup>1</sup>President/CEO, Southcentral Foundation, Anchorage, United States; <sup>2</sup>Vice President of Organisational Development and Innovation, Southcentral Foundation, Anchorage, United States.

#### Background and aims

Southcentral Foundation (SCF) provides primary care for approximately 65,000 Alaska Native and American Indian people in Alaska's Cook Inlet region. In 1998, SCF implemented a reform effort to engage patients in their own healthcare and focus on strong relationships between patients and providers. The purpose of this study was to investigate whether patients who are empanelled (chose to be assigned) to a primary care provider (PCP) perceive that their relationship with that provider is important to improving their health, to evaluate the quality of that relationship and to measure the impact of patient and provider factors on the quality of the relationship and on health outcomes.

#### Methods

We carried out a survey with 2,126 empanelled patients in SCF's high-volume primary care clinics to investigate what patient and PCP factors, if any, influence the relationship between a PCP and a patient to improve health outcomes. We also measured match rate (the percentage of time the patient sees their chosen provider for primary care visit), and used the Consultation and Relational Empathy (CARE) Measure,<sup>1</sup> to assess the quality of the patient-provider relationship. We hypothesised that these factors would positively influence health outcomes, and we used multilevel regression analysis to analyse data

collected at more than one level to understand multiple relationships.

#### Results

Of the sample participants, 1,988 (93.5%) agreed that a relationship is important to improve health outcomes; 1,632 (76.8%) reported having a relationship with their PCP, and this figure was substantiated through analysis of the CARE Measures submitted by those patients. Of patients who self-reported a strong health rating, 83.1% ( $n=621$ ) reported having a relationship with a PCP, compared to 73.3% ( $n=1,011$ ) of those who did not report a strong health rating. The mean number of emergency department visits for those who responded with at least "Agree" on all items of the CARE measure was 1.9 ( $SD=2.4$ ) compared to a mean of 2.4 ( $SD=4.3$ ) for those who did not respond with at least "Agree" on all items.

#### Discussion

Patients believe that having a relationship with a PCP is important to their health, and results suggest that strong relationships between providers and patients may help patients achieve better health outcomes. Many providers lack the skills for relationship-building, so training to increase provider skills in this area and help them foster and maintain relationships with their patients is crucial. This study can serve as a basis for future research; a longitudinal study of patients with a chosen PCP is recommended. A similar study of a different patient population could also provide useful comparative data. Limitations to the research were that only patients who visited the clinics were included in the sample and that SCF has had a unique position as a relationship-focused organisation for more than 15 years, which may impact the generalisability of the research findings.

**Declaration of conflict of interest**  
None.

#### Acknowledgements

The authors acknowledge the assistance of Matthew Doogan with editing this abstract.

#### References

1. Mercer SW, McConnachie A, Maxwell M, Heaney D, Watt GC. Relevance and practical use of the Consultation and Relational Empathy (CARE) measure in general practice. *Fam Pract.* 2005; 22(3):328–334. doi: 10.1093/fampra/cmh730

### Using patients' experiences of adverse events to improve health service delivery and practice

Walton M,<sup>1</sup> Iedema I,<sup>2</sup> Manias E,<sup>3</sup> Kelly P,<sup>1</sup> Smith-Merry J,<sup>1</sup> Jorm C,<sup>1</sup> Harrison R<sup>1</sup>

<sup>1</sup>School of Public Health, Sydney Medical School, University of Sydney;

<sup>2</sup>Agency for Clinical Innovation & University of Tasmania; <sup>3</sup>Department of Nursing, University of Melbourne & Deakin University

#### Background and aims

Adverse events (AEs) can be described as injuries "related to medical management, in contrast to complications of the disease".<sup>1</sup> Evidence of the patient experience of AEs is fundamental to creating effective health policy and service responses, yet we lack this knowledge. Our research objective was to provide the first large-scale cohort investigation of the experiences of patients in New South Wales hospitals in relation to AEs.

#### Methods

A survey was developed based on previous validated patient experience survey tools and administered to a sample of 20,000 recently hospitalised patients from the 45 & Up databank.<sup>2,3</sup> The sample was identified by the Centre for Health Record Linkage using data linkage. The survey captured quantitative and qualitative data regarding: (1) patients' experiences in hospital; (2) the nature and frequency of any AEs experienced; (3) the impact of AEs on patient outcomes; (4) whether the patient experienced an open disclosure process (formal or informal); (5) whether the patient made a complaint or initiated legal action.

