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Adult cochlear implant recipients and meningitis in New Zealand: are patients receiving the recommended immunisations?

Scott Mitchell, Lesina Nakhid-Schuster, Michel Neeff

This work looks at how many adults who have had a cochlear implant, which is a device surgically implanted into the hearing organ to help with hearing, had received the vaccinations that are recommended and fully funded in New Zealand to try and reduce their risk of meningitis (a rare but potentially life-threatening infection of the linings around the brain). We found that only 4.4% of adult cochlear implant recipients had received the 'full' recommended vaccination schedule.

Utilisation and maintenance of high-intensity statins following acute coronary syndrome and coronary angiography: opportunities to improve care (ANZACS-QI 26)

Andrew J Kerr, Sirisha Mitnala, Mildred Lee, Harvey D White

This study captured virtually all New Zealand patients discharged from hospital after a heart attack or unstable angina who had been considered appropriate for invasive heart treatments. A key pillar of management for these patients is optimal cholesterol lowering with high-intensity statin medication. We found that 79% of people were discharged with a high-intensity statin, although only 36% received the maximum recommended dose. However, by a year post-discharge only 60% were receiving high-intensity statin, and one in five people received no statin. We discuss the opportunities in-hospital, and post-discharge in general practice, to improve survival and quality of life, by improving utilisation of evidence-based secondary prevention medications.

The role and measurement of patient activation in the management of long-term conditions in New Zealand

Claire Budge, Melanie Taylor, Chiquita Hansen, Folole Fai, Materoa Mar

This article provides information about patient activation, a concept defined as people's knowledge skills and confidence to manage their own health. After describing previous research about how patient activation is measured, and the reliability and validity of the measurement instrument, we present data from a study carried out in New Zealand. Our results were similar to those found internationally and we conclude that patient activation is a useful tool for research and clinical practice in Aotearoa.

Incidence of venous thromboembolism after total hip, total knee and hip fracture surgery at Waitemata District Health Board following a peer-reviewed audit

James S Millar, Carlene MM Lawes, Bill Farrington, Penny Andrew, Peter Misur, Eileen Merriman, Matt Walker

Blood clots in the legs (deep vein thrombosis, or DVT) or lungs (pulmonary emboli, or PE), are rare side effects of lower-limb surgery. The rates of these blood clots in patients undergoing hip and knee replacement, and hip fracture surgery at Waitemata District Health Board were analysed. Comparing the years 2006–2010 and 2013–2016, DVT rates have decreased from 2.3% to 1.5% and PE rates from 0.9% to 0.6%. We believe this decrease is due to better collaboration between the orthopaedic and haematology departments, more widespread use of drugs that prevent blood clots, and interventions aimed at improving patients' recovery after surgery to enable them to walk sooner.

Vitamin D concentrations in New Zealanders with and without inflammatory bowel disease: do they differ?

Hannah Morton, Kevin C Pedley, Robin JC Stewart, Jane Coad

People with inflammatory bowel disease (IBD), which occurs at a high prevalence in New Zealand, are at increased risk of vitamin D deficiency. This study investigated the vitamin D status of people with IBD (Crohn's disease and ulcerative colitis) recruited from various towns throughout New Zealand and compared them to healthy controls. The main findings suggest that vitamin D status may be associated with disease severity in Crohn's disease.

Food taxes and subsidies to protect health: relevance to Aotearoa New Zealand

Nick Wilson, Lisa Te Morenga, Sally Mackay, Sarah Gerritsen, Christine Cleghorn, Amanda C Jones, Boyd Swinburn

The hazardous and obesogenic food environment are major contributors to health loss in Aotearoa New Zealand. In this article we report that each one of the 14 recent systematic reviews on the food tax and/or subsidy topic since 2015, find that such interventions have favourable impacts from a health perspective. The New Zealand-specific evidence we considered (12 studies since January 2010) is less definitive, but the pattern of results is consistent with the international evidence. Given this overall picture, the New Zealand Government should consider such tax/subsidy interventions, potentially starting with a UK-style sugary drinks industry levy.

Politicians: please work together to minimise the spread of COVID-19

David Murdoch, Michael Addidle, Hanna-Sofia Andersson, Brendan Arnold, Michelle Balm, Jackie Benschop, Bryan Betty, Mark Birch, Max Bloomfield, Cheryl Brunton, Andrew Burns, Stephen Chambers, Lynley Cook, Simon Dalton, Harvey Duncan, Juliet Elvy, Richard Everts, Joshua Freeman, Nigel French, Kate Grimwade, David Hammer, David Hayman, David Holland, Ben Hudson, Paul Huggan, Susan Jack, Rosemary Ikram, Matthew Kelly, Iain Lamont, Michael Maze, Gary McAuliffe, Stephen McBride, Sarah Metcalf, Susan Morpeth, Arthur Morris, Samantha Murton, Ramon Pink, Alan Pithie, Martin Pitout, Patricia Priest, Nigel Raymond, Kerry Read, Stephen Ritchie, Matthew Rogers, Philip Schroeder, Susan Taylor, James Taylor, Mark Thomas, Arlo Upton, James Ussher, Anja Werno, Siouxsie Wiles

With COVID-19 now present in New Zealand, and the situation a genuine health emergency, it is essential our political leaders work together and use their influence to minimise the impact of the virus in our community.

As a group of more than 50 of the country's leading infectious disease and public health scientists and professionals, we ask the following of our political leaders:

Although it is election year, we urge politicians resist the temptation to scaremonger in an attempt to score points in the media. Instead, they should use their moments in the spotlight to amplify messages of our health system's preparedness and how New Zealanders can individually make a difference at this critical time. A cross-party parliamentary task force on COVID-19 could be one way to ensure this cooperation happens in a timely and productive fashion.

The level of fear around COVID-19 is high. New Zealanders are being bombarded with information and misinformation about this new viral disease. When people are scared or ill-informed, they aren't at their best. When they are well-informed they can make a huge difference both as individuals and as members of the wider community. This is very true with COVID-19, where every person practising good hygiene and cough etiquette can radically impact the spread of this disease. If the virus spreads further throughout our communities, and authorities ask people to limit social contact and self-isolate, cooperation with these necessary measures will play a crucial role in minimising COVID-19's spread and protecting the most vulnerable among us.

Teams of infectious disease, public health and primary care experts are advising the Government on the best way to deal with this threat. Specialist members of our country's health system have been preparing for such a scenario for many years and their plans are being put into action, and tweaked where necessary as new information comes to hand. These experts are monitoring the rapidly changing situation, looking to what is being done and what is working in other countries, and giving pragmatic, evidence-based advice on a regular basis. Politicking and criticising these professionals who are working hard on behalf of the country does nothing more than undermine them and public confidence in our system. It is the media's role to report on matters of public interest and concern, and we ask politicians to leave this task to them and instead show leadership in spreading essential information.

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It is essential to work together in times of crisis. We need our politicians to avoid cluttering the media landscape with political messages and undermining the life-saving information coming from the government, health professionals, scientists and public health officials.

Competing interests:
Nil.

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Mentally ill people in our prisons are suffering human rights violations

Erik Monasterio, Susanna Every-Palmer, Julie Norris, Jackie Short, Krishna Pillai, Peter Dean, James Foulds

"The true measure of any society can be found in how it treats its most vulnerable members"

Mahatma Gandhi

The prison population increase in Aotearoa New Zealand

Successive governments have ignored the effect that rapid growth in the prison population has had on health and equity in Aotearoa New Zealand. The clinical directors from all the regional forensic services consider this an unacceptable health equity and human rights crisis, requiring an urgent Government response. This editorial is based on the authors' direct experiences of providing clinical care in Aotearoa New Zealand's prisons over many years and analysis of key epidemiological data.

The New Zealand prison population has more than doubled since 2000[1] despite little change in serious crime over that period.[2,3] The population sits at just over 10,000. Māori are overrepresented in the criminal justice system, and Māori women especially so. Over 50% of prisoners and 60% of female prisoners are Māori, despite Māori being 16.5% of the total population.[1] The majority of prisoners (93%) are male, but in the five years to 2017, the female prison population increased by just over 56%, rising nearly three times as fast as the male population for the same period.[1] Rates of incarceration in Aotearoa New Zealand are the fifth highest among 36 Organisation for Economic Cooperation and Development (OECD) countries, and are about 30% higher per capita than Australia.[2]

In 2018, the New Zealand Justice Minister described prisons as "stretched to breaking point".[3] The Government has targeted a 30% reduction in the prison population by 2030. To meet this target, New Zealand's justice system will need to be rebuilt.

