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***Erysipelothrix rhusiopathiae* bacteraemia in an immunocompromised host: the unexpected complication of a crustacean altercation**

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Differing protocols of managing adult diabetic ketoacidosis outside of the intensive care unit make no difference to the rate of resolution of hyperglycaemia and acidosis

Geoffrey Braatvedt, Alfred Kwan, Will Dransfield, Catherine McNamara, Cameron Schauer, Steven Miller, Manish Khanolkar

This study has shown that different protocols of insulin infusion in people with type 1 diabetes admitted to hospital with ketoacidosis made no difference to the rate of resolution of the metabolic abnormalities, nor length of hospital stay.

A cost-effectiveness analysis of the Prediabetes Intervention Package (PIP) in primary care: a New Zealand pilot programme

Deborah Connor, Kirsten Coppell, Andrew Gray, Trudy Sullivan

A prediabetes programme delivered by primary care nurses—called the Prediabetes Intervention Package (PIP)—was piloted in Hawke's Bay in New Zealand. The costs and benefits of PIP were com-pared with standard care. We found that the intervention is likely to be a cost-effective weight loss strategy for preventing or delaying progression to type 2 diabetes in people with prediabetes.

The cost of diabetes-related hospital care to the Southern District Health Board in 2016/17

Kirsten J Coppell, Shaun J Drabble, Janine A Cochrane, Rosemary A Stamm, Trudy A Sullivan

We estimated the cost of diabetes hospital admissions to Dunedin Hospital and Southland Hospital for the year 2016/17. There were 6,994 separate diabetes-related hospital admissions costing a total of NZ$41M. Where diabetes was coded as the main reason for hospitalisation the cost was NZ$2.2M, compared with NZ$8M if diabetes was coded as the main underlying reason for hospitalisation. A high proportion of the costs were attributed to cardiovascular disease, eye disease and diseases of the musculoskeletal system, many of which were related to diabetes. Hospital costs are likely to increase as the number of people diagnosed with diabetes increase unless interventions that target preventable diabetes-related admissions and programmes to prevent diabetes are prioritised.

An open-label feasibility study of repetitive transcranial magnetic stimulation (rTMS) for treatment-resistant depression in the New Zealand healthcare context

Kate EM Godfrey, Suresh D Muthukumaraswamy, Cathy M Stinear, Nicholas R Hoeh

A significant number of people in New Zealand struggle with clinical depression. People with clinical depression have difficulties with employment, maintaining healthy relationships, and have an in-creased risk of suicide. Many people with clinical depression do not respond to treatments with multi-ple trials of medications and talk therapy. Treatment with repetitive transcranial magnetic stimulation (rTMS) is established as a helpful and safe treatment which is widely available internationally. Addi-tionally, rTMS can be safely used in combination with other types of treatment. However, currently the majority of the New Zealand population has almost no realistic way to access rTMS treatment. This study demonstrates that treatment with rTMS can be helpful and safely provided to many people in New Zealand struggling with clinical depression.

The burden of alcohol-related presentations to a busy urban New Zealand hospital emergency department

Georgina Svensen, Bridget Kool, Sarah Buller

This study analysed electronic patient records for adult alcohol-related presentations (ARPs) to a major New Zealand emergency department (ED) for a 12-month period (November 2017 to October 2018). Among 73,381 presentations, 7% were alcohol-related, the majority were male (65%) and aged 20–39 (52%). ARPs were more frequent at night, during the weekends, public holidays and over the summer months. Sixteen percent of injury-related presentations were alcohol-related. ARPs commonly arrived at the ED via emergency services and had a longer length of stay than non-ARPs. Continued public health efforts are required to implement preventative strategies for alcohol-related harm in the ED and society as a whole.

Wintertime Vitamin D status and its related risk factors among children living in Auckland, New Zealand

Maryam Delshad, Kathryn L Beck, Cathryn A Conlon, Owen Mugridge, Marlena C Kruger, Berit P Jensen, Jing Ma, Pamela R von Hurst

This study showed that approximately one-third of our population had vitamin D less than recommendations. Poor vitamin D status during childhood can affect long-term health, so opportunities to intervene during childhood should be pursued. A strong consideration should be given to the high-risk children for whom supplementation may be considered especially in wintertime.

Gender and the surgical profession

Angela D McGregor

Gender imbalance exists in the surgical profession, meaning there are more barriers for women to overcome when pursuing a career in surgery compared to men. The gender imbalance is rooted in our stereotypical expectations of men and women and is perpetuated by extensions of these expecta-tions. It is important for the surgical profession to address this gender imbalance so that the character-istics, values and experiences women bring to surgery can be combined with those of men. That way we can appreciate our differences, learn from and build on each other's strengths, and work together for improved patient care.

On personal responsibility in medicine

Helen Ker

It's commonly said that doctors treat patients only to send them back into the environments that made them sick. Encouraging patients to make behavioural changes despite these environmental in-fluences is very difficult, and is hindered by our narrow working construct of personal responsibility, largely based on the Western construct of self-contained individualism. Through recognising alterna-tive notions of self and responsibility, while advocating to improve the social determinants of health, we can better enable patients to enjoy greater health.

The motivations behind science denial

Alan McLintic

Cultural cognition is where we view information through the lens of our worldview. Worldview is a term for our deeply engrained values and beliefs that give us our sense of self, group identity and how we want society to be.

New Zealand needs a comprehensive interpreting service

Ben Gray

Abdelfattah Qasem was one of the people killed in the Christchurch Mosque shootings. He worked as an interpreter. In response to the shootings there has been considerable commitment expressed to make New Zealand a more inclusive society. Current provision of interpreting services is woefully inadequate and it is common for people with limited English proficiency to receive inferior care. Establishing a fully funded national interpreting service would be a fitting way to mark Abdelfattah Qasem's passing and a tangible way to improve the integration of minority communities into New Zealand.

A measles epidemic in New Zealand: Why did this occur and how can we prevent it occurring again?

Nikki Turner

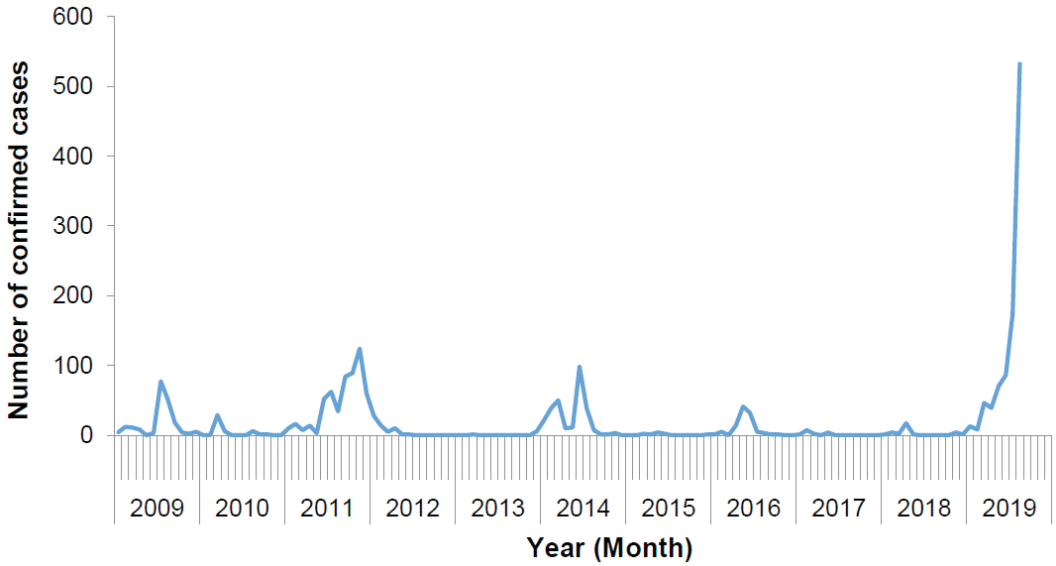
In 2019 New Zealand has seen an upsurge in measles cases, the largest for more than two decades (refer Figure 1). From 1 January to late September 2019 there have been over 1,500 confirmed cases of measles notified across the country of which over a third have been hospitalised.[1] As of September, rates of measles in New Zealand were the second highest in the Western Pacific region at 152.4 per million, with only the Philippines having higher rates at 612.1 per million.[2] With multiple imports and more than 12 recognised outbreaks in the first five months of this year affecting most regions,[3] this should appropriately be called an epidemic.

The pattern of the epidemic

Cases have been reported in 16 of the 20 DHBs. The majority of these have been controlled with effective public health measures. However, in the Auckland metropolitan region, particularly centred on the Counties Manukau District Health Board (DHB) area numbers from two separate outbreaks quickly multiplied and overwhelmed the ability of public health services to control the spread.

Up to September more than 80% of the notifications in 2019 have been from the Auckland metropolitan region, of which over two-thirds have been from Counties

Figure 1: Number of measles notifications Jan 2019 to Sept 2019.



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Manukau DHB. The burden of disease is highest in young infants, particularly under two years of age, followed by teenagers and young adults under the age of 30 years. Disease in those over 50 years of age is rare with only 25 (1.6%) notifications to date. Māori and Pacific ethnicity populations are disproportionately affected with Pacific being 36% and Māori being 39.6% of those hospitalised.[1] The circulating genotype distribution are B3 (62%) and D8 (38%).[1] These are the dominant circulating genotypes in this region with B3 in the Philippines, D8 in Vietnam and a mix of the two in other countries in the region.[2] By vaccination status, nearly 14% of non-hospitalised cases were fully vaccinated (includes children under four years with only one dose), while a significantly lower number at 6.7% of hospitalised cases were fully vaccinated. This is consistent with other literature showing that secondary vaccine failure is associated with modified clinical illness.[4]

A September report on the 380 hospitalisation cases in the Auckland metropolitan region records an overall hospitalisation rate of 36%, but up to 52% in infants under four years of age and highest in the Counties Manukau DHB. Nearly half of all hospitalised were Pacific ethnicity.[5] Overall 22% were considered complicated and included three cases of encephalitis all in young children, 65 with pneumonia, most in young children and five pregnant women of whom two had second trimester fetal losses. There have been no deaths reported to date. While not as high as these rates, previous New Zealand outbreaks have also noted higher rates than other international figures; in the 2013/2014 outbreak, 23% were hospitalised.[6] These hospitalisation rates are higher than most internationally quoted figures of around 10–20%. Internationally published figures report around 30–40% of cases results in one or more complications with the highest rates in children, pregnant women and adults over 20 years of age. These are consistent with this current pattern.[7,8] The one exception is the high encephalitis rate,[9] however the numbers are small.

Background

Prior to 2019, the most recent measles epidemic in New Zealand occurred in 1991 with an estimation of tens of thousands of cases, followed by 1997 with 2,169 cases notified. Since then smaller outbreaks have occurred in 2009, 2011, 2014 and 2016, with the largest of these being in 2011 with 489 cases.[9]

New Zealand was verified by the World Health Organization (WHO) as having eliminated endemic measles in October 2017 having demonstrated the absence of local transmission for more than three years. However, it was acknowledged at the time there was was at risk of further transmission due to known immunity gaps across the population.[10]

New Zealand historically had low immunisation coverage in the childhood programme with significant equity gaps. In 1991 less than 60% of children were fully immunised for all the Schedule vaccines by two years of age with only 42% of Māori and 45% of Pacific children fully immunised. Gains were made over the following years so that by 2005, 77% of children were fully immunised by the two years milestone.[11] These historic figures suggest there are likely to be large numbers of young and mid-life adults with inadequate or no immunity to measles. The amount of catch up that may have occurred is unknown, and records are frequently absent. Confirming the immunity gap, a 2014/2015 serosurvey showed systematically lower measles seropositivity for the birth cohort from 1980 to 1999 with IgG seropositive or equivocal sitting at around 83.4 to 84.6%.[3]

The National Immunisation Register (NIR) was instigated for birth cohorts from 1995. This shows MMR coverage of dose one (MMR1) was around 86% for those born in 2006 rising to over 93% in 2012, but in more recent years a 1–2% drop off. The second dose MMR (MMR2) rose from 82.6% for those born in 2006 to 87.7% for the 2012 cohort. Coverage rates got close, but have never reached the national target of 95%, and equity gaps persist by ethnicity, socioeconomic status and region.[3]

Most of the historic low coverage was due to a poorly performing immunisation programme.[11] This had possibly been aggravated with some loss of confidence in the late 1990s following the discredited hypothesis of a link between MMR and autism. The current New Zealand programme performs well, although not fully reaching the national coverage targets,

with slippage in the past three years in the infant programme, particularly for Māori.[12] A lot of media attention has been given to the possibility of increasing fears creating hesitancy and delay in accepting vaccination both internationally and in New Zealand. Current data on those who chose to decline vaccination on the NIR record has increased by 1 to 1.5% since 2017, suggesting a small added contribution.

So why do we have so much measles?

Overall, the world has made tremendous progress in the control of measles and by 2019, 43% (89 of the total 194 countries) have achieved elimination status. Deaths from measles have decreased by 80% from 2000 to 2017 as a result of vaccination, averting about 21 million deaths since 2000. Despite this progress, vaccination coverage has levelled off in the past eight years and since 2017 there has been a resurgence of measles with outbreaks in many parts of the world and resultant importations to many countries.[13] Reasons for these outbreaks include increased conflict and migration, climate change, increasing inequities in wealth, health and security, alongside increasing circulation of misinformation leading to distrust and reduced vaccination uptake.[14]

In the New Zealand context there are two clear issues. Firstly, the historically low immunisation coverage has left a legacy of large numbers of adolescents and young adults who are under or unimmunised, particularly of Māori and Pacific ethnicity, often with unknown immunisation records. Secondly, recent declines in immunisation coverage and increasing equity gaps in the infant programme. The pattern of this epidemic and the groups most affected is consistent with these two concerns. However, there are particularly high rates of hospitalisation for Pacific communities. Other broader issues are likely to be a factor such as insecure and crowded housing and high-density urban settings in Auckland.

The hospitalisation rate is surprisingly high. However, except for encephalitis, the rates of complications are in line with international expectations, and there have been no deaths to date despite over 1,500 cases. Reasons for hospitalisation are more likely to include differences in how New Zealand healthcare identifies and admits cases compared to other countries. There is no evidence that the viruses in circulation are behaving differently from elsewhere. While secondary vaccine failure is seen, it does appear to modify the severity of the clinical illness.

Management strategies

The New Zealand National Verification Committee (NVC) for measles and rubella was established in 2016. In May 2017 they reported there was a need to *ensure that existing significant pockets of susceptible individuals are identified and immunised to avoid or minimise further outbreaks…* and *to undertake MMR catch up/supplementary immunisation activities*.[10] A further report in July 2019 reported that the New Zealand elimination status was threatened due to the *lack of progress with closing immunity gaps, multiple measles importations and outbreaks in many parts of New Zealand*.[3]

The recent reduction in coverage rates and increase in equity gaps for the childhood programme is of considerable concern. The environment locally and internationally is changing. Immunisation is primarily delivered in general practice. The effects from socioeconomic deprivation have entrenched and increased in some New Zealand communities, creating issues such as crowding and housing instability. This affects enrolment, engaged relationships and ease of access to general practice. There can be challenges for working parents in accessing general practices usually only open weekdays. Internationally and locally, issues around trust—trust in governments and trust in health services—appear to be growing, amplified by social media connectivity. Outreach services report increasing challenges for families in accessing services, at times alongside some elements of fear and mistrust. The root causes are multifactorial and the solutions needs to be innovative, flexible and responsive to both health sector and community challenges.[15]

It is not easy to close historic immunity gaps. A national campaign targeting adolescents and young adults requires significant planning and resourcing. This includes adequacy of vaccine supply; increased support to busy front-line health service delivery; improved vaccination accessibility such as more use of pharmacy, pop-up clinics and occupational health providers.

Furthermore, this age group are notoriously hard to reach, motivate and vaccinate—they tend to have other life priorities! Calls for action can create overvaccination in 'worried well' and still miss the unvaccinated.

The community response to this epidemic has been strong and positive. For example, the national Healthline phone line reported an overall call volume up by 60–80%, the child Healthline (Plunketline) a nearly 50% increase and the healthcare provider line (0800 IMMUNE) 160% increase through the peak of the epidemic.[16] For a single issue these are dramatic call volume increases. Vaccine demand has also been very strong with the Ministry of Health reporting nearly a doubling of vaccine uptake in the months of August and September resulting in vaccine shortage.[17] International examples have shown public attention and support can be used to assist strengthening of national immunisation programmes.[18]

Conclusions

With a mixture of increased transmission from international sources, ease of travel and a population with recognised immunity gaps, the current measles epidemic in New Zealand was not surprising. Ensuring better protection for the New Zealand community and moving towards effective long-term measles elimination will require both innovative new thinking to strengthen the current immunisation programme, particularly focusing on our higher-risk populations groups, alongside an active national approach to systematically close the immunity gaps in teenagers and young adults. This year the New Zealand population has demonstrated a high demand for measles-containing vaccine, an opportunity to build upon. Measles currently remains a risk both to the New Zealand population and also to our Pacific neighbours. There are clear opportunities to build on the strong public and health sector response to address these issues.

Competing interests:
Nil.

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Differing protocols of managing adult diabetic ketoacidosis outside of the intensive care unit make no difference to the rate of resolution of hyperglycaemia and acidosis

Geoffrey Braatvedt, Alfred Kwan, Will Dransfield, Catherine McNamara, Cameron Schauer, Steven Miller, Manish Khanolkar

ABSTRACT

AIMS: To compare the outcome of people with type 1 diabetes admitted to the general ward with diabetic ketoacidosis (DKA) to two hospitals in Auckland, using different protocols of care.

METHODS: North Shore Hospital uses a UK weight-based, ketone centric protocol while Auckland Hospital uses a protocol based on glucose measurements only. All notes of people over 16 years of age admitted to the general wards with DKA to these hospitals in one year were reviewed and their outcome compared.

RESULTS: Forty-one admissions in 35 people with DKA at Auckland Hospital were compared to 30 admissions in 26 people with DKA at North Shore Hospital. The degree of ketoacidosis and hyperglycaemia on admission was similar at the two hospitals. The duration of insulin and 10% dextrose infusions was similar but the total number of units of insulin infused and rate of dextrose given per hour were higher at North Shore, with similar rates of hypokalaemia and hypoglycaemic events at each site. The rate of resolution of hyperglycaemia and acidosis did not differ. The length of stay of patients was similar at the two hospitals.

CONCLUSIONS: The frequent measurement of bedside ketones did not result in more rapid resolution of DKA compared to relying on glucose measurements alone.

Diabetic ketoacidosis (DKA) is a life threatening, complex metabolic disorder complicating diabetes and is a common cause for admission to acute medical units. We have previously[1] reported our 23-year experience of managing DKA at Auckland Hospital which extended observations from a previous report.[2] This showed that there was a substantial reduction in length of stay (LOS) and need for intensive care unit (ICU) admission with low in-hospital mortality over the two decades of observation, although high rates of subsequent readmission with DKA has remained an issue.[3] Over this time there was no change in the DKA management protocol, which remained "glucose centric", in that decisions regarding insulin administration were made primarily based on current blood glucose rather than pH or ketone concentrations. The severity of acidosis and degree of hyperglycaemia at presentation was less in recent years,[1] perhaps reflecting more widespread use of long-acting analogue insulins, better education of people with diabetes and easier access to care among other factors, which

could have contributed to the reduced need for ICU admission and shorter LOS. However, the LOS and ICU admission rate for those with severe DKA (pH ≤7.1) also showed a trend downwards in recent years, suggesting factors other than the DKA management protocol itself have led to the improvement in outcomes, such as better immediate care in the emergency department.

In 2011, the Joint British Diabetes Societies Guideline for the Management of DKA was published[4] with a shift away from a glucose-centric protocol to a more ketone-centric one, with the recommendation to commence a weight-based, fixed dose insulin infusion until ketosis (measured at the bedside) clears. The evidence that this approach results in better outcomes is however limited.[5] A recent UK audit of the ketone-based protocol in managing 50 episodes of DKA[6] showed that hypokalaemia occurred in 46% of people, but that the protocol for potassium replacement was not followed well. Forty percent of people had a hypoglycaemic episode at a median time of approximately 13 hours after the insulin infusion was started, despite 80% correctly receiving IV dextrose as per protocol. Moreover, the switch from IV to subcutaneous insulin was appropriately managed in only 34% of cases. This study suggests that the protocol may not be easy to follow and does not prevent hypokalaemia and hypoglycaemia, possibly due to a relatively high insulin infusion rate. New Zealand is currently attempting to develop a nationally agreed protocol of care for the management of DKA with some advocating the adoption of the UK protocol of care.

Following publication of the new UK DKA guideline, North Shore Hospital in Auckland changed their DKA management protocol from a glucose-centric one to the UK, more ketone-centric protocol in 2012. This allowed a comparison in outcome of people subsequently admitted with DKA treated at the two hospitals using two different protocols.

The optimal rate of correction of the metabolic abnormalities of DKA is unclear. Very rapid correction of hyperglycaemia can lead to hypoglycaemia and hypokalaemia, and the resulting rapid change in osmolality can increase the risk of cerebral oedema especially in children.[7–9] We were thus interested in comparing the performance of the two protocols in correcting the metabolic abnormalities of people admitted with DKA to the two hospitals in the year following implementation of the new protocol at North Shore Hospital.

Methods

The management of all people aged 16 years and over admitted with DKA from 1 January 2013–31 December 2013 to both hospitals were compared. DKA was defined as a laboratory glucose ≥11.1mmol/l, **and** pH ≤7.30 **and** bicarbonate ≤15mmol/l **and** raised beta hydroxybutyrate ≥3mmol/l. Each episode of DKA was treated as a discrete event, and thus could include multiple episodes in an individual person. A beta hydroxybutyrate >8mmol/l is reported as ">8" at Auckland Hospital's laboratory. The admissions of people who were pregnant, had end-stage renal disease, had type 2 diabetes, or who were transferred from another hospital or who were admitted to ICU (as they may have initially been treated by a different insulin infusion protocol) were excluded from the analysis. ICU admission criteria is similar between the two hospitals, with admission to ICU guidelines including significant haemodynamic or electrolyte abnormalities or pH <7.1 as noteworthy criteria.

The two hospitals serve similar sized populations of about 400,000 (although Auckland Hospital has a higher proportion of people of non-European ethnicity), are both teaching hospitals attached to Auckland Medical School, and share a pool of general medicine registrar trainees who rotate through each hospital for 6–12 months at a time. People domiciled in the catchment area of each hospital are admitted directly to that hospital. Substantial training of emergency department, medical and nursing staff in the use of the new protocol was undertaken during 2012 at North Shore Hospital, and 16 ketone meters for measuring ketones at the patient's bedside were purchased (at a cost of $69 each). Ketone measurement at Auckland hospital was done in the main laboratory, but at North Shore only the admission ketone value was measured in the laboratory, with all subsequent tests being done using the point-of-care meter at the bedside.

The Auckland hospital protocol encourages the continuation of long-acting insulin in those already on such an insulin. The insulin infusion rate is based on the prevailing glucose concentration rather than on patient weight or ketone concentration. The usual default first scale insulin infusion rate typically starts at 6 to 12 units per hour. If the hourly measured capillary glucose levels are not falling, the rate of insulin infused per hour is rapidly escalated by protocol and on a variable scale. Insulin continues to be infused alone until the capillary glucose falls to <15mmol/L, when insulin is continued and 10% dextrose IV is added at 80ml per hour. Fluids containing varying concentrations of potassium are also infused according to the person's renal function and potassium concentration. Once the person is eating and drinking and glucose values are stable, subcutaneous insulin is commenced and the insulin infusion is weaned to stop. While the protocol recommends "frequent" measurement of venous bicarbonate, potassium and pH, there is no requirement to repeat the measurement of plasma beta-hydroxybutyrate after the initial diagnosis of DKA.

The North Shore protocol states that people should be given subcutaneous long-acting insulin (0.25units/kg) on the evening of admission or the next morning, and the IV insulin infusion commenced at 0.1 unit per kg of body weight/hr as soon as possible. Adjustments to the rate of infusion (1 unit per hour, every hour) are based on a combination of hourly measurements of ketones and glucose using point-of-care meters at the bedside. Venous blood gas analysis is recommended five times in the first 24 hours. When the glucose falls to <14mmol/l, 10% dextrose is commenced at 100ml/hr and both infusions are continued until the ketones have cleared and the patient is eating and drinking.

People admitted to both hospitals with DKA are seen first either by the emergency department staff before being transferred to the general medical "team of the day", or they can be admitted directly to general medicine if the GP calls ahead of their arrival. They are managed in the acute assessment areas until stable, and then transferred to a general medical ward (or ICU). Specialist diabetes nurse or diabetologist involvement in patients' care is by referral from the general medical team and recommended in all cases within 24 hours of admission but is only available during the week (Monday to Friday) and within normal working hours (0800–1700). Acute admissions at Auckland Hospital were allocated at 0800 each morning between four teams that were on a roster to receive all acutely admitted general medical patients (including those with DKA) admitted within the previous 24 hours. Of the 30 physicians contributing to the roster at that time, six had specific advanced training in diabetes; people with DKA were thus not specifically directed to a team with a diabetologist. At North Shore Hospital there were 15 general medical teams, with four including a physician with specific advanced training in diabetes; newly admitted patients were allocated between three teams at 0800 each day.

All people in New Zealand have a unique National Health number, which facilitates retrieval of their medical records. All records and investigations at Auckland hospital are stored electronically, but hard copy notes are still in use at North Shore (although letters and all investigations are also stored electronically). The notes (hard copy or electronic) of people admitted with DKA were examined and all relevant data extracted in retrospect.

The primary end points of interest were rate of correction of acidosis and hyperglycaemia with secondary end points being incidence of hypoglycaemia, hypokalaemia and length of stay (LOS).

Audit studies such as this are approved by the local hospital ethics committees.

Results

Data was compared using t, Chi squared, or Mann-Whitney tests and results are presented as mean ±SD (range) or when not normally distributed, as median (95% CI or range). Forty-one admissions of 35 people admitted to Auckland Hospital met all the criteria for inclusion in the study. A further 17 admissions were excluded: two were transferred from another hospital during the admission, six were admitted to ICU and nine had type 2 diabetes. Thirty admissions of 26 people were included at North Shore Hospital. A further 13 admissions were

Table 1: Details of admissions of people with type 1 diabetes presenting with DKA to Auckland City and North Shore Hospitals in 2013.

	Auckland n=41	North Shore n=30	P value
Age years	25 (22–30)	24 (19–28)	0.6
% female	51	57	0.61
Duration diabetes months #	84 (56–111)	116 (77–157)	0.28
Newly diagnosed %	17	7	0.20
Daily insulin dose #	65 (52–72)	54 (46–64)	0.07
Ethnicity European %	44	70	0.03
Pacific %	15	7	0.3
Maori %	29	17	0.24
Other %	12	6	0.4
Glucose mmol/l	29±8.8	30±11.2	1
pH	7.16 (7.08–7.20)	7.12 (7.1–7.18)	0.98
Bicarbonate mmol/l	11.8±3.9	10.2±5.6	0.16
Anion gap	26.6±4.8	32±6.1	0.0005
Beta hydroxybutyrate mmol/l	56 %>8	40% >8	0.28
Lactate mmol/l	2.9 (2.5–3.31)	2.6 (1.76–3.5)	0.47
Admitted Saturday/Sunday %	24	33	0.41
Admitted 8am–5pm %	51	67	0.18
HbA1c in previous six months mmol/mol	95±27 (n=32)	94±22 (n=26)	0.9
HbA1c during admission mmol/mol	119±32 (n=19)	104±13 (n=7)	0.24
Creatinine (range) umol/l	110±35 (44–201)	93±31 (52–157)	0.04
Systolic BP mmHg	137±25	135±19	0.71
Diastolic BP mmHg	77±13	73±15	0.23
Sodium (range) mmol/l	135±3.3 (129–142)	133±3.4 (120–138)	0.02
Potassium (range) mmol/l	5.0±1.1 (3.1–7.70)	4.84±0.95 (3.3–7.1)	0.5

Data are mean ± SD (± range) or median (95% CI of median). # = in those with known diabetes at presentation.

excluded: six were transferred from another hospital and seven were admitted to ICU.

The severity of biochemical derangement and clinical features of the people admitted to ICU was very similar between the hospitals; pH 7.04±0.1 Auckland vs 7.02±0.14 North Shore, glucose 35.3±8.8 vs 41.5±21mmol/l, anion gap 32±5.3 vs 32±10 and lactate 4.2±1.95 vs 3.2±1.8mmol/l. This suggests that the criteria for admission to ICU were very similar and that patients looked after on the general medical wards at the two hospitals were of similar acuity.

On admission, the degree of hyperglycaemia, pH and bicarbonate were not different between the two cohorts, but the anion gap was larger at North Shore, suggesting a greater degree of ketosis (Table 1). A similar proportion of people were admitted after-hours or at the weekend. There were more people of European ethnicity admitted with DKA to North Shore than to Auckland Hospital (Table 1), reflecting the different ethnic makeup of the two catchment areas. In the 12 months before this study, 11 (31%) of the people admitted to Auckland Hospital had had 22 admissions with DKA and nine (36%) of the people admitted to North Shore Hospital had had 12 admissions with DKA (data not shown).

Insulin omission/error or non-adherence was the cause of DKA admission in 41% of people at Auckland Hospital and 27% at North Shore, infection in 20% and 37% and alcohol or drug excess in 10% and 13% respectively. A variety of miscellaneous causes of DKA was identified in the remaining people.

Treatment of DKA—Table 2 and Figures 1 and 2

The number of bedside glucose measurements done in the first 24 hours was higher at the North Shore site but slightly more venous blood gases were done at the Auckland site. By protocol, very few ketone measurements beyond those obtained at baseline were done at Auckland Hospital. A total of 385 point of care tests for ketones were done in the 30 DKA admissions at North Shore Hospital. As seen in Figure 2, the concentration of ketones in people admitted to North Shore fell rapidly to the target of <0.5mmol/l.

In some people, there was considerable delay in starting IV insulin after presenting to hospital at both sites, and especially at the Auckland site. Review of the admissions with the longest delays revealed a variety of reasons, including misjudging the severity of DKA in a person whose glucose was "only 18mmol/l", not considering DKA in two people with newly presenting diabetes admitted with "gastroenteritis", and delay in considering DKA in a person with known type 1 diabetes as "the patient looked well". For some admissions there was no clear reason for the delay and this may have reflected the busyness of the emergency department at the time.

The volume of saline infused in the first four hours was similar between the two hospitals but more saline was infused in the subsequent 20 hours at the North Shore site. The amount of IV insulin infused was greater at North Shore in the 4–24-hour period, consistent with the protocol, but the duration of insulin infusion was very similar between the two sites. In the first 24 hours of admission, 20 of 41 people at Auckland had a protocol scale change (minor adjustment up or down) and three people had three or more scale changes. At North shore only three people had their initial infusion rate changed in the first 24 hours.

In the first 24 hours of admission after DKA, 25 of 41 people at Auckland and all 30 people at North Shore received a long acting subcutaneous injection of insulin (usually glargine). A similar amount of potassium was infused in the first 24 hours at both sites. The numbers of patients with documented hypokalaemia (<3.5mmol/l) was similar between the two sites. Although very few were <3mmol/l, the proportion of patients with a potassium at any time <3.5mmol/l, was relatively high at both sites suggesting the protocol for infusion of potassium needs updating. Two patients had a glucose <4mmol/l at any one time at Auckland and three at North shore. All were minor and easily corrected.

The time when 10% dextrose was started after admission and the duration of the infusion was no different between the sites. The volume and rate of infusion of 10% dextrose was however higher at North Shore by protocol. The rate of fall of glucose, and rise of pH and bicarbonate were nearly identical at the two sites (Figure 1).

The number of people with DKA seen as an inpatient by a specialist diabetologist was significantly higher at the Auckland site. This may account for the trend seen in weekend discharge rates between the hospitals as the inpatient review by the diabetes team only occurs during working hours Monday–Friday.