#### Results

Seven thousand six hundred and sixty-one patients responded with completed surveys. Of these, 7% reported an adverse event. Clinical process or procedure (29%) and medication (18%) AEs were most common. Fifty-eight percent of patients considered the harm they experienced to be moderate or severe. Only 17% of those reporting an event reported formal open disclosure in which the organisation arranged a meeting to discuss the adverse event.

Those who received formal open disclosure generally described this favourably.

**Conclusions**

Minimising harm to patients is a challenge for health services and providers. Our data demonstrates that patients can identify adverse events occurring in their care and could contribute to identifying events, providing contextual information and identifying quality of care issues if this data is routinely collected along with other incident monitoring.

**Declaration of conflict of interest**  
None.

**References**

1. World Alliance for Patient Safety (WHO). WHO draft guidelines for adverse event reporting and learning systems: from information to action. Geneva, Switzerland: WHO Publications; 2005.
2. Walton M, Smith-Merry J, Harrison R, Manias E, Iedema R, Kelly P. Using patients' experiences of adverse events to improve health service delivery and practice: protocol of a data linkage study of Australian adults age 45 and above. *BMJ Open*. 2014 Oct 1; 4(10):e006599.
3. 45 and Up Study Collaborators, Banks E, Redman S, Jorm L, Armstrong B, Bauman A, et al. Cohort profile: the 45 and up study. *Int J Epidemiol*. 2008 Oct; 37(5):941-7.

**Open for better care: lessons from a national patient safety campaign**

Price L,<sup>1</sup> York D,<sup>2</sup> Bohm G,<sup>3</sup> Stolarek I<sup>4</sup>

<sup>1</sup>Director of Communications, Health Quality & Safety Commission;

<sup>2</sup>Programme Manager, Health Quality & Safety Commission;

<sup>3</sup>Principal Advisor Quality Improvement, Health Quality & Safety Commission; <sup>4</sup>Medical Director, Health Quality & Safety Commission

**Background and aims**

Launched in May 2013, the Health Quality & Safety Commission's Open for Better Care campaign aimed to reduce patient harm by promoting

simple interventions known to make a difference to patient safety. The campaign focused on reducing harm from falls, infection prevention and control, safe surgery, medication safety and developing emerging clinical leaders for quality and safety.

**Methods**

Each area of focus was coordinated nationally with the health sector choosing from, or suggesting, a range of interventions and activities and implementing these regionally. All 20 district health boards participated in the campaign, as well as the primary and aged-care sectors. Each campaign topic ran over a six-month period, both highlighting a particular patient safety area and facilitating a local and regional focus on improving capability and sustainability.

Quality and safety markers were put in place to help evaluate the campaign and determine whether the desired changes occurred. Process measures included percentage of patients with a falls risk assessment and care plan and percentage given timely hip and knee replacements as well as percentage of surgical teams using all three parts of the surgical safety checklist. These quality and safety markers continue beyond the campaign's conclusion to measure sustainability and results are updated quarterly.

**Results**

Evaluation of the campaign examining both quantitative and qualitative measures has highlighted where the campaign has made a difference, what resonated with participants and where it could have been improved. Evaluation involved key informant interviews, tracking of web hits, document review and ongoing monitoring of quality and safety markers.

Regional improvement networks that were established have been strengthened as a result of the campaign. Specifically, the strong clinical leadership observed across all topic areas has contributed to local spread and sustainability of local patient safety initiatives. Sustainability continues to be monitored through the quality and safety markers. For example, there has been a significant reduction in in-hospital fractured neck of femur for the seventh quarter up to June 2016 and this continues to be monitored, along with other process and outcome measures.<sup>1</sup>

**Discussion**

Patient safety campaigns can effect sustainable change. The ongoing monitoring of quality and safety markers, and the continuation of regional networks provide evidence for this. A multi-layered approach, engaging both 'hearts and minds' is more likely to deliver real improvement. Key to the success of this campaign was strong local buy-in and dedicated clinical leadership.

This campaign has contributed to raising the profile of patient safety in New Zealand and galvanised support. The spread of campaign activities and messages continues beyond the life of the campaign. The implications for patients include reinforcing a system that focuses on quality and patient safety. For true sustainability to occur, local leadership at all levels will be essential to keep this focus.

**Declaration of conflict of interest**  
None.

**References**

1. Health Quality & Safety Commission New Zealand. Quality and safety markers [website]. Updated 30 June 2016. Available from: <http://www.hqsc.govt.nz/our-programmes/health-quality-evaluation/projects/quality-and-safety-markers/>

**URL:**

<http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2017/vol-130-no-1450-17-february-2017/7169>