Growth in the prison population has far exceeded resources to manage the wide and complex range of health problems common in this population. It leaves prisoners with acute health needs far worse off than the rest of the population, especially those suffering from serious mental illness. The growth has contributed to a serious—arguably scandalous—mental health crisis with few options for relief in sight.

Prisoners' health needs: a vulnerable population

Most people in prison have been exposed to childhood trauma and other adverse childhood experiences. Prisoners also experience poor physical health, and while their physical health typically improves in prison,[4] excess morbidity and mortality extends well beyond prison release.[5]

Two important prison studies conducted 15 years apart in Aotearoa New Zealand show rates of mental disorder and substance use disorder are very high and climbing, with over 90% of the prison population having a lifetime diagnosis of a mental health or substance use disorder.[6,7] These studies also show that psychotic symptoms are far more common in prison than in the general community.[6]

The rise of methamphetamine[8] has also strained mental health services in our prisons. One in eight prisoners has a current dependence on stimulants.[6] Since methamphetamine increases the risk of psychosis,[9] remand prisoners who have been using methamphetamine heavily are often acutely mentally unwell, and some need psychiatric hospital admission.

While the public may not see prisoner welfare as a national priority, there are important reasons to promote the health of people in prison. First, the Government has an obligation under Te Tiriti o Waitangi to

protect the rights of Māori. As described, rates of imprisonment are particularly high for Māori.[1] The disproportionate incarceration, and the added barriers to adequate healthcare and dislocation from whānau for Māori in prison, are in blatant breach of Te Tiriti o Waitangi. In The third article of Te Tiriti o Waitangi, the Principle of Equality constitutes a guarantee of legal equality between Māori and other citizens of Aotearoa New Zealand, and assurance of equal access to social rights. The right to equal healthcare has been ratified in international human rights frameworks (such as the Convention of the Elimination of all forms of Racial Discrimination and the Declaration on the Rights of Indigenous People), of which Aotearoa New Zealand is a signatory.Te Tiriti o Waitangi also established a partnership, which imposes on the partners the duty to act reasonably and in good faith.

Second, The Government has the responsibility under the Bill of Rights Act 1990 to protect prisoners' rights, including the Right 23 (5) to be treated with humanity and dignity, and Right 9 not to be subjected to cruel treatment.

Furthermore, as a signatory to the 2008 United Nations Convention on the Rights of Persons with Disabilities (CRPD), the Government has a duty to enable access to healthcare services for people with a disability, including serious mental illness, in custody.

Third, health outcomes and offending outcomes are inextricably linked. Good quality healthcare is an essential component of rehabilitation and it leads to lower re-offending.[2] Most stays in prison are short, therefore improving the health of people in prison benefits the population in general.

Finally, providing mental healthcare is particularly important for this vulnerable group since suicide rates among people in prison are high[10,11] and they remain elevated after release.[12]

Prison mental healthcare has not kept up

The Department of Corrections has increased resources to provide additional support and treatment to prisoners with mild to moderate mental distress/illness and addictions, including with the projected building of a 100-bed mental health facility in Waikeria Prison.[13,14] However, these beds will not meet the needs of prisoners with serious mental illness and will only service the Waikato area. So far, the increase in the prison population has been met by little increase in prison capacity or funding for specialist mental health services in prisons. For example, 610 extra prison beds have been planned in Christchurch (a potential 50% increase in prisoner population) by the middle of 2020 with no extra specialist mental health resources.[15]

Forensic psychiatric services' ability to meet the demand for acute psychiatric care for those with serious mental illness has become unsafely stretched. This has resulted in forensic services throughout the country placing prisoners needing immediate psychiatric inpatient treatment on waitlists, rather than admitting them to hospital—as would occur if they were in the community. Involuntary treatment cannot legally be enforced in prison. It is the authors' clinical experience that not infrequently this leads to acutely unwell prisoners, including those with a severe acute psychosis, waiting untreated for weeks under 23-hour per-day solitary lockdown in Intervention and Support Units (ISU). Those prisoners often cannot keep up even basic self-care, they pose risks to themselves and others from symptoms of their illness, and some show extremely disturbed or aggressive behaviours. This is more than lack of access to healthcare while in custody—in the language of the CRPD, this is inhuman and degrading treatment.

An unannounced inspection of Christchurch Men's Prison by the Office of the Ombudsman in 2017 found serious human rights breaches.[16] It described the ISU (then known as the At Risk Unit, or "ARU") as lacking even basic amenities such as furniture, adequate toilets and access to natural light or fresh air. The austere conditions were felt to breach United Nations Standard Minimum Rules for the Treatment of Prisoners (the Mandela Rules[17]). The Ombudsman also noted that the average waiting time for transfer from the ARU to a Forensic bed was four weeks. Most concerning, despite the Ombudsman

pointing out these human rights breaches, there has been no meaningful change in the two years since the Ombudsman's report; in fact, if anything the situation has worsened.

How to fix the prison problem

Prisoners lack an effective public voice—currently sentenced prisoners cannot vote. A planned amendment to the Electoral Act 1993 (The Electoral Amendment Bill) will give voting rights to those serving sentences less than three years, but it is yet to be seen whether this will help alter the dire state of our prisons. Furthermore, political opposition to this amendment suggests it may not survive a change of government.

The main determinant of criminal behaviour is inter-generational social disadvantage including effects of colonisation on Māori. Institutional racism and cultural insensitivity are some of the root causes of the overrepresentation of Māori within prisons.[18] A 2014 United Nations Working Group on Arbitrary Detention found that systemic bias existed against Māori at all levels of the criminal justice system.[19] Proximal causes of serious crime include poverty, inequality, exposure to family violence and trauma, substance use and destabilising peer influences. At a population level, none of these risk factors can be easily removed in the short term. However, in the long-term the common underlying risk factors might be addressed via programmes which support the health and wellbeing of vulnerable children and their families.[20] This will require a consistent all-of-government approach, free of the forces of partisan agendas.

Many other processes play a role in determining how many people are in prison. A systematic political agenda of "tough on crime" has led to substantially longer sentences. At the same time, the Bail Amendment Act 2013 has contributed to a 50% increase in the number of people remanded in custody comparing the 2013/2014 and 2018/2019 fiscal years, yet no new services or funding have been provided for this high-needs group, even though this outcome could have been foreseen. Coherence between law, policy and funding decisions is vital.

Specialist Mental Health Courts (as run successfully, for example, in South Australia[21] and other Australian states) do not exist in Aotearoa New Zealand. However, they make sense as a response to the increasing number of people with serious mental illness who are remanded to prison on minor charges. In many cases offending in this group can be traced to general adult mental health services' lack of resources to assertively treat these people or provide the level of care necessary for successful diversion, as a result of decades of decay from under-funding.

Likewise, there is good evidence to support Drug Courts,[22] which have been piloted successfully here since 2012 but have not yet been rolled out throughout the country. While these specialised courts are more expensive to run, they provide more flexible sentencing options for people who have offended, including alternatives to prison.

Finally, the Government Inquiry into Mental Health and Addiction[23] was a chance to put forward solutions to some of the problems mentioned in this article. However, the Inquiry did not focus on the needs of those with serious mental illness, instead laying out plans to expand mental health services to cater for a much larger and less severely unwell group of people—a shift in ideology which has alarmed leading academic psychiatrists.[24] The Inquiry's comparative neglect of people with serious mental illness is hard to understand while Aotearoa New Zealand is still failing in its obligations to this group.

We urge the Government to act now to protect the human rights of people in prison with disabilities including serious mental illness. A strong and immediate government response is needed to end the serious human rights abuses which are being inflicted on vulnerable people in this country's prisons.

Competing interests:
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Adult cochlear implant recipients and meningitis in New Zealand: are patients receiving the recommended immunisations?