The LOS was similar at the two sites. One patient was readmitted to Auckland hospital within 48 hours of discharge (not with DKA) and there were five readmissions of four patients in days 3–28 at Auckland and four readmissions of four patients at North Shore, with a variety of medical illnesses (data not shown). No patients were readmitted with DKA within 28 days.

One patient died from overwhelming sepsis 40 hours after their admission at North Shore.

Table 2: Treatment and outcome of people with type 1 diabetes admitted to Auckland and North Shore hospitals with DKA in 2013.

	Auckland n=41	**North Shore n=30**	**p value**
Number glucose checks in 24hrs (range)	19±3.2 (13–25)	23±4.7 (12–30)	<0.001
Number blood gases in 24 hrs (range)	7±3 (1–13)	5.7±2 (2–9)	0.04
Number ketone checks in 24 hrs #	3	385	<0.001
IV potassium infused (mmol) 0–4 hrs	9.4±13	13.1±16	0.29
4–8 hrs	13±11.3	19±19	0.10
8–24 hrs	45±33	43±25	0.78
Number potassium <3.5 mmol/l 0–6 hrs	29%	20%	0.4
6–12 hrs	41%	47%	0.6
12–18 hrs	24%	27%	0.8
18–24 hrs	20%	32%	0.3
Time to start insulin infusion (min)	41 (30–76) Range 3–420	37 (25–49) Range 7–155	0.55
Duration insulin infusion (hrs)	22 (18–28) Range 7–84	26 (18–32) Range 2–145	0.6
Units insulin infused IV 0–4 hrs	21±16	23±9	0.54
4–8 hrs	21±10	27±10	0.01
8–24 hrs	47±26	75±44	0.001
Volume saline IV (L) 0–4 hrs	2.54±1.44	2.36±1.1	0.57
4–8 hrs	1.19±0.75	1.6±0.74	0.03
8–24 hrs	2.1±1.04	3.44±2.8	0.006
Time 10% dextrose IV given after admission (hrs)	4.7 (3.9–6.1)	3.7 (3–5.2)	0.2
Volume 10% IV dextrose (L)	1.32 (1–1.5)	1.8 (1.5–2)	0.04
Duration (hr)	17.2 (14–24)	19.8 (15–25)	0.86
Rate (ml/hr)	75 (61–79)	84 (76–114)	0.002
% seen by diabetes nurse as inpatient	71	60	0.34
% seen by specialist diabetologist	59	30	0.02
% discharged Saturday/Sunday	12	27	0.11
Length of stay (hrs)	64 (47–77)	41 (27–57)	0.11

Data are mean ± SD (range) or median (95% CI). # after the initial admission baseline measurement.

Figure 1: Glucose, pH, bicarbonate and potassium concentrations of people with DKA admitted to Auckland City Hospital (n=41) and North Shore Hospital (n=30) and treated with an intravenous insulin infusion.

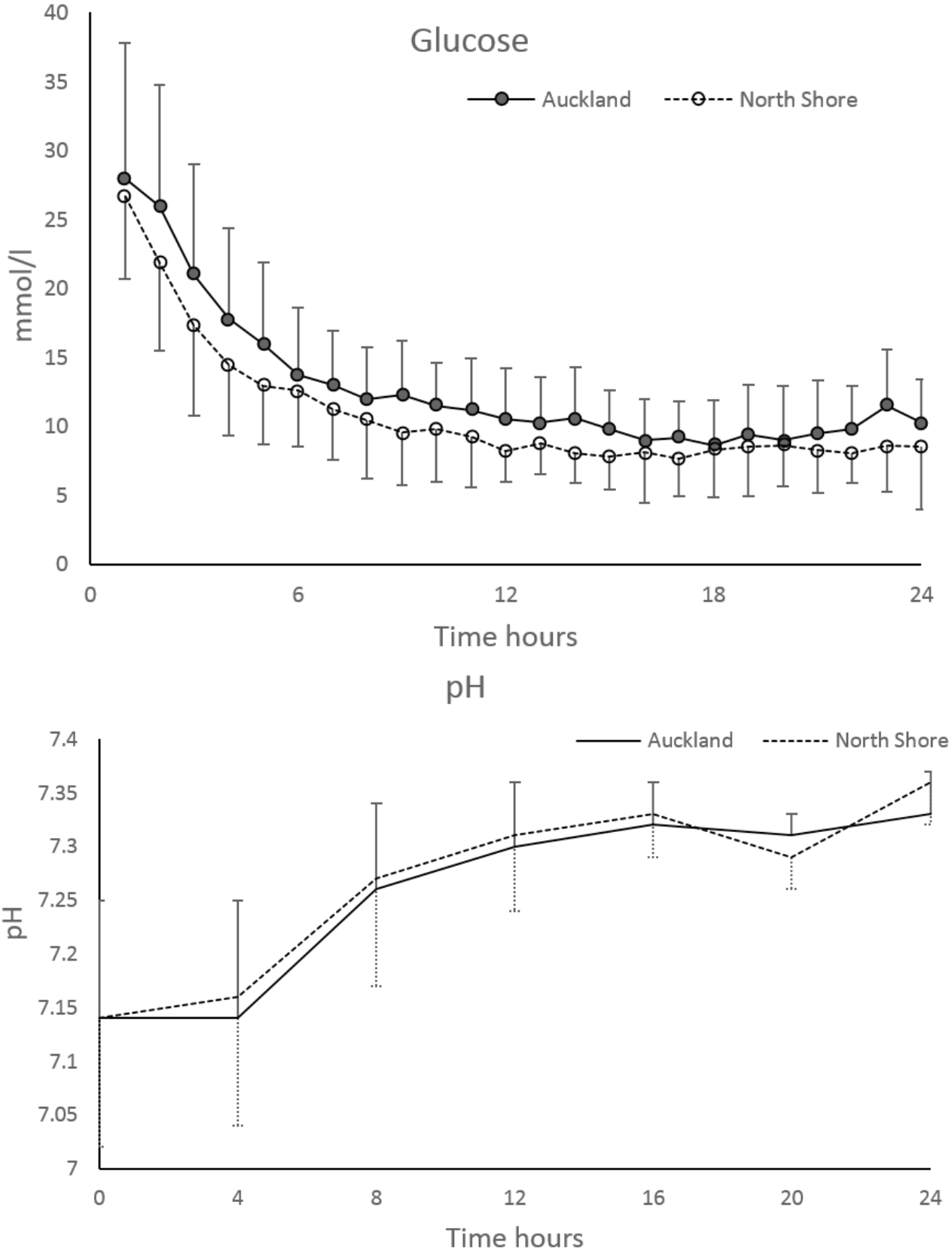
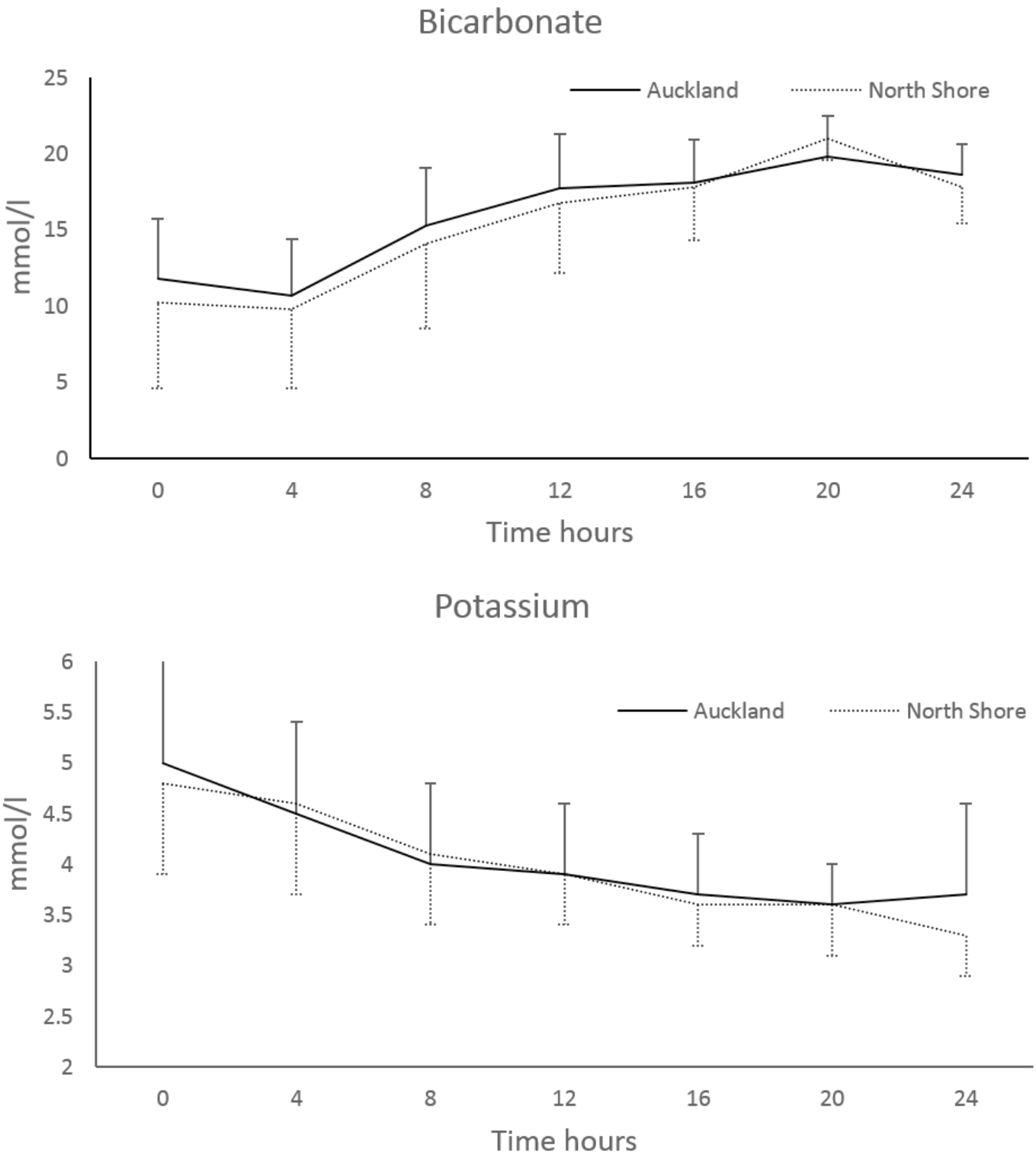
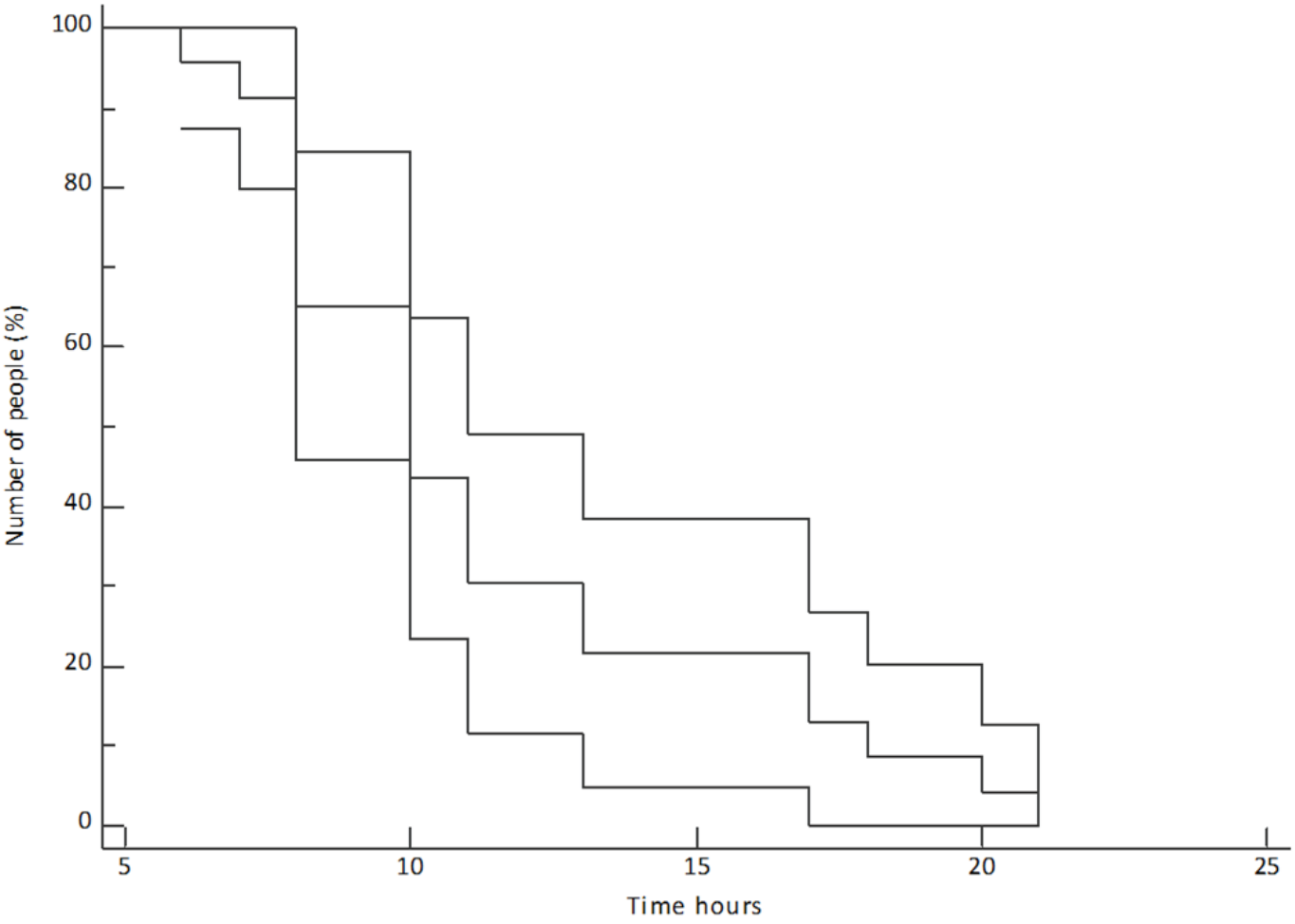


Figure 1: Glucose, pH, bicarbonate and potassium concentrations of people with DKA admitted to Auckland City Hospital (n=41) and North Shore Hospital (n=30) and treated with an intravenous insulin infusion (continued).



Data are mean ± SD.

Figure 2: Kaplan Meier graph showing time for ketone concentration to reach <0.5mmol/l in 30 episodes of DKA at North Shore hospital.



Data are mean ± 95%CI.

Discussion

Previous studies have shown that following protocols of care for complex disorders such as DKA improves outcome and many different protocols for management of DKA have been published.[10–15] There is consensus that relatively low-dose IV insulin infusion, along with vigorous fluid resuscitation and close monitoring of patients is important. Recent studies from the UK[6,16] have shown, however, that standard protocols for the management of DKA are in fact poorly followed. The recent UK Guidelines [4] 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 any large and robust randomised controlled trials examining if this has advantages for patient outcomes that matter—mortality and length of stay.

There were few overall differences in the outcome in the first 24 hours of people admitted with DKA between the two hospitals in our study. Both hospitals have considerable room for improvement in the time to start an insulin infusion. At Auckland Hospital, 40% of people did not receive a subcutaneous injection of long-acting insulin

in the first 24 hours of admission despite the protocol encouraging its use. There were no patients on sodium–glucose co-transporter inhibitors at the time of this study to account for possible insidious presentations of DKA. The admission weight of people with DKA was not routinely measured (16 of 30 admissions North Shore, 18 of 41 admissions Auckland) and thus the dose of insulin infused per hour at North Shore was based only on a bedside estimate of weight. Both protocols saw a number of patients develop hypokalaemia but this was mild and easily corrected. There were very few episodes of hypoglycaemia at both sites, and all were mild. The protocol at North Shore hospital stipulates that the rate of infusion of 10% dextrose is 100ml/hr, but this was not followed in many patients (Table 2) for unknown reasons. This may have led to some confounding of results.

By protocol the North Shore patients had many bedside point-of-care measurements of ketones done in the first 24 hours (mean 13 per patient). There was no evidence this resulted in any advantage in the improvement in metabolic parameters in the first 24 hours between the two sites. At both sites patients had multiple measurements of glucose by finger prick performed (nearly every hour) as well as frequent venous samples for blood gases and potassium, with likely resultant disruption on their ability to rest or sleep. The additional measurement of ketones invariably adds to nursing time as well as incurring added expense (just over $2.00 per ketone strip—New Zealand Scientific and Medical supplier, personal communication).

Limitations of this study include its retrospective nature and the number of exclusions of admissions due to transfer of patient care from outlying hospitals, or ICU admission. The model of funding in New Zealand is that people where possible need to be cared for in the hospital nearest to their residence. A similar number of people were cared for in ICU but we excluded these admissions, as the ICUs have their own protocols for management of DKA.

Conclusion

This study has shown very similar rates of resolution of DKA in the first 24 hours after admission, despite the use of two different protocols, with little evidence of any added value in the frequent bedside measurement of ketones. Furthermore, there was no difference in the length of stay between the two hospitals.

Competing interests:
Dr Braatvedt reports affiliation with Eli Lilly and Novo Nordisk outside the submitted work.

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A cost-effectiveness analysis of the Prediabetes Intervention Package (PIP) in primary care: a New Zealand pilot programme

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ABSTRACT

AIMS: To estimate the cost-effectiveness of the Prediabetes Intervention Package (PIP), a multilevel primary care nurse-delivered prediabetes lifestyle intervention programme was piloted in Hawke's Bay, New Zealand. The goal of the intervention was weight loss and prevention of progression from prediabetes to type 2 diabetes.

METHODS: A cost-effectiveness evaluation was conducted from a health funder perspective using 2015 NZ$ with costs and per kilogram (kg) weight change at six months analysed at an individual participant level. Missing six-month data were imputed using multiple imputation adjusted for baseline characteristics. Change in weight was calculated following intention-to-treat principles. Three lower-cost scenarios were modelled.

RESULTS: Using multiple imputation and bootstrapping, there was a statistically significant median difference in weight between the intervention and control groups of 1.87kg (95% CI 0.54, 3.15) at six months. The incremental cost-effectiveness ratio (ICER) was NZ$170.90 (95% CI 100.37, 553.93) per 1kg of weight loss. ICERs for the lower-cost scenarios ranged from NZ$95.33 (95% CI 56.12, 308.36) to $NZ120.74 (95% CI 71.04, 391.60).

CONCLUSION: The primary care nurse-delivered PIP intervention is likely to be a cost-effective weight loss strategy for preventing or delaying progression to type 2 diabetes in people with prediabetes.

Diabetes imposes significant personal and economic costs.[1–2] These include the costs for the management of diabetes itself, and the associated complications.[3] As diabetes is a long-term condition, costs accrue over time. Identifying people at risk of developing diabetes and preventing, or at least delaying, progression to diabetes is therefore worthwhile from a personal, health system and societal perspective.[4–6]

Clinical trials have shown that lifestyle interventions can prevent progression from prediabetes to type 2 diabetes.[7–8] Generally, these interventions were resource intensive, employing a relatively expensive specialist workforce, such as dietitians and exercise physiologists and considered too expensive to implement in real-world settings.[9] More affordable, sustainable approaches are required, such as utilising primary care nurses.[6,10]

Few published studies have utilised primary care nurses to deliver diabetes prevention lifestyle interventions[6] and, to our knowledge, none have published economic evaluations. The New Zealand-based Prediabetes Intervention Package (PIP) in primary care study was a six-month pragmatic non-randomised pilot study [ACTRN12615000806561] designed to address workforce capacity and cost challenges.[11] The aim of the intervention

was to provide those with prediabetes and a body mass index (BMI) >25kg/m^2 and their *whānau* (family group) with an understanding of the principles of healthy eating to empower them to make healthy dietary choices and facilitate weight loss. The aim of this study was to estimate the cost-effectiveness of the primary care-based PIP lifestyle intervention.

Methods

A cost-effectiveness analysis of the PIP study was conducted from a health funder perspective on an intention-to-treat basis. Costs associated with the programme intervention were considered, with costs and per kilogram (kg) weight change analysed at an individual participant level. Incremental cost-effectiveness ratios (ICERs) comparing the difference in cost between PIP and usual care and the difference in weight between the groups were calculated. Three alternative cost scenarios were modelled.

In brief, the PIP study, which has been previously described,[11] was conducted in general practices and community settings in two neighbouring provincial cities in the Hawke's Bay region. In New Zealand, primary medical care is delivered by general practitioners (GPs) in mostly group practices. Almost all general practices have government capitation funding with varying levels of patient co-payment, and most belong to a primary health organisation (PHO), which is responsible for providing essential primary healthcare services to an enrolled population. Four intervention practices were located in one city and four control practices in the other.

Participants

Eligible participants were non-pregnant adults aged ≤70 years with newly diagnosed prediabetes according to the New Zealand diagnostic criteria (HbA1c 41–49mmol/mol (5.9–6.6%) or fasting plasma glucose 6.1–6.9mmol/L)[12] with a BMI above 25kg/m^2, not prescribed Metformin and able to communicate in English. Recruitment occurred between August 2014 and April 2015. All participants provided informed written consent. The study was approved by the Northern A Health and Disability Ethics Committee, New Zealand (Ethics reference: 14/NTA/114).

Intervention

The intervention, informed by a literature review of lifestyle interventions and behaviour change theory, sought to provide participants and their family/*whānau* with an understanding of healthy eating principles and enhance empowerment around dietary choices. The six components were:

1. Primary care nurse and community nurse intervention training and dietitian support.
2. Individualised patient dietary assessment, goal setting and dietary advice at an initial 30 minute dietary session followed by 15-minute appointments at 2–3 weeks, three months and six months for reassessment, support and advice.
3. Key messages and consistent individualised opportunistic reminders based on participants' three dietary goals, which were recorded in the patient management system.
4. Nutritionally supportive general practice environment where pamphlets, magazines and posters provided appropriate and consistent dietary messages.
5. Referral to a community-based group nutrition education course consisting of six weekly sessions of 1–1.5 hours each.
6. Written patient resources, the main one being the Diabetes New Zealand booklet, *Diabetes and healthy food choices*.[13]

Usual care

Primary care nurses at control practices provided prediabetes dietary advice in their usual way according to the 2013 national Prediabetes Advice interim recommendations.[14] This typically consisted of unstructured advice using routinely available dietary pamphlets. Patients were followed up at intervals deemed appropriate depending on the goals and plan agreed with their nurse, usually at 3–6 months.

Physical activity

All participants were given standard physical activity advice, that is, 30 minutes of physical activity of moderate intensity on most, if not all, days of the week.

Data

Data collected as part of routine primary care practice included demographic and medical details, lifestyle information (smoking, alcohol, diet and physical activity), blood pressure, anthropometric measures (height, weight and waist circumference) and laboratory measures (HbA1c, total cholesterol, HDL-cholesterol and triglycerides).

Outcomes

In the pilot study, after adjustment, there were positive differences in the intervention group compared to the control group for most of the clinical and laboratory measures (see Coppell et al 2017 for further detail[11]) including for weight (kg) and glycated haemoglobin (HbA1c). Participants who completed the six-month intervention lost a mean 1.3kg while those in the control group gained a mean 0.9kg ($p<0.001$); HbA1c decreased by a mean 1.3mmol/mol in the intervention group and increased by a mean 0.5mmol/mol in the control group ($p<0.096$). As the only statistically significant outcome in the pilot study was weight, the outcome measure used in this economic evaluation was change in weight in kilograms (kgs) at six months.

Measurement of costs

Costs were calculated in New Zealand dollars ($US1=$NZ1.48, February 2019) for the intervention and control groups at the participant level, using data from the Health Hawke's Bay Primary Health Organisation for the 2014–15 year. All resources needed to deliver the pilot intervention programme including training of practice and community nurses, dietetic support for practice nurses, written patient resources and pamphlets, magazines and posters for waiting rooms were included. Intervention costs included the total time cost for the practice nurses based on four visits (90 minutes), and patient resources such as the Diabetes New Zealand diet and diabetes booklet.[13] Usual care costs included the time cost for two practice nurse visits at baseline and six months. Overhead costs were excluded as the administration and overhead cost associated with two to four visits for a relatively small number of participants spread over eight practices was considered to be minimal. Research-specific costs such as the time taken to obtain informed written consent were excluded.

Cost per participant was based on the number of visits, except where costs were fixed, in which case costs were apportioned regardless of the number of visits. For instance, the community nutrition programme consisted of six sessions of 60–90 minutes' duration, and was funded under a fixed price contract. As the total cost was based on the number of expected participants, the cost was allocated equally to the 85 intervention participants irrespective of attendance.

Three alternative scenarios were costed where the costs of nurse training and/or community education were reduced, given the cost of delivering the intervention programme in routine practice is likely to be lower than the cost of the pilot programme. For example, the set-up costs associated with training practice nurses to implement the intervention are high initially, but over time these costs will reduce as refresher training replaces full training for practice nurses familiar with the programme. Furthermore, attendance at community education sessions was shown to be relatively low (approximately 50% attendance), and a differently structured, less costly programme could be offered.

Statistical analysis

Cost-effectiveness analysis was undertaken from a health funder perspective, following intention-to-treat principles. Missing data were imputed using multiple imputation with chained equations with the imputation model estimating weight at six months based on age, sex, Māori ethnicity, baseline weight and family history of diabetes. Following each imputation, a bootstrap sample was taken with 10,000 samples used to obtain means, medians, and 95% confidence intervals (using percentiles), and to construct incremental cost-effectiveness planes and cost-effectiveness acceptability curves. This analysis was repeated for the three alternative scenarios where costs were reduced. Discounting was not used as the duration of the trial was less than one year. Analyses were conducted using Stata version 15.1 and R 3.5.1 using the BCEA package (version 2.2.6).[15]

Results

The demographic characteristics and diabetes-related co-morbidities for the 157 participants enrolled at baseline are shown in Table 1. The two groups were similar. Almost one-third self-identified as Māori and 40% had a family history of diabetes.

Table 1: Demographic characteristics and diabetes-related co-morbidities of participants at baseline prior to receiving the intervention.

Characteristic	Control (n=72)	Intervention (n=85)	*P* value†
Age (years)			0.72
≤49	15	15	
50–64	56	49	
≥65	29	35	
Sex			0.08
Female	39	54	
Male	61	46	
Ethnicity			0.46
Māori	31	32	
NZ European and Other	67	61	
Pacific	3	7	
Family history of type 2 diabetes			1.00
Yes	40	40	
No	60	60	
Hypertension			0.26
Yes	55	45	
No	45	55	
Ischaemic heart disease			0.78
Yes	11	8	
No	89	92	
Stroke			0.70
Yes	3	6	
No	97	94	
Non-alcoholic fatty liver disease			0.17
Yes	7	1	
No	93	99	
Gout			0.38
Yes	13	12	
No	51	73	

Data are % unless otherwise indicated. †For continuous measures, two-sample t-test where residuals were normally distributed, assuming or not assuming equal variance based on Levene's test, and Mann-Whitney-Wilcoxon otherwise; for categorical variables, Chi-squared test where at least 80% of cells have expected counts of 5 or above, Fisher's Exact test otherwise.

Table 2: Costs per participant in 2015 NZ$ for the intervention and control groups.

Item	Cost (NZ$)	Cost per participant (NZ$)*
Intervention costs		
Four visits to practice nurse: 90 minutes at $33 per hour	49.50	
Patient resources (educational material)	5.40	
Group education (six sessions)	188.24	
Practice nurse training and dietitian support⁺	106.14	349.28
Usual care cost		
Three 15-minute visits to practice nurse: 45 minutes at $33 per hour	24.75	24.75

*Based on participants attending all assessments. The mean cost is lower in Table 3 as costs per participant were adjusted based on the number of assessments received.
⁺Based on each practice nurse managing six patients in a six-month period.

These data are a subset of those presented elsewhere (11) in "Table 2 Demographic characteristics and diabetes-related co-morbidities of participants at baseline and six months".

Not all participants completed all assessments. Of the 72 participants attending control practices, 66 participants had a six-month assessment, and of the 85 participants attending intervention practices, 14 participants had a baseline assessment only, four participants had a baseline and three-month assessment, and 67 participants had all three assessments.

The cost per participant for the intervention and control groups are detailed in Table 2. The per participant cost for the intervention was NZ$349.28. The practice nurse training and dietitian support cost of NZ$106.14 included nurse time costs, cost for the community educators and dietitian, administration, resource cost and room hire, and assumed each nurse managed six patients in a six-month period. This cost is expected to reduce to NZ$58.44 in subsequent intakes as refresher training is substituted for full training and dietetic support. The cost per participant for the control group was NZ$24.75.

The summary results of the economic evaluation (and the three cost scenarios) are presented in Table 3.

The mean cost per participant was NZ$24.07 for usual care and NZ$344.43 for the intervention, giving a mean difference of NZ$320.36 between groups. The median weight change after imputation for the usual care group was a 0.79kg (95% CI -0.02, 1.63) weight gain and for the intervention group a 1.08kg (0.01, 2.06) weight loss (median difference of 1.87 kg, 95% CI 0.54, 3.15 kg), attenuating the difference from completers only. The ICER for the pilot study was NZ$170.90 (95% CI 100.37, 553.93) per 1kg of weight loss.

When the community education costs are halved (all else being the same), the mean cost per participant is NZ$250.31 (Scenario A). When refresher training replaces full training (all else being the same), the mean cost per participant is NZ$296.73 (Scenario B), and when both refresher training replaces full training and the community education costs are halved, the mean cost per participant is NZ$202.61 (Scenario C).

The point estimate and bootstrapped estimates of incremental cost and incremental weight change for each scenario are shown in the cost-effectiveness planes in Figure 1A. The ICER for the lowest-cost scenario (Scenario C) is NZ$75.57 lower than the full-cost scenario (Scenario A) at NZ$95.33 ($56.12–$308.36). The cost-effectiveness acceptability curves for each scenario are shown in Figure 1B. If the willingness to pay for a 1kg reduction in weight is NZ$250 for example, the PIP intervention has a 0.81 probability of being cost-effective which increases to 0.96 for the lowest-cost scenario.

Table 3: Cost-effectiveness results (2015 $NZ).

Scenario	Mean cost ($)	Bootstrapped incremental weight reduction (kg) (95% CI)	Bootstrapped incremental cost ($) (95% CI)	Bootstrapped ICER ($) (95% CI)
***Median weight (kg) (95% CI) change at six months** Control 0.79 (–0.02, 1.63) Intervention –1.08 (–2.06, –0.01)	24.07			
Intervention Full practice costs, full training and support costs, full community education costs	344.43	1.87 (0.54, 3.15)	320.47 (318.43, 322.40)	170.90 (100.37, 553.93)
Scenario A Full practice costs, full training and support costs, half community education costs	250.31	1.87 (0.54, 3.15)	226.35 (224.31, 228.28)	120.74 (71.04, 391.60)
Scenario B Full practice costs, refresher training and full support costs, full community education	296.73	1.87 (0.54, 3.15)	272.77 (270.73, 274.70)	145.52 (85.54, 471.67)
Scenario C Full practice costs, refresher training and full support costs, *half* community education costs	202.61	1.87 (0.54, 3.15)	178.65 (176.61, 180.58)	95.33 (56.12, 308.36)

*Calculated following multiple imputation and bootstrapping.

Figure 1: Cost-effectiveness results for the PIP programme.