Scott Mitchell, Lesina Nakhid-Schuster, Michel Neeff

ABSTRACT

AIM: To investigate if adult cochlear implant (CI) recipients have received the recommended immunisations as compared to current guidelines and to report instances of meningitis within this population. **METHODS:** Telephone interview of CI recipient's general practitioner (GP) surgeries for details regarding immunisations received. Subsequent reporting of immunisation rates of adult patients, under the care of the Northern Cochlear Implant Programme (NCIP) in New Zealand, when compared to the recommended guidelines from the Immunisation Advisory Centre (IMAC) and rates of meningitis of CI recipients are presented. **RESULTS:** It is recommended to immunise against the most common organisms causing meningitis, Streptococcus pneumoniae and Haemophilus influenzae type b (HiB), as well as influenza. Data for 135 CI recipients over the last five years was complete. 14.8% of patients had received a full pneumococcal immunisation schedule. 11.9% had received a HiB immunisation and 62.2% an influenza vaccination. No patient had developed meningitis following CI insertion. **CONCLUSION:** This paper highlights clear issues with the immunisation of adult CI recipients.

A cochlear implant (CI) consists of two parts: an external part, the processor, worn behind the ear like a hearing aid which communicates through the scalp via a magnetic coil with the implant proper, the receiver/stimulator, which includes an active electrode array which traverses through the mastoid, middle ear and enters the scala tympani of the inner ear through the round window or a drilled cochleostomy near to the round window.[1] In response to auditory stimuli, this electrode array provides direct electrical stimulation of neurons in the auditory nerve. This gives the recipient a perception of sound which aids speech recognition. Bacteria may directly migrate from the middle ear into the cochlea and labyrinth in a similar manner as reported by Friedmann and Arnold when describing meningitis caused by spreading infection in acute or chronic otitis media.[2] Here organisms infiltrate the cochlear turns along the electrode, enter Schuknecht's bony channels and follow perineural or perivascular pathways into the internal auditory canal.[2,3] Direct haematological spread of infection to the cochlea resulting in micro-abscess formation and onward spread to the internal auditory meatus has also been theorised.[3] Whatever the mechanism, it is well documented that there is a greater incidence of meningitis in both adult and paediatric CI recipients when compared to the general population as a whole.[4–8] Additional risk factors of meningitis have been documented, including inner ear malformations with or without cerebrospinal fluid (CSF) leak, persistent CSF leak after CI surgery, history of a ventriculoperitoneal

Table 1: Immunisation schedule for adult cochlear implant patients adapted from the New Zealand Immunisation Advisory Centre.[9]

Pneumococcus		**Haemophilus influenza B (HiB)**		**Influenza**	
Age	**Vaccine**	**Age**	**Vaccine**	**Age**	**Vaccine**
>18 years ↓	One dose of PCV 13 (PREVENAR 13)* *Followed by:* 23PPV (PNEUMOVAX 23) One dose, at least eight weeks AFTER previous Prevenar 13	2 months to under 65 years	HIBERIX One dose either pre- or post-CI	9 years to <65 years >65 years	INFLUVAC TETRA One dose—yearly vaccination INFLUVAC TETRA Annual vaccination
Minimum of five years after last Pneumovax 23 vaccination ↓	PNEUMOVAX 23 One further dose				
>65 years of age	PNEUMOVAX 23 One further dose once over age 65				

*If 23PPV has already been given (prior to any doses of PCV13) to adults aged 18 years and older, wait at least one year before administering PCV13.

shunt and the use of cochlear implants with positioner devices (which were withdrawn in 2002).[10] As a result of this increased risk, most developed countries including the US,[11,12] the UK,[13] Canada[14] and Australia[15] recommend vaccination against the most common causative organisms. The recommendations from the New Zealand Immunisation Advisory Centre include vaccination against pneumococcus, Haemophilus influenzae and influenza.[9] A summary of the schedule can be seen in Table 1.

Aims

We aim to report the rate of adult cochlear implant patients, under the care of the Northern Cochlear Implant Programme, who received their immunisations as per the schedule recommended by the Immunisation Advisory Centre (IMAC) in New Zealand. We also aim to report any recorded occurrences of post-implantation bacterial meningitis within this patient population.

Methods

Following approval from the northern cochlear implant programme, a retrospective study investigating adult patients who had received unilateral or bilateral cochlear implantation between September 2013 and September 2018 was undertaken searching the database of the Northern Cochlear Implant Programme. Demographic data including the date of implantation were identified. Telephone interviews were completed with general practitioner (GP) surgeries, obtaining patient information on type and date of any immunisations received, contraindication to immunisations and any hospital admissions for suspected or confirmed meningitis. Simple statistical analysis was completed using Microsoft Excel software.

Results

A total of 189 adult CI recipients were identified within the Northern Cochlear

Table 2: Vaccination rates and timing of vaccination in relation to cochlear implantation.

Received vaccination?	PCV 13 (Prevnar 13)	PPV 23 (Pneumovax 23)	HiB (Hiberix)	Influenza (Influvac Tetra)
NO	95 (70.4%)	81 (60%)	119 (88.1%)	51 (37.8%)
YES	40 (29.6%)	54 (40%)	16 (11.9%)	84 (62.2%)
Timing of vaccination?				
Vaccinated before CI	18 (13.3%)	28 (20.7%)	6 (4.4%)	Yearly prior to CI—38 (28.1%) One dose prior to CI—6 (4.4%)
Vaccinated after CI	22 (16.3%)	26 (19.3%)	10 (7.4%)	Yearly after CI—10 (7.4%) One dose after CI—13 (9.6%)
Unknown timing				No date recorded but confirmed had vaccination—17 (12.6%)

Implant database over the five-year period. Complete data was collected for 135 (71.4%) patients, which were included in the analysis.

Fifty-four patients in total were excluded as they had no known GP (42 patients), GP practice uncontactable despite multiple attempts (10 patients), patient had changed GP with unknown new GP (one patient) or had deceased following implantation from cause unrelated to surgery or bacterial meningitis (one patient).

The average patient age was 61 years (median 65.3, range 20.4–93.7 years) with 51% male and 49% female. No patient had any relative or absolute contraindications to receiving vaccinations.

The results for percentages of patients having received vaccination can be seen in Table 2.

With regards to pneumococcal vaccination, 29% had received the PCV 13 vaccine and 40% the 23 PPV. Only 14.8% (15 patients) had received both of these vaccinations, as per the Immunisation Advisory Centre recommendations.

Overall, 4.4% (six patients) were fully immunised in accordance with the New Zealand Immunisation Advisory Centre guidelines.

In terms of timing of vaccination of patients who received their vaccinations, 45% (18/40) PCV 13, 52% (28/54) PPV 23, 38% (6/16) HiB and 52% (44/84) influenza, received the vaccinations before their implantation surgery.

No cases of meningitis were reported in our patients.

Discussion

Overall, meningitis is a rare complication of cochlear implantation and the rates of meningitis are low. CI recipients are, however, at an increased risk of meningitis.[4–8] A recent review of 18 studies combining 5,324 CI patients identified nine cases of meningitis with an incidence in this population of 0.2%.[8] It should be noted that meningitis caused by various serogroups of the more common organism, Neisseria meningitides bacteria, are not implicated in CI associated infections and that meningitis in CI recipients is more in keeping with an invasive pneumococcal infection. The reported 'all-age' rate of pneumococcal meningitis in New Zealand in 2016 was 0.9 per 100,000 population,[16] which is clearly lower than the most recent 2017 notification rate for meningococcal disease of 2.3 cases per 100,000 population.[17] During implantation, surgeons aim to limit the risk of meningitis by minimising insertion trauma, packing the insertion site, choice of device, providing prophylactic antibiotics around the insertion period, promptly treating any post-implantation acute ear infections and vaccinating the recipient.[18] There is debate about the direct effectiveness of vaccination in preventing meningitis, specifically in cochlear implant recipients,[18] but there is clear evidence that vaccination decreases pneumococcal meningitis incidence, morbidity and mortality. An example of this is a recent study in Brazil, highlighting a 50% reduction in pneumococcal meningitis incidence and 69% reduction in mortality after introduction of a PCV10 vaccination programme.[19] There is also

sufficient evidence from population-based studies, including studies from New Zealand, that vaccination is effective in reducing 'invasive pneumococcal disease', which includes meningitis.[12,20–22] In a recent large double-blind placebo controlled study, vaccination was shown to prevent adult community-acquired pneumonia and reduce the presence of S. pneumoniae sub-types from sampled 'sterile' sites within the study population.[23] There have also been population-based studies with evidence to suggest a sustained additive effect of pneumococcal and influenza vaccination in preventing all-cause mortality and hospitalisation in the elderly population.[24–26] This suggests that combined vaccination can be potentially more effective than single vaccination alone.

The optimal timing of vaccination for cochlear implant recipients is unclear,[12] but in most cochlear implant programmes it is preferred that candidates receive the vaccination prior to implantation.[11–15]

There are few articles on vaccination against Streptococcus pneumoniae in cochlear implant patients specifically.[27] Vaccination rates published from the US seem to range from 49% to 99%,[28,29] but a recent Polish study of 740 patients showed a vaccination rate of 49.2% in children and 5.5% in adults. Overall, our results compare unfavourably with the rates reported in the literature. In our review no patient contracted meningitis so far, but this potential risk needs to be managed by improving vaccination rates. Poor rates of general vaccination uptake are a worldwide public health problem[30] and comments upon the potential barriers to vaccination must be made. In New Zealand, prior to the latest guidelines from 2017, only the pneumococcal vaccine was recommended outside of the routine childhood vaccination schedule, but this was not publicly funded and therefore a cost to the patient. Hence, socioeconomic considerations may have contributed to the low vaccination rate in our population.

After 1 July 2017, CI recipients were added to the special group schedule, with full funding for vaccination available.[9] In general, health practitioners may not have been aware of this change in guidelines. In a recent study from the US, it was reported that "cochlear implant providers have a high awareness of vaccination guidelines, but less detailed knowledge of age-specific recommendations". Most had the primary care provider give the recommended vaccinations.[31] The current practice of the NCIP is similar, in that general practitioners are sent a copy of the vaccination eligibility on discharge following implantation. It is therefore surprising that there is low rate of vaccination. Other barriers to vaccination may have played a part. These have been defined and discussed in the literature and include lack of physician recommendations, mistaken patient assumptions such as "healthy people do not need immunisation", concerns about side effects, fear of autism, fear of needles or objections based on moral or religious grounds,[31,32] but the degree to which these may have impacted upon our patient population and our results is difficult to estimate.

Strategies to improve vaccination uptake have been investigated for other vaccination programmes. A recent systematic review, based principally on papers from the US, reviewed potential interventions to increase influenza vaccination rates in high-risk children including: multi-component strategies, letter reminders, telephone recall, letters plus telephone calls, educational tools and year-round scheduling, among others. There was good evidence for the effectiveness of reminder letters, but weak evidence for the effectiveness of other strategies.[33] Further studies from the US have investigated the effects of using an 'immunisation verification protocol' to increase the recording and uptake of vaccines. This identified that those patients who were required to document immunisation status before surgery had the highest rates of compliance.[34] Unfortunately, to the authors' awareness, there were no other specific studies investigating methods for increasing vaccination uptake within this specific group of patients or how effective these methods might be. Practically, within New Zealand, the adoption of reminder letters to both patients and vaccination providers is potentially both attainable and manageable following implantation. This may increase awareness among medical professionals and patients. Similarly, disseminating relevant research or updates among relevant media platforms, such as publication in

journals, may also highlight a need for changes in practice. Having vaccinations before implantation or as a prerequisite to implantation is not likely to be feasible for various reasons, including the point that patients typically may fall into the 'specially funded' criteria only after implantation. Equally, central funding for implants is fluctuant and delaying any patient's implantation to wait for immunisations is not necessarily beneficial. This is in-line with international practice; such as that in the UK where "immunisation should not delay implantation".[13]

Limitations and future work

The retrospective nature of this work leads to potential issues regarding documentation of interventions. It is possible that individuals received vaccination, but this was not documented. Given the number of patients involved, it is clear that patients lost to follow up may have influenced the results of this work.

The Northern Cochlear Implant Programme have taken measures to address the low vaccination rate in our patient cohort. General practitioners will be contacted regarding the availability and requirement of these vaccinations. There will be systematic improvements to our services to ensure vaccination rates are monitored for the adult and paediatric population of cochlear implant recipients.

A review of the vaccination rate, specifically in the paediatric CI population, is underway.

Summary

The vaccination rate of CI patients implanted by the Northern Cochlear Implant Programme in New Zealand is low, but fortunately no CI recipient has been identified as having had a CI-related meningitis infection as yet. Systematic approaches with an initial focus on dissemination of new guidelines are required to increase the vaccination rates in our CI users.

Competing interests:
Dr Neeff is the chairperson of the Northern Cochlear Implant Programme Advisory Group. The Northern Cochlear Implant Programme (NCIP) is a publicly funded programme for profoundly deaf children and adults in the northern region of New Zealand.

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Utilisation and maintenance of high-intensity statins following acute coronary syndrome and coronary angiography: opportunities to improve care (ANZACS-QI 26)

Andrew J Kerr, Sirisha Mitnala, Mildred Lee, Harvey D White

ABSTRACT

AIMS: A key pillar in the medical management of patients after an acute coronary syndrome (ACS) is the early initiation and maintenance of "high-intensity" statin therapy to lower low-density lipoprotein cholesterol (LDL-C) and to improve clinical outcomes. The aim of this study was to describe the New Zealand utilisation of high-intensity statin therapy in the first year post-ACS.

METHODS: 19,867 New Zealand patients (≥20 years), discharged post-ACS event (2015–2017) were identified from the All New Zealand ACS Quality Improvement (ANZACS-QI) registry and anonymously linked with the national pharmaceutical dataset to identify statin dispensing early (0–3 months) and late (9–12 months) post-discharge. "High intensity" statin was subdivided into the New Zealand guidelines recommended dose (80mg atorvastatin) and "other high-intensity" statin (atorvastatin 40mg, simvastatin 80mg). All other statin doses were classified as "low/medium dose".

RESULTS: Seventy-nine percent were initially dispensed high-intensity statins. Thirty-six percent of the overall cohort received 80mg atorvastatin and 43% a lower "other high-intensity" statin. A further 13% received a medium/low dose and 8% no statin. By 12 months, 29% were dispensed atorvastatin 80mg, 36% another high dose, 14% a low/medium dose and 21% no statin. Only 14% of those initially on 80mg atorvastatin had a statin dose reduction. After multivariable adjustment, the risk of discontinuation was the same for those started on atorvastatin 80mg compared with "other high dose", and lower than for those started on a low/medium dose. Few patients (6.2%) had statins started, or dose up-titrated post-discharge. There is clinically unexplained variation in the use of the highest atorvastatin 80mg dose between district health boards (range 15% to 65%).

CONCLUSIONS: Eight in 10 ACS patients were dispensed a high-intensity statin at discharge, but only 36% received the guidelines-recommended dose of 80mg of atorvastatin. By one year, one in five patients discharged on a statin were not receiving it. There are opportunities to improve longer-term LDL-C reduction and clinical outcomes through dosage optimisation and improved medication maintenance.

Acute coronary syndrome (ACS) is one of the leading causes of death in New Zealand and worldwide. Over 15,000 patients are admitted to New Zealand hospitals with an acute coronary syndrome (ACS) every year and the one-year case fatality is 20%.[1] LDL lowering using 3-hydroxy-3-methyl-glutaryl-CoA (HMGCoA) inhibitors (statins) following an acute coronary syndrome has been shown to reduce the risk of heart attack, stroke and all-cause mortality.[2] Trials of higher-dose statin therapy compared with lower-dose therapy have been demonstrated to further reduce major vascular events in patients with ACS or stable coronary artery disease.[3,4] The early

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period following an ACS is a critical stage of coronary heart disease; with a high risk of recurrent events and death. The early initiation and maintenance of "high-intensity" statin after ACS is recommended for all patients without contraindications, regardless of initial low density lipoprotein-C (LDL-C) values.[5–14] This recommendation comes with the proviso that the use of lower-intensity statin therapy should be considered in patients at increased risk of adverse effects with high-intensity statin therapy, such as in the elderly, patients diagnosed with hepatic or renal impairment, or in the case of a potential risk of drug-drug interactions with other essential concomitant therapies. Prior studies report that only two-thirds of patients who present with an ACS are maintained on a statin at one year[15] and three years'[16] post-discharge. But to date, no studies have reported the use of high-intensity therapy in New Zealand post-ACS.