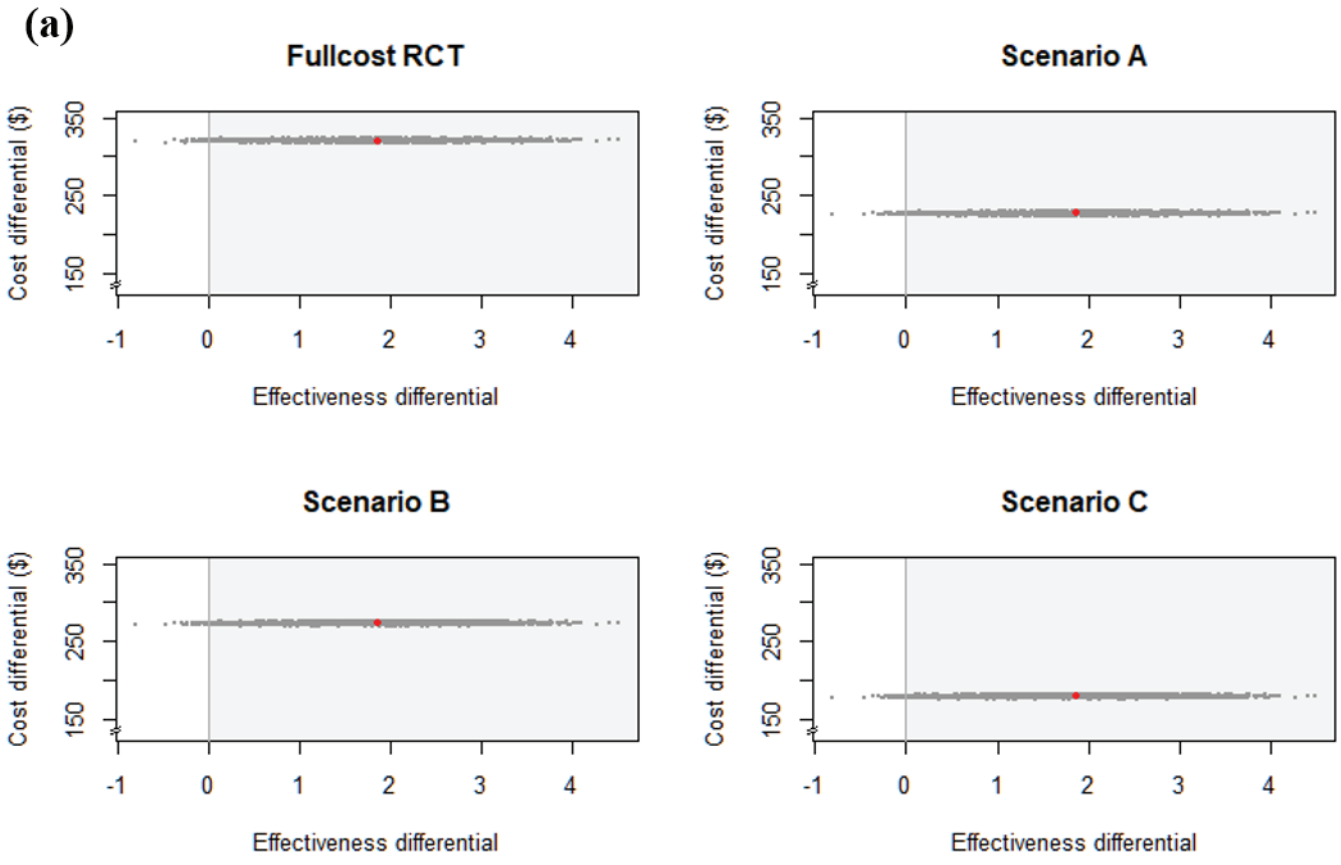

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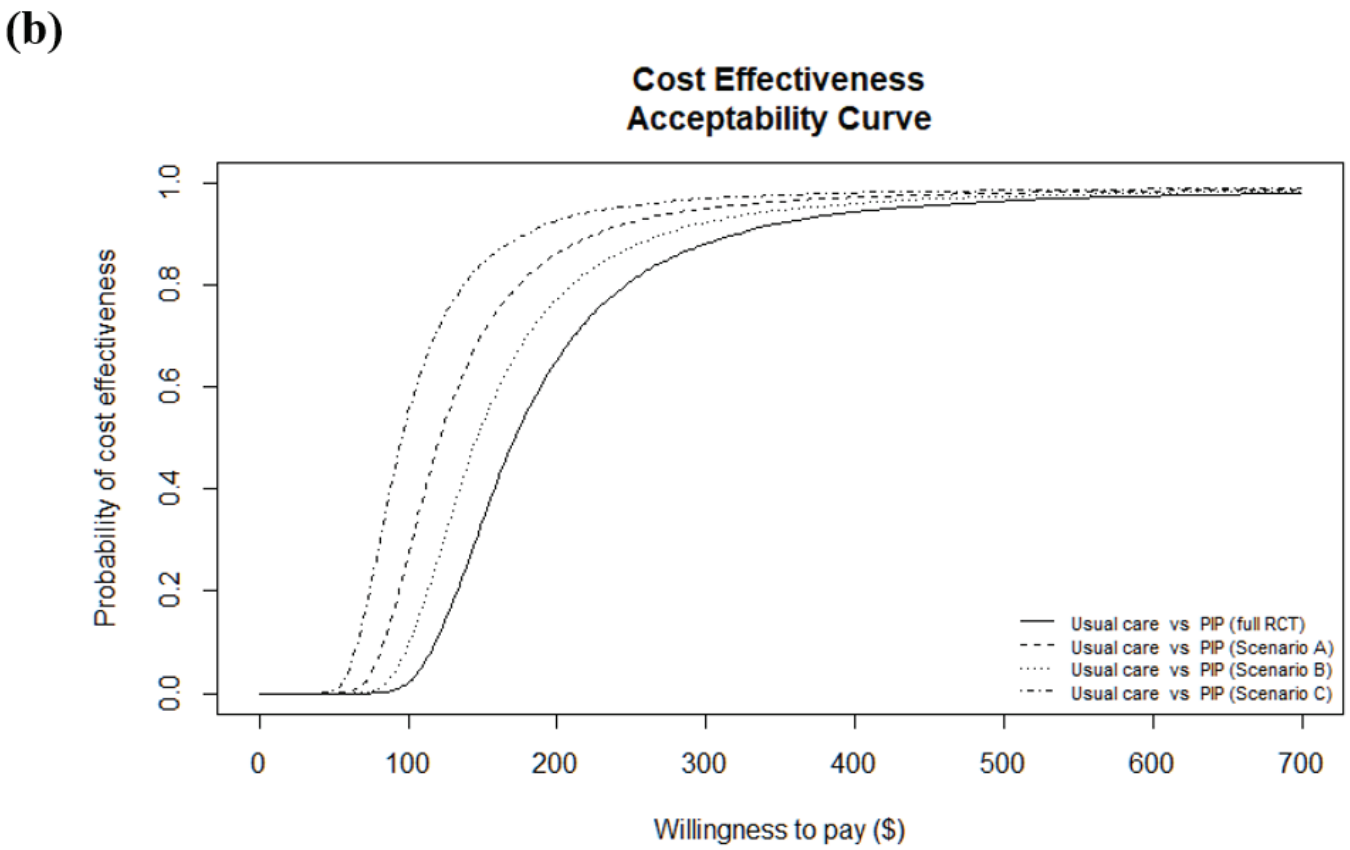
Figure 1: Cost-effectiveness results for the PIP programme (continued).



(a): Incremental cost-effectiveness plane. Larger solid point = estimates used to calculate the ICER; grey points = bootstrapped replicates of incremental costs and incremental weight loss. (b): Cost-effectiveness acceptability curves of reducing weight (kgs) by one unit. Solid black line = probability that the PIP is cost-effective; dashed and dotted lines = probability of Scenarios A, B and C being cost-effective in practice. Costs are reported in 2015NZ$.

Discussion

Diabetes is a global epidemic that accrues significant costs to the health system.[1–2] Clinical trials that have shown lifestyle interventions can prevent progression from prediabetes to type 2 diabetes[7–8] are cost-effective in the short- and long-term,[4] and in many cases probably cost-saving in the longer term.[16–18] How these clinical trial lifestyle interventions are translated into real-world settings influences the cost and effectiveness of such programmes.[16]

The PIP lifestyle intervention pilot programme was implemented in busy general practices in New Zealand. The cost-effectiveness evaluation, from a health funder perspective, showed a mean cost of NZ$170.90 (95% CI 100.37, 553.93) per 1kg of weight loss with a lower-cost scenario estimated at NZ$95.33 (95% CI 56.12, 308.36). The probability of being cost-effective at a willingness to pay of NZ$250 per 1kg of weight loss was 0.81 and 0.96, respectively. The probability of the intervention being cost-effective increases significantly if costs are reduced. For example, at an acceptability threshold of NZ$150 for a 1kg reduction in weight, the PIP intervention has a 0.34 probability of being cost-effective, whereas the lowest-cost scenario has a probability of 0.84. As a weight loss of 1kg reduces the risk of diabetes among those with prediabetes by 16%,[19] any weight loss is likely to be value for money in terms of preventing future costs associated with the treatment of diabetes and its complications.

The mean cost per intervention participant was NZ$344.43, of which 55% was for the community group nutrition programme. Given almost half of the intervention participants did not attend the community group education, the programme could be modified to be less costly, thereby reducing per patient cost. The health professional group who delivers the lifestyle programme also influences programme cost-effectiveness with physicians and specialists such as dietitians typically costing more.[4,20–21] Primary care nurses have infrequently been employed to deliver diabetes prevention programmes, yet they have the potential to

deliver a cost-effective diabetes prevention programme.[22] In the PIP study, specifically trained practice nurses competently delivered the programme and did not require ongoing support from a dietitian after their first 6–12 months of delivering the intervention.[11] Further, ongoing support from healthcare professionals has been shown to be effective in helping patients maintain weight loss.[12,24] As primary care nurses typically have an ongoing trusting relationship with their patients,[6,25] and many of those with prediabetes have co-morbidities requiring treatment,[11] primary care nurses are in an ideal position to provide ongoing guidance and support for weight loss and maintenance.

There are no comparable published economic evaluations of prediabetes lifestyle intervention programmes in New Zealand. Internationally, although not directly comparable to the PIP study due to its size and employment of private weight loss providers, the programme that is most similar to the New Zealand-based PIP programme is the NHS Diabetes Prevention Programme in England, a structured 9–12 month prediabetes lifestyle intervention programme.[5] An impact analysis of this programme to determine cost implications found the per patient medium-end average cost was £270 per participant enrolled (high end £350; low end £155),[5] which was deemed cost-effective. This equates to an approximate medium-end cost of NZ$518 per participant, higher than the per participant cost of NZ$344.43 in the PIP study. As mentioned, although the two studies are not directly comparable, it suggests the PIP programme could potentially be viewed as cost-effective by NHS-standards.

Other alternatives for encouraging weight loss are commercial programmes such as Weight Watchers and Jenny Craig. A within-trial cost-effectiveness analysis of a randomised controlled trial comparing Weight Watchers to standard care in populations in Australia, the UK and Germany concluded that relative to standard care, Weight Watchers was cost-effective over one year from a health sector perspective.[26] In Australia the cost per 1kg of weight loss was US$122 (NZ$181), which is higher than the PIP programme per participant cost of NZ$170.90. Similarly, when Weight Watchers, Jenny Craig and three weight management pharmaceutical products were compared in a US-based study,[27] Weight Watchers was the most cost-effective with an ICER of US$155 (NZ$230) per 1kg weight loss (based solely on subscription costs). The ICER for Jenny Craig's programme (including subscription costs and incremental food costs) was US$338 (NZ$501) per 1kg of weight loss. Although Weight Watchers was considered cost-effective, there are several important differences between commercial programmes and the PIP programme. First, people who choose commercial weight loss programmes are willing to join and able to pay. It is likely therefore that they are different to those who participated in the PIP programme, many of whom had very low food budgets. Second, Weight Watchers predominantly uses group-based sessions delivered at set times in a public setting by trained peers, and clinical advice is not given or available.

Economic studies that have included future healthcare costs show people who participated in an intervention lifestyle programme used significantly less healthcare resources than people who received standard care.[28] Indeed, a lifestyle intervention can be cost-saving. For example, the Diabetes Prevention Program (DPP) 10-year follow-up study found direct medical costs (including emergency department visits, outpatient and hospital admissions) were lowest in the lifestyle intervention group compared with the control and Metformin groups, and were in fact cost-saving.[29] Lifestyle changes reduce the impact, or risk of developing, a number of co-morbidities such as cardiovascular disease, and some cancers.[30] As co-morbidities were common among PIP participants,[11] eg, hypertension (50%) and dyslipidaemia (40%), it is likely the ICER is an over-estimate as it does not include the potential future lower health system costs, on average, for the intervention group. Also not quantified in this study, but captured in the qualitative study[25] are the positive consequences that could impact on future healthcare costs; for example, one intervention participant improved their asthma control, and for others, their positive lifestyle changes extended to other family members.[31] Valuing and including these wider health benefits would further enhance the cost-effectiveness of the programme.

This economic evaluation used actual costs, rather than estimates, and results were based on the realities of primary care, where patient attendance is not 100%. For instance, in this study about half of the participants attended the group community education sessions and 15% of participants did not attend their final six-month appointment. Evaluating a lifestyle programme conducted in a real-world setting highlights what works well and what could be changed, enabling different cost scenarios to be modelled for future roll-out.

Studies have shown that lifestyle programme intervention effects persist after the trial period.[29] Therefore, the six-month time horizon for this study may have underestimated the benefits of the PIP programme. Similarly, this evaluation did not include societal costs, indirect medical costs or future health use costs or the flow-on benefits to participants' families,[31] which may also underestimate the cost-effectiveness. As this was a pilot study, the sample size was not sufficient for sub group (eg, age, ethnicity) analyses to be undertaken. Although multiple imputation can mitigate the effect of missing data, we do not know if there was a difference in the likelihood of excess weight gain between intervention and control group participants who dropped out of the study. As data were collected during primary care consultations in a busy environment, there was insufficient time to collect additional data to calculate QALY gains, limiting comparisons with other studies using QALYs. The lack of a published willingness to pay threshold in New Zealand means it is difficult to determine whether resulting ICERs would be considered value for money by the New Zealand health funder.

In conclusion, this study indicates that the six-month Prediabetes Intervention Package in primary care programme as implemented in Hawke's Bay is likely to be a cost-effective weight loss strategy for preventing or delaying progression to type 2 diabetes in people with prediabetes, with additional health gains beyond diabetes prevention.

Competing interests:
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The cost of diabetes-related hospital care to the Southern District Health Board in 2016/17

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ABSTRACT

AIM: To estimate the cost of diabetes-related hospital admissions to the Southern District Health Board for the year 2016/17.

METHODS: Unidentified data with an ICD-10-AM diagnostic code for any type of diabetes were obtained for admissions to Dunedin and Southland Hospitals. Each admission was categorised according to whether the diabetes diagnostic code was listed first, second or subsequently, and by diagnostic group within each of these three categories. The case weight for each admission was multiplied by the 2016/17 cost weight value of NZ$4,824.67.

RESULTS: There were 6,994 separate hospital admission events. The total cost was NZ$40,986,618. Admissions where diabetes was the primary, secondary or subsequent diagnosis cost NZ$2,214,172, NZ$8,057,235 and NZ$30,697,210, respectively. More than 80% of admissions were for those aged 55 years and over. Ketoacidosis was the most common primary reason for admission (n=103) among those with type 1 diabetes, costing NZ$349,892. When diabetes was not the primary or secondary diagnosis, the most common primary diagnosis was a circulatory system disease, costing NZ$8,181,324. The mean (SD) cost per admission where the primary diagnosis was coronary artery disease with and without diabetes diagnostic codes was NZ$10,407 ($20,694) and NZ$8,657 ($11,347), respectively.

CONCLUSIONS: The annual cost of diabetes-related hospital admissions is substantial. Monitoring the cost of diabetes to DHBs should be prioritised, along with implementation of interventions that reduce preventable diabetes-related hospital admissions, and new diabetes cases.

Diabetes is an increasingly common non-communicable disease associated with high personal and health system costs. The global prevalence of diagnosed diabetes was estimated to be 8.4% among adults aged 18 years and over in 2017, and this is projected to increase to 9.9% by 2045.[1] In New Zealand, the prevalence of diabetes among those aged 15 years and over was 7.0% in 2008/09,[2] and this is likely to have increased over the last 10 years alongside the increasing prevalence of obesity.[3] In 2017, there were an estimated 245,680 New Zealanders with diagnosed diabetes.[4]

An estimated 12% of global health expenditure is spent on diabetes.[5] In 2017 this cost was more than US$850 billion.[6] Health system costs of diabetes include the management of diabetes itself, as well as the associated complications such as cardiovascular disease, diabetic retinopathy, renal failure and peripheral vascular disease. Compared to individuals without diabetes, those with diabetes are more likely to be hospitalised for any reason,[7] and a large portion of health system costs for diabetes is attributable to hospital care.[8]

In New Zealand, while direct pharmaceutical costs for diabetes are known and monitored annually ($79.7 million in 2018),[9] there are few published data on the other health system costs associated with diabetes.

A now decade-old report by PricewaterhouseCoopers (PWC), first produced in 2001 and updated in 2007, estimated diabetes cost NZ$540 million (NZ$1=US$0.66 as at 10 June 2019) in 2007, and forecast that diabetes would cost between NZ$1–1.2 billion by 2016/17.[10,11] The cost to the Canterbury District Health Board (DHB) for hospital admissions in 2007, where diabetes was recorded as the primary or secondary diagnosis, was estimated at NZ$10.1 million.[12] At Counties Manukau DHB, cardiovascular disease, diabetes or both, were the primary reasons for 13% of hospitalisations for those aged 15 years and over, yet together contributed to 46% of the total inpatient hospitalisation cost of NZ$101 million in 2008.[13] This cost increased to NZ$151 million, if pharmaceuticals and laboratory services were also included, of which NZ$83 million was for those with diabetes. For the period 2007–2014, an estimated 5.3% of total health expenditure in New Zealand was for type 2 diabetes alone.[14]

The aim of this study was to estimate the cost to the Southern DHB of diabetes-related inpatient admissions to Dunedin Hospital and Southland Hospital for the 2016/17 financial year (1 July 2016–30 June 2017).

Methods

Data for all admissions to Dunedin Hospital and Southland Hospital with any International Classification of Diseases (ICD) diabetes diagnostic code were obtained from the Southern DHB for the financial year ending June 2017. For the study period, all diabetes-related inpatient hospital admissions were coded using the ICD 10th Revision Australian Modification (ICD-10-AM) codes.[15] The ICD-10-AM diabetes codes used in this study were E10 Type 1 diabetes, E11 Type 2 diabetes, E13 Other specified diabetes, E14 Unspecified diabetes, O240-244 Pre-existing diabetes in pregnancy and O249 Diabetes diagnosed in pregnancy. As well, data for *all* admissions with a primary diagnosis of coronary artery disease (ICD-10-AM codes I20-I25) during the same 12-month period, irrespective of diabetes status, were obtained.

A unique non-identifying number was assigned to each admission. The data accessed for each admission included: admission and discharge dates, date of birth, sex, ethnicity, hospital (Dunedin or Southland), all ICD-10-AM codes for all diagnoses associated with each admission, the order of the diagnoses according to the primary, secondary or subsequent reason(s) for admission, the Diagnosis-Related Group (DRG) codes, the primary procedure and associated code (where applicable) and the case weight. Case weights measure the relative complexity of the treatment(s) given to patients during their hospital admission.[16] Differences in case weights reflect the resources needed for each admission, such as number of days in hospital or the length of time in the operating theatre. For example, in this study the case weight for a coronary bypass with invasive cardiac investigation and reoperation was 12.3 compared with 0.22 for congestive heart failure.

Data analysis

Data were for separate admission events. The diabetes admission data were categorised into three categories according to whether the diabetes diagnostic code was listed first (primary diagnosis), second (secondary diagnosis) or subsequently ('other' diagnosis). A primary diagnosis was considered to be the main reason for the hospital admission and a secondary diagnosis was the main *underlying* reason for the hospital admission. Other diagnoses were any other listed conditions, but these were not necessarily in any order of priority. Therefore, for example, where a single admission event had nine ICD codes listed and diabetes was neither the primary or secondary diagnosis, whether the diabetes code was listed fifth or seventh or eighth, the order had no bearing on the relative importance of diabetes in relation to that hospital admission.

Where diabetes was coded as the primary reason for hospital admission, these admissions were further categorised according to the type of complication, such as renal complications or neurological complications. Admissions where diabetes was the secondary or an 'other' diagnosis were categorised according to the primary diagnosis ICD-10-AM code into the main disease groups, for example, C00-D48 Neoplasms, I00-I99 Diseases of the circulatory system. The coronary artery disease admission data were categorised into those with a diabetes diagnostic code listed, and those without.

The cost of each admission was calculated by multiplying the case weight by the 2016/17 cost weight value of NZ$4,824.67. Separate admission costs were summed for each of the different diagnostic categories, as described above.

As the demographic data related to admissions and not individuals, matching data based on birthdate, sex, ethnicity and type of diabetes was undertaken to estimate the number of individuals who were hospitalised during the study period.

Māori consultation was undertaken with both the Ngāi Tahu Research Consultation Committee, University of Otago and the Southern DHB before commencing this study. Ethics approval was obtained the Human Research Ethics Committee of the University of Otago (HD17/066). Data were analysed in Excel (Microsoft) and Stata SE 15 (StataCorp).

Results

The total number of diabetes-related admissions to Dunedin and Southland Hospitals for the year ended 30 June 2017 was 6,994. These admissions were for an estimated 3,615 individuals with diabetes. This means that some individuals had multiple admissions and were included more than once in the demographic data relating to admissions. The median number of diagnoses per admission was six, with a range of 1–73.

In Table 1 the demographic characteristics are presented by admission event. The demographic characteristics of the estimated 3,615 individuals are shown in Appendix Table 1. Among these individuals, 59.4% had one admission, 20.7% had two admissions and 19.9% had three or more admissions, with one having 31 admissions. Of the 6,994 admissions, nearly 65% were in Dunedin Hospital, and 80% were for patients aged 55 years and older. Diabetes was the primary diagnosis for only 7% of admissions, of which 31% were associated with type 1 diabetes. In contrast, diabetes was listed as an 'other' diagnosis for 58% of admissions, and nearly 86% of all admissions were associated with type 2 diabetes.

The estimated total cost of all diabetes-related admissions was NZ$40,968,618, with 75% of this cost associated with admissions where diabetes was listed as an 'other' diagnosis (Table 1). As summarised in Table 2, the total cost of admissions relating to diabetes as a primary diagnosis was NZ$2.2 million, of which 68% (NZ$1.5 million) was associated with type 2 diabetes. Ketoacidosis was the most frequent reason for admission (n=103) among those with type 1 diabetes at a cost of NZ$349,892. Where type 2 diabetes was the primary diagnosis, admissions with the highest cost were attributable to peripheral circulatory complications (NZ$394,944) and type 2 diabetes with multiple complications (NZ$698,062).

As detailed in Table 3, the total cost of admissions where diabetes was the secondary diagnosis was slightly over NZ$8 million. The disease categories with the highest cost were 'diseases of the eye and adnexa' (NZ$1.2 million), followed by 'diseases of the circulatory system' (NZ$1.16 million), 'diseases of the musculoskeletal system and connective tissue' (NZ$992,883) and 'neoplasms' (NZ$655,339). Most of the total cost (NZ$30.7 million) of all diabetes-related admissions was for admissions where diabetes was not coded as a primary or secondary diagnosis. Of this, NZ$8.2 million was primarily for diseases of the circulatory system, and NZ$4.5 million for reasons coded as 'injury, poisoning and certain other consequences of external causes', with more than NZ$2 million for each of the following categories: 'factors influencing health status and contact with health services', 'diseases of the digestive system', 'diseases of the respiratory system' and 'neoplasms'.

There were 1,573 admissions with a primary diagnosis of coronary artery disease, of which 371 (24.9%) also had a diabetes diagnostic code. The mean (SD) length of stay for those with a diabetes code was 4.1 (8.3) days compared with 3.0 (5.0) days for those without a diabetes code. Similarly, the mean (SD) cost per admission with and without diabetes codes was NZ$10,407 ($20,694) and NZ$8,657 ($11,347), respectively.

Table 1: Demographic characteristics and cost of admissions to Dunedin and Southland hospitals for the 2016/17 financial year by primary, secondary and 'other' diabetes diagnosis.

	All diabetes-related admissions	**Primary diabetes diagnosis**	**Secondary diabetes diagnosis**	**'Other' diabetes diagnosis**
	n (%)	n (%)	n (%)	n (%)
Total admissions	6,994 (100%)	474 (6.8%)	2,448 (35.0%)	4,072 (58.2%)
Total cost, NZ$	$40,968,618	$2,214,172	$8,057,235	$30,697,210
Hospital				
Dunedin	4,513 (64.5%)	281 (6.2%)	1,534 (34.0%)	2,698 (59.8%)
Southland	2,481 (35.5%)	193 (7.8%)	914 (36.8%)	1,374 (59.8%)
Sex				
Male	3,693 (52.8%)	298 (8.1%)	1,277 (34.6%)	2,118 (57.4%)
Female	3,301 (47.2%)	176 (5.3%)	1,171 (35.5%)	1,954 (59.2%)
Age groups				
<15y	59 (0.8%)	38 (64.4%)	14 (23.7%)	7 (11.9%)
15–24y	117 (2.5%)	74 (41.8%)	44 (24.9%)	59 (33.3%)
25–34y	250 (3.6%)	39 (15.6%)	95 (38.0%)	116 (46.4%)
35–44y	299 (4.3%)	21 (7.0%)	114 (38.1%)	164 (54.9%)
45–54y	566 (8.1%)	48 (8.5%)	241 (42.6%)	277 (48.9%)
55–64y	1,068 (15.3%)	55 (5.2%)	412 (38.6%)	601 (56.3%)
65–74y	1,827 (26.1%)	103 (5.6%)	562 (35.7%)	1,072 (58.7%)
75+y	2,748 (39.3%)	96 (3.5%)	876 (31.9%)	1,776 (64.6%)
Ethnicity				
NZ Māori	503 (7.2%)	39 (7.8%)	160 (31.8%)	304 (60.4%)
Pacific Islander	215 (3.1%)	10 (4.7%)	83 (38.6%)	122 (56.7%)
European	6,020 (86.1%)	411 (6.8%)	2,100 (34.9%)	3,509 (58.3%)
Asian	154 (2.2%)	5 (3.3%)	68 (44.2%)	81 (52.6%)
Other	47 (0.7%)	8 (17.0%)	15 (31.9%)	24 (51.1%)
Unknown	55 (0.8%)	1 (1.8%)	22 (40.0%)	32 (58.2%)
Diabetes type				
Type 1 diabetes	664 (9.5%)	208 (31.3%)	223 (33.6%)	233 (35.1%)
Type 2 diabetes	5,988 (85.6%)	248 (4.1%)	2,098 (35.0%)	3,642 (60.8%)
Other diabetes	166 (2.4%)	11 (6.6%)	42 (25.3%)	113 (68.1%)
Diabetes in pregnancy	176 (2.5%)	7 (4.0%)	85 (48.3%)	84 (47.7%)

Table 2: Cost of admissions to Dunedin and Southland hospitals where diabetes was the primary diagnosis sorted by ICD-10-AM† diabetes code for the year to 30 June 2017.

ICD-10-AM† diabetes code	Diagnosis description	Admissions n (%)	Expenditure NZ$ (% of total cost)
E10.0	Type 1 diabetes with coma	0	-
E10.1	Type 1 diabetes with ketoacidosis	103 (21.7%)	349,892 (15.8%)
E10.2	Type 1 diabetes with renal complications	0	-
E10.3	Type 1 diabetes with ophthalmic complications	18 (3.8%)	12,563 (0.6%)
E10.4	Type 1 diabetes with neurological complications	0	-
E10.5	Type 1 diabetes with peripheral circulatory complications	2 (0.4%)	20,425 (0.9%)
E10.6	Type 1 diabetes with other specified complications	50 (10.6%)	123,938 (5.6%)
E10.7	Type 1 diabetes with multiple complications	5 (1.1%)	23,977 (1.1%)
E10.8	Type 1 diabetes with unspecified complications	0	-
E10.9	Type 1 diabetes without complications	30 (6.3%)	78,540 (3.6%)
	Subtotal type 1 diabetes	208 (43.9%)	609,335 (27.6%)
E11.0	Type 2 diabetes with coma	7 (1.5%)	51,731 (2.3%)
E11.1	Type 2 diabetes with ketoacidosis	10 (2.1%)	49,528 (2.2%)
E11.2	Type 2 diabetes with renal complications	3 (0.6%)	11,917 (0.5%)
E11.3	Type 2 diabetes with ophthalmic complications	63 (13.3%)	85,106 (3.8%)
E11.4	Type 2 diabetes with neurological complications	2 (0.4%)	17,403 (0.8%)
E11.5	Type 2 diabetes with peripheral circulatory complications	34 (7.2%)	394,944 (17.8%)
E11.6	Type 2 diabetes with other specified complications	63 (13.3%)	96,285 (8.9%)
E11.7	Type 2 diabetes with multiple complications	62 (13.1%)	698,062 (31.5%)
E11.8	Type 2 diabetes with unspecified complications	0	-
E11.9	Type 2 diabetes without complications	4 (0.8%)	6,122 (0.3%)
	Subtotal type 2 diabetes	248 (52.3%)	1,511,098 (68.1%)
E13	Other specified diabetes mellitus	7 (1.5%)	61,994 (2.8%)
E14	Other unspecified diabetes mellitus	4 (0.8%)	6,122 (0.3%)
O24	Diabetes mellitus in pregnancy	7 (1.5%)	25,623 (1.2%)
Total		**474**	**$2,214,172**

†International Classification of Diseases 10th Revision Australian Modification (ICD-10-AM).

Table 3: Costs of admissions to Dunedin and Southland hospitals where diabetes was a secondary or 'other' diagnosis categorised by ICD-10-AM† primary diagnostic category for the year to 30 June 2017.

ICD-10-AM† diagnostic codes	Diagnostic category description	Secondary diabetes diagnosis, NZ$ (% of total cost)	'Other' diabetes diagnosis, NZ$ (% of total cost)
A00-B99	Certain infectious and parasitic diseases	113,886 (1.4%)	1,151,209 (3.7%)
C00-D48	Neoplasms	655,339 (8.1%)	2,104,443 (6.9%)
D50-D89	Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	76,668 (0.9%)	263,920 (0.9%)
E00-E90	Endocrine, nutritional and metabolic diseases	329,624 (4.1%)	323,819 (1.0%)
F00-F99	Mental and behavioural disorders	160,466 (2.0%)	914,046 (3.0%)
G00-G99	Diseases of the nervous system	257,858 (3.2%)	412,580 (1.3%)
H00-H59	Diseases of the eye and adnexa	1,222,924 (15.2%)	141,428 (0.5%)
H60-H95	Diseases of the ear and mastoid process	42,265 (0.5%)	35,323 (0.1%)
I00-I99	Diseases of the circulatory system	1,161,453 (14.4%)	8,181,324 (26.7%)
J00-J99	Diseases of the respiratory system	386,887(4.8%)	2,272,344 (7.4%)
K00-K93	Diseases of the digestive system	566,448 (7.0%)	2,570,626 (8.4%)
L00-L99	Diseases of the skin and subcutaneous tissue	226,505 (2.8%)	499,039 (1.6%)
M00-M99	Diseases of the musculoskeletal system and connective tissue	992,883 (12.3%)	1,962,082 (6.4%)
N00-N99	Diseases of the genitourinary system	582,134 (7.2%)	947,623 (3.1%)
O00-O99	Pregnancy, childbirth and the puerperium	349,414 (4.3%)	461,957 (1.5%)
Q00-Q99	Congenital malformations, deformations and chromosomal abnormalities	14,355 (0.2%)	4,222 (0.01%)
R00-R99	Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified	602,493 (7.5%)	1,189,118 (3.9%)
S00-T98	Injury, poisoning and certain other consequences of external causes	18,828 (0.2%)	4,470,724 (14.5%)
Z00-Z99	Factors influencing health status and contact with health services	296,805 (3.7%)	2,791,385 (9.1%)
Total		**$8,057,235**	**$30,697,212**

† International Classification of Diseases 10th Revision Australian Modification (ICD-10-AM).