"High-intensity" has been variously defined by either the type of statin/dose used in the randomised controlled clinical trials or by the pharmacological effects of statin type/dose on mean LDL-C levels. While the American College of Cardiology/American Heart Association (ACC/AHA) definition of high-intensity statin is based on statin/type and dose,[11] the European Society of Cardiology (ESC) definition includes any statin type/dose which can reduce mean LDL by 50%.[17] However, in the ESC guideline, data is presented showing the statin type/doses which achieve this LDL reduction, which align well with the ACC/AHA definition. Three of the five more intense versus less intense clinical trials used 80mg of atorvastatin in the "high-intensity" arm,[18–20] which lowers LDL-C by approximately 50%.[14] Two trials used simvastatin 80mg as the more intense statin arm,[21,22] a dose which is no longer recommended due to an excess of myositis, and has a Food and Drug Administration (FDA) black box warning not to prescribe this dose of simvastatin. In one trial a small percentage received atorvastatin 40mg[20] and this dose has been included within the ESC and AHA/ACC "high-intensity" statin definition.[11,17] Moderate low/medium doses are expected to reduce LDL-C by 30–50%.

Since 2015 the All New Zealand Acute Coronary Syndrome Quality Improvement (ANZACS-QI) registry has captured data on over 95% of New Zealand patients hospitalised for ACS who underwent coronary angiography. By linking ANZACS-QI registry data to national routine datasets, both initial statin dispensing (type and dose), and subsequent maintenance, can be tracked.[23]

The aim of this study is therefore to describe the contemporary New Zealand use of high-intensity statin therapy after discharge post-ACS, its maintenance in the first year and the determinants of high-dose statin maintenance at one year.

Methods

Cohort

New Zealand residents (aged over 20y) hospitalised with ACS between 1 Jan 2015 to 31 Dec 2017 and who underwent coronary angiography were identified from the ANZACS-QI registry. Only patients who were alive 30 days after discharge were included to ensure the completeness of early dispensing data. Only the first presentation per person in the three-year period was included.

The ANZACS-QI registry is a web-based electronic database that captures a mandatory dataset in ACS patients who underwent a coronary angiogram, which includes patient demographics, admission ACS risk stratification, cardiovascular risk factors, investigations and management, inpatient outcomes and medications prescribed at discharge. Details regarding the ANZACS-QI programme and registry data collection have been previously reported.[24] The registry is subject to monthly auditing to ensure capture of >95% of all patients admitted with suspected ACS who are investigated with coronary angiography, and annual audit to check the accuracy of data entry.

Definitions

ACS included myocardial infarction (MI) or unstable angina. MI was defined according to the contemporary universal definition.[25] Variables used for this study included age, sex, ethnicity, prior statin use, diabetes, smoking status, history of cardiovascular disease (CVD), coronary intervention (coronary artery bypass grafting (CABG)) or percutaneous coronary intervention (PCI) and a global estimate of in-hospital mortality post-ACS (the Global Registry of ACS (GRACE) score).[26] The GRACE score is reported categorically as low (<1%),

medium (1 to <3%) or high (>=3%) as recommended by the ESC. History of CVD was defined as a diagnosis, prior to the current event, of MI/angina, stroke/TIA, peripheral vascular disease or prior radiological evidence of vascular disease. PCI/CABG Sociodemographic variables and residency status were derived from the linked national dataset. For patients in whom more than one ethnic group was recorded, ethnicity was prioritised, in accordance with health sector protocols, in the following order: indigenous Māori, Pacific, Indian and New Zealand European/Other (NZEO).[27] Socioeconomic deprivation was assessed by the NZDep13 score,[28] a census-based small area 10-point index of deprivation based on the person's domicile. Prior statin use was derived from the national pharmaceutical claims dataset and was defined as having a statin dispensed during the 90-day period prior to index admission. Concomitant medications dispensed at discharge which should be used with caution in combination with statins were also identified. Because most high-dose statin use was with atorvastatin, we have reported the co-dispensing of medications identified as having a potentially major interaction with that agent.[29,30] This group comprises cyclosporin, diltiazem, verapamil, digoxin, erythromycin, clarithromycin, gemfibrozil, ketoconazole, itraconazole and ritonavir.

Statin dispensing analysis

Statin type and dose dispensing were obtained early (within three months of hospital discharge) and late after discharge (in the last three months of the year after discharge) using anonymised linkage to the national pharmaceuticals dataset in order to identify and describe the patients who were initiated on high-dose statins, and those who were maintained on maximum-dose statins at one year.

"High-intensity statin" was defined according to the ACC/AHA definition.[11] In New Zealand this comprises atorvastatin 40mg or 80mg, or simvastatin 80mg. For this analysis we chose to divide "high intensity" into the clinical trial high-intensity dose of 80mg atorvastatin recommended in the New Zealand guidelines[6,7] and "other high-intensity" statin (atorvastatin 40mg, simvastatin 80mg). Rosuvastatin is not publicly funded in New Zealand so use is very low. All other statin type/doses combinations were considered low/medium dose.

Data linkage

Medication dispensing data, hospitalisation and mortality data required for assessment of medication maintenance were obtained by individual linkage to routine national datasets as previously described.[24] An encrypted version of the National Health Index Number (NHI), a unique identifier assigned to everyone who uses health and disability support services (>98% of the population),[31] was used to anonymously link in-hospital ANZACS-QI patient records to the national datasets. Dispensing of cardiovascular medications was identified from the national Pharmaceutical Collection (PHARMS), which is jointly administered by the New Zealand Ministry of Health (MOH) and the Pharmaceutical Management Agency of New Zealand. PHARMS contains data regarding government-subsidised medications dispensed by community pharmacies nationwide, which encompasses statin medications relevant to these analyses.[32] By 2009, 96% of dispensing episodes were reliably identifiable by NHI numbers. (S Ross, MOH, personal communication). ANZACS-QI validation in the national cohort has shown a high level of statin dispensing capture in PHARMS. In the 2015 to 2017 cohort, similar to the current study cohort, 95% of ACS patients were recorded as prescribed a statin in the ANZACS-QI registry and 93% were recorded as dispensed in PHARMS (unpublished data, manuscript in preparation).

Statistical analysis

Descriptive statistics for continuous variables were summarised as mean with standard deviation, median with inter-quartile range (IQR) and range. Categorical data are reported by frequency and percentage. For continuous variables, comparisons between groups were performed by the Student's T-test. For categorical variables, Pearson's chi-squared test was used.

Three multivariable log-binomial regression models were constructed to investigate the variables associated with (i) initial dispensing of 80mg atorvastatin, (ii) statin dose reduction by one year in those initially dispensed 80mg of statin post-dis-

charge, and (iii) statin discontinuation by one year in those initially receiving any dose of statin. The relative risks (RRs) with accompanying 95% confidence intervals (CIs) of covariates were estimated. The covariates adjusted for were age, sex, ethnicity, smoker, diabetes, prior CVD, history of congestive heart failure (CHF), statin three months prior to admission, LDL-C level at baseline, coronary revascularisation, common concomitant medications, district health boards (DHB) and initial statin dose (model iii only).

All P-values reported were two tailed and a P-value <0.05 was considered significant. Data were analysed using SAS statistical package, version 9.4 (SAS Institute, Cary, NC).

Ethics

Linkage of the ANZACS-QI and national datasets has been approved by the National Multi Region Ethics Committee (MEC/07/19/EXP).

Results

The demographics, risk factors, clinical presentation and inpatient management for the 19,867 patient cohort are shown in Table 1. Overall, 92% of patients were dispensed a statin within the first three months post-discharge. Thirty-six percent of patients were dispensed 80mg atorvastatin and 43% "other high-intensity" statin dose, predominantly atorvastatin 40mg. Seventy-nine percent of patients were therefore dispensed high-intensity statins. There were a further 13% who received a low/medium dose and 8% received no statin.