Discussion

The prevalence of diabetes and the costs of treating this disease continue to rise both internationally and in New Zealand.[6] The number of people with diabetes on the New Zealand Virtual Diabetes Register was 187,860 in 2010 increasing to 245,680 in 2017, and from 12,002 to 14,355 in the Southern DHB area over the same period.[4] The annual cost of diabetes medicines and diabetes management (which includes blood glucose monitoring metres and testing strips) are monitored by PHARMAC and the cost of these treatments was NZ$74.5 million for the year to 30 June 2017,[17] increasing by 7% to NZ$79.9 million for the year to 30 June 2018.[9] In contrast, diabetes hospital and primary care costs are not regularly monitored in New Zealand. We sought to estimate the cost of diabetes-related hospital admis-

sions to Dunedin Hospital and Southland Hospital for the 2016/17 financial year. This cost was NZ$41 million (for a resident population of about 320,640),[18] of which NZ$2.2 million was for admissions where diabetes was the primary diagnosis, and NZ$8 million where diabetes was the secondary diagnosis. These costs are most likely to be an underestimate, as it is recognised that diabetes is under-reported on hospital admission data in New Zealand and overseas.[19–21] The degree of under-reporting in this study is not known.

There are few published studies in New Zealand with which to compare our results. Sheerin estimated the cost to the Canterbury DHB of hospitalisations where diabetes was the primary or secondary diagnosis.[12] This cost was NZ$10.1 million in 2005/06, which is the same cost associated with primary and secondary diabetes diagnoses estimated in our study. However, in addition to the two studies being over 10 years apart, there are other differences which limit direct comparisons. The population for the Canterbury region is about twice that for the Otago/Southland area. In 2006, the population for Canterbury was 521,832 and the combined population for the Otago/Southland region was 284,673.[22] There are also methodological differences. First, our study included hospitalisations for pre-existing or newly-diagnosed diabetes in pregnancy, unlike the Canterbury study.[12] Second, we included all disease categories where diabetes was the secondary diagnosis, whereas the Canterbury study only included those codes which were considered to be directly related to known complications of diabetes-acidosis (E87.2), diseases of the nervous system (G45-63), diseases of the eye and adnexa (H25-41), diseases of the circulatory system (I20-75), diseases of the genitourinary system (N10-23), and preparatory care for dialysis (Z49.0).[12] Primary reasons for admission such as osteomyelitis where diabetes is an underlying contributing factor or lower limb amputations for instance, were not included in the Canterbury study, resulting in an underestimate of costs.

As illustrated in our study and the Canterbury study,[12] diabetes is typically not considered the primary reason for hospitalisation, yet is frequently coded as the secondary diagnosis or a co-morbid condition. This was particularly evident among those aged 65 years and over, for whom diabetes was recorded as a secondary (31.4%) or 'other' (62.3%) diagnosis for most admissions. These admissions for this age group cost 65% of the total NZ$41 million cost. This is almost the same as the US, where 64% of hospital inpatient expenditure is for those aged 65 years and over.[23]

Health system costs of individuals with diabetes are greater than that of those without diabetes, irrespective of whether diabetes is the primary, secondary or subsequent diagnosis. In Australia, the annual cost (including medications, hospitalisations and ambulatory services) of a patient with diabetes is 2.3 times more than a patient with normal glucose tolerance (A$4,390 compared with A$1,898; (NZ$1=A$0.95 as at 10 June 2019).[24] In New Zealand, while data are limited, the total additional cost to the Counties Manakau DHB for those with diabetes compared with those without diabetes who were hospitalised during 2007 was NZ$66 million.[13] A significant part of the additional cost for patients with diabetes is due to longer hospital stays,[25] as we demonstrated in this study for admissions where coronary artery disease was the primary diagnosis. Similarly, a case control study examining lower limb cellulitis risk factors conducted at Auckland City Hospital found type 2 diabetes was associated with a significantly longer hospital stay compared with those without diabetes (median 5.3 vs 3.0 days, P<0.001) regardless of age and ethnicity.[26] Moreover, in this Auckland study, those with type 2 diabetes (20% of the identified cases) were more likely to be re-admitted further increasing hospital costs. The average annual cost of lower limb cellulitis was estimated to be A$4.2 million of which A$1.4 million was for type 2 diabetes patients.

Clinical coding practice is governed by rules and conventions to ensure consistency and accuracy of information. These rules are updated over time. In the eighth revision of the ICD-10-AM, it became a requirement to code diabetes whenever a patient with diabetes is hospitalised to recognise that on average they require a higher (more expensive) standard of care.[15] However, unless diabetes is a primary or secondary diagnosis, it is not prioritised in the list of subsequent codes. As such, it is not possible

to determine the extent to which diabetes contributes to the reason for admission. In our study, how much diabetes contributed to the NZ$30.7 million cost where diabetes was listed as an 'other' diagnosis was not able to be determined, but it does, however, highlight that hospital costs for those with diabetes are substantial.

Diabetes is a risk factor for many diseases, and while it may be the underlying or a contributing cause, this is not always reflected in hospital admission or mortality data. Indeed, diabetes may not be recorded at all.[19,25,27] In 2007 in Scotland, only 59% of hospital admissions for people known to have diabetes prior to admission had a diabetes code recorded.[19] Because of this under-recording, the health system cost of diabetes can be underestimated. In our study, a disease of the circulatory system (including ischaemic heart disease, stroke and peripheral vascular disease) was the most common primary diagnosis, and the cost of these hospital admissions was NZ$9,342,777 or 23% of the total diabetes-related admission cost. For most of these admissions diabetes was not recorded as either the primary or secondary diagnosis. Indeed, diabetes was recorded as a secondary diagnosis in only 12% of admissions where a circulatory disease was coded as the primary diagnosis, yet diabetes is often the underlying cause of many cardiovascular disorders.

A limitation to the scope of the study was the inclusion of hospital inpatient costs only. Including the cost of out-patient appointments, retinal screening, home renal dialysis, private hospital admissions, pharmaceuticals prescribed outside the index admission and primary care consultations would provide a more complete description of the health system cost of diabetes. Also, data from the smaller hospitals in the Southern DHB region (Oamaru, Balclutha, Clutha and Queenstown) were not available at the time of sourcing the main dataset, although the number of diabetes-related admissions at these hospitals is likely to be relatively small.

This study estimated that the cost of diabetes in 2016/17 to the Southern DHB amounted to NZ$10 million when diabetes was classified as a primary or secondary diagnosis. However, the actual cost of diabetes to the Southern DHB far outweighs this value. Attributing costs related to diseases of the circulatory system to diabetes, and other diabetes-related co-morbidities remains challenging. Given the continued increase in the prevalence of diabetes in New Zealand, monitoring the cost of diabetes to DHBs should be prioritised, along with the implementation of interventions that target preventable diabetes-related hospital admissions, and diabetes prevention intervention programmes.

Appendix Table 1: The demographics and diabetes type for the total number of admissions to Dunedin and Southland hospitals and the estimated number of individuals for the 2016/17 financial year.

	All diabetes-related admissions	**Individuals with one or more diabetes-related admission**
	n (%)	n (%)
Total	6,994	3,615
Hospital		
Dunedin	4,513 (64.5%)	2,424 (67.1%)
Southland	2,481 (35.5%)	1,191 (33.0%)
Sex		
Male	3,693 (52.8%)	1,804 (49.9%)
Female	3,301 (47.2%)	1,811 (50.1%)
Age groups		
<15y	59 (0.8%)	40 (1.1%)
15–24y	117 (2.5%)	85 (2.4%)
25–34y	250 (3.6%)	161 (4.5%)
35–44y	299 (4.3%)	200 (5.5%)
45–54y	566 (8.1%)	319 (8.8%)
55–64y	1,068 (15.3%)	565 (15.6%)
65–74y	1,827 (26.1%)	886 (24.5%)
75+y	2,748 (39.3%)	1,359 (37.6%)
Ethnicity		
NZ Māori	503 (7.2%)	249 (6.9%)
Pacific Islander	215 (3.1%)	133 (3.7%)
European	6,020 (86.1%)	3,041 (84.1%)
Asian	154 (2.2%)	117 (3.2%)
Other	47 (0.7%)	29 (0.8%)
Unknown	55 (0.8%)	46 (1.3%)
Diabetes type		
Type 1 diabetes	664 (9.5%)	326 (9.0%)
Type 2 diabetes	5,988 (85.6%)	3,060 (84.7%)
Other diabetes	166 (2.4%)	89 (2.5%)
Diabetes in pregnancy	176 (2.5%)	140 (3.9%)

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An open-label feasibility study of repetitive transcranial magnetic stimulation (rTMS) for treatment-resistant depression in the New Zealand healthcare context

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ABSTRACT

AIM: Major depressive disorder (MDD) poses a significant and growing burden on the New Zealand population. It is a leading cause of disability, and resistance to currently offered treatments is common. Repetitive transcranial magnetic stimulation (rTMS) is a treatment offered internationally demonstrating good efficacy and few reports of side effects. It is an intervention that requires daily visits to a clinic over a period of at least four weeks. This study aimed to investigate the effectiveness and acceptability of offering rTMS as a treatment for MDD in the setting of New Zealand healthcare systems.

METHOD: This was a naturalistic, open-label pilot study in which 30 patients with moderate-to-severe treatment-resistant MDD were treated with a course of rTMS (10 Hz) daily over the left dorsolateral prefrontal cortex for four weeks (20 sessions). Primary endpoint was response to treatment, stratified into non-responder, partial responder or responder based on the Montgomery–Åsberg Depression Rating Scale (MADRS) at the end of treatment compared to baseline (<25% reduction, 25–50% reduction, and >50% reduction respectively). Participant remission was also noted as reaching a score of ≤10.

RESULTS: Thirty participants completed the full course of treatment (16 women, mean age 47y, range 19–77y), with a mean baseline MADRS of 32.0 (range 21–48). Twelve participants were classified as responders, six as partial responders, and 12 as non-responders. Of the responders, nine were in remission at the end of treatment. Minimal side effects were reported.

CONCLUSION: Daily sessions of rTMS were successfully administered and were effective in treatment-resistant MDD. The treatment was accessible and well tolerated by the majority of the study participants and should be made available to MDD patients in New Zealand as a treatment option.

Major depressive disorder (MDD), as described in the DSM 5, is the most prevalent mental health disorder in New Zealand. It affects at least 5.3 % of the population and consequently poses a substantial health, social and economic burden.[1] A particularly concerning subpopulation of MDD is the treatment-resistant (TR) class, defined as those patients who are unresponsive to at least two adequate courses of different antidepressant treatments. This group makes up approximately 30% of the population with MDD.[2,3]

Current treatment options for the TR population in New Zealand are further medication trials or non-pharmacological treatments such as cognitive behavioural therapy or electroconvulsive therapy (ECT). Remission rates in TR patients have been found to drop with each failed course of antidepressant medications, therefore further medication trials generally have poor response rates.[4] ECT is an alternative to further medication trials offered in New Zealand and has a higher success rate with TR populations.[5] However, there are disadvantages associated with ECT such as the risk of cognitive side effects, the need for general anaesthetic and the stigma associated with use of ECT for mental health conditions.

Over the past two decades, numerous randomised and sham-controlled trials summarised in multiple meta-analyses have established the efficacy of repetitive transcranial magnetic stimulation (rTMS) for the treatment of MDD.[6–8] In addition to being a viable alternative for those that cannot tolerate or are unresponsive to pharmacological treatments, rTMS also has the advantage of not having adverse effects upon metabolic and sexual functions as do many pharmacological antidepressants.[2] rTMS has a good tolerability profile for patients receiving treatment. The most frequent side effects are focal pain, headache, neck-ache and scalp discomfort, all of which are generally mild and very responsive to analgesics.[9–11] The patient remains awake during the therapy, and afterwards may immediately continue with their daily activities, without needing time to recover from treatment sessions. rTMS is an FDA-approved treatment for depression in the US,[12] and the Royal Australian and New Zealand College of Psychiatrists recommends that rTMS should be available in public and private mental health services for the treatment of MDD.[13,14]

New Zealand is currently behind in the uptake and availability of rTMS as a treatment for MDD, when compared to countries in the EU, Australia, Canada and the US. At the time of writing, aside from this study, only one psychiatrist has offered rTMS on a fee for service basis in New Zealand. Therefore, the majority of the large New Zealand TR population does not have access to rTMS. This limited access is not surprising given that the majority of funding for mental health care is publicly funded through locally unique pathways that prioritise emergency care over longer-term mental health conditions. Additionally, these services often do not provide newer treatments due to perceived cost burden.

For these reasons, the feasibility of offering a rTMS service for patients who might be referred in a New Zealand context requires evaluation. This study piloted the use of rTMS for participants referred from secondary and tertiary healthcare services and evaluated the real-world efficacy and acceptability of rTMS.

Methods

Participants

This open-label clinical study was approved by a Health and Disability Ethics Committee, and participants were recruited from the Auckland District Health Board and Waitematā District Health Board catchment areas. Written informed consent was obtained from all participants. Recruitment was primarily from Community Mental Health Centres in the central Auckland region. Recruitment and treatments ran from April 2016 to September 2019.

In order to be included in the study, participants were aged over 18 years and had a primary diagnosis of unipolar major depressive disorder (MDD) or bipolar affective disorder (BPD). Current depressive episode was a minimum of three months in duration, and at least moderate severity, determined by a MADRS score of ≥20. Additionally, they must have had an insufficient response to at least two adequate courses of antidepressant treatments. The adequacy of each treatment course was assessed using the Antidepressant Treatment Response Questionnaire,[15] and medical records when available.

Participants were also screened for a variety of exclusion criteria during a screening risk assessment conducted by a consultant psychiatrist. Participants with a history of psychosis, any unstable medical or neurologic condition, or imminent risk of suicide as determined by the Columbia-Suicide Severity Rating Scale (CSSRS) and a clinical interview were excluded from the study. Additionally, participants were excluded if they had planned or probable major changes to psychotropic medication,

planned use of ECT, any substance abuse or dependence in the previous six months, or contraindications to rTMS assessed using the University of Auckland safety checklist (eg, history of epilepsy, metallic head implants). Prior to starting treatment, participants were not withdrawn from any current medications, but the dose was required to remain stable for four weeks prior to treatment, and for the duration of the four weeks of treatment. Each participant completed a drug urine test and a pregnancy test when appropriate.

TMS

TMS was delivered via a figure of eight coil using a Magstim Rapid stimulator (MagStim Company, Dyfed, UK) for the first seven participants, and a Neurosoft Neuro-MS/D stimulator (Neurosoft, Russia) for the remainder of the participants. Resting motor threshold (RMT) was measured during the screening process, before each participant was confirmed to receive treatment. Surface electromyography (EMG) was recorded from the right first dorsal interossei (FDI). The coil was positioned over the left motor cortex, at the optimal site for eliciting responses in the FDI muscle. RMT was determined using established methods,[16] as the minimum intensity sufficient to produce a response in the target muscle 50% of the time. Participants with a RMT >80% maximum stimulator output (MSO) were withdrawn from the study due to safety and discomfort of treatment at higher intensities.

rTMS treatment

Participants received daily treatment of rTMS for four weeks. A standard course of rTMS in clinical practice is four to six weeks of daily treatment.[12] For treatment, the figure of eight coil was positioned over the left dorsolateral prefrontal cortex (DLPFC) using the Beam F3 method.[17] rTMS was administered at 120% of RMT, intermittent 10Hz bursts, for 4,000 stimuli per day. RMT was re-measured weekly using methods detailed above to check treatment was administered at a safe intensity for the individual. If threshold changed by more than 5% MSO from when last measured, the treatment session was postponed until the patient was reviewed by a medical practitioner.

This initial daily treatment was then followed by an optional three-month maintenance period. Maintenance treatment is not currently standard in clinical practice, however, it was offered as an option to the participants aiming to prolong response and evaluate potential feasibility in the New Zealand context. Methods and results of maintenance treatment can be found in the Appendix.

Clinical evaluation

Depressive symptomology assessed by the Montgomery–Åsberg Depression Rating Scale (MADRS).[18] The MADRS was administered at baseline, halfway through treatment (at the end of week 2), and at completion of treatment (at the end of week 4). Primary endpoint was extent of clinical response, stratified into three categories based on difference in MADRS score between baseline and end of treatment; responders, partial responders and non-responders. As reduction in MADRS score indicates improvement in depressive symptomology, responders were defined as those who demonstrated a ≥50% reduction, partial responders as those with a 25–49% reduction and non-responders as those with <25% reduction. Remission was also evaluated as a secondary endpoint, classified as a MADRS score of ≤10/50.

Results

A total of 39 participants were recruited, and 30 completed the rTMS treatment (16 female, mean age 46.7y, age range 19–77y, five left-handed). Mean baseline MADRS score was 32.0 (range 21–48), and mean RMT was 43.4% MSO (range 29–68%). Of the eight participants who did not complete treatment, two participants were not eligible as they had RMTs above 80%. Four participants withdrew prior to beginning treatment, two due to the time commitment required, one was postponed to reach medication stability but reached remission on new medications, and one failed to respond to invitation to participate following screening. Two participants withdrew during the treatment, one due to onset of an unrelated mental health crisis requiring acute care, and the other because of time commitment and reduced accessibility due to change in employment. Demographic information for the 30 participants who completed treatment is presented in Table 1.

Table 1: Participant demographics.

ID	Diagnosis	Age	Gender	Ethnicity	Responder	No. failed treatments	Referral source	Psychiatric comorbidities	Current CNS medications
#01	MDD	60–69	M	NZ European	Yes	4	OAIPU	GAD	Clonazepam, Lithium, Lorazepam, Mirtazapine, Olanzapine, Venlafaxine, Zopiclone
#02	BPD	40–49	F	NZ European/Māori	No	4	CMHC	None	Carbamazepine, Clonazepam, Gabapentin, Ibuprofen, Melatonin, Metoclopramide, Omeprazole, Paracetamol, Promethazine, Tramadol, Venlafaxine, Zopiclone
#03	MDD	60–69	F	NZ European	Partial	3	CMHC	GAD	Escitalopram, Quetiapine
#04	MDD	50–59	M	NZ European	Yes	3	CMHC	None	Aripiprazole, Fluoxetine, Methylphenidate, Quetiapine, Zopiclone
#05	MDD	40–49	F	NZ European	Yes	3	CMHC	ADHD	Aripiprazole, Atomoxetine, Buproprion, Dexamethasone, Fluoxetine, Levothyroxine, Zopiclone
#06	MDD	60–69	M	NZ European	Partial	>2	CMHC	None	Aspirin, Lorazepam, Mirtazapine, Nortriptyline, Olanzapine
#07	MDD	40–49	F	NZ European/ Tongan/Fijian	Partial	5	CMHC/PS	Chronic anxiety	Clonazepam, Zopiclone
#08	BPD	20–29	M	Indian	No	7	CMHC	None	Clonazepam, Lithium, Venlafaxine, Zopiclone
#09	MDD	30–39	X	NZ European/Māori	Yes	11	CMHC	ADHD	Clonazepam, Gabapentin, Methotrexate, Methylphenidate, Quetiapine, Riandron
#10	MDD	50–59	M	NZ European	Partial	2	CMHC	None	Quetiapine, Temazepam, Zopiclone
#11	MDD	70–79	F	European/Australian	No	2	OAIPU	GAD	Clonazepam, Diazepam, Melatonin, Mirtazepine, Quetiapine, Venlafaxine
#12	MDD	40–49	M	European	Yes	2	CMHC	ADHD	Codeine, Lorazepam, Mirtazapine, Venlafaxine
#13	MDD	20–29	F	NZ European	No	2	CMHC	None	Amitriptyline, Methotrexate, Promethazine
#14	BPD	60–69	M	European/Māori	No	3	MMH	None	Lithium, Methylphenidate, Olanzapine
#15	MDD	30–39	F	NZ European	No	>4	CMHC/PP	None	Buproprion, Lamotrigine, Lithium, Thyroxine, Venlafaxine
#16	MDD	30–39	F	Chinese/Singapore	Partial	>8	CMHC	GAD, Social anxiety	Buproprion, Lorazepam
#17	BPD	50–59	F	NZ European	No	>7	PP	None	Buproprion, Lamotrigine, Clonazepam
#18	MDD	50–59	F	NZ European	Yes	>7	CMHC	Social anxiety	Venlafaxine, Olanzaoine, Clonazepam
#19	MDD	60–69	F	NZ European	No	10	CMHC	None	Lorazepam, Temazepam, Quetiapine, Zopiclone, Apripiprazole
#20	MDD	30–39	M	NZ European/Māori	Partial	3	CMHC	GAD	Venlafaxine, Buproprion
#21	MDD	50–59	M	NZ European	Yes	7	PP	None	Tranylcypromine, Cilazapril, Felodipine, Lamotrigine, Clonazepam, Celecoxib
#22	MDD	18–19	M	NZ European	No	3	PS	None	Methylphenidate
#23	MDD	18–19	F	NZ European/Māori	No	2	GP	GAD, PTSD	Quetiapine, Zopiclone, Panadol
#24	MDD	40–49	M	NZ European	No	>10	PP	ADHD, social anxiety	Quetiapine, Gabapentin, Dexamphetamine
#25	MDD	60–69	F	Bengali	No	3	CMHC	ADHD, PTSD	Venlafaxine, Mirtazapine, Buproprion, Quetiapine, Gabapentin, Magnesium
#26	MDD	50–59	M	Samoan/European	Yes	6	PP	None	Mirtazepine, Melatonin, Zopiclone
#27	MDD	40–49	F	NZ European	Yes	9	PP	None	Venlafaxine
#28	MDD	30–39	F	NZ European	Yes	4	GP	PTSD, social anxiety	Paroxetine, Buproprion
#29	MDD	40–49	M	NZ European/Māori	Yes	7	PP/PS	None	Quetiapine, Lorazepam
#30	MDD	70–79	F	NZ European	Yes	3	PP	None	Sertraline, Quetiapine, Methylphenidate

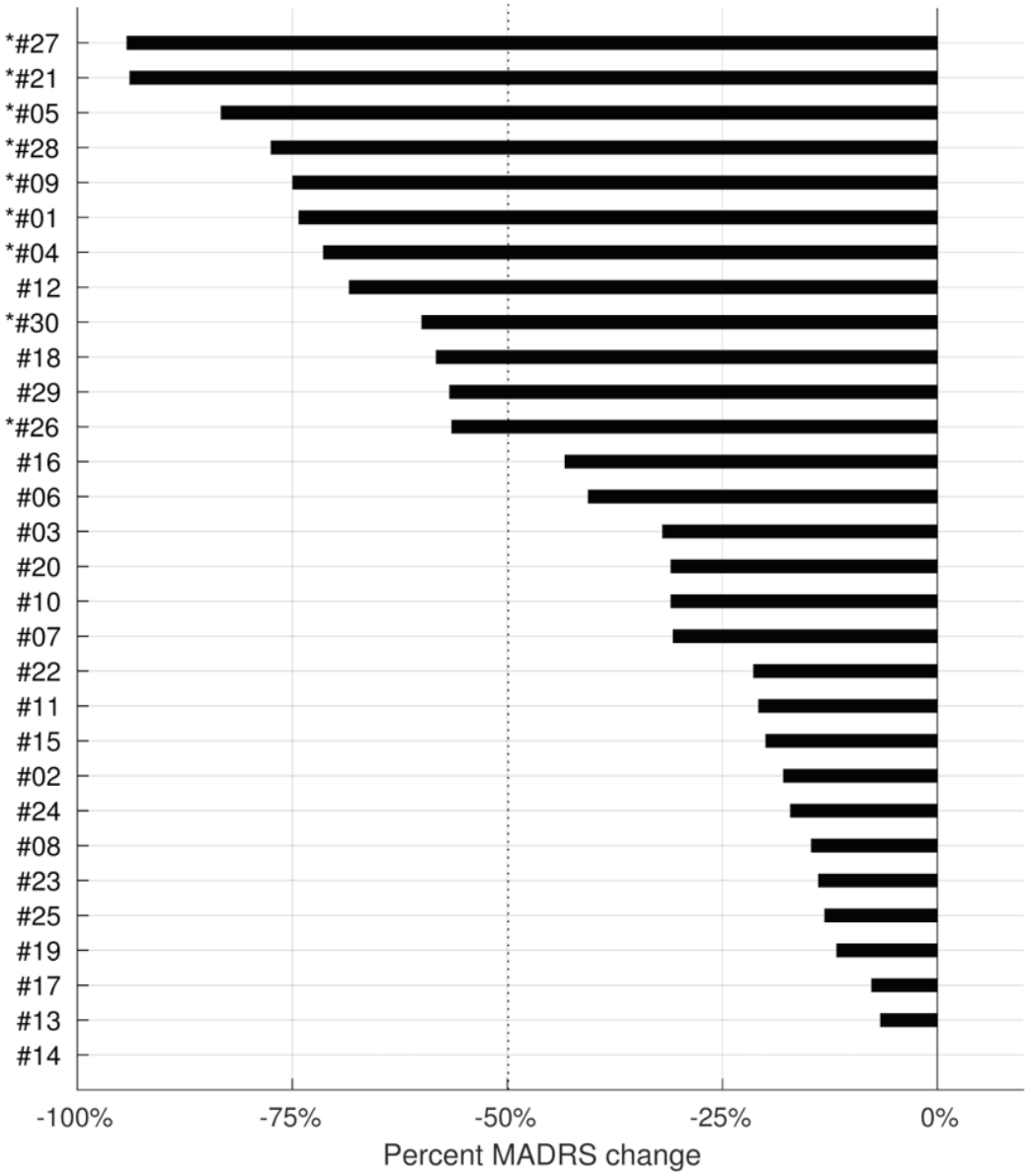
Abbreviations: ADHD, attention deficit hyperactivity disorder; BPD, bipolar disorder; CMHC, community mental health centre; CNS, central nervous system; F, female; GAD, generalised anxiety disorder; GP, General Practitioner; ID, patient identification number; M, male; MDD, major depressive disorder; MMH, Māori mental health; NZ, New Zealand; OAIPU, old age inpatient psychiatric unit; PP, private psychiatrist/psychologist; PS, previous study; X, transgender.

Of the 30 participants who completed treatment 12 were classified as responders. This demonstrates a response rate of 40%. Another six were classified as partial responders, and 12 as non-responders. Proportional improvement in MADRS scores for individual participants demonstrating responder status can be seen in Figure 1.

Remission rates were also evaluated during treatment. Nine participants reached remission over the duration of this study. One participant (#01) was in remission halfway through treatment (week 2), with the other eight in remission at the end of treatment (week 4). Individual MADRS scores are presented in Figure 2 with remission indicated as a score ≤10.

No serious adverse events were reported. The treatment was tolerable throughout every session for 26 of the 30 participants. The remaining four participants reported intolerable discomfort upon starting the treatment and were unable to complete their first session at the treatment intensity of 120% of RMT. All four reported pain at the site of stimulation, and one additionally

Figure 1: Percent MADRS score change for individual participants from baseline to end of week 4. A ≥50% improvement classified as response, indicated by dashed line.



*Participant reached remission.

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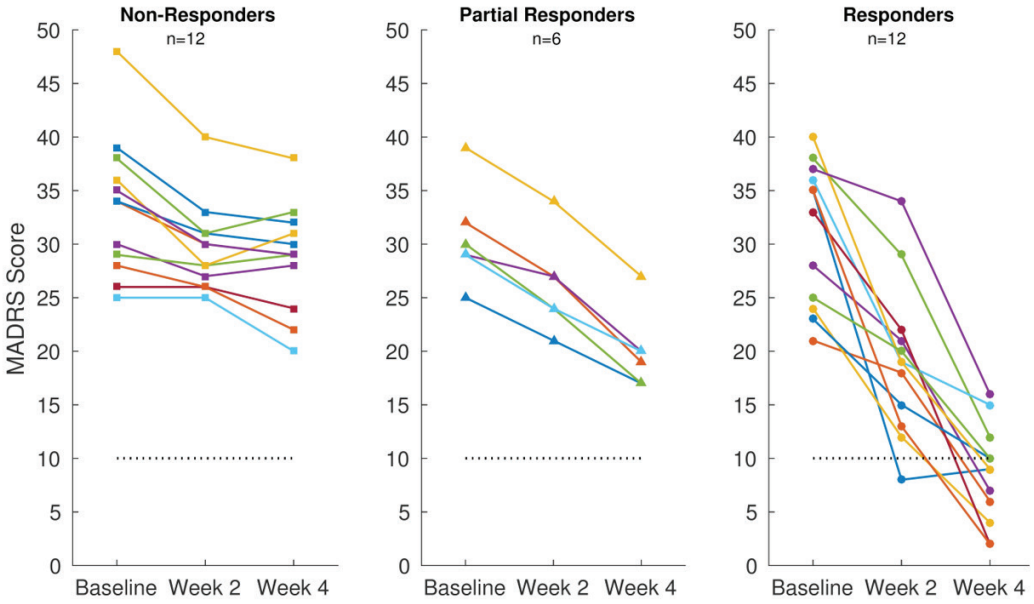
Figure 2: Individual MADRS scores for non-responders, partial responders, and responders to rTMS at baseline, week 2, week 4 (at the completion of treatment).



Dashed line indicates remission (MADRS score ≤10).

reported intolerable jaw movement due to facial nerve activation. Stimulation intensity was then lowered and incrementally increased to maximum tolerability within the session, and across treatment days until an intensity of 100% RMT or higher was tolerable. In three cases (#04, #09, and #19), within two weeks 120% RMT was tolerable and remained so for the remainder of the treatment. In one case (#17), a maximum tolerability of 105% RMT was reached and therefore was treated at this intensity for the duration. Treatment sessions administered below 100% RMT were not counted, therefore as required additional sessions were added to reach 20 sessions at ≥100% RMT. Headache immediately after treatment was infrequently reported and was not a significant burden to the participants. Treatment with paracetamol if necessary was advised.

Discussion

This pilot study demonstrated that rTMS is both an acceptable and efficacious treatment option that could be more widely used in New Zealand. A limitation of the current study when evaluating efficacy is the lack of a sham control group. As the study was open-label the clinical response data will contain a mixture of clinical response to the rTMS treatment regimen, placebo effect, environmental effects from coming to daily treatment sessions, and regression to the mean. However, the clinical efficacy of rTMS is well established in multiple meta-analyses,[6–8] and our data reflect the real-world effects clinicians might expect to see in practice.

In general, the response rate of 40% was perhaps slightly lower than has been previously reported. For example, in a recent multicentre analysis of a population of 130 Australian patients a response rate of 55 % was reported.[19] This may reflect the relatively small sample size, or the naturalistic sample that had many failed treatments with a number of psychiatric co-morbidities often with multiple concomitant CNS medications (see Table 1). In particular, because the treatment was locally novel many of the referrals received were from clinicians for their most TR patients. Like response to further medication trials, it has been demonstrated that the success rate of rTMS therapy declines with the number of failed pharmacological treatments,[20] and response rates are higher in less treatment-resistant patients.[21] However there is some evidence that this decline is less in rTMS when compared to further medication trials.[22] This study also included participants with BPD in a depressed state (note the four patients with BPD were non-responders).

Nevertheless, the fact that acceptability of the treatment was so high with such a complex TR sample that has been difficult to successfully treat is encouraging for its use as a patient service. We note that two patients who were actually treated from an inpatient ward were able to be discharged from the inpatient setting during the treatment. This indicates that some patients can have large functional improvements that meaningfully transform their lives with large economic benefit. We also note that another limitation of our study is that being a pilot trial with a fixed treatment protocol we were limited to offering four weeks of treatment. However, partial responders who were improving but not yet responders at week four may have benefitted from a six week treatment protocol, and in clinical practice extending for an additional two weeks may be practical.

We note here some pragmatic observations based on our experiences. Relevant to New Zealand, our study sample included six patients who self-identified as Māori and two identified with a Pacific Island ethnicity. Interviews with patients conducted after treatment suggested no cultural issues with the treatment but identification of the head as tapu is an important cultural consideration. Two participants who were unable to be treated due to high thresholds highlight the need for appropriate screening before guaranteeing the treatment as an option. First testing if RMT was below 80% of MSO reduced the burden of disappointment when treatment could no longer be offered. Finally, for rTMS provided in an outpatient setting, basic considerations such as availability of public transport, ease of parking and general physical accessibility for 20-plus daily sessions are important. These factors combined with robust screening, resulted in low dropout rates, with only one participant discontinuing treatment after beginning the initial phase.

No participants dropped out of the study due to intolerable side effects. If used as a proxy measure of acceptability, this indicates that the acceptability of the treatment was good. Additionally, the majority of patients (25/30) elected to receive the additional maintenance treatment sessions, four of whom did not demonstrate a clinical response to the initial or maintenance treatment, further indicating the acceptability and tolerability of the treatment sessions, even in the absence of clinically measured benefit. It is also worth noting, newer iterations of rTMS including theta burst TMS offer more efficient treatment with similar response rates in the TR population, which could improve the practicalities and patient tolerability of daily sessions.[23]

Cost is an important consideration for making rTMS more available in New Zealand. rTMS machines are relatively inexpensive at approximately ~$70,000NZD, but the ongoing operator and occupancy costs need to be considered. However, several studies have shown that after two treatment failures rTMS is less costly relative to further pharmacotherapy or ECT.[24–26] Of course cost-effectiveness in the New Zealand context would require a New Zealand specific cost-analysis, which is beyond the scope of this work.

In summary, rTMS therapy has distinct need in light of the New Zealand Mental Health Inquiry and the potential for new patient services to be commissioned in mental health services. Providing an alternative to psychological and pharmacological treatments for those who are treatment resistant or unable to tolerate antidepressant medications will augment patient options and potentially improve outcomes for one of New Zealand's leading disease burdens.[1] There have already been developments in rTMS, which will likely improve the efficient delivery of rTMS for mental health conditions.

Appendix

Maintenance treatment

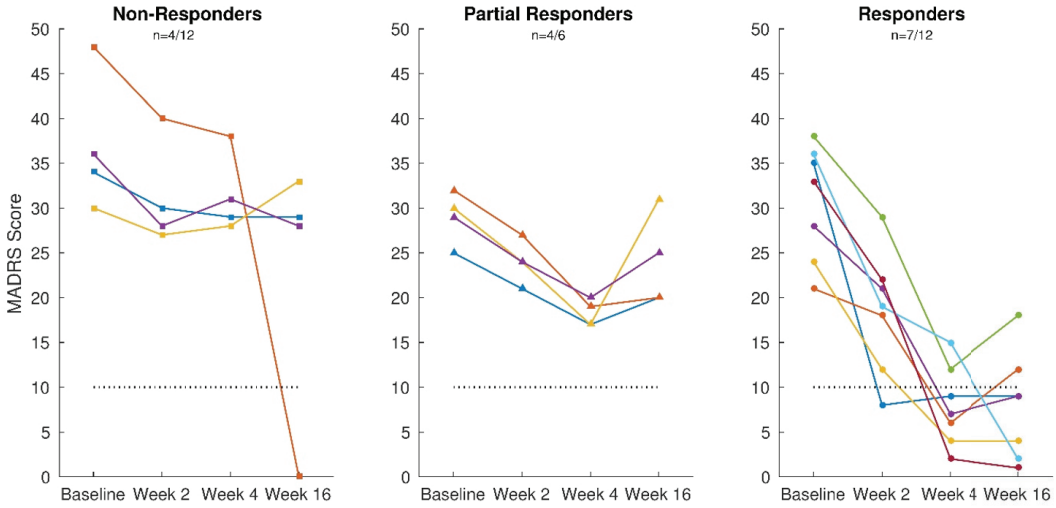
Methods

The initial 20 days of rTMS treatment was followed by an optional three-month maintenance period using the same treatment parameters. Frequency of treatment sessions however tapered off, with maintenance consisting of treatment once weekly for one month (weeks 5–8), then treatment once fortnightly for one month (weeks 8 and 12), and then a final session at the end of the third month (week 16). During maintenance RMT was measured at each session using methods detailed previously. Clinical symptomology was assessed using the MADRS upon the completion of maintenance treatment at week 16. Differences in depressive scores across time were compared using Wilcoxon signed ranks test. A p value of <0.05 was considered statistically significant.

Results

At the time of writing this manuscript, 15 participants had completed maintenance treatment, with another five in progress. Five participants elected not to receive maintenance treatment due to lack of clinical response. An additional five participants (two partial responders, two non-responders) who began maintenance elected to withdraw during the maintenance phase due to lack of noticeable further benefit. MADRS scores for the 15 patients who completed maintenance treatment are shown in Appendix Figure 1.

Appendix Figure 1: Individual MADRS scores for non-responders, partial responders, and responders to rTMS at baseline, week 2, week 4 (at the completion of treatment) and week 16 (at the completion of maintenance treatment. Dashed line indicates remission (MADRS score >10).



MADRS scores at end of maintenance were not significantly different from those at the end of initial treatment for the 16 participants who completed maintenance treatment ($p>0.05$). Tolerability was good with no reports of pain at the site of stimulation, or any other side effects. All sessions were able to be conducted at 120% RMT. Maintenance treatment was found to be tolerable and no additional practical considerations were found to those of initial daily treatment.

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The burden of alcohol-related presentations to a busy urban New Zealand hospital emergency department

Georgina Svensen, Bridget Kool, Sarah Buller

ABSTRACT

AIMS: This cross-sectional observational study presents a focused analysis of alcohol-related presentations (ARPs) to a major New Zealand emergency department (ED) with the aim of describing and comparing the profile and outcomes of these presentations.

METHODS: A secondary analysis of 12 months (November 2017 to October 2018) of electronic patient records of adult (≥15 years) presentations to the Auckland City Hospital Adult ED. The primary area of interest was patient's alcohol-related status. Additional information reviewed included: patient demographics, and features of the presentation such as time of presentation, triage category and discharge disposition.

RESULTS: Among 73,381 presentations, 7% (n=5,130) were alcohol–related, the majority were male (65%) and aged 20–39 (52%). ARPs were more frequent at night, during the weekends, public holidays and over the summer months. Sixteen percent of injury-related presentations were alcohol-related. ARPs commonly arrived at the ED via emergency services and had a longer length of stay than non-ARPs.

CONCLUSIONS: The findings from this study highlight the burden of alcohol misuse on the ED. Continued public health efforts are required to implement preventative strategies for alcohol-related harm in the ED and society as a whole.

The consumption and harmful use of alcohol is associated with significant global health burden, resulting in physical, psychological and social impacts. The harmful use of alcohol is estimated to cause 5.3% of all deaths globally, and account for 5.1% of all Disability Adjusted Life Years.[1] In New Zealand, mortality and morbidity rates associated with alcohol differ by sex and ethnicity. There are twice as many alcohol-related deaths in men than women. The age-standardised alcohol-related mortality rate for Māori (New Zealand's indigenous population) is two and a half times that of their non-Māori counterparts.[2]

The most recent Ministry of Health (MoH) National Health Survey (2017) found 20% of adults engage in 'hazardous drinking'.[3] Hospital emergency departments (EDs) often bear the brunt of alcohol-related harm.[3,4] According to a 2017 survey of Australasian EDs, at peak times, at least 12% of New Zealand ED presentations are there as a result of harmful alcohol use.[5] Alcohol-related presentations (ARPs) commonly occur in the weekends and are more likely to be late at night or in the early hours of the morning, typically presenting as injuries which are often serious and potentially life threatening.[6]

Alcohol is the most commonly reported factor involved in aggression experienced in the ED.[4,7] A 2014 survey of Australasian ED staff found that 98% of staff in Australia and 92% in New Zealand reported experiencing alcohol-related verbal and physical

aggression in the workplace, with 68% experiencing verbal aggression at least once a week.[4] ED patients with ARPs pose a risk to their own health, but also divert time and resources from other patients.[4,8]

In order to effectively target prevention strategies to reduce ARPs and their deleterious consequences, evidence regarding the characteristics of those who present to the ED with an ARP is required. Since November 2017, in response to the MOH's guidelines (National Non-admitted Patient Collection), Auckland Hospital has been collecting information on the number of ARPs to the ED. The aim of this study is to describe and compare the profile and outcomes of those who present to ED with an ARP to those who present with a non-ARP.

Methods

This cross-sectional observational study used a descriptive analytical approach to examine routinely collected anonymised data of adult (≥15 years old) presentations to the Auckland Hospital Adult ED from 1 November 2017 to 31 October 2018. Auckland District Health Board serves a population of over 500,000 people. The adult (>15 years) ED is a busy urban ED and sees approximately 70,000 adult presentations per annum.

In response to the MOH's mandate to complete an 'alcohol involved field' on all patients, all Auckland Hospital ED clinical staff are required to determine "is alcohol associated with this presentation?". This can be filled out by any member of staff including triage nurses, patients' nurses, charge nurses or doctors. It is a mandatory field in the patient management system and the visit can not be completed until the question is completed. There are four available alcohol consumption response options recorded:

- Yes: associated with this presentation
- Secondary: a consequence of others' consumption
- No: not directly associated with the presentation
- Unknown: not known or could not be determined

In determining whether alcohol is associated with a presentation, both acute and usual use of alcohol are considered. For the purposes of this analysis, the reason for presentation was classified as one of the four categories used by Egerton-Warburton et al: injuries, mental health, intoxication and multiple medical conditions.[8] An ARP was defined as per the criteria used by Egerton-Warburton et al, staff received training on the use of these categories. Staff were directed to ask about alcohol involvement if patients presented with an injury or overdose, if staff did not ask about alcohol ingestion then they were directed to record the response as 'unknown'. To remind staff to ask about alcohol-related presentations it was incorporated into the nursing assessment pro forma. For all other presentations, staff used their judgement regarding alcohol involvement.

In addition to the alcohol field, the variables of interest included: patient demographics (age, gender description, domicile description and code, New Zealand deprivation index—an area level measure, ethnicity and New Zealand residency status), temporal features of the presentation (date, time, day of week, season and public holiday), and presentation characteristics (presentation type, arrival mode, triage category code, discharge type and ED length of stay). The alcohol question response variable was classified into three categories: alcohol-related (Yes or Secondary), not alcohol-related and unknown.

Descriptive statistics were used to summarise the data. Ethics approval for the study was obtained from the University of Auckland Human Participants Ethics Committee (Reference 022208) and institutional approval from the Auckland District Health Board (Reference A+8299). All data was de-identified and stored in password-protected files.

Results

During the 12-month period reviewed, there were 73,381 presentations to the Auckland Hospital ED. The overall study population was 51% male and 54% were of New Zealand European or 'Other' ethnicity. Among the 73,381 presentations in the study period, the alcohol-related status was recorded as 'not known' in 5% (n=3,387). In these cases, either the alcohol status was unable to be ascertained by staff (n= 2,313) or the information was missing (n=1,074).

Table 1: Distribution of the demographics of Auckland Hospital Emergency Department presentations by alcohol status (column percentages).

Variables	Total n (%)	Alcohol-related n (%)	Not alcohol-related n (%)	Not known n (%)
Total presentations	73,381 (100%)	5,130 (7.0%)	64,864 (88.4%)	3,387 (4.6%)
Gender*				
Female	35,698 (48.3%)	1,787 (34.8%)	34,383 (53.0%)	1,512 (44.6%)
Male	37,682 (51.4%)	3,343 (65.2%)	30,280 (47.0%)	1,875 (55.4%)
Age (in years)				
15–19	4,871 (6.6%)	605 (11.8%)	3,991 (6.2%)	275 (8.1%)
20–29	16,197 (22.1%)	1,731 (33.7%)	13,502 (20.8%)	964 (28.5%)
30–39	11,849 (16.2%)	903 (17.6%)	10,227 (15.8%)	719 (21.2%)
40–49	8,867 (12.1%)	739 (14.4%)	7,679 (11.8%)	449 (13.3%)
50–59	9,186 (12.5%)	564 (11.0%)	8,206 (12.7%)	416 (12.3%)
60–69	8,149 (11.1%)	316 (6.2%)	7,581 (11.7%)	252 (7.4%)
70–79	6,997 (9.5%)	182 (3.6%)	6,642 (10.2%)	173 (5.1%)
80–89	5,399 (7.4%)	71 (1.4%)	5,211 (8.0%)	117 (3.5%)
90+	1,866 (2.5%)	19 (0.4%)	1,825 (2.8%)	22 (0.7%)
Ethnicity				
Māori	7,905 (10.8%)	860 (16.8%)	6,465 (10.0%)	580 (17.2%)
NZE other**	39,614 (54.0%)	3,141 (61.2%)	34,689 (53.5%)	1,784 (52.7%)
Asian	15,614 (21.3%)	585 (11.4%)	14,363 (22.1)	666 (19.7%)
Pacific peoples	10,248 (14.0%)	544 (10.6%)	9,347 (14.4%)	357 (10.5%)
NZDep2013 index of deprivation				
1–2 least deprived	11,636 (15.9%)	772 (15.1%)	10,446 (16.1%)	418 (12.3%)
3–4	14,505 (19.8%)	889 (17.3%)	13,053 (20.1%)	563 (16.6%)
5–6	15,765 (21.5%)	1,173 (22.9%)	13,963 (21.5%)	629 (18.6%)
7–8	10,712 (14.6%)	729 (14.2%)	9,519 (14.7%)	464 (13.7%)
9–10 most deprived	18,696 (25.48%)	1,450 (28.3%)	16,053 (24.8%)	1,193 (35.2%)
New Zealand residency				
No	9,111 (12.4%)	630 (12.3%)	7,769 (12.0%)	712 (21.0%)
Yes	64,270 (87.6%)	4,500 (87.7%)	57,095 (88.0%)	2,675 (79.0%)

* One record missing = gender unspecified.
** NZE other = European, Middle Eastern/Latin American/African, other ethnic groups and ethnicities.

Seven percent of all presentations were alcohol-related, and 65% of these were male. Among all ARPs, the majority were aged 20–29 years old (34%) followed by those aged 30–39 years (18%) (Table 1). The highest proportion of ARPs among all presentations were found in those aged 15–19 years. This group covered a five-year age range but accounted for 12.4% of ARPs compared with 7% overall. Māori were over represented, accounting for 17% of ARPs but only 8% of the Auckland DHB population.[9] NZDep2013, an area level deprivation measure, was used to analyse the socioeconomic deprivation status of ARPs.[10] Twenty-eight percent of ARPs were in the most deprived (9–10) groups compared with only 25% of non-ARPs. There was no difference in the prevalence of ARPs by New Zealand residency status.

Table 2: Distribution of the timing of Auckland Hospital Emergency Department presentations by alcohol status (column percentages).

Variables	Total n (%)	Alcohol-related n (%)	Not alcohol-related n (%)	Not known n (%)
Total presentations	73,381 (100%)	5,130 (7.0%)	64,864 (88.4%)	3,387 (4.6%)
Time of day				
Day (0700–1459)	28,446 (38.8%)	906 (17.7%)	26,182 (40.4%)	1,358 (40.1%)
Evening (1500–2259)	30,860 (42.1%)	1,717 (33.5%)	27,787 (42.8%)	1,356 (20.0%)
Night (2300–0659)	14,075 (19.2%)	2,507 (48.9%)	10,895 (16.8%)	673 (19.9%)
Weekend or weekday				
Monday–Thursday	51,232 (69.8%)	2,692 (52.5%)	46,150 (71.1%)	2,390 (70.6%)
Friday–Sunday	22,149 (30.2%)	2,438 (47.5%)	18,714 (28.9%)	997 (29.4%)
Public holiday				
No	70,958 (96.7%)	4,914 (95.8%)	62,773 (96.8%)	3,271 (96.6%)
Yes	2,423 (3.3%)	216 (4.2%)	2,091 (3.2%)	116 (3.4%)
ANZAC day	221 (0.3%)	15 (0.3%)	200 (0.3%)	6 (0.2%)
Auckland Anniversary	229 (0.3%)	19 (0.4%)	200 (0.3%)	10 (0.3%)
Boxing Day	196 (0.3%)	24 (0.5%)	170 (0.3%)	2 (0.1%)
Christmas Day	238 (0.3%)	20 (0.4%)	210 (0.3%)	8 (0.2%)
Easter Monday	200 (0.3%)	10 (0.2%)	187 (0.3%)	3 (0.1%)
Good Friday	183 (0.3%)	20 (0.4%)	154 (0.2%)	9 (0.3%)
Labour Day	230 (0.3%)	22 (0.4%)	196 (0.3%)	12 (0.4%)
New Year's Day	270 (0.4%)	48 (0.9%)	208 (0.3%)	14 (0.4%)
2nd January	230 (0.3%)	16 (0.3%)	187 (0.3%)	27 (0.8%)
Queen's Birthday	209 (0.3%)	12 (0.2%)	183 (0.3%)	14 (0.4%)
Waitangi Day	217 (0.3%)	10 (0.2%)	196 (0.3%)	11 (0.3%)
Season				
Spring	18,091 (24.7%)	1,188 (23.2%)	15,993 (24.7%)	910 (26.9%)
Summer	18,493 (25.2%)	1,510 (29.4%)	16,182 (25.0%)	801 (23.7%)
Autumn	18,379 (25.1%)	1,253 (24.4%)	16,274 (25.1%)	852 (25.2%)
Winter	18,418 (25.1%)	1,179 (23.0%)	16,415 (25.3%)	824 (24.3%)

Table 3: Distribution of the characteristics of Auckland Hospital Emergency Department presentations by alcohol status (column percentages).

Variables	Total n (%)	Alcohol-related n (%)	Not alcohol-related n (%)	Not known n (%)
Total presentations	73,381 (100%)	5,130 (7.0%)	64,864 (88.4%)	3,387 (4.6%)
Injury related presentation				
Yes	20,188 (27.5%)	3,308 (64.5%)	16,060 (24.7%)	820 (24.2%)
Female	9,223 (12.6%)	1,153 (22.5%)	7,717 (11.9%)	353 (10.4%)
Male	10,965 (14.9%)	2,155 (42.0%)	8,343 (12.9%)	467 (13.8%)
Arrival mode to the emergency department				
Aeroplane or helicopter	435 (0.6%)	45 (0.9%)	351 (0.5%)	39 (1.2%)
Ambulance	20,716 (28.2%)	2,518 (49.1%)	17,226 (26.6%)	972 (28.7%)
Own transport	51,151 (69.7%)	2,405 (46.9%)	46,497 (71.7%)	2249 (66.4%)
Police	567 (0.8%)	157 (3.1%)	301 (0.5%)	109 (3.2%)
Unknown	512 (0.7%)	5 (0.1%)	489 (0.7%)	18 (0.5%)
Triage category				
1 (life threatening)	2,951 (4.0%)	426 (8.3%)	2,119 (3.3%)	406 (12.0%)
2	13,753 (18.7%)	678 (13.2%)	12,761 (19.7%)	314 (9.3%)
3	28,195 (38.4%)	2,010 (39.2%)	25,160 (38.8%)	1,025 (30.3%)
4	26,883 (36.6%)	1,944 (37.9%)	23,526 (36.3%)	1,413 (41.7%)
5 (least urgent)	1,573 (2.1%)	72 (1.4%)	1,291 (2.0%)	210 (6.2%)

During the day (7am–2.59pm), ARPs accounted for 3% of all presentations compared to 6% during the evening (3pm–10.59pm) and 18% at night (11pm–6.59am) (Table 2). ARPs accounted for 5% of weekday presentations (Monday to Thursday) compared to 11% of presentations on the weekend (Friday to Sunday). ARPs were more common on public holidays, with New Year's Day having the greatest proportion of ARPs (18%) of all the public holidays, followed by Boxing Day (12%), compared to 7% of all presentations being alcohol-related in the study period. ARPs were more likely to occur (29%) over the summer months (December, January, February) compared to other seasons.

More than one in seven injury-related presentations (n=3,308) during the study period were alcohol-related, accounting for 65% of all ARPs (Table 3). Alcohol-related injury presentations (ARIPs) were more commonly males (20% of all injury presentations cf. 13% for females). Half (53%) of ARPs arrived at the ED via emergency services, compared with only 28% of non-ARPs. Eight percent of ARPs were classified as having life-threatening conditions (Australasian Triage Scale [ATS] 1) compared with only 3% of non-ARPs.

Using the total length of stay recorded for individuals from presentation to the ED until either admission to the hospital or discharge, we determined the median and interquartile range for both ARPs and non-ARPs. ARPs had a median ED length of stay of five hours (interquartile range [IQR] 3h10m–7h19m) compared to a median stay of 2 hours and 58 minutes (IQR 2h42m–5h40) for non-ARPs. The majority of both ARPs and non-ARPs were discharged home from the ED, however, ARPs were more likely to self-discharge (6% cf. 2%), leave the ED prior to treatment (5% cf. 2%) and be forcibly removed (0.1% cf. 0.01%) compared with non-ARPs (Table 4).

Table 4: Distribution of discharge type descriptions of Auckland Hospital Emergency Department presentations by alcohol status (column percentages).

Variables	Total n (%)	Alcohol-related n (%)	Not alcohol-related n (%)	Not known n (%)
Total presentations	73,381 (100%)	5,130 (7.0%)	64,864 (88.4%)	3,387 (4.6%)
Discharge type				
Home	43,100 (58.7%)	4,047 (78.9%)	36,701 (56.6%)	2,352 (69.4%)
Routine discharge from ED	38,510 (52.5%)	3,453 (67.3%)	34,027 (52.5%)	1,029 (30.4%)
Self-discharge from ED	1,834 (2.5%)	321 (5.7%)	1,366 (2.1%)	147 (4.34)
Forcibly removed	23 (0.03%)	7 (0.1%)	4 (0.01%)	12 (0.4%)
Patient did not wait	2,733 (3.7%)	205 (5.2%)	1,304 (2.0%)	1,164 (34.4%)
Admitted	28,806 (39.3%)	1,020 (19.9%)	26,914 (41.5%)	872 (25.8%)
Transfer to another healthcare facility	650 (0.9%)	51 (1.0%)	507 (0.8%)	92 (2.7%)
Deceased*	791 (1.1%)	11 (0.2%)	714 (1.1%)	66 (2.0%)
Unknown	34 (0.1%)	1 (0.02%)	28 (0.04%)	5 (0.2%)

* Death could have occurred in ED or as an inpatient.

Discussion

This observational study has quantified the prevalence of ARPs to the Auckland ED and described the profile and outcomes of these presentations compared to those with non-ARPs. The findings have provided important insight into the role alcohol plays in a large urban New Zealand ED. Over the 12-month period reviewed, alcohol played a role in 7% of presentations, equating to 5,130 patients. Strengths of the study include its size, over 73,000 patients, the completeness of data relating to the role alcohol played in the admission, and the 12-month data review period which enabled analysis of temporal factors.

It is likely that the 7% prevalence of ARPs among all presentations to the ED found in the current study is an underestimate. This finding is at the lower end of the range of previous study findings on the role of alcohol in the ED, which identified alcohol involvement in between 5–20% of all ED presentations.[11,12] In the 2013 study performed on a Saturday in December across multiple Australasian EDs, Egerton Warburton et al found up to 14% of presentations were alcohol-related.[8] However, their study index time was 0200 in the morning when there is more likely to be ARPs. Similarly, the study by Indig et al evaluating staff attitudes and perceptions of the impact of alcohol in the ED, attributed up to 18% of weekday ED presentations as alcohol-related.[13] The comparatively lower prevalence found for ARPs at Auckland ED may in part reflect the 12-month duration of this study, which is longer than many of the studies reviewed, demonstrating variations in the prevalence of ARPs at certain times of the year. The exception to this is the study by Indig et al, which looked at the prevalence of alcohol or drug (AOD) presentations to Australian EDs over a 24-month period, and found that 5% of presentations were alcohol related, similar to our findings.[11] While the study period and time of data collection were factors that could influence the number of presentations recorded as alcohol-related, the likely exclusion of presentations that are not immediately related to alcohol consumption (coded as 'secondary' alcohol use) such as those of chronic alcohol use and related problems could further explain the lower than expected prevalence.

Sixteen percent of injury-related presentations in the present study were alcohol-related, lower than the 21% found in the 2018 study of alcohol and injury among attendees to the Auckland ED by Kool et al.[14] However, the Kool et al study assessed alcohol consumption using an interviewer-administered World Health Organization Emergency Room Collaboration Analysis Project (WHO/ERCAAP) questionnaire, in which breath alcohol and patient self-report were used to assess alcohol consumption. This comprehensive approach is likely to more accurately represent the burden of alcohol in the ED in the context of injury-related presentations. The use of a screening tool such as the Alcohol Use Disorders Test (AUDIT) in the ED and or blood/breath alcohol would increase the precision of our estimates.[15] However, the present study made use of data collected as part of the recently mandated MOH 'alcohol involved field'. Future studies could look to validate the information collected in this manner with a validated tool such as the AUDIT.

Males comprised almost two-thirds of ARPs to the ED in this study. This is consistent with findings from Whitlam et al's study of 1,000 ARPs to New South Wales (NSW) EDs in Australia that identified 64% of these presentations as male.[16] This skewed distribution of gender among ARPs is confirmed in the published literature, as are trends in age of presentations to the ED.[11,17–19] In accordance with our data showing the highest proportion of ARPs found in young adults (<29 years), both Stewart et al[19] and Muscatello et al[20] found that patients with ARPs were commonly aged between 16 and 25 years. However, the surveillance study by Muscatello et al looking at all acute ARPs from NSW EDs identified that while the highest rates of ARPs were among males and young adults, in the 10-17 year-old age group, females represented a higher proportion of ARPs compared to their male counterparts. Māori were over represented among ARPs in the current study, accounting for 17% of ARPs while only representing 8% of the Auckland DHB population.[9] The 2018 study by Kool et al looking at acute alcohol involvement in injury-related presentations (those who had consumed alcohol within six hours of presentation) to the Auckland ED demonstrates similar findings, with Māori accounting for 15% of all ARIPs, a higher proportion than that found for Pacific, Asian or NZE/Other ethnicities.[14] Our findings highlighted some discrepancies between ARPs and socioeconomic status, with a higher proportion of those in more deprived areas (NZDep 9–10) found for ARPs compared to non-ARPs. No studies were located in the published literature that had reported on ARPs by socioeconomic deprivation.

ARPs to the ED showed temporal patterns in our data that are supported in the published literature, confirming that ARPs are more common late at night, [6,12,16,19,21,22] in the weekends[6,12,19] and during the summer months,[23] likely to reflect the drinking culture of the community. The study by McLay et al found that over one week in December 2014, up to 33% of ED presentations between 12am and 4am were alcohol related.[22] Similarly, Hides et al found in a study of young adults presenting to the ED with an injury that the majority of ARIPs occurred between 10pm and 5am and more than half presented on the weekends.[24] The retrospective surveillance study in rural coastal towns of Australia by Coomber et al highlights the seasonal trends in alcohol consumption and consequential ARIPs to the ED, where the prevalence of these increased over the summer months of the year compared to others, particularly for males.[23]

Previous research confirms our findings in relation to the distribution of triage category and arrival methods for ARPs, where the majority of presentations were triaged into a more urgent category and arrived by EMS to the ED. For example, both McLay et al[22] and Egerton-Warburton et al[18] found that the majority (57–59%) of ARPs were assigned a more serious triage category, classed on the Australasian Triage Scale as categories 1–3. In addition, across two surveillance studies by Indig et al it was reported that ARPs were more likely to arrive by ambulance to the ED and require police or hospital security staff involvement upon presentation in comparison to non-ARPs.[12,25]

In the present study, almost two-thirds of ARPs were injury-related. We did not have data regarding the specific type of injury mechanism. The prospective study by McLay et al describing the profile of ARPs to a Perth ED found that individuals with ARPs were more likely than non-ARPs to have an

injury or mental health diagnosis.[22] Further literature supports injury-related diagnoses as the most common among ARPs.[18,25] While ARIPs make up a significant portion of the alcohol-related harm in the ED, future research should be inclusive of all ARPs in the context of both acute and usual alcohol use to ensure that the full extent of the role alcohol plays in the ED is captured.

The negative impacts of ARPs on patients' own health outcomes as well as that of other patients in the ED are well documented.[4,18] The current study found that ARPs to the Auckland ED had longer length of stays than non-ARPs. Moreover, ARPs were more likely to be forcibly removed, self-discharge or leave prior to treatment compared to non-ARPs. Further analysis into how these presentations impact the ED is provided in the study by Butler et al that looks at the effects of drug and alcohol use among patients in a hospital ED.[17] These individuals were more likely to cost more per presentation and stay longer if admitted.

The present study was not designed to evaluate the impact ARPs have on the ED with regards to clinicians' working environment and the effect of these presentations on other patients, both significant areas when assessing the role alcohol plays in the department. A survey of Wellington hospital ED staff by Gunasekara et al in 2011 reported that ARPs negatively impacted the ED in a multitude of areas, from increasing the workload and waiting times to negatively affecting the staff mood and care of other patients.[26] Furthermore, up to 85% of respondents in the study felt that no suitable measures were in place in the ED to handle ARPs and their impact on the ED. A survey completed by ED clinicians across Australasia found that 98% and 92% of respondents had experienced verbal and aggression respectively from an alcohol-affected patient in the last year.[4] ARPs divert resources away from other patients and add strain to the ED, diminishing job satisfaction for ED staff and affecting the quality of care for all.[4,7,13,26,27]

The study findings should be considered in light of some limitations. We have no available information regarding the reliability of our data in relation to the role alcohol played in the admission. As data collection on ARPs had recently been introduced into the ED prior to our study, the definition of an ARP was developed prior to implementation, however this was a new concept for staff and may not have been utilised rigorously. While there were guidelines in place to aid the clinician, informed judgements were made which may have introduced measurement error. Ascertainment bias may have also occurred for patients who appeared intoxicated and were assumed to have an ARP rather than other drugs being primarily involved in their presentation. The alcohol-related question encompasses 'secondary' alcohol involvement, which may not have been elicited by the ED team therefore result in an underestimate of the secondary involvement of alcohol in ED presentations. This study is not population-based as Auckland Hospital is one of the three major admitting public hospitals in the Auckland region, which may limit generalisability of the findings. However, in light of the similar drinking culture across New Zealand and Australia, this study may have some external validity when looking at the impact alcohol has on EDs in general.

We were unable to identify any relationship between reasons for presenting to the ED and alcohol involvement in our study. The 'presenting complaint' free text field in our data was completed by ED triage nurses and describes the reason for a patient's presentation to the ED. However, due to the widely varying codes and descriptions used for this field, we were unable to identify any consistent trends using this data. Injuries are more clearly identified at triage and did allow analysis of ARIPs.

The present study highlights the burden of alcohol misuse on the ED. There is some evidence that the implementation of alcohol screening and brief intervention (SBI) programmes in the ED setting may be effective in reducing the harmful use of alcohol and other drugs.[28] A 2017 systematic review assessing the effectiveness of SBI in the ED setting found variable evidence where almost half of the studies failed to show an intervention effect for the outcome of alcohol consumption reduction, however there may be subgroups that have improved outcomes.[29] A recent study by Patson et al looking at the feasibility of SBI in the ED

found that this technique provided potential benefits for the patients with ARPs, their families and their nurses, however application of the SBI may create potential challenges for the ED with regards to an already immense staff workload and high patient to clinician ratio.[30]

Our findings and those of similarly published studies emphasise the need for continued public health efforts to implement preventative strategies for alcohol-related harm in the ED and society as a whole. Raising awareness of the harms associated with alcohol through media and targeted programmes, along with evidence-based alcohol policies are among some of the most effective preventative approaches.[8,31] Marketing restrictions, regulating the availability of alcohol, and modifying the drinking context using community-based solutions also offer the opportunity to reduce alcohol-related harm, and in doing so relieve the burden of alcohol in the healthcare sector.[31] A study by Connor et al examining alcohol outlet density in New Zealand found the density of outlets is associated with increased binge drinking and alcohol-related harm.[32] These findings reinforce the need for local area alcohol policies to address the concentration of bars and off-licences in New Zealand. In 2012, the Sale and Supply of Alcohol Act (SAAA) was introduced which had as its key objective the minimisation of harm from excessive or inappropriate consumption of alcohol. However, a study by Randerson et al investigating perceived changes in the alcohol environment before and after the implementation of the SAAA found the Act's introduction had little impact on the alcohol environment during the period reviewed (2013–15).[33] The findings of the present study confirm the need for continued efforts to develop effective national policy to reduce the harms associated with harmful alcohol use.

Competing interests:
Nil.