Those dispensed the maximal 80mg atorvastatin rather than another high-intensity dose were more likely to be younger, be men, be non-European, be current smokers and have an MI rather than unstable angina. They also had higher mean LDL-C, better renal function, more obstructive coronary artery disease and were more likely to have had coronary revascularisation by PCI or CABG. Similar variables differentiated those dispensed 80mg atorvastatin compared with a low/medium dose and no statin although the differences were more marked. Eigh percent of patients were discharged on a medication with the potential to interact with atorvastatin and although they were less likely than others to receive the 80mg dose nearly a quarter of these patients (n=384) were on the 80mg dose.

Who gets 80mg atorvastatin early post discharge? (Table 2, Figure 1)

On multivariable analysis, those who received the maximum 80mg dose of atorvastatin, as opposed to lower doses of statin, were more likely to be younger, male, smoke and have no history of CHF, normal renal function and coronary revascularisation. They were more likely to have been already on statin therapy and to have higher LDL-C cholesterol. Co-prescription of medications with known interactions with statin was also associated with lower use of 80mg atorvastatin.

There was wide variation in the use of the guidelines recommended atorvastatin 80mg dose between DHBs, with use ranging from 15% to 65% of patients. Even after adjustment for covariates, the RR for receiving 80mg statin ranged from as low as 0.29 (95% CIs, 0.22–0.39) to 1.05 (95% CIs, 0.96–1.15).

Dispensing of statin at one year post-discharge (See Table 3, Figure 2)

By one year after discharge, 625 (3.2%) of the study cohort had died leaving 19,242 in the one-year dispensing cohort. In the final quarter of the year post-discharge, 29.2% were dispensed atorvastatin 80mg, 35.7% "other high dose", 14.1% a low/medium dose and 21.1% were not dispensed a statin. 70.5% of those discharged with atorvastatin 80mg continued that dose at one year. Only 14.2% of those initially on 80mg atorvastatin had a dose reduction. A similar number of patients initially dispensed atorvastatin 80mg and other high dose received no statin at one year. Only a small proportion of patients (6.2%) had statins started or dose up-titrated post-discharge.

Multivariable model predictors of high-dose statin maintenance and statin discontinuation by one year. (Appendix Table 1,2)

Table 1: Characteristics according to statin dispensing.

	Overall, n (col %) (N=19,867)	Statin dispensed within three months post*discharge, n (row %)				P-value		
		High intensity (n=15,647, 79%)		Low/medium intensity (n=2,640, 13%)	No statin dispensed (n=1,580, 8%)			
		Atorvastatin 80mg (n=7,085, 36%)	Other high intensity (n=8,562, 43%)			Ator 80mg vs other high intensity	Ator 80mg vs low/ medium intensity	Ator 80mg vs no statin
Type of statin dispensed early post-discharge						-	-	-
Atorvastatin	17,103 (86.1)	7,085 (41.4)	8,509 (49.8)	1,509 (8.8)	0 (0)			
Simvastatin	765 (3.9)	0 (0)	53 (6.9)	712 (93.1)	0 (0)			
Pravastatin	419 (2.1)	0 (0)	0 (0)	419 (100)	0 (0)			
No statin dispensed	1,580 (8.0)	0 (0)	0 (0)	0 (0)	1,580 (100)			
Age (years)						<.01	<.01	<.01
Mean (SD)	65.3 (11.7)	61.9 (11.1)	66.3 (11.5)	69.6 (10.9)	67.5 (12.9)			
Age (years)						<.01	<.01	<.01
<55	3,848 (19.4)	1,901 (49.4)	1,428 (37.1)	255 (6.6)	264 (6.9)			
55–<70	8,325 (41.9)	3,285 (39.5)	3,549 (42.6)	949 (11.4)	542 (6.5)			
70–<80	5,397 (27.2)	1,563 (29.0)	2,427 (45.0)	926 (17.2)	481 (8.9)			
80+	2,297 (11.6)	336 (14.6)	1,158 (50.4)	510 (22.2)	293 (12.8)			
Sex						<.01	<.01	<.01
Male	13,795 (69.4)	5,382 (39.0)	5,904 (42.8)	1,647 (11.9)	862 (6.2)			
Female	6,072 (30.6)	1,703 (28.0)	2,658 (43.8)	993 (16.4)	718 (11.8)			
Ethnicity						<.01	<.01	<.01
Māori	2,187 (11.0)	852 (39.0)	951 (43.5)	224 (10.2)	160 (7.3)			
Pacific	905 (4.6)	458 (50.6)	296 (32.7)	95 (10.6)	55 (6.1)			
Indian	876 (4.4)	485 (55.4)	262 (29.9)	92 (10.5)	37 (4.2)			
Other Asian	554 (2.8)	245 (44.2)	200 (36.1)	76 (13.7)	33 (6.0)			
European/other	15,345 (77.2)	5,045 (32.9)	6,853 (44.7)	2,152 (14.0)	1,295 (8.4)			
NZDep01						<.01	0.01	<.01
Least deprived (1–3)	5,000 (25.2)	1,764 (35.3)	2,155 (43.1)	657 (13.1)	424 (8.5)			
Intermediate (4–7)	8,416 (42.4)	2,865 (34.0)	3,714 (44.1)	1,153 (13.7)	684 (8.1)			
Most deprived (8–10)	6,374 (32.1)	2,423 (38.0)	2,673 (41.9)	822 (12.9)	456 (7.2)			
Missing	77 (0.4)	33 (42.9)	20 (26.0)	8 (10.4)	16 (20.8)			
NZDep13						<.01	0.01	<.01
Least deprived (1–3)	4,941 (24.9)	1,760 (35.6)	2,107 (42.6)	653 (13.2)	421 (8.5)			
Intermediate (4–7)	7,919 (39.9)	2,748 (34.7)	3,465 (43.8)	1,073 (13.6)	633 (8.0)			
Most deprived (8–10)	6,948 (35.0)	2,552 (36.7)	2,975 (42.8)	908 (13.1)	513 (7.4)			
Missing	59 (0.3)	25 (42.4)	20 (25.4)	6 (10.2)	13 (22.0)			

Table 1: Characteristics according to statin dispensing (continued).

	Overall, n (col %) (N=19,867)	Statin dispensed within three months post discharge, n (row %)				P-value		
		High intensity (n=15,647, 79%)		Low/medium intensity (n=2,640, 13%)	No statin dispensed (n=1,580, 8%)			
		Atorvastatin 80mg (n=7,085, 36%)	Other high intensity (n=8,562, 43%)			Ator 80mg vs other high intensity	Ator 80mg vs low/ medium intensity	Ator 80mg vs no statin
Statin dispensed prior to ACS admission						<.01	<.01	<.01
Atorvastatin 80mg	1,035 (5.2)	901 (87.1)	88 (8.5)	10 (1.0)	36 (3.5)			
Other high dose	2,656 (13.4)	718 (27.0)	1,799 (67.7)	69 (2.6)	70 (2.6)			
Low/medium dose	4,453 (22.4)	1,198 (26.9)	1,641 (36.9)	1,500 (33.7)	114 (2.6)			
No	11,723 (59.0)	4,268 (36.4)	5,034 (42.9)	1061 (9.1)	1,360 (11.6)			
LDL-C, n (%) mmol/L						<.01	<.01	0.01
<2	4,346 (21.9)	1,484 (34.1)	1,859 (42.8)	749 (17.2)	254 (5.8)			
2–<3	5,767 (29.0)	2,088 (36.2)	2,442 (42.3)	778 (13.5)	459 (8.0)			
≥3	7,638 (38.4)	3,016 (39.5)	3,203 (41.9)	772 (10.1)	647 (8.5)			
Missing	2,116 (10.7)	497 (23.5)	1,058 (50.0)	341 (16.1)	220 (10.4)			
LDL-C (with prior statin) mmol/L						<.01	<.01	<.01
<2	3,247 (39.9)	1,110 (34.2)	1,431 (44.1)	645 (19.9)	61 (1.9)			
2–<3	815 (32.5)	958 (36.2)	1,134 (42.9)	493 (18.6)	59 (2.2)			
≥3	398 (15.7)	513 (40.2)	478 (37.5)	216 (16.9)	68 (5.3)			
Missing	978 (12.0)	236 (24.1)	485 (49.6)	225 (3.3)	32 (3.3)			
LDL-C (without prior statin) mmol/L						0.42	0.09	<.01
<2	1,099 (9.4)	374 (34.0)	428 (38.9)	104 (9.5)	193 (17.6)			
2–<3	3,123 (26.6)	1,130 (36.2)	1,308 (41.9)	285 (9.1)	400 (12.8)			
≥3	6,363 (54.3)	2,503 (39.3)	2,725 (42.8)	556 (8.7)	579 (9.1)			
Missing	1,138 (9.7)	261 (22.9)	573 (50.4)	116 (10.2)	188 (16.5)			
eGFR ml/min1.732						<.01	<.01	<.01
0–<30	606 (3.1)	118 (19.5)	257 (42.4)	155 (25.6)	76 (12.5)			
30–<60	4,735 (23.8)	1,297 (27.4)	2,152 (45.4)	822 (17.4)	464 (9.8)			
60+	14,525 (73.1)	5,669 (39.0)	6,153 (42.4)	1,663 (11.4)	1,040 (7.2)			
Missing	1 (0.01)	1 (100)	0 (0)	0 (0)	0 (0)			