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Wintertime Vitamin D status and its related risk factors among children living in Auckland, New Zealand

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ABSTRACT

AIM: To investigate vitamin D status and its determinants in school-aged children living in Auckland, New Zealand.

METHODS: Healthy children (n=507) aged 8–11 years were recruited from six primary schools to include a range of ethnicities and sociodemographic characteristics. Finger-prick blood spots were collected and analysed for capillary 25-hydroxyvitamin D (25(OH)D). Weight and percentage of body fat (%BF) were measured using the InBody 230 (Biospace Co. Ltd., Seoul, Korea). Information related to ethnicity, skin colour, physical activity and sun exposure were sought from parents through a questionnaire.

RESULTS: Mean±standard deviation (SD) 25(OH)D concentration were 64±21 nmol/L, with 31% of the population presenting with 25(OH)D≥75nmol/L, 41% 50–75nmol/L and 28%<50nmol/L. Capillary 25(OH)D was significantly higher in New Zealand European compared to all other ethnic groups (75±20nmol/L, P<0.001). As expected, children with dark/brown skin colour had lower 25(OH)D levels compared to other skin colour categories (51±18nmol/L, P<0.001). Using multiple logistic regression analysis, determinants of 25(OH)D were %BF and ethnicity.

CONCLUSION: Approximately one-third of this population had 25(OH)D<50nmol/L. Determinants of a 25(OH)D<50nmol/L included %BF and ethnicity. Wintertime serum 25(OH)D was highly variable. There are some children at high risk of 25(OH)D<50nmol/L for whom supplementation may be considered.

Vitamin D is a secosteroid and an essential nutrient, which has a crucial role in the absorption of calcium from the intestine, regulation of serum calcium and bone health.[1] Observational and clinical studies suggest there is a relationship between vitamin D deficiency and insufficiency and skeletal health problems (eg, rickets, metabolic bone disease and hypocalcaemia) during childhood.[2,3] Therefore an adequate 25(OH)D concentration is considered important for ensuring bone health during childhood and later in life.

In New Zealand, a small quantity of vitamin D comes from a limited number of foods that naturally contain vitamin D (salmon, herring, tuna and mackerel). There is a limited range of foods including milk and yoghurt, which are sometimes fortified with vitamin D,[4] although there is no mandatory fortification in New Zealand.[4] Therefore, it would be hard to reach acceptable blood levels of vitamin D through diet alone. However, endogenous synthesis through sunlight exposure is the major source of vitamin D for most people living in New Zealand since diet alone is not adequate to meet recommended 25(OH)D concentrations.[4] Nevertheless, the efficacy of cutaneous synthesis of vitamin D can be influenced by various factors including geographic latitude, season, time of day,[5] ethnicity,[6] obesity,[7] waist circumference,[8] age[9] and gender.[10] Furthermore, some other conditions such as skin pigmentation, use of sunscreens, exposed body surface and exposure duration,[11] and restricted sunlight exposure habits (eg, clothing)[12] can affect the cutaneous vitamin D synthesis.

Unfortunately, there are limited data available regarding the vitamin D status and risk factors for vitamin D deficiency in New Zealand children.[6,13,14] Therefore, the aims of the present study were to assess wintertime vitamin D status in New Zealand children living in Auckland and to identify related risk factors for deficiency.

Methods

Study participants

In this cross-sectional study, we recruited children aged 8–11 years from six Auckland, New Zealand primary schools (one from north, two from east, two from south, and one from central) (in August 2016 and 2017—late winter in southern hemisphere). We originally approached schools through a collaboration of primary school science teachers and asked for expressions of interest. We then endeavored to recruit schools specifically to include a range of sociodemographic levels and ethnicities. The study protocol was approved by the Human Ethics Committee of Massey University (Southern A; approval no. MUHECN 16/42). All children and their parents provided written informed consent prior to participating in the study. The study was run in agreement with the Declaration of Helsinki.[15] Children who were apparently healthy were sought. Children were ineligible when they met the following exclusion criteria 1) a history of any disease affecting vitamin D metabolism (eg, cardiac, kidney or liver disease) or 2) a history of any long-term medication use (eg, steroids) 3) having had any surgical implants, metal screws or similar, or 4) having a cast. All data collection from the children took place at their schools on one occasion.

Data collection

Sample size

Children were stratified by gender (two groups), ethnicity (six categories), and skin colour (four groups) and logistic regression used to determine contribution of risk factors for vitamin D deficiency. A sample of 10–15 per factor per group is the standard requirement for regression analysis. Therefore: 2*6*4*10–15 = 480–720 participants.[16]

Anthropometric measurements

Information about participants' weight and height were collected. Children were asked to remove their shoes to measure their height (to an accuracy of 0.1cm) using a Seca 213 portable stadiometer. Body weight was recorded in minimal clothing to the nearest 0.1kg through InBody 230 (Biospace Co. Ltd., Seoul, Korea). Body mass index (BMI) was derived by dividing weight (kg) by the square of height (m^2). We calculated BMI adjusted-for-age as described by Cole et al (2000 and 2007).[17,18] Percentage of body fat (%BF) was measured by bioelectrical impedance analysis (BIA), the InBody 230 (Biospace Co. Ltd., Seoul, Korea) according to the standard procedure provided by the manufacturer. The Inbody 230 has been validated in the same population (the abstract in proceedings).

Healthy and demographic questionnaires

Parents were asked to identify their child's ethnic group. Ethnicity was categorised as 1) European New Zealanders, 2) Māori, 3) Pacific, 4) South Asian and 5) Chinese/Korean/Southeast Asian, and 6) other ethnicity. If participants reported more than one ethnic group, then based on the priority system,[19,20] the child was assigned to one ethnic groups. The following ethnicity prioritisation was used for analytical purposes: Māori>Pacific>South Asian> Chinese/Korean/Southeast Asian>European New Zealanders>Other. For instance, if Māori was one of the reported groups, then the child was allocated to Māori group. Parents were asked to classify their child's skin colour. Skin colour was categorised as 'fair', 'medium', 'olive' or 'dark/brown'.

Information about the sunlight exposure, applying sunscreen, and the frequency of using sunscreen was obtained from a self-reported parents' questionnaire. Parents were asked to identify which parts of the body were usually exposed to sunlight, and what the usual sunscreen usage was.

Physical activity was evaluated with the short version of the "International Physical Activity Questionnaire (IPAQ)".[21] Three types of activity were assessed: walking, moderate-intensity and vigorous-intensity

activities. Data collected using IPAQ were categorised into three levels: low, moderate and high.[21]

Dried blood spot sampling and vitamin D assay

The dried blood spot (DBS) method was used, as it is a minimally invasive and convenient technique for children. A trained researcher used a single-use safety lancet to collect sufficient capillary blood from the child's finger. Blood spots were collected onto a Whatman filter paper card within a pre-marked area. The sample cards were air dried at room temperature before being placed into a sealed plastic bag. All the DBS cards were refrigerated and stored at 4°C until sent to the Canterbury Health Laboratories for further analysis.

Measurement of 25(OH)D in dried blood spots was performed by a newly developed method utilising two-dimensional liquid chromatography tandem mass spectrometry (2D LC-MS/MS) following methanol and hexane extraction of the dried blood spots. Capillary calculated serum concentrations of 25(OH)D were determined by assuming a hematocrit of 0.39. The method has been thoroughly validated for research applications.[22]

Statistical analysis

The computer software statistical package program SPSS version 24 (IBM Corporation, New York, NY, USA) and R statistical package, version 2.15.1 (R Foundation for Statistical Computing, Vienna, Austria, http://www.R- project.org/) were used to analyse the data. The variables were tested for normality using the Kolmogorov-Smirnov test and Shapiro-Wilk (S-W) tests and for homogeneity using the Levene's test.

The data were normally distributed so parametric tests were used. For baseline characteristics and measurements, continuous variables are expressed as mean±standard deviation (SD) (Table 1) and categorical as number and percentages (n(%)) (Table 2). Pearson's correlation coefficients were used to test relationships between continuous variables. The independent T-test, ANOVA test and Chi-squared test were used to compare 25(OH)D levels between groups.

The association between 25(OH)D status (<50nmol/L) and potential determinants were tested using univariate analysis. The following independent variables (all with a *P*-value of<0.20) were included in the model: age, %BF, gender, physical activity and ethnicity. Age and %BF were entered into the model as continuous variables. All other variables were treated as categorical variables. Reference categories were being male, having normal physical activity and being of New Zealand European ethnicity. Body Mass Index, body part sun exposure and sunscreen user did not meet the screening criteria (*P*-value≥0.20) so were not included as they were unlikely to contribute to a model contacting other potential determinants of 25(OH)D<50nmol/L. Interaction between ethnicity and skin colour did not pass the initial screen, therefore only ethnicity entered to the model. There was not any collinearity between variables. Forward stepwise multiple logistic regression analysis with the entry criterion set at *P*-value<0.05 was used to determine which variables to include in the final model.

Results

A total of 507 children (237 (47%) boys) participated. The main characteristics of the study population group are presented in Table 1. The distribution of ethnic groups was: 191 (38%) New Zealand European, 64 (13%) Māori, 108 (21%) Pacific, 47 (9%) South Asian, 65 (13%) Chinese/Korean/Southeast Asian and 32(6%) of others not specified. The mean age was 10±1 years (range 8–11 years). 25(OH)D concentrations were negatively associated with age (*P*<0.05), weight, BMI and %BF (*P*<0.01). Most participants were normal (86%) with a mean BMI of 19 kg/m^2. High physical activity levels were reported in 36% of participants. The most common skin colour was 'medium' (40%) with 'olive' (29%) ranking second. Our results showed 90% of children exposed only legs; arms and legs; face and arms and legs to the sunlight. The majority used sunscreen only in summer time (87%) and 75% applied sunscreen on their only legs; arms and legs; face and arms and legs.

Capillary (calculated) serum concentrations of 25(OH)D ranged from 9–123nmol/L. Mean±SD 25(OH)D concentration was 64±21nmol/L. The prevalence of 25(OH)D<50nmol/L, 50–75nmol/L, and ≥75nmol/L was 28, 41 and 31%, respectively (Figure 1).

Table 1: Characteristics of population group (n=507) and Pearson correlation between 25(OH)D and continuous variables.

	Mean±SD	Correlation coefficient (*r*)	*P*-value
Age (years)	10 (1)	-0.123	0.006
Weight (kg)	39 (11)	-0.178	<0.0001
Height (cm)	144 (8)	-0.020	0.656
BMI (kg/m^2)	19 (4)	-0.201	< 0.0001
Body fat percentage	22 (9)	-0.233	< 0.0001
25(OH)D (nmol/L)	64 (21)	-	-

SD standard deviation.

The relationship between 25(OH)D levels and their potential determinants are summarised in Table 2.

Mean 25(OH)D did not differ significantly between boys and girls (66±20 vs 62±21nmol/L; *P*>0.05), or by levels of physical activity. However, mean 25(OH)D was significantly higher in normal compared with overweight/obese children (65±21 vs 55±18nmol/L; *P*<0.005). New Zealand European children had the highest mean 25(OH)D concentrations of 75±20nmol/L and South Asian children had the lowest 49±20nmol/L. Participants reporting 'dark/brown' skin colour had lower mean 25(OH)D levels (51±18nmol/L) compared to the other skin colours. Body parts exposed to sunlight, use of sunscreen, frequency of use of sunscreen and body parts where sunscreen applied were significantly associated with 25(OH)D (Table 2).

Figure 1: Prevalence of 25(OH)D status (<50nmol/L, 50–75nmol/L and ≥75nmol/L).

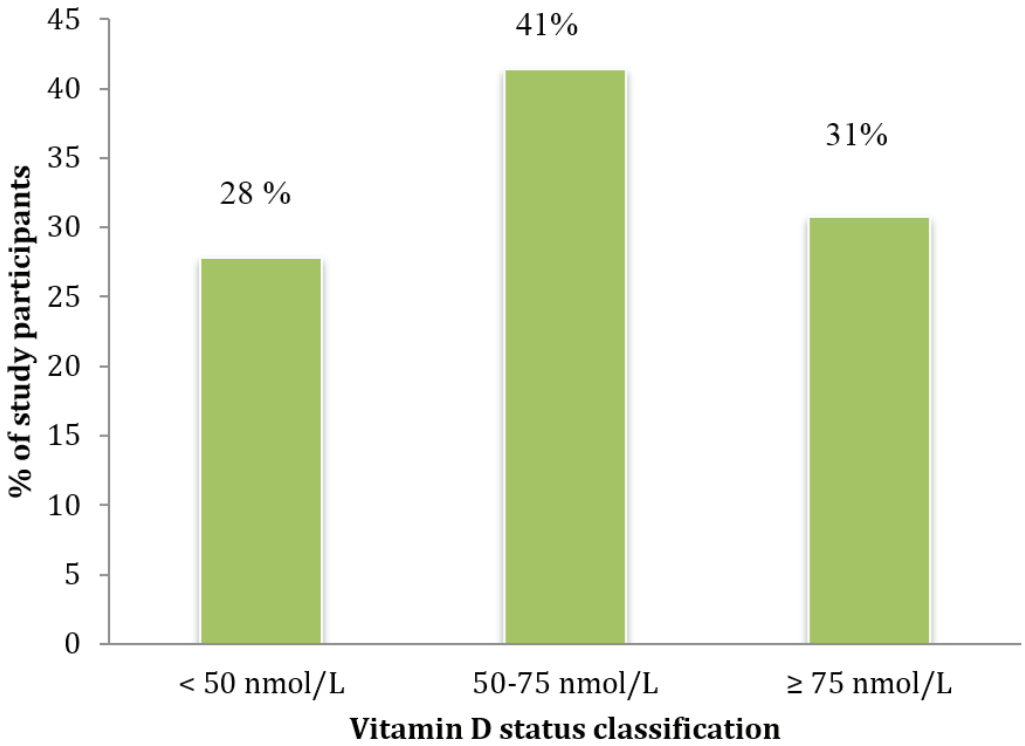

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Table 2: 25(OH)D levels and its related determinants.

	n (%)	25(OH)D, nmol/L mean±SD	*P*-value[1]	25(OH)D <50nmol/L n (%)	25(OH)D 50–75nmol/L n (%)	25(OH)D ≥75nmol/L n (%)	*P*-value[2]
Gender			0.190				0.140
Boys	237 (47)	66 (20)		56 (24)	105 (44)	76 (32)	
Girls	270 (53)	62 (21)		85 (31)	105 (39)	80 (30)	
BMI status[3]			<0.005				<0.004
Underweight	26 (5)	65 (23)		6 (23)	9 (35)	11 (42)	
Normal weight	434 (86)	65 (21)		114 (26)	179 (41)	141 (33)	
Overweight and obese	47 (9)	55 (18)		21 (45)	22 (47)	4 (8)	
Physical activity levels			0.627				0.129
Low level	176 (35)	65 (21)		45 (26)	70 (40)	61 (35)	
Moderate level	146 (29)	63 (22)		49 (34)	52 (36)	45 (31)	
High level	185 (36)	64 (19)		47 (25)	88 (48)	50 (27)	
Ethnic group			<0.0001				
New Zealand European	191 (38)	75 (20)		22 (11)	73 (38)	96 (50)	<0.0001
Māori	64 (13)	66 (19)		20 (31)	23 (36)	21 (33)	
Pacific	108 (21)	57 (17)		37 (34)	56 (52)	15 (14)	
South Asian	47 (9)	49 (20)		29 (62)	13 (28)	5 (11)	
Chinese/Korean/Southeast Asian	65 (13)	57 (15)		21 (32)	33 (51)	11 (17)	
Other	32 (6)	57 (23)		12 (8)	12 (37)	8 (25)	
Skin colour group			<0.0001				
Fair	58 (11)	66 (22)		14 (24)	24 (41)	20 (34)	<0.0001
Medium	199 (40)	69 (20)		37 (19)	83 (42)	79 (40)	
Olive	146 (29)	65 (19)		36 (25)	64 (44)	46 (31)	
Dark/brown	104 (20)	51 (18)		54 (52)	39 (37)	11 (11)	
Body part exposed to sun			0.001				<0.001
Only face; only arm; face and arm	53 (10)	54 (21)		28 (53)	15 (29)	10 (19)	
Only legs; arms and legs; face, arms and legs	454 (90)	65 (20)		113 (25)	195 (43)	146 (32)	
Sunscreen user			0.003				0.099
Yes, all year	31 (6)	63 (24)		9 (29)	12 (39)	10 (32)	
Yes, only summer	441 (87)	65 (21)		116 (26)	184 (42)	141 (32)	
No	35 (7)	53 (18)		16 (46)	14 (40)	5 (14)	
Frequency sunscreen use			<0.0001				0.001
Always	205 (40)	69 (22)		46 (22)	83 (40)	76 (37)	
Sometimes	241 (48)	62 (19)		67 (28)	102 (42)	72 (30)	
Rarely and never	61 (12)	54 (17)		28 (46)	25 (41)	8 (13)	
Body part apply sunscreen			<0.0001				0.060
Only face; only arm; face and arm	88 (17)	65 (22)		25 (28)	31 (35)	32 (37)	
Only legs; arms and legs; face, arms and legs	382 (75)	65 (21)		100 (26)	163 (43)	119 (33)	

[1]Independent T-test (for two categories) and ANOVA test (more than two categories), [2]Chi-squared test, [3]Cole et al (2000) and Cole et al (2007).
SD standard deviation.

Logistic regression analysis results on 25(OH)D and its determinants are shown in Table 3. There was a significant inverse association between %BF and an increased odds ratio (OR) of 25(OH)D<50nmol/L (OR 0.96, CI 95%: 0.94–0.99, *P*<0.001). All other ethnic groups compared to reference group (New Zealand European) were at higher risk of 25(OH)D<50nmol/L (OR from 0.07 to 0.28, *P*<0.0001).

Table 3: Results of stepwise linear regression identifying determinants of 25(OH)D<50 nmol/L.

Variable		25(OH)D<50nmol/L vs 25(OH)D≥50nmol/L)		
	B (SE)	OR[1]	95% CI for OR	P-value
Body fat percentage	-0.03 (0.01)	0.96	0.94, 0.99	0.008
Ethnicity group				
(NZ European vs Maori)	-1.26 (0.39)	0.28	0.13, 0.60	0.001
(NZ European vs Pacific)	-1.39 (0.35)	0.24	0.12, 0.49	< 0.0001
(NZ European vs South Asian)	-2.54 (0.40)	0.07	0.03, 0.17	< 0.0001
(NZ European vs Chinese/Korean/ Southeast Asian)	-1.58 (0.37)	0.20	0.09, 0.42	< 0.0001
(NZ European vs other)	-1.80 (0.45)	0.16	0.06, 0.40	< 0.0001

Following independent variables were included in the model: age, body fat percentage, gender, physical activity and ethnicity.
[1]Change in odds of 25(OH)D<50nmol/L occurring for each unit change in determinant variable. If >1.0, as determinant variable increases, odds of 25(OH)D<50nmol/L increase. If <1.0, as determinant variable increases, odds of 25(OH)D<50nmol/L decrease.
R^2 =0.22 (Hosmer and Lemeshow), 0.12 (Cox and Snell), and 0.17 (Nagelkerke). Model X^2=65.49.
CI confidence interval; *OR* odds ratio; *NZ* New Zealand.

Discussion

There are limited data available regarding wintertime vitamin D status and its related determinants in New Zealand children. Approximately one-third of the study population had 25(OH)D≥75nmol/L and about one-third had 25(OH)D<50nmol/L. Body fat percentage and ethnicity were the strongest predictors of 25(OH)D<50nmol/L, while many established contributors to vitamin D status (eg, gender, physical activity) were not associated with 25(OH)D<50nmol/L in this population.

There is not a worldwide consensus about acceptable vitamin D concentrations. Different cutoff points have been set for populations based on the relationship between vitamin D status and various criteria such as parathyroid hormone (PTH) levels, intestinal calcium absorption and bone mineralisation. It has been shown that 25(OH)D levels less than 50nmol/L may cause hypo-calcemia and secondary hyperparathyroidism in children.[23] Based on New Zealand consensus, aiming for 25(OH)D≥50nmol/L seems prudent.[4] In our study, nearly one-third (31%) of participants had 25(OH)D≥75nmol/L, 41% had 25(OH) D=50–75nmol/L and 25(OH)D<50-nmol/L was detected in approximately one-third (28%) of children. Our data are comparable to previous studies in children and adolescents.[6–8,24–27] For instance, Alemzadeh et al,[24] using the same cutoff points for vitamin D categories, showed 32% and 41.7% of 6–18 year olds (49 Caucasian, 39 Hispanic and 39 African American) had 25(OH)D<50nmol/L and between 50 and 75nmol/L, respectively. There are a few studies that have investigated the status of 25(OH)D among the New Zealand paediatric population. Rockell et al[6] used 37.5nmol/L 25(OH)D as the cut-off value and found a high prevalence of 25(OH)D insufficiency 41%, 59% and 25% among Māori, Pacific and New Zealand European school-age children aged 5–14 years, respectively. Recently, Cairncross[13] collected capillary blood spots late-winter to early spring and measured 25(OH)D in 1,329 preschool children (2–4 years old). Their results showed (86) 7% and (642) 48% of children had vitamin D deficiency (<25nmol/L) and insufficiency (<50nmol/L) respectively. Therefore, further demonstrating that wintertime 25(OH)D concentrations are of concern for some New Zealand children.

Previous studies have suggested a significant inverse association between vitamin D status and fat mass, due to vitamin D being a fat-soluble vitamin.[28,29] Our results showed an inverse correlation between weight, BMI and %BF and vitamin D status. Children

with normal BMI showed significantly higher 25(OH)D compared to overweight/obese children (65nmol/L vs 55nmol/L). Wortsman et al,[29] suggesting obesity is associated with vitamin D insufficiency since vitamin D can be deposited in adipocytes, and therefore its bioavailability decreases. Also, in our logistic regression analysis, having a higher %BF increased the chance of having 25(OH)D<50nmol/L after accounting for age, gender and physical activity.

In our study New Zealand European children had higher 25(OH)D concentration (75 nmol/L) compared with all other ethnic groups. Rockell et al[6] demonstrated the prevalence of vitamin D insufficiency (<37.5nmol/L) was 38, 58 and 23% in Māori, Pacific and New Zealand European children aged 5–14 years, respectively. Cairncross[13] found that the prevalence of vitamin D deficiency (<25 nmol/L) was 9 and 23% in Māori and Pacific children, respectively, compared with 3% in New Zealand European children. We found 11% of New Zealand European children, 31% of Māori children and 34% of Pacific children had 25(OH)D less than 50nmol/L. The highest prevalence of 25(OH)D<50nmol/L was in South Asian children (62%) with a mean of 49nmol/L. Results from the logistic regression analysis demonstrated ethnicity is a predictor of 25(OH)D<50nmol/L and South Asian children had the highest odds of 25(OH)D<50nmol/L compared to the New Zealand European group. In a review paper, Akhtar[30] reported that vitamin D deficiency (20–50nmol/L) is highly prevalent among South Asian population. A possible reason for these results can be related to differences in skin colour among different ethnic groups. In the present study, most New Zealand European children (56%) reported 'medium' skin colour while 36% of Māori children reported having 'olive' skin colour. About half (47%) of Pacific children and more than half (57%) of South Asian children had dark/brown skin colour. The negative association of vitamin D status with skin colour is well documented in previous studies.[26,27] We found that children reporting a 'dark/brown' skin colour had significantly lower 25(OH)D concentrations than children reporting a 'fair' skin colour (51nmol/L vs 67nmol/L). It is suggested that the cutaneous synthesis of vitamin D is lower in individuals with a darker skin colour due to the ultraviolet radiation beta (UVβ) photons being less absorbed through melanin pigmentation.[31]

Contrary to previous studies,[10,32–34] and in line with Vierucci et al[7] and Avagyan et al,[25] we did not find a significant difference in 25(OH)D concentration between boys (66nmol/L,) and girls (62nmol/L). It has been proposed that gender differences in vitamin D are related to BMI status.[28,29] In the current study, there was not a significant difference in BMI between boys (mean 18kg/m^2) and girls (mean 19kg/m^2).

It has been established that sedentary lifestyle and less physically active people have lower 25(OH)D values.[35] Less physically active people usually spend less time outdoors and therefore have limited opportunity for sun exposure, with an increased risk of obesity. In this study we did not find a significant relationship between vitamin D status and physical activity. Also, the mean of BMI across all three physical activity levels did not differ significantly.

In the literature, several factors have been postulated that affect cutaneous synthesis of vitamin D through sun exposure, such as body surface area, time of the day, season, latitude, degree of skin pigmentation and sunscreen use.[36,37] In this study, we asked parents to identify which parts of the body were usually exposed to sunlight, frequency of use and where sunscreen was applied. Our data confirm the prevalence of vitamin D deficiency is lower in children who exposed more surface of their body (only legs; arms and legs; face and arms and legs) to the sunlight.

It is suggested that properly applying sunscreen with a sun protection factor (SPF) of 30 can decrease cutaneous synthesis of vitamin D by as much as 95–99%.[36] In our study, similar to Cairncross et al,[14] our participants who reported that they applied sunscreen and were more frequent users, had higher 25(OH)D concentrations compared to the other groups. An explanation for these findings may be because of the lack of parents' knowledge about how to apply sunscreen. Furthermore, we did not ask parents or children how often they renewed application of sunscreen each day and the SPF of the sunscreen used. The amount of sunscreen applied[38] and how often

it is re-applied influences its effectiveness. We could also speculate that the children who were using sunscreen were going out in the sun, but those who were not applying sunscreen were not exposing themselves.

In this study, we observed that 28% of the study population presented with 25(OH) D values<50nmol/L. An effective strategy is needed to prevent vitamin D status less than 50nmol/L and its related health consequences among children. Increasing sun exposure, especially in high-risk children (overweight/obese, darker skin colour, specific ethnic groups) should be the first-line treatment. For most people living in New Zealand, exposure to the sun is the major source of vitamin D.[4] The body is able to synthesise sufficient vitamin D with adequate skin sunlight exposure.[39] However, there is no evidence regarding the safe threshold level of ultraviolet (UV) radiation exposure from the sun without increasing risk of skin cancer.[4] A balance between avoiding excessive sun exposure to prevent skin cancer, and enough sun exposure to achieve adequate vitamin D levels, is required.[4] The New Zealand Ministry of Health recommends a daily walk or some other form of outdoor physical activity in the early morning or late afternoon between the summer months of September and April.[4] Between May and August (winter in New Zealand), except at high altitudes or near highly reflective surfaces (eg, snow or water), sun protection is generally not recommended. During this time exposure to direct sunlight, especially in the hours around noon when ultraviolet radiation beta (UVβ) levels are highest, will be enough for adequate cutaneous production of vitamin D.[4] However, pediatricians should monitor children's sun exposure and vitamin D levels in high-risk subjects, including those who are overweight/obese, have dark skin colour or who are of South Asian ethnicity. These groups may benefit from vitamin D supplementation.[4]

Strengths and limitations

One of the limitations of this study is that our results are not necessarily transferable to other populations since we recruited only healthy children living in Auckland, New Zealand. Also we recruited children based on their schools' collaboration, so we cannot claim that our participants are representative of all children living in Auckland. For instance, the proportion of Pacific and Asian groups seem high while the Māori on the low side. However, we tried to include a range of sociodemographic levels and ethnicities. This study was a cross-sectional study at the end of winter, and lacked longitudinal assessment of vitamin D status, therefore we could not consider the effect of seasonal variation on vitamin D status. In addition, we collected some information regarding vitamin D risk factors (eg, body exposure area, use of sunscreen, physical activity) through self-reported questionnaires, so recall bias could have influenced our results. Although the DBS method is minimally invasive for measuring vitamin D and previous studies have used it,[14] it has not been specifically validated in children. Despite these limitations, this study had some notable strengths and suggested a general pattern of vitamin D status and its related risk factors among school-age children living in Auckland, New Zealand. We recruited a large sample of healthy children from a broad range of sociodemographic and ethnic backgrounds. Also we used %BF, which is a better indicator of fatness in an individual than BMI.

Conclusion

Our data indicates an association between winter vitamin D status and its related factors in children living in Auckland, New Zealand. 25(OH)D<50nmol/L was present in about one-third of our participants. Ethnicity and %BF were the only significant predictors of 25(OH)D<50nmol/L, with no association shown between gender, physical activity and 25(OH)D in our population. Being overweight and obese, being from South Asian ethnicity, having darker skin colour and exposing less surface of the body to sunlight significantly affected vitamin D status. Poor vitamin D status during childhood can affect long-term health, so opportunities to intervene during childhood should be pursued. A strong consideration should be given to the high-risk children and production of cutaneous vitamin D through sunlight exposure.

Competing interests:
Nil.

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Gender and the surgical profession

Angela D McGregor

ABSTRACT

There is ongoing discussion in the literature about the impact of gender in the surgical profession. This article explains the social expectations of men and women and how these create and perpetuate the gender imbalance. Correcting this imbalance is discussed in the context of supporting a positive, inclusive culture change within surgery.

In 2015 the surgical profession attracted a frenzy of media attention when a female vascular surgeon commented in an interview that female surgical trainees should "accept unwanted sexual advances because coming forward could ruin their careers".[1] The uproar regarding sexual harassment, bullying and discrimination in surgery that followed compelled the Royal Australasian College of Surgeons (RACS) to review the culture of surgery. The subsequent Diversity and Inclusion Plan identified, among other things, that there is gender inequity in surgery.[2] Men can ascend the surgical hierarchy with fewer barriers than females and these need to be addressed to ensure men and women have the same opportunity regarding a career in surgery.

This viewpoint article will discuss the gender imbalance in surgery through three questions:

1. Why does a gender imbalance exist in the surgical profession?
2. what are they key issues that perpetuate this imbalance? and
3. why should the imbalance be addressed?

The overall purpose is to help individual surgeons understand how and why the gender disparity is perpetuated, and to support a positive, inclusive culture change within surgery.

Why does a gender imbalance exist in the surgical profession?

A key issue underpinning the gender discrepancy in surgery is the concept of gender itself, and how this plays out in the social milieu. 'Gender expression' is a socially constructed phenomenon that informs how a person should act, dress, behave and interact, and is based on traditional gender roles.[3] This essay will use the term 'gender' to mean gender expression, as defined above. While there are different ways to express gender, this article will focus on the binary of man/woman.

There is no difference in competence between men and women in surgery,[4] but there is a difference in the social construction, or *what we expect* of men and women. Traditional, stereotypical characteristics associated with men is that they are decisive, independent and goal orientated, and those associated with women are that they are community minded, caring and sensitive.[5,6] In traditionally male-dominated occupations, male values are maintained as the standard for success for both sexes,[7–9] therefore in surgery those characteristics deemed as important for a surgical career align with male characteristics. When a male strives for professional achievement, he meets our stereotypical expectation of him, and *both men and women* like him more, the result being that at each step of his

career he is encouraged, congratulated and positively reinforced.[5,10] In contrast, women are expected to be community minded, caring and sensitive, so when she strives for professional achievement by aspiring to become a surgeon she violates our stereotypical expectation of her. The result is that she is liked less by both men and women and it becomes more difficult to ascend the surgical hierarchy. Competence and likeability are positively correlated for men, but inversely correlated for women.[5,10]

What are they key issues that perpetuate this imbalance?