Table 1: Characteristics according to statin dispensing (continued).

	Overall, n (col %) (N=19,867)	Statin dispensed within three months post discharge, n (row %)				P-value		
		High intensity (n=15,647, 79%)		Low/medium intensity (n=2,640, 13%)	No statin dispensed (n=1,580, 8%)			
		Atorvastatin 80mg (n=7,085, 36%)	Other high intensity (n=8,562, 43%)			Ator 80mg vs other high intensity	Ator 80mg vs low/ medium intensity	Ator 80mg vs no statin
Current smoker	3,893 (19.6)	1,781 (45.7)	1,610 (41.4)	309 (7.9)	193 (5.0)	<.01	<.01	<.01
Diabetes	4,609 (23.2)	1,663 (36.1)	1,846 (40.1)	769 (16.7)	330 (7.2)	<.01	<.01	0.03
Initial Killip Class I II–IV	 18,146 (91.3) 1,721 (8.7)	 6,675 (36.2) 510 (29.6)	 7,797 (43.0) 765 (44.5)	 2,361 (13.0) 279 (16.2)	 1,413 (7.8) 167 (9.7)	<.01	<.01	<.01
Type of ACS STEMI NSTEMI Unstable angina	 4,973 (25.0) 11,471 (57.7) 3,423 (17.2)	 2,281 (45.9) 3,943 (34.4) 861 (25.2)	 2,061 (41.4) 5,002 (43.6) 1,499 (43.8)	 389 (7.8) 1,571 (13.7) 680 (19.9)	 242 (4.9) 955 (8.3) 383 (11.2)	<.01	<.01	<.01
History of CVD	7,033 (35.4)	2,087 (29.7)	2,849 (40.5)	1,324 (18.8)	773 (11.0)	<.01	<.01	<.01
History of CHF	738 (3.7)	158 (21.4)	318 (43.1)	163 (22.1)	99 (13.4)	<.01	<.01	<.01
COPD	1,809 (9.1)	504 (27.9)	803 (44.4)	309 (17.1)	193 (10.7)	<.01	<.01	<.01
GRACE risk score <1% 1–<3% ≥3% Missing	 5,318 (26.8) 8,051 (40.5) 6,494 (32.7) 4 (0.02)	 2,092 (39.3) 2,946 (36.6) 2,045 (31.5) 2 (50.0)	 2,262 (42.5) 3,366 (41.8) 2,933 (45.2) 1 (25.0)	 579 (10.9) 1,096 (13.6) 964 (14.8) 1 (25.0)	 385 (7.2) 643 (8.0) 552 (8.5) 0 (0)	<.01	<.01	<.01
CAD >50% on angiogram No obstructive CAD Single/double VD Three VD and/or LMS >50%	 2,722 (13.7) 11,448 (57.6) 5,696 (28.7)	 677 (24.9) 4,448 (38.9) 1,960 (34.4)	 1,061 (39.0) 4,999 (43.7) 2,502 (43.9)	 471 (17.3) 1,341 (11.7) 828 (14.5)	 513 (18.8) 661 (5.8) 406 (7.1)	0.02	<.01	<.01
Coronary procedure PCI CABG Medical management	 11,538 (58.1) 2,424 (12.2) 5,905 (29.7)	 4,695 (40.7) 796 (32.8) 1,594 (27.0)	 4,863 (42.1) 1,245 (51.4) 2,454 (41.6)	 1,312 (11.4) 282 (11.6) 1,046 (17.7)	 668 (5.8) 101 (4.2) 811 (13.7)	<.01	<.01	<.01
Concomitant discharge medications with interaction potential	1,639 (8.3)	384 (23.4)	695 (42.4)	342 (20.9)	218 (13.3)	<.01	<.01	<.01

ACS = Acute coronary syndrome; CABG = coronary artery bypass grafting; CAD = Coronary Artery Disease; CHF = Coronary Heart Failure; COPD = chronic obstructed pulmonary disease; CVD = Cardiovascular disease; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein-cholesterol; LMS = Left main STEMI; NSTEMI = Non ST elevation myocardial infarction; PCI = percutaneous coronary intervention; STEMI = ST elevation myocardial Infarction; VD = Vessel disease.

Table 2: Multivariable model for 80mg atorvastatin at discharge vs all other statin type and dose.

	Relative risk (95% CI)	**P-value**
Age (per 10 years increase)	0.85 (0.83–0.86)	<.01
Sex		
Male	1.18 (1.13–1.23)	<.01
Female	Reference	-
Ethnicity		
Māori	1.03 (0.97–1.09)	0.35
Pacific	1.00 (0.94–1.07)	0.96
Indian	0.98 (0.92–1.05)	0.64
Other Asian	0.94 (0.86–1.02)	0.15
Euro/Other	Reference	-
Current smoker		
Yes	1.08 (1.03–1.12)	<.01
No	Reference	-
Diabetes		
Yes	1.01 (0.97–1.06)	0.64
No	Reference	-
Prior CVD		
Yes	0.97 (0.93–1.01)	0.13
No	Reference	-
History of CHF		
Yes	0.81 (0.71–0.93)	<.01
No	Reference	-
Statin three months prior to admission		
Yes	1.05 (1.01–1.10)	0.03
No	Reference	-
LDL-C (mmol/L)		
<2	Reference	-
2–<3	1.05 (1.00–1.11)	0.05
3+	1.08 (1.03–1.14)	<.01
eGFR (ml/min/1.73m2)		
0–<30	Reference	-
30–<60	1.67 (1.41–1.98)	<.01
60+	1.77 (1.50–2.09)	<.01
Revascularisation		
Yes	1.22 (1.17–1.28)	<.01
No	Reference	-
Common concomitant medications		
Yes	0.83 (0.76–0.90)	<.01
No	Reference	-

Table 2: Multivariable model for 80mg atorvastatin at discharge vs all other statin type and dose (continued).

District health board		
Counties Manukau	Reference	
Northland	1.05 (0.96–1.15)	0.28
Auckland	1.03 (0.98–1.08)	0.22
Waitemata	0.71 (0.65–0.78)	<.01
Bay of Plenty	0.38 (0.33–0.44)	<.01
Lakes	0.52 (0.45–0.61)	<.01
Tairawhiti	0.62 (0.51–0.75)	<.01
Taranaki	0.29 (0.23–0.36)	<.01
Waikato	0.41 (0.37–0.46)	<.01
Capital and Coast	0.34 (0.29–0.40)	<.01
Hawke's Bay	0.33 (0.28–0.40)	<.01
Hutt Valley	0.29 (0.24–0.36)	<.01
MidCentral	0.60 (0.53–0.69)	<.01
Nelson Marlborough	0.93 (0.84–1.02)	0.13
Whanganui	0.29 (0.22–0.39)	<.01
Wairarapa	0.40 (0.30–0.52)	<.01
Canterbury	0.52 (0.47–0.57)	<.01
South Canterbury	0.80 (0.68–0.94)	0.01
Southern	0.43 (0.38–0.48)	<.01
West Coast	0.53 (0.41–0.68)	<.01

CHF = Congestive heart failure; CVD = Cardiovascular disease; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein-cholesterol.