These stereotypical expectations perpetuate the gender imbalance because it is important to be liked for career progression. Gaining a mentor or advocate who will make introductions and endorse the aspiring surgeon depend in part on the trainee being liked: since competence and likeability are positively correlated for males, as a male ascends the surgical hierarchy these endorsements flourish.[5,6] Compounding this is that people are judged in comparison to the traditional stereotypical expectations of them. Men who are communal may be accused of being 'wimpy' or 'soft' and women who are assertive may be accused of being 'bossy' or 'domineering';[6] a male surgeon who has tantrums in the operating room is characterised as 'temperamental' or 'high strung', whereas a woman surgeon who throws a 'doctor fit' is described as a 'bitch'.[11] This puts women in a double-bind. On the one hand, they need to display the male-aligned characteristics seen as important in surgery to progress their career, but this violates what is expected of them as women and they face a social backlash that in turn impedes their career progression.[5]

These gendered social expectations determine what is acceptable behaviour by men and women so strategies that are employed by men are not always helpful if done by a woman. In response to the surgeon having a tantrum/doctor fit, nurses in the operating room tend to pay scrupulous attention to the male surgeon yet tend to become slow and sulky when this behaviour comes from the female surgeon.[11] For aspiring male surgeons there is no discordance between social expectation and career, whereas aspiring female surgeons must negotiate the discord between female stereotypes and surgeon qualities, and the strategies that will make this negotiation successful. One way to do this is by learning from role models, however many specialties have a paucity of female surgeons, which can make learning these strategies difficult.[12] In addition, if there is only one or very few female surgeons in a specialty, they are perceived as representing what all women must be like to be successful. If the junior doctor doesn't see themselves reflected in this small cohort they may discount a career in surgery.[12] In contrast, junior male doctors have a much greater number of male surgeons who they can look to for role modelling.

Another factor that perpetuates the gender imbalance is that when a profession is dominated by a single group, the dominant group becomes invisible.[13] Surgery is a male-dominated profession and this phenomenon can be observed. For example, searching the literature using keywords 'gender' and 'surgery' return articles that almost solely focus on women. Yet men have a gender, and to progress a career in surgery a female must be selected by the people, mainly men, who are already there. A study from the American Association of Oral and Maxillofacial Surgeons (OMS) has investigated the male perception of women in residency programmes or as practice associates.[14] Fifty-five percent of programme directors, 28% of male residents and 56% of male surgeon practitioners who were approached agreed to participate in this study. Ninety-eight percent of programme directors, 82% of residents and 91% of practicing surgeons agreed that women were as capable to practice OMS as men. While it is pleasing that almost all the programme directors believed women and men were equally capable it is disappointing to note that one in five residents thought women were not as capable as men. Reasons for this included that women lacked adequate physical strength, lacked emotional strength, did not work as fast as men or did not want to work as many hours as men. It would have been particularly interesting if this study had compared patient outcomes. If male OMS surgeons had better patient outcomes, then training programmes could

be tailored to ensure females are acquiring the necessary skills. If females had better patient outcomes, perhaps those reasons identified by their male counterparts as being negatives may in fact be advantageous, and training programmes could be tailored to improve the practice and skill of males. For example, one female surgeon reported she spent more time communicating with patients and their families,[11] meaning she may not "work as fast", but if this were to result in lower rate of complications, mortality or other such outcomes this could be reinterpreted as working more safely and efficiently. This does provide evidence that there is some bias against women in the OMS field, and more research is needed to ascertain if this is isolated to OMS or if it represents widespread beliefs among male surgeons. If the latter is true these beliefs must be addressed in order to improve gender diversity.

When there is a particularly dominant group, such as males in the surgical profession, the lack of diversity and world views means that the system reflects the dominant group.[13] In effect, it becomes a system that is designed by men, for men. This is reflected in the criteria for selection onto many surgical and education training programmes. The process for selection includes an examination, references, an interview and submission of a curriculum vitae. From the documents I was able to access it appears that there is a focus on 'service' or surgical performance: technical skill, work experience, clinical scenarios and publications/presentations. There appears to be little evidence of points being *directly* awarded for displaying collaboration, compassion, respect or integrity, the other four Values of the Royal Australasian College of Surgeons (service is the fifth value).[15] While it is imperative that surgical performance is of the highest standard, formal assessment of these other values may reduce potential institutionalised barriers to female career progression, and enable stereotypical female characteristics and strengths to be *formally* regarded as positive traits for surgeons. It should be emphasised that I had limited access to documentation and that those with full access to the selection criteria, process and other relevant information could add to this topic and may have a different perspective on it.

Perpetuating the gender imbalance is the expectation that women will have children, and that the female will be the primary caregiver. Indeed, in her Presidential Address for the Association of Academic Surgery, Dr Caprice Greenberg[16] states "the conceptualisation of the issues facing women in surgery are almost exclusively considered to relate to parenting and work-life balance". Although cultural shifts are occurring, this remains the reality for many women: women do 80% of housework and childcare;[5] perceive as having to choose between career and family, including deferring having a family due to work commitments or because it is perceived as a detriment to their career.[17,18] When compared to the average population fewer women in surgery have children, indicating that the choice between family and work may be being made.[17,19,20] Women in surgery who were married, and women in surgery with children reported being more emotionally exhausted than single women and women without children,[21,22] presumably because women are supporting their spouse and children. Finally, male surgeons don't recommend some surgical specialties to women because of the conflict between work and family life.[23]

In contrast, men in surgery have children at a rate equitable to the non-surgical male population,[17,19] play a smaller role in housework and childcare, and those men in surgery who are in a committed relationship or who have children are significantly less emotionally exhausted than men without.[21,22] This suggest that men in surgery do not have to choose between career and family but are able to have both comfortably, indeed it is protective. It is important to acknowledge that parental leave does place challenges on those left to provide cover: "If we have a female colleague [who] decides to take six months of maternity leave, all of a sudden my schedule goes from one in seven to one in six. Or there's more [operating room] time that needs to be filled…".[24] Institutions should employ additional staff but if this does not happen these challenges may make it more difficult for colleagues to support parental leave, and therefore perpetuate the gender imbalance.

To truly move forward, the conversation about the gender imbalance in surgery

needs redirecting. Firstly, surgery cannot claim to attract the best and brightest if the talent pool is reduced by half,[25] so working out how career and family can be facilitated would help recruit the best candidates. Secondly, 40% of female surgeons do not have children[16] so solely focusing on the balance between children and career detracts from the many other barriers faced by women. Thirdly, conceptualising surgery, parenting and work-life balance as solely being an issue for women reinforces the current gender stereotypes and perpetuates the existing gender imbalance. Interestingly, many men now regard the trend towards gender equity in the workplace as a positive shift, and there is a changing attitude from men towards wanting to and enjoying being more active in fulfilling family responsibilities.[24] However, men wanting to take leave for family reasons and step outside their gender expectations may experience a professional and social backlash, creating a barrier for men in surgery to do this. Parental leave is a legal right in New Zealand and supporting new fathers to access this would start to shift the gender stereotypes for men, would help to balance gender expectations regarding career and family, and would better reflect the changing attitudes of men. Fourthly, the conversation needs to be redirected so that non-parental extracurricular activities are included as legitimate reasons for leave. Burnout is prevalent throughout the medical profession and is particularly high in the surgical specialties.[26,27] When asked to identify what doctors would like to spend their leisure time doing, exercise, travel and time with family were the top three activities,[27] so the ability for aspiring surgeons to take leave for such activities could help reduce burnout. Men and women are increasingly matching specialty choice to lifestyle, so support for extracurricular activities would also reflect the changing mentality around 'live to work'.[24,28]

Why should the imbalance be addressed?

Addressing the gender imbalance in surgery reaches beyond the widely acknowledged and publicised 'gender equity for women' rhetoric to the very core of healthcare. Indeed, there is evidence to suggest that there may be a difference in patient outcomes depending on whether the surgical intervention is performed by a male or female surgeon. Three pertinent studies assessing this will be discussed here. A retrospective matched cohort analysis compared patient outcomes between male and female surgeons.[29] Female surgeons were identified, and a corresponding male surgeon was matched 1:1 according to procedure, volume of these procedures performed by the surgeon in the preceding year, surgeon age, hospital, patient age, patient sex and patient comorbidities. After matching, more than 52,300 patients were included in this study. Rates of hospital readmission and complications were similar, but patients undergoing elective surgery who were operated on by a female surgeon had a significantly lower likelihood of death within 30 days of surgery.

The authors explained these findings by suggesting that female surgeons provide care that is more congruent with guidelines (less risk taking), is more patient-centred and involves superior communication (for example a greater willingness to collaborate, including a lower threshold for asking for a consultant opinion on a case). They also suggested that there are fewer barriers for men to overcome to become surgeons compared to women, resulting in female surgeons being more skilled, more motivated and harder working.

Interestingly, the authors advised that "these results do not support the preferential selection of a surgeon…". If a large, well-conducted study demonstrated a surgical technique, componentry or implant that offered a significantly lower likelihood of death, surgeons would almost certainly want to adopt this new product or at least investigate further, so this statement seems inconsistent. Yet it is appropriate that the authors advise this, because rather than turn the gender conversation into a male versus female win-or-lose binary, it is crucial to build on each other's strengths and work together to maximise patient care.

In the field of physicians, Tsugawa et al[30] analysed 30-day mortality of over 1.5 million hospitalisations and readmissions. Patients were comprised of a 20% random sample of Medicare fee-for-service beneficiaries ≥65 years who were hospitalised with a medical condition between 2011–2014. Patients were quasi-randomised to physicians based

on work schedules. Patients treated by female physicians had a statistically significant lower 30-day mortality and 30-day readmission than those patients treated by male physicians. This difference persisted across eight common medical conditions and across a range of illness severity. Confounders such as patient and physician characteristics and different hospitals were accounted for. A strength of this study is the random sampling of patients that will reduce selection bias, and that older patients with greater comorbidities were included, however the US system of healthcare with its fee-for-service must be considered when assessing generalisability to the New Zealand healthcare system.

Jerant et al (2013) researched whether the gender of a patient's usual source of healthcare was associated with healthcare utilisation and mortality.[31] This was a prospective observational study and data was obtained from the US Medical expenditure panel surveys, between 2002–2008. Respondents were aged ≥18 years, and a total of 21,365 respondents' data was analysed. They found there was no difference between the gender of healthcare providers and total expenditure, number of office visits, emergency visits, hospitalisations or mortality.

Conclusions

The gender imbalance in the surgical profession finds its roots in our social expectations of men and women. Improving gender diversity will help to break down these rigidly held expectations and support women to pursue a career in surgery, encourage men to participate more fully in their family responsibilities, and allow both men and women to achieve their overall life aspirations. By doing this, surgery will undergo a positive culture change and will continue to attract the best and brightest to this prestigious career.

The positive impact of increasing women in surgery comes from combining the different characteristics, values and experiences women bring to surgery with those of men. With this approach we can appreciate our differences, learn from and build on each other's strengths, and work together for improved patient care.

Competing interests:
Nil.

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On personal responsibility in medicine

Helen Ker

ABSTRACT

Enabling patients to make lasting behavioural change is one of the enduring challenges doctors face. The success of our medical treatment and preventative health measures relies heavily on our patients exercising personal responsibility, however, we rarely question our assumptions about what this looks like and how it is best enabled in clinical practice. Theoretical models of the individual and their ensuing responsibility are important because they influence the nature and expectations of the relationships we engage in with our patients as well as influencing how we interpret patient behaviour. Individuals with an ensembled construction of individualism and ensuing locus of responsibility, whether culturally or socioeconomically influenced, may not respond as well to the dominant Western construct of personal responsibility we commonly assume of our patients. We must broaden our notions of what constitutes an individual and their locus of responsibility in order to reduce inequities within our treatment outcomes.

Healthcare is an individually distributed commodity. Patients go to their doctors, often alone or with another person for their appointments and treatment. Doctors don't (except on rare occasions) visit their patients or treat them within their personal environments. Patients are given advice, usually within 15 minutes and sent home to act on this. As doctors we assume that once we provide a patient with the information they need, they will proceed to make a rational decision in their own best interest. We consult family members as an adjunct, but the implications of our healthcare decisions on the wider group are rarely considered during treatment planning. I know this because, as a newly trained doctor, I have not been taught how I might approach any of these aspects of healthcare delivery differently.

What I have experienced is one of the enduring challenges clinicians face: enabling patients to make lasting behavioural change. The DNA (did not attend) stamp chews through ink pads on the clinic reception desk with one in three Māori patients not attending a urologic outpatient clinic in 2017.[1] Adherence to medications has been reported as low as 50% in Counties Manukau DHB.[2] Māori, Pasifika and people with a low socioeconomic status are significantly overrepresented in these statistics which is reflected in the poorer health outcomes among these groups.[2] Most doctors recognise the barriers preventing people from accessing healthcare such as time, financial pressures and stress, but more often than not missed appointments, non-adherence and adverse health behaviours are explained away as a failure of personal responsibility.

Our working model of personal responsibility is largely based on the Western sociohistorical construct of self-contained individualism. Here the self is a singular, unbound identity that is theoretically disconnected from others and the environment. It follows that we hold the individual singularly responsible for their actions and that we expect these actions to align with the individual's best interests. Contrast this with an alternate construction of the individual that psychologist EE Sampson has termed ensembled individualism, which is traditionally dominant among Māori and Pasifika cultures and also noted to be more common among those living in neighbourhoods of lower socioeconomic status.[3–6] Here the self/non-self-boundary is more fluid. The self is defined in relation to others and the environment does not exist as a whole without these relationships. This notion lends itself to a wider locus of control and responsibility.

Self-determination is fostered within the context of a wider system, including family members and place. Obligations are not necessarily to do what is in the individual's self-interest but to act in the best interest of the many relationships that contribute to the whole. Responsibility lies within a wider context, and for this reason may also appear more elusive to the clinician.

The theoretical models of the individual and their ensuing responsibility are important because they influence the nature and expectations of the relationships we engage in with our patients as well as influencing how we interpret patient behaviour. This includes what we tell ourselves when people don't turn up for clinic appointments, how we discuss our patients with our colleagues, and what we ask of our patients after they leave our clinic and hospital rooms. Failure to recognise that there are alternative constructs of self as well as a related locus of responsibility can lead to premature labelling of people and their actions irresponsible. This undermines the possibilities we have in medicine of enacting lasting behavioural change and encouraging people towards healthier ways of living.

There are insidious and explicit ways in which self-contained individualism (and ensuing expectations of personal responsibility) is valued and fostered in our healthcare system over ensembled individualism. The historical basis of the system is telling. Formalised healthcare as we now know it was imported by Anglo-Saxon colonists and remained largely exclusionary of Māori (the major population group in New Zealand with a different cultural basis of self) until fewer than 3.5 generations ago. While the system has certainly become more open and self-aware, the underlying expectations of self-contained individualism and responsibility have not diversified to become more inclusive of other ways of being and approaching illness. Because the system works well for the majority of New Zealanders whose notion of the individual aligns with Western values there has been little impetus for this to change. For most New Zealanders these underlying assumptions are entirely appropriate, and they thrive in a system predicated on a similar notion of responsibility and self.

Within ensembled individualism, healthcare is more likely to be seen as a shared commodity. In Māori culture for example, health is viewed as a taonga (treasure) inherited from ancestors. Responsibility for the health of an individual lies within a wider group, usually the whānau and the hapū, not the individual alone. A surgeon from Syria recently told me that it is very common for patients with cancer not to know they have cancer, but for their family to be told and manage their health decisions on their behalf. Occasionally Chinese families in New Zealand request a similar approach. For people with an ensembled sense of individualism, whereby group loyalties and relationships impact directly on one's experience of health and illness, the way in which we may best enable our patients to exercise responsibility may require a different approach to that of the self-contained individual.

I recently met a woman in the emergency department who had brought in her two-month-old baby with apnoeic episodes. Talking with her I learnt she was living in temporary housing, had not received any formal antenatal or postnatal care, was feeding her two-month-old baby solid food, and was sleeping next to him in the same bed. While the apnoea was probably related to a viral infection that would clear soon, this mother and her baby warranted admission to hospital because of the social contract we have in medicine to protect those at risk, even if the risk is not directly due to a medical problem.

Our default medical approach was to tell this woman that her behaviour was wrong and attempt to educate her on how to change it within the hospital setting. We rarely invested time talking to her about what her own barriers were to enacting change, what she thought would improve her situation or what her family and support networks also thought and needed. We treated her as though her world stopped at the four walls of her hospital room—and it didn't work. Throughout her admission I frequently heard nurses complaining about what they deemed to be her irresponsible behaviour, complaining, for example, that they caught her sleeping in bed with her baby despite having been given a pēpi-

pod. (A pēpi-pod is a flax-woven basket in which a baby can sleep on the bed with their whānau within a protected space. It was developed as part of a successful public health initiative to reduce the rates of sudden infant death syndrome, particularly among Māori infants.) Only through exercising personal responsibility would this mother be able to enact long-term change, but it was clear that our current attempts to enable this were inadequate. From what I had gathered through talking with her, her sense of right and wrong was informed by those close to her, determined by a predominantly relational sense of self, yet we engaged with her as a context-less, self-contained individual, assuming rational knowledge would simply enable her to change her situation and approach to parenting.

A better approach would have first involved recognising that there is another way possible. This often fails to happen in the hospital where we rarely stop to examine the underlying assumptions that inform our treatment approaches. The next step would have involved forming a relationship with her based on mutual trust, respect and the recognition of a shared goal; namely the health of her baby. Exploring the different ways of achieving this, rather than simply assuming, would have enabled us to establish a closer relationship and a more realistic sense of the path forward. The way forward would very likely have meant involving others—family, and important relationships close to the patient who all contribute to a shared sense of responsibility for the child's upbringing. Most importantly perhaps, underlying all of this is a greater appreciation of diversity and different lived experiences without judgement.

None of this is to excuse irresponsible or neglectful parenting, but to realise that irresponsibility is an explanation of exclusion. We must first be sure we have understood how it is that someone sees themselves and their locus of responsibility, where their duties of responsibility lie, and the barriers they subjectively experience in exercising these. We enter dangerous territory when we label people irresponsible, especially as those in a position of power with a duty to care.

The colonially-imported assumptions of personal responsibility are losing their effectiveness as our population diversifies and we become increasingly aware of the repetitive failures of our system to enact long-term behavioural change. With a sincere and concerted effort to widen our understanding of how we may best enable our patients to take responsibility for their health we can achieve more in our medical consults. We must approach each person with a willingness to understand their situation not apply our judgement to it. We must ask how we can help rather than assume. Family and wider support networks should be more routinely involved in decision making. This involves respecting alternative views of health and knowledge systems. For many the advice of a grandmother may be far more powerful and meaningful than that of a health professional. We must be able to appreciate this and use this knowledge skilfully. Without diversifying our approach, we inadvertently harm those most in need of effective healthcare through misunderstanding what it means to them to be cared for and belong.

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The motivations behind science denial

Alan McLintic

ABSTRACT

Across the world highly educated, science-literate parents are refusing to have their children vaccinated against contagious diseases. Decades of international, peer-reviewed climate science is being dismissed as institutional conspiracy. Activists troll and harass scientists who come to unfavourable conclusions in pet areas such as genome modification, childhood memory recall, chronic fatigue syndrome and even the hazards of smoking. And somewhat legitimising this behaviour are rising numbers of populist leaders who pour scorn on whichever science is inconvenient to their popularity. Science denial is tangible on a day-to-day basis and has measurable detriment to health and education. This article outlines the key research underpinning its ideological, psychological and pragmatic motivations.

In 2007 a Minneapolis special education survey raised concerns within the Somali community about a predisposition for autism. This was a signal for anti-vaccination activists to swoop and cultivate the perennial scapegoat, MMR vaccine.[1] Subsequent analysis did not show a greater Somali susceptibility to autism but by that time MMR had been demonised and vaccination rates fell from 92% in 2004 to 42% by 2014.[2] Autism rates did not change over this period but children became vulnerable to measles. The first outbreak was in 2011 and the second, in 2017, was one of the largest US outbreaks of measles in the last two decades. Despite this 'lesson', there is burgeoning vaccine hesitancy in the US and outbreaks have, improbably, become rallying points for activists to allege vaccine injury and institutional cover-up.[3,4] The pattern is repeated worldwide. The incitement of vaccine distrust is a causal factor in the European and Asian measles epidemics as well as rubella outbreaks in Japan and diphtheria in Spain.[5–7] Measles was declared eradicated from New Zealand in 2017 but has returned in increasing numbers of unvaccinated children. While vaccine hesitancy is multifactorial, home-grown scaremongering and faux immunisation advice are reckless contributors to this problem.[8,9]

Vaccine hesitancy involves broad issues of accessibility, complacency and trust.[10] This article focuses on one element of the latter, the rejection of stable scientific evidence and advice. Why do activists and well-informed parents dismiss the imperative of childhood vaccines when the evidence has never been better understood, better documented and better communicated?

The same is true of anthropogenic global warming (AGW). The evidence that humans are warming the planet is overwhelming and is the long-term consensus of virtually the entire expert scientific community researching in that area.[11,12] Yet highly educated, science-literate people will not accept AGW and are often scornful of the science and scientists behind it.

Certain areas of science have become a battleground of fact between scientific expertise and lay opinion. It is here that we find 'science denial': the dismissal, outside peer review and relevant expertise, of a scientific consensus where the science is characteristically perceived as a threat.[13] Arguments are therefore often accompanied by attacks on the integrity of the scientists themselves.[14] How widespread is this problem? US national science reports from 2018 show the majority of Americans view

scientific research as beneficial, they trust scientists to tell the truth and they trust scientists to report findings accurately.[15,16] These findings have been stable over four decades and are mirrored in New Zealand and UK surveys.[17,18] The data don't support a general 'war on science' but they do identify a minority who do not trust scientists and who reject their findings. This group can be very vocal and influence policy and community health.

The knowledge deficit model

Do those who reject the scientific consensus simply have a poor understanding of the relevant science? This is called the knowledge deficit model and its importance is that, if science denial stems from a lack of knowledge, better education and communication should improve trust in science. This is borne out in several international surveys that show a weak but consistent relationship between general science knowledge or education attainment and more positive attitudes to science.[15,16,18–20] Thus education may improve trust in science.

The problem is that when surveys look at attitudes in specific areas of science the relationship breaks down. American surveys show those with more science knowledge have greater trust in the science of GM food safety, but an international meta-analysis found the relationship was weaker unless that knowledge related to biology or genetics.[19,21,22] Science knowledge is only a medium factor in predicting attitude to AGW or evolution and a weak factor in predicting attitudes to vaccine safety.[19,22] The role education plays in vaccine acceptance varies between regions. In The Netherlands, Greece, Nigeria and Pakistan, education increases vaccine acceptance, while in China, Bangladesh, Israel, and the US, higher education can be a potential barrier to acceptance.[10] Negative correlations are reported in other fields. For certain religious and conservative demographics, attitudes to embryo research, genetic testing, Big Bang theory, evolution and AGW deteriorate as knowledge and education increase.[19,20,23]

Thus, in these areas of science, and they are common areas for science denial, educational attainment and science knowledge are not necessarily predictive of greater trust in the scientific consensus.

Science denial is a misleading term. People don't deny Boyle's Law, they reject science in selected areas such as evolution, AGW, vaccine safety and GM food safety. This has been called the motivated rejection of science and most instances are explained by four overlapping components: cultural cognition, conspiracy ideation, free-market ideology and political populism.[24]

Cultural cognition

When people lack the time or the expertise to form a real understanding of a science proposal, the strongest influence on their viewpoint tends to be their cultural worldview. These are deeply engrained values and beliefs that give us our sense of self, group identity and how we want society to be. Science proposals are viewed through that lens and it is a self-reaffirming lens because, if the science threatens the worldview, it will tend to be the science that is dismissed rather than the worldview adjusted.[25,26] This results in certain areas of science being culturally, politically and religiously polarised.[25]

Kahan compared subjects' science knowledge with the probability of getting the answer right to some basic science questions.[27] With general science questions there was a strong relationship: the greater the science knowledge, the more likely a correct answer. But when subjects were asked whether AGW was real, the relationship barely reached statistical significance. Science knowledge was no longer predictive of the correct answer. When those subjects were grouped according to political outlook the influence of worldview became clear: those on the Left were agreeing and those on the Right were disagreeing. In the same year Americans were asked if Earth warming is due to human activity. Seventy-one percent of Democrats said yes in contrast to only 27% of Republicans.[22]

What was particularly revealing in the Kahan study was that, as science knowledge increased, so did the polarisation; the Left became more supportive and the Right became more resistant to AGW. Drummond and Fischoff examined the effects of political and religious identity on the acceptance of core science findings including AGW, evolution and Big Bang theory.[20] In each of these areas extreme Conservatives and religious fundamentalists adopted positions

counter to established scientific evidence and the greater their science education, the greater was their resistance to that science.

This is the opposite of what we would expect from the knowledge deficit model. The accepted explanation is that the greater your education and science literacy, the more adept you become in interpreting information in ways to support your world view.[20,28] Consequently, if a science finding feels threatening, the greater your education, the greater your resistance can become. This is bad news for the role that facts and education might have in preventing or correcting science denial.

In another example, individuals with culturally polarised views on the risks of mandatory human papillomavirus (HPV) vaccination were provided with balanced arguments.[25] Everyone could now see each other's point of view. It might be expected that this would move groups towards middle ground but the opposite occurred. The participants became more polarised because they had quickly dismissed conflicting material and been reinforced by new information that bolstered their established position.

In summary, certain areas of science are culturally polarised and minds made up in these areas are not usually swayed by more facts and knowledge. Indeed, giving more facts can harden resistance by amplifying the competing positions.[23,26] When parents were provided corrective information on MMR/autism links or images of children sick with measles, parents with the least favourable views on vaccination hardened their intentions not to vaccinate.[29] There is no single population that has an anti-science leaning, the demographics depend on which science position is being denied.[15] The rejection of stem cell research, evolution, Big Bang theory and AGW are associated strongly with religious and conservative-right ideologies.[15,20] Public attitudes to GM food vary between regions as do the demographics of those who distrust messages regarding its safety. In the US, GM food safety is not consistently politically polarised while in Europe distrust is more common among the political left.[22] Active distrust in vaccines has a bias towards the young in US surveys but does not polarise to left or right politics.[22] The exception is opposition to HPV vaccination which has a conservative bias.[25] The politicisation of science is not a new phenomenon, but this is a time when scientific expertise is being openly derided across mass media in favour of politically flavoured opinion. Gauchat looked at 40 years of American data and found that while the public trust in science was stable over that time, political polarisation has increased.[30] This was because of a shift in conservative stance. In the 1970s it was the conservatives who had the greatest trust in science but now they are the group in which there is least trust. This is attributed to conservative aversion to increasing amounts of science with regulatory implications, particularly those that increase institutional interference and constrain free-choice.[25,30]

Conspiracy thinking

Science denial, especially vaccine distrust and AGW rejection, is strongly associated with another style of thinking, conspiracy ideation.[24,31–34] The salient features of a conspiracy are that it is a *covert* plot by a powerful *authority* in order to accomplish a *sinister goal*.[33,35]

When someone rejects an established scientific consensus they frequently explain the consensus in terms of a conspiracy among scientists. Vaccination programmes are regularly attacked by activists as schemes for profit and mass control in which institutions have suppressed evidence of harm.[34] The origins and treatment of HIV/AIDs has a long history of denial and conspiracy, and even in 2013 12% of Americans believed it credible that the CIA deliberately infected African Americans with HIV.[36] The same survey found 37% believed drug companies pressure the FDA to suppress natural cures for cancer and 20% believed corporations were preventing health officials from revealing links between cell phones and cancer.[36] Climate science imperatives are regularly explained as conspiracies. A 2016 poll found that 40% believed it possible that global warming was a myth concocted by scientists.[37]

Explaining a science position as a conspiracy is not usually an isolated consideration. Conspiracy ideation is an ideological view of how the world works that has a distrust of authority at its core.[35] It is a psychological disposition that is amplified by anxiety, lack of social control

or a sense of social ostracism.[32,33,35] In this context the conspiracies instil a sense of valour, morality and superior insight.[31,32] These are powerful, enabling motivations and drive a style of reasoning that tends to deride conventional wisdom and protect the conspiracy at all costs. This is sustained by a heightened tendency for a number of cognitive biases so that strong empirical and statistical evidence is ultimately dismissed in favour of anecdote and anomaly with complete resistance to falsification.[31,32]

Conspiracy theories perpetuated in mass media may have an insidious effect on our behaviour. The mere exposure to conspiracy theories can induce a sense of disillusionment and disengage people from behavioural changes such as reducing their carbon footprint or vaccinating their children.[35,38,39] Why would you vaccinate your children if it were a government plot?

Pragmatic: profit and populism

Science findings are also dismissed for pragmatic reasons. The last hundred years have shown numerous corporations dismissing science that threatened profits. Most notable has been the tobacco industry, which has a long history of attacking research linking smoking to cancer and other health problems. Corporate strategies to discredit research and researchers are well-described elsewhere but there is one tactic worth highlighting because it is an insidious manipulation of public understanding, and that is corporate campaigns specifically designed to make people feel uncertain.[40,41]

Robert Proctor was the first historian to testify against the tobacco industry. A focal point of his research was an account of tobacco strategies to induce public uncertainty over the ill-effects of smoking. In doing so he coined the term agnotology, the study of the construction of public ignorance and doubt through propaganda and misleading scientific publication.[42] Internal memos from tobacco companies revealed this to be an explicit corporate strategy and it is a tactic at the heart of much organised climate science denial. The Heartland-funded NIPCC report, "Why Scientists Disagree About Global Warming" is a text dedicated to this cause.[43]

These tactics are effective. The perception of even a small amount of scientific disagreement makes people think that no one knows the truth.[44] Activists therefore work hard to inflate the size of science dissent.[41,45] Over 97% of the scientific community researching in climate science agree that AGW is real and the consensus has been in place for decades.[11,45] Despite this, only a minority of Americans believe that most scientists are in agreement on AGW.[46,47] Organised disinformation has led to a perception that there is no consensus on AGW. The opposite is true.

A second, pragmatic reason for attacking established science is that doing so can garner votes for politicians and, in particular, those advancing a populist mandate. Populist leaders claim they will prioritise the interests and culture of the native populace and deliver them from the impositions of previous establishment elites. Scientists and academics are often among those proposed elites so there are votes to be won on an anti-intellectual platform that derides the apparent tyrannies of vaccine schedules and carbon footprint regulations. This rhetoric has accompanied a global rise in populist governments espousing anti-AGW, anti-vaccine and other anti-science messages.[7,48,49] WHO cite the politicisation of immunisation as a growing component of vaccine hesitancy, and several European measles epidemics have been linked explicitly to populist campaigns.[7,48]

Solutions

Corporate attacks on science that are motivated by profit have solutions in public awareness, investigative journalism, legislation and prosecution by citizen-centric governments that drive policies best for health and humanity rather than inducements for re-election.

When individuals reject a widely-recognised scientific consensus they usually do so not through lack of education or facts, but from culturally engrained instincts that are reinforced by distrust of experts and authority and varying degrees of conspiracy ideation. For the most part those opinions are resistant to facts and, if the individual is an activist whose identity is tied up in the cause, nothing is likely to change his or her

mind. But what should we say to those who are sitting on the fence? Vaccine hesitancy is multifactorial and WHO emphasises that the key local barriers must be identified and targeted specifically.[10] This article does not try to address all those issues but there is advice from communication research on encountering science denial in general. The acceptance of science propositions is more likely when information is given in the form of narratives and examples rather than by pushing facts.[24] The first paragraph of this article is one such narrative. If there is a single fact worth emphasising, it is to emphasise the expert scientific consensus. The perception of even the smallest amount of disagreement within the science community tends to cast doubt on the science findings for individuals.[44] For this reason activists work hard to promote a notion of science discord and narratives must redress that.

If people are to change their minds they need to be able to do so without losing their sense of ideological or group identity. There is therefore better acceptance of a message if it comes from the same cultural group, or by educators emphasising common interests and empathising with common fears.[50] The acceptance of AGW by conservatives and evangelicals is greater when the message is delivered by conservatives and evangelicals, and vaccination rates among the Minneapolis Somali have greatly improved because the message to vaccinate is now coming from leaders in that community.[50,51]

Facts may not turn around a committed science denier, but education, communication and public engagement demonstrably improve public trust in science, and concerted efforts are being made in these areas. The result is that the majority of the public still value science, trust scientists and want rational science decision making. It is this majority that we must hope will ultimately restore public preference for fact and truth and prevent science deniers being elected to power.

Naomi Oreskes, an American science historian, suggests scientists should also be less coy and start talking about so-called 'contentious' areas such as AGW as scientific fact.[45] There is a tendency to preface public information with philosophical caveats regarding the provisional nature of science knowledge and its inherent uncertainties. While this is philosophically accurate, it is also the case that we do have knowledge of AGW, vaccine safety, Big Bang theory and evolution that are facts by any standard. Furthermore, the crucial issue is not so much what qualifies as a scientific fact but what is the best source of science information on which to make reliable decisions today? That has to be replicated, peer-reviewed science findings that are the long-term consensus of the expert scientific community working in that field. If we are not to make decisions based on that source, what should we base them on?

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New Zealand needs a comprehensive interpreting service

Ben Gray

Abdelfattah Qasem was killed in the Masjid Al Noor mosque on 15 March.[1] He worked as an Arabic interpreter. For survivors in the Mosque community, particularly those from a refugee background with limited English proficiency (LEP), his loss will be keenly felt. It would not have been possible to provide high-quality trauma care after the attacks without professional interpreting services. It is particularly hard to provide psychological services to LEP people without a professional interpreter. The national response to the killings has been heartening, with a commitment to valuing diversity and including minority populations within our community. However, this is aspirational and much work has to be done before we can achieve these goals. In this context The Human Rights Commission on "Give Nothing to Racism" is an important initiative.

New Zealand accepts 1,000 refugees (to be increased to 1,500 from July 2020)[2] per year through our refugee quota system and many others from a refugee-like background through programmes such as the family reunification scheme. Many of these people do not speak English when they arrive. To settle successfully they need interpreter services at least initially for everything they do.

We have tolerated what I view as a form of institutional racism for too long; we do not as of right now provide a professional interpreter for LEP patients when they need. While this paper is predominantly focused on the health sector, the issues raised are clearly relevant to every aspect of society if people with limited English are going to be able to fully participate in our community.

Our study in 2011[3] found that none of the 21 LEP patients presenting to ED had the benefit of a professional interpreter and 65% of surveyed clinicians used interpreters half the time they were needed or less.

The main nationally available service has been the government-run telephone interpreting service Language Line. It has interpreted around 54,000 conversations a year and provided services to most government departments, DHBs and PHOs. The costs were subsidised but not free. This number has not changed in the last six years. Given that at the last census there were 80,000 people who only spoke a language other than English and many more have limited English proficiency, this is a tiny drop in the bucket of need. Language Line will cease to operate on 30 September 2019. Mr Qasem's employer Interpreting New Zealand provides on-site services in Wellington Christchurch and Nelson and telephone services nationally 24/7. Decypher provides on-site and telephone interpreting based in Hamilton. The three Auckland DHBs have their own interpreter services.[4] Funding for health interpreter services comes out of health budgets and of course falls disproportionately more to some providers than others.

It is time that our attitudes to the provision of interpreting services changed. Surely every clinician would acknowledge that it is impossible to provide good care without getting a good history and being able to negotiate an agreed management plan. If the patient is of LEP this cannot be done without an interpreter.

The Code of Patient Rights[5] in right 5 guarantees a right to effective communication:

1. *Every consumer has the right to effective communication in a form, language and manner that enables the*

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> *consumer to understand the information provided. Where necessary and reasonably practicable, this includes the right to a competent interpreter.*

Despite submissions to update this right it has remained the same since the inception of the code. I would argue that for a patient with LEP, many of the other rights in the code are not available without a professional interpreter. It is impossible for me as a clinician to determine the "competence" of an interpreter without an outside arbiter. By definition I do not understand the language, so how could I judge their competence? Interpreting is a skilled task and the ethical requirements are the same, if not more so, as those that apply to the doctor. For some consultations this can only be addressed by employing a professional interpreter who is trained and accredited in a transparent way and who is a member of a professional organisation that ensures adherance to a code of ethics. Rights 6 and 7, the right to full information and to be able to give informed consent are unmeetable for an LEP patient without a professional interpreter. A clinician could not ascertain whether an LEP patient understood the information provided without a professional interpreter and for significant procedures there would be significant medicolegal risk in proceeding without an interpreter. The code needs to be amended to "*the right to a professional interpreter*".

A specific anomaly is that ACC will only fund an interpreter once a case manager has been appointed. They do not provide funding for an interpreter for the initial consultation following an accident. At that consultation the patient signs a consent form. Without a professional interpreter that consent cannot be valid.

The Ministry of Business Innovation and Employment has a Language Assistance Services Project in progress that is considering these issues.[6] Their recommendations if implemented would significantly improve the availability of interpreters. They have just announced[7] the establishment of a 24/7 telephone interpreting service starting on 16 September, which is a huge improvement on the previous Language Line which was only available during business hours. At the time of writing there are important issues that are not resolved.[8] A particular problem is that the new provider is to provide services to organisations eligible to use collaborative contracts under the New Zealand Government Procurement. Primary Health Organisations are not eligible for this, although (outside of Auckland) they are major current users of Language Line. While the project plan[6] includes a recommendation that "The Ministry of Health consider in conjunction with DHBs a consistent approach to the funding of interpreters in the primary care sector throughout the country", there has been no announcement regarding progress on this recommendation.

The proposed model of a provider that contracts with multiple 'clients' who provide services to LEP patients has large transaction costs compared with a centrally funded service that can be accessed without cost by those publicly funded or contracted services that need them, as is the model for the Auckland primary care interpreting service.[4] There has been much criticism[9] of our large number of DHBs and PHOs and the costs these incur. Our study of interpreter policies showed that all DHBs have policies on interpreter use, but there was a wide variation in quality with some clearly not following recommended practice.[10] Rolling out a funding model is an opportunity to use a consistent and more efficient model.

As discussed in the chapter in Coles Medical Practice on interpreting[11] there will be times when a telephone interpreter is not sufficient so we need to develop capacity in on-site interpreters; a task that is plausible in the main centres but much more difficult if we settle refugees in smaller provincial centres.

It is time we followed the Australian lead. They have had a federally funded Translating and Interpreting Service[12] that has provided free telephone and on-site interpreting services for the past 46 years. They have a training and accreditation system to ensure adequate standards of interpreting and ethics.

There is also a lot of work to be done by the professions on working with interpreters, for we know from the Australian experience[13] that even if there is a long-standing fully funded service it is not automatically used. While there are times when working with a family member may be acceptable,[14] we need to develop the

clinical skill of determining what form of language assistance is needed for each consultation.[11] There will always be many consultations where a professional interpreter is the required option.

The Christchurch shootings have highlighted that some of the minority communities in New Zealand are not able to be "one of us" because of some of the barriers to better engagement with the wider community. Issues of racism, religious intolerance and discomfort with difference are challenging to address, even if a lot of money were put into programmes. By contrast, a comprehensive fully funded language assistance service is entirely doable and would make a significant difference. It is important that there are tangible changes resulting from the outpouring of goodwill that occurred. A comprehensive centrally funded language assistance service would be a fitting memorial for Abdelfattah Qasem and tangible proof that we as New Zealanders genuinely value diversity and wish to enable all people irrespective of language ability to participate fully in our society.

Competing interests:
Nil.

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Erysipelothrix rhusiopathiae bacteraemia in an immunocompromised host: the unexpected complication of a crustacean altercation

Eben Jones, Peter Burrell, Tony Barnett, David Lyons-Ewing, Elisabeth Nuttall

Zoonotic infections are infrequently considered and subsequently under-diagnosed. Here we report a case of *Erysipelothrix rhusiopathiae* infection as a means of highlighting the importance of both considering and investigating for zoonotic infections in patients presenting with infective symptoms.

Case report

A retired 57-year-old man presented to Nelson Hospital with a three-day history of fevers, nausea, headaches, myalgia and lethargy. History revealed no localising symptoms of infection, and he reported no recent foreign travel. His past medical history included psoriatic arthritis and diabetes mellitus. His medications included prednisone, methotrexate and etanercept.

On examination, his temperature was 35.7°C, blood pressure was 80/60mmHg, and he was noted to have a 5x7cm, purple, crusted lesion on the dorsum of his left wrist (Figure 1). The lesion was not cellulitic. The patient had no murmur or peripheral stigmata of infective endocarditis.

Figure 1: Skin lesion on the patient's wrist.

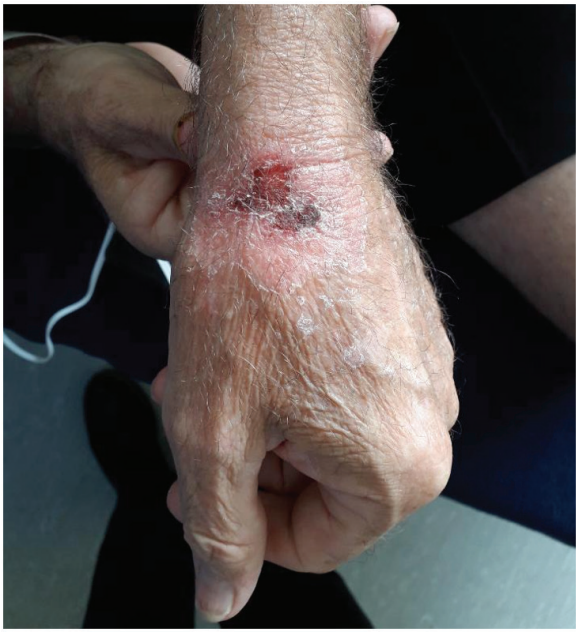

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Table 1: Initial investigations.

	Admission value	**Reference ranges**
Haematology		
Haemoglobin	144	130–175g/L
White cell count	**13.2**	4.0–11.0x10^9/L
Neutrophil count	**11.5**	1.9–7.5x10^9/L
Lymphocyte count	**0.6**	1.0–4.0x10^9/L
Biochemistry		
Sodium	**136**	135–145mmol/L
Potassium	**5.7**	3.5–5.2mmol/L
Lactate	**2.73**	0.5–1.6mmol/L
CRP	**124**	0-5mg/L
Imaging		
Chest x-ray	No focal consolidation	
Microbiology		
Blood cultures	Sent prior to the administration of antibiotics	
Urine microscopy	Leucocytes: 21–50x10^6/L Red cells: <10x10^6/L	

The working diagnosis of sepsis of unknown source with associated adrenal insufficiency was treated with 4L of IV crystalloid, IV ceftriaxone and IV hydrocortisone.

Following 20 hours of incubation the anaerobic blood cultures were reported to be growing fine Gram-negative bacilli (Figure 2). Two days later, the organism was identified as *E. rhusiopathiae* by MALDI-TOF mass spectrometry.

Figure 2: An example of the Gram-variable nature of *E. rhusiopathiae*.[1]

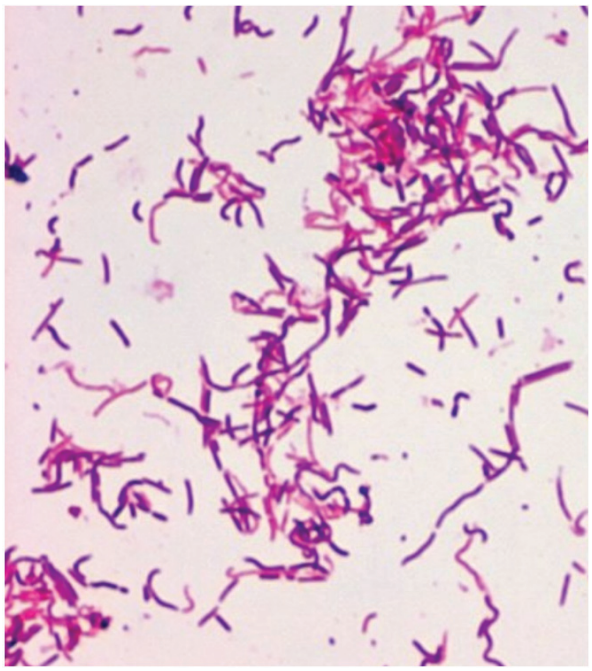

Figure 3: The culprit crustacean.

Further questioning revealed that the patient had suffered small puncture wounds to his left wrist while handling salt-water crayfish in Kaikoura five days prior to the onset of symptoms (Figure 3). His family also kept sheep and chickens, but he denied recent contact with these animals.

Considering this bacterium's association with infective endocarditis, a trans-thoracic echocardiogram was performed, which demonstrated no vegetations. Due to the patient's rapid clinical recovery, subsequent negative blood cultures and lack of stigmata of endocarditis, a trans-oesophageal echocardiogram was not performed. The antibiotic regimen was rationalised to a seven-day course of oral amoxicillin. At follow-up, the patient was asymptomatic and felt that he had made a full recovery.

Discussion

Erysipelothrix rhusiopathiae is a facultatively anaerobic non-spore-forming Gram-positive bacillus. It has Greek etymology, and combines the terms *erythros* (red), *pella* (skin) and *thrix* (thread-like). It was first isolated by Robert Koch in 1876, and is recognised as a zoonotic pathogen in humans.[2]

E. rhusiopathiae is hosted by a range of wild and domesticated mammals, birds, amphibians and marine species, including crayfish.[3] Human infection is associated with occupational and recreational exposures to animals and their excretions, with case reports clustered among farmers, butchers and fish-handlers.[4–6] In this case, the likely source of infection was a puncture wound sustained while handing a salt-water crayfish. This case underlines the importance of considering zoonotic exposures when confronted with a septic patient with no clear source.

Human disease most commonly presents as a well-defined violaceous lesion (erysipeloid) that normally resolves without treatment.[7] *E. rhusiopathiae* bacteraemia is substantially rarer, but commonly results in a severe clinical illness, associated with endocarditis in over one-third of cases.[8] Specific risk factors for systemic illness in this case included diabetes mellitus and immunosuppression. Additionally, a variety of focal disease has been rarely reported, including central nervous system infection, osteomyelitis, septic arthritis, liver abscess and intra-abdominal abscess.[9]

This case serves to illustrate the potential for opportunistic zoonotic infections in immunocompromised individuals, and some of the pitfalls experienced when diagnosing human disease caused by *E. rhusiopathiae.* Gram-stain may yield a Gram-variable result due to poor retention of the stain.[10] Indeed, the provisional report in this instance was of a Gram-negative organism. Secondly, since many Gram-positive bacilli that grow in blood cultures represent sample contamination, some labs may not proceed to fully characterise these organisms. Combined, these factors may contribute to delayed or under-diagnosis of this clinically significant organism.

Competing interests:
Nil.

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Due diligence on today's cannabis—a response

Bob McCoskrie

A response to "What we know, and don't know, about cannabis, psychosis and violence",[1] Boden and Spittlehouse (26 July 2019).

In May 2019, as calls for the legalisation of cannabis grew ever louder, and with the upcoming referendum on legalisation in 2020, Family First launched a petition asking the government to first investigate the possible link between cannabis and violence. Emphasis on 'possible'. We raised the issue for good reason.

Over the past couple of decades, studies around the globe have found that higher levels of THC—the active compound in cannabis—is strongly linked to psychosis, schizophrenia, and violence.

And with increasing THC levels being found in marijuana products consumed via edibles, vaping and dabbing, the risk is growing. In Colorado the average THC content of all tested flower in 2017 was 19.6%, and for concentrated extract products, 68.6%.[2] Potency rates can now be as high as 99.9%.[3]

Researchers have studied alcohol and violence for generations, proving that alcohol is a risk factor for domestic abuse and assault. Far less work has been done on cannabis. And that's effectively the work that we'd like to see done—before we move to legalise it. We would argue that the evidence is already building.

A recent study in *The Lancet* concluded that "*people who smoked marijuana on a daily basis were three times more likely to be diagnosed with psychosis… For those who used high-potency marijuana daily, the risk jumped to nearly five times.*"[4,5]

This follows research last year which found that frequent marijuana use was associated with intimate partner violence,[6] similar to a 2011 study.[7]

Research published in 2016 in the journal *Psychological Medicine* concluded that continued cannabis use is associated with seven-fold greater odds for subsequent commission of violent crimes.[8]

As with all research, there are limitations in the studies mentioned above. But those same limitations also apply to studies which say there is no association.

The United Nations Office on Drugs and Crime (UNODC) summed up the issue in their 2012 report, saying that the increasing potency of cannabis can increase psychotic symptoms in regular users.[9]

The new paper "*Cannabis use and violence in patients with severe mental illnesses: A meta-analytical investigation*" is the most comprehensive survey yet on the issue.[10] Findings showed a moderate cannabis-violence association in severe mental illness. What's also striking is how recent most of the papers examined are—10 of the 12 papers are in the last decade, and 7 of the 12 since 2016.

Just this year, more than 40 clinicians, researchers and scientists from Massachusetts, including many from Harvard Medical School, released a Statement of Concern, highlighting negative effects of THC—"*Increased risk of serious mental health problems including acute psychosis (eg, hallucinations, delusions), paranoia, schizophrenia, depression, anxiety and suicide, with growing scientific evidence that daily use of high THC products bring greater risk*".[11]

They highlighted 2018 research from the Copenhagen University Hospital which found that "*41% of those who experience cannabis-induced psychosis later convert to schizophrenia.*"[12]

Recently in Vermont, the Department of Mental Health warned legislators, stating, "*…multiple studies have linked regular*

cannabis use to an estimated doubling of the risk of a psychotic illness... Violent behaviour as a result of cannabis-induced paranoia and other psychotic symptoms is also an increasing concern".[13]

In Maryland, neuroscientist Christine Miller warned legislators, "*The causal link between marijuana use and the development of psychosis is quite simply the most well-replicated, high-impact finding in schizophrenia research today. Given current use rates and the strong potency of the drug available, it stands to be responsible for a larger proportion of schizophrenia cases than any other established factor...*"[14]

In the same way that there is some real evidence that components of marijuana can be made into medicine, there is building scientific evidence suggesting that components of the plant can at times lead to mental illness, at times severe, that can lead to violence.

It is interesting that Boden and Spittlehouse agree with Family First that "*more research is needed on the possible linkages between cannabis exposure and violence*."

We are simply asking for research and scientific consensus, before moving forward as a country with a change this massive. We believe this to be a responsible and thoughtful way to move forward.

Competing interests:
Nil.

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Clarification: a rebuttal to 'The value of frenotomy for ankylo-glossia from a parental perspective'

Graham J Sharpe

I must clarify a matter raised in the rebuttal to my letter published in the *NZMJ*.[1]

The authors refer to a paper published under my name in the Sri Lankan Journal of Anaesthesiology.[2] This paper was published without my knowledge. I was not involved in any part of its research, data collection, writing or publication, and I have made no claims for its authorship, such as inclusion in my CV. I became aware of it, quite by accident, some time in 2013. I accept that the authors of the rebuttal could not have known this.

Competing interests:
Nil.

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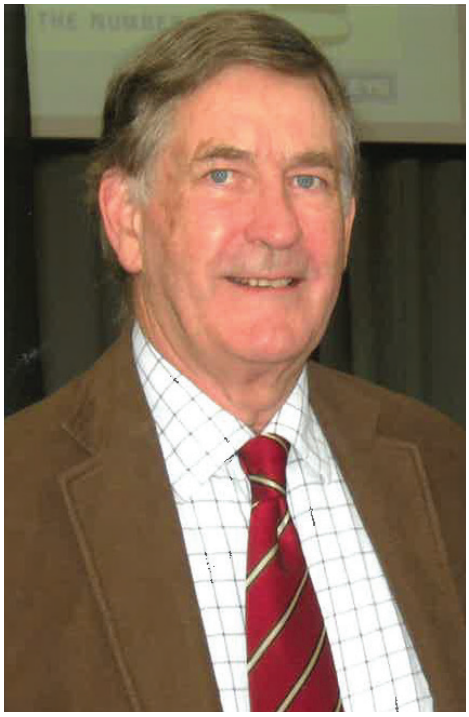
Kevin Vincent Marriott

8 April 1940–17 May 2018



MB, CHB, Otago 1965

Kevin Marriott was born in Christchurch, the third child of six, a son of the Four Square grocer in New Brighton. He was a very able youngster, brought up as a good Catholic and for a time expected to join the priesthood although he had other ideas, dux of his school, an able 1st fifteen rugby player, and the only person known personally to the writer who was able to break the string used for tying up parcels of groceries in those days with a flick of his hand. He proudly related the story of an early school report which said: place in class, first; achievement, first; comment, could do better.

He chose to go to Medical School in Dunedin, progressed easily through the years but surprisingly failed his final examinations. By the time he sat and passed special exams all the house surgeon positions in New Zealand were taken except for one, and he ended up in Waipukurau in 1965, which he then aptly called the last place in New Zealand. He did some time in Waipukurau as a junior, moved to Hastings as a medical registrar, developed his skills as a very able internist and anaesthetist, and then took up a position back in Waipukurau as a part-time GP and part-time anaesthetist and physician at Waipukurau Hospital, replacing the late Des Dickson who moved on to Palmerston North as a cardiologist.

He remained in Waipukurau throughout his working life, partly as a GP and partly as a senior medical officer at Waipukurau Hospital to which he was devoted. He was not a born GP and tolerated much of the work and many of his patients because it was necessary to make the system work, but he never suffered fools gladly and to him there were quite a few fools around. He had a self-selected group of patients who liked him and his style and whom he enjoyed in turn, and for these he could never do too much and was always available, and many became firm personal friends. His

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particular interest was in the associated hospital work, especially anaesthetics and cardiology, he had a broad and a deep understanding of medicine and he formed and maintained strong positive relationships with consultant colleagues in Hastings to the benefit of all. Having been at times Medical Superintendent, with the closure of Waipukurau Hospital in 2000 he resumed a low key role in general practice until he retired in 2009 much to his own relief and that of his colleagues.

Kevin married early and had five children with his first wife, Margaret. While Margaret probably brought up the family largely single-handed, he was very proud of his children and when he had time he used to tramp with them in the Ruahines and visit farming friends and beaches around Central Hawke's Bay. At the same time he had cultivated tastes and enjoyed the finer things of life, especially classical music and opera, fine wine, spirits of juniper and other drinks sometimes in too much quantity, read widely, remembered what he read and frequently referred to The Department of Useless Information. In particular he had a love of the English language, was rarely without a dictionary to hand and was an expert in crossword puzzles, especially the one in the Guardian Weekly which he had airmailed to him.

In those days rural medicine was a field of particularly heavy personal commitment by all those involved, he was the long-term backbone of the local hospital services and he was on call continuously for two weeks in three over many years. Inevitably this took a toll on his personal life and his relationships. After his first marriage foundered he had other relationships and later married Diana and later again Harriet with whom he had his sixth child and third son. Harriet stood by him to the end, throughout the ravages of the dementia which afflicted both his parents and which he had feared for many years, until he finally died peacefully in Wellington. He had a small largely family funeral in Wellington and a large memorial service in Waipukurau, which was well attended by friends, colleagues and former patients. He is survived by Margaret and Diana and Harriet, and Catherine, David, Rachael, Jeremy, Abigail and James, and eight grandchildren.

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Antony Todd Young

15 August 1951–7 September 2019

It is with great sadness that we acknowledge the recent death of Dr Tony Young.

Tony was the son of Dr Ted and Prue Young. Ted was a Christchurch GP who practiced for many years from his home in Barrington Street.

Tony attended Somerfield and Cathedral Grammar primary schools and then went on to Christ's College with a scholarship. At school he showed his outstanding academic ability being Dux of Christ's College and being in the top 10 in New Zealand in the New Zealand Scholarship exams in his last year.

Having left school Tony went down to Dunedin to study Medicine. He was in Selwyn College for three years and in his last year was President. In his fourth Year he came up to Christchurch in the inaugural year of the Christchurch Clinical School.

He graduated in 1975 and was a house surgeon at Christchurch Hospital. Not being quite ready to settle down, he did some general practice in Australia before heading off through Asia on the old "Hippie Trail", eventually arriving in London. After working briefly in England he returned to Christchurch to take up a post as a radiology registrar. He did his basic training here, being awarded the FRANZCR in 1983, along with the HR Sear prize for the best candidate in the final examination. This was followed by fellowships at the University of Minnesota Hospital in the US, and the Hammersmith Hospital in the UK from 1982 until 1985.

While working enormous hours in the intervention suites in America, Tony somehow also found time to co-author many of his 36 scientific publications. During his career he also presented at 10 scientific meetings.

Tony and his family returned to Christchurch where he was a consultant radiologist from 1986 until his retirement in 2016. We were very lucky to have him return to Christchurch as he could probably have obtained a job anywhere in the world.

He was the first true interventional radiologist in the South Island and during his career oversaw the growth of this subspecialty to the point where it now plays a pivotal role in the management of many surgical and medical patients. In addition to pioneering many procedures that are now commonplace, Tony fostered and enhanced the roles of radiographers and radiology nurses with the aim of improving patient care, outcomes and efficiency.

Tony's wonderful sense of humour and enthusiasm for life made him very popular wherever he worked. His surgical and anaesthesia colleagues very much enjoyed his theatre banter, while at the same time admiring his wonderful interventional abilities. His relaxed approach with no great fanfare meant patients felt very comfortable under his expert care.

Tony was an active member of RANZCR, holding various positions over the years and was also the managing radiologist of Christchurch Radiology Group, and subsequently Pacific Radiology for 17 years. During his years at the helm of these groups he oversaw a huge expansion, with Pacific Radiology becoming one of the leading Radiology groups in Australasia.

Tony was hugely talented, he was both highly intellectual and extremely practical and was at his best dealing with high-risk, complex cases. He was extremely approachable with a "can do" attitude, cheerfully accepting further cases, no matter how great the work load.

Tony's radiology legacy is a very strong interventional radiology department, the younger radiologists whom he inspired with his skill and enthusiasm and the many MRTs and nurses who became integral members of the team.

Outside of work, Tony's happy place was in the Marlborough Sounds, surrounded by friends and family. He loved being on the water whether it was waterskiing, fishing, diving or just relaxing with a glass of wine. Tony, along with his wife Judith, was very generous in sharing his special place and his large group of friends were the beneficiaries of this. He also loved travelling and he and Judith spent six months travelling around Europe after he retired from Christchurch Hospital.

Tony Young was a very special man. The combination of a huge intellect, huge generosity, huge enthusiasm for life, a wonderful sense of humour, great practical skills and a desire to always help out if asked, made him very unique. He set a great example to all of us to try and follow.

In all aspects of his life Tony was of course very much supported by his amazing wife Judith. he could not have achieved everything he did without her. Her support also included nursing him at home during his final illness—something he very much wanted.

As well as Judith, Tony is survived by his children George, Alex and Harriet and grandchildren Oliver and Harry who he very much adored and was very proud of.

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Upadacitinib as monotherapy in patients with active rheumatoid arthritis and inadequate response to methotrexate

Methotrexate is regarded as being the first-line treatment in the management of rheumatoid arthritis. However, one-half to two-thirds of patients do not achieve satisfactory disease control.

Upadacitinib, which is an oral Janus Kinase selective inhibitor, has been shown to be helpful in combination with methotrexate for patients who do not respond adequately to methotrexate. In this study its potential value is assessed when it is used in such patients as the sole agent.

It was found that monotherapy showed statistically significant improvements compared with the use of upadacitinib and methotrexate in patients who had shown an inadequate response to methotrexate.

***Lancet* 2019; 393:2303–11**

Effect of systolic and diastolic blood pressure on cardiovascular outcomes

The relationship between outpatient systolic and diastolic blood pressure and cardiovascular outcomes remains unclear and has been complicated by recently revised guidelines with two different thresholds (>140/90mmHg and >130/80mmHg) for treating hypertension.

In this study the researchers use data from 1.3 million adults to determine the effect of the burden of systolic and diastolic hypertension on a composite outcome of myocardial infarction, ischaemic stroke or haemorrhagic stroke over a period of eight years.

The researchers report that although systolic BP elevation had a greater effect on outcomes, both systolic and diastolic hypertension independently influenced the risk of adverse cardiovascular events regardless of the definition of hypertension (>140/90 or >130/80).

NEJM 2019; 381:243–51

Stress-related disorders and risk of cardiovascular disease

This population-based, sibling-controlled cohort study was carried out in Sweden. The stress-related disorders included were post-traumatic stress disorders, acute stress reactions, adjustment disorders and other stress reactions.

The incidence of serious cardiovascular disease in over 100,000 patients with stress-related disorders was compared with the incidence in over 10,000 unaffected full siblings of these patients. A comparison was also made with over 1,000,000 matched unexposed people from the Swedish general population. During a 27-year follow-up the incidence of any cardiovascular disease in the three cohorts was 10.5, 8.4 and 6.9 per 1,000 patient years respectively.

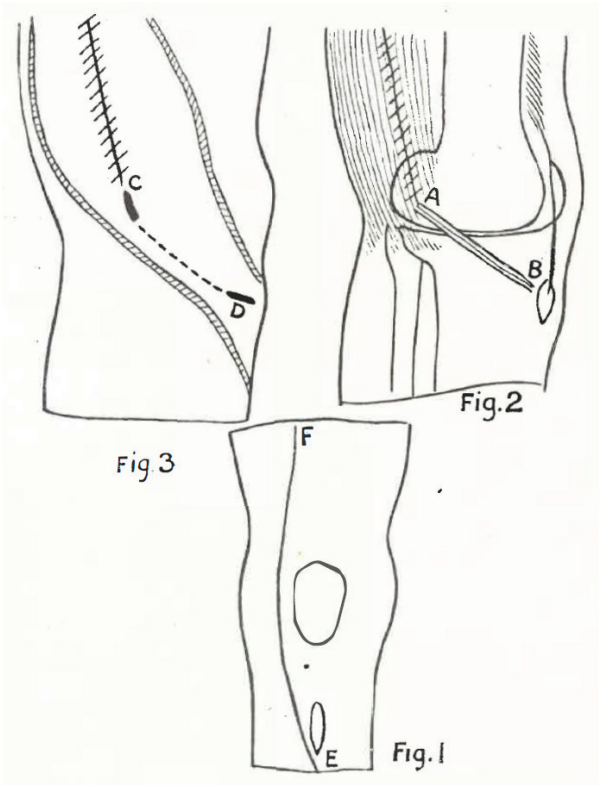
It was concluded that stress-related disorders are robustly associated with the subsequent incidence of serious cardiovascular disease.

***BMJ* 2019; 365:l1255**

URL:
http://www.nzma.org.nz/journal/read-the-journal/all-issues/2010-2019/2019/vol-132-no-1504-25-october-2019/8033

Operation to Replace the Most Important Function of the Anterior Crucial Ligament of the Knee Joint when Rupture of the Ligament has Occurred

By JOHN CRAIG, F.R.C.S.I., Consulting Surgeon to Gisborne Hospital



A new external lateral ligament is formed in the following way. An incision is made beginning at lower and inner side of the tubercle of the tibia and carried upward and outward by the outer border of patella, then upwards for about 3½ inches, E.F. The outer flap is dissected up including the superfical fascia. The fascia lata is disclosed and a strip about ¼ inch in breadth and of sufficient length to reach just beyond the tubercle of the tibia is dissected up from the point, marked A. in Fig. 2. This is situated just above the junction of the posterior and inferior borders of the outer condyle. The free extremity of this band is now passed between the fascia and skin of the outer flap, entering at the point marked C. in Fig. 3, and traversing the flap for about 2 inches, then emerging at point marked D. It is then carried through a hole drilled through the tubercle of the tibia at upper part and quite superficially. The end

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is turned up and stitched in that situation by two fine silver wire sutures. The gap in the fascia lata is now closed, and the skin united according to the technique of the surgeon. Ruptured anterior crucial ligament causes frequent partial posterior dislocation of the lower end of femur, a matter of great discomfort and some danger to the patient. My attention was drawn to this subject by a returned soldier affected by this accident. I could only find operations in literature that had the inside of the joint for their objective. The criticisms on those operations were not of a character to encourage their adoption. I therefore experimented with dry bones by tacking a tape in various situations on the condyles of a femur and upper part of tibia. I found by placing one in the situation marked A. and B. in Fig. 2, that you could flex the femur easily, but it did not allow any backward displacement. Now the mechanical side of the puzzle being solved, the next item was how to make such a ligament from the anatomic structures in relation to the knee joint. Such a one, I believe, we have in that part of the fascia lata which is attached strongly to the femur in the very place where we want it for our operation. I believe that the suggested operation is sound mechanically, anatomically, and physiologically. I fail to see where it is likely to disappoint surgeon or patient. It has the great advantage of extreme simplicity, nor could it reasonably be a cause of danger to the joint. The various tendons passing the joint received my consideration, and might be made to serve the purpose.

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