Figure 1: Percentage of patients who receive high-dose statin at discharge in each district health board area. The high-intensity statin is subdivided into 80mg atorvastatin (dark bar) and other high intensity (lighter bar).

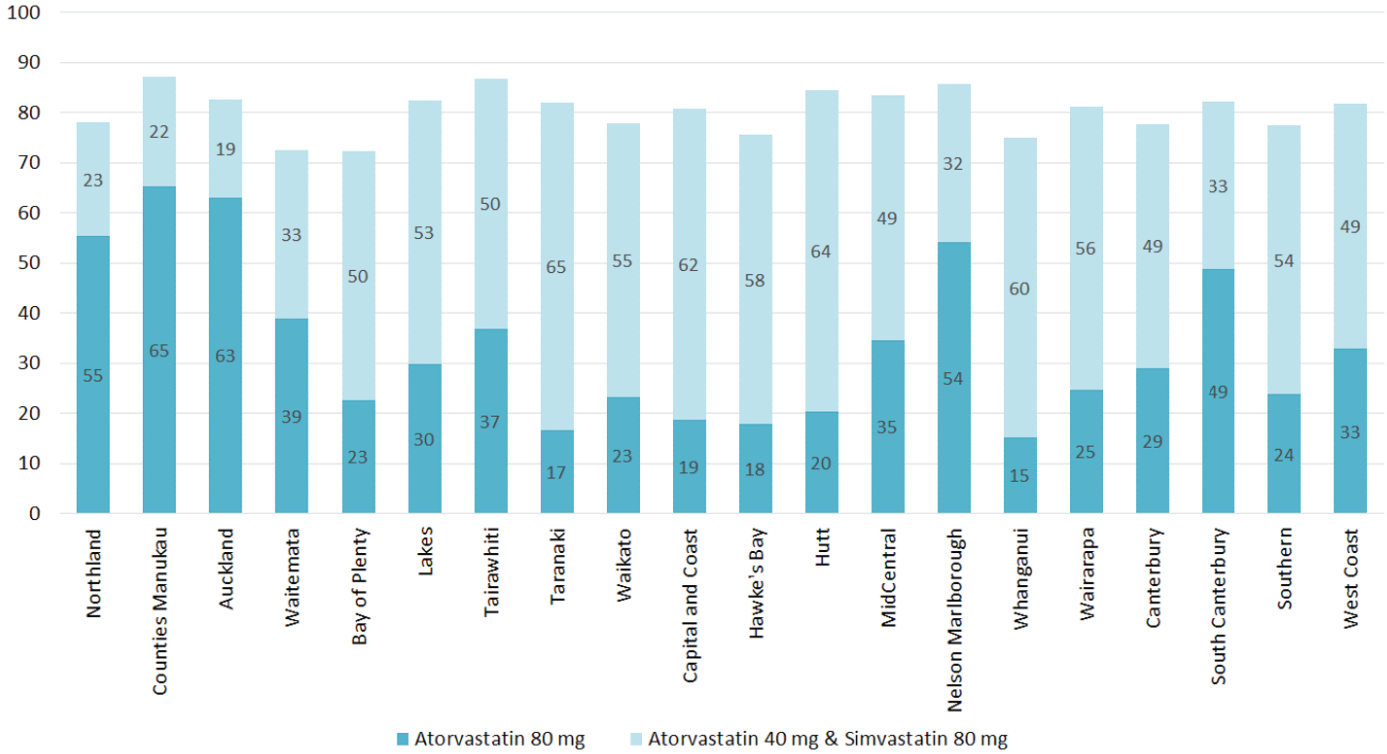


Table 3: Comparison of early vs one-year statin dispensing.

Early statin dispensed	Late statin dispensed				
	Atorvastatin 80mg (%)	Other high dose 80mg (%)	Low/medium dose 80mg (%)	No statin 80mg (%)	Total
Atorvastatin 80mg	4,893 (70.5)	660 (9.5)	328 (4.7)	1,055 (15.2)	6,936 (36.0)
Other high dose	602 (7.3)	5,837 (70.4)	576 (6.9)	1,279 (15.4)	8,294 (43.1)
Low/medium dose	66 (2.6)	287 (11.4)	1,705 (67.7)	461 (18.3)	2,519 (13.1)
No statin	50 (3.3)	76 (5.1)	106 (7.1)	1,261 (84.5)	1,493 (7.8)
Total	5,611 (29.2)	6,860 (35.7)	2,715 (14.1)	4,056 (21.1)	19,242 (100)

Other high dose = atorvastatin 40mg and simvastatin 80mg; low/medium dose = other doses of statins.

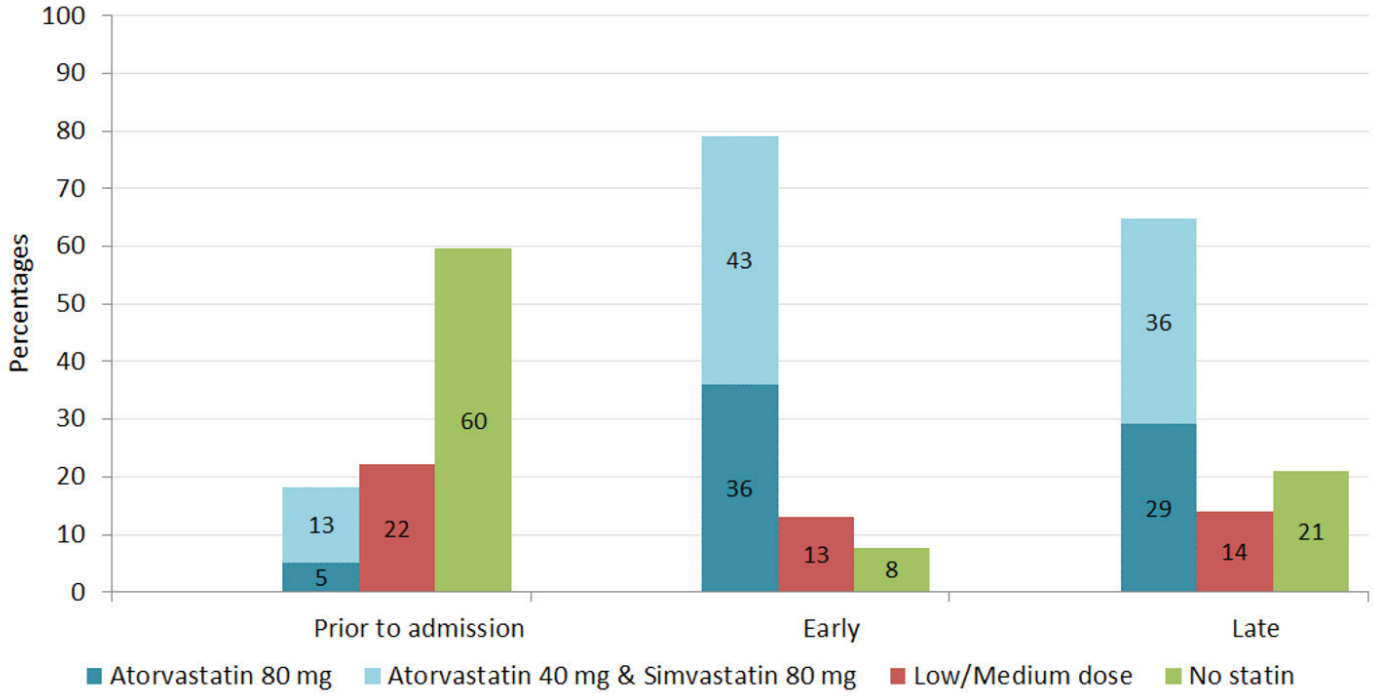
Who receives 80mg of atorvastatin by 1 year? (see Appendix Table 1)

On multivariable regression the strongest predictor of being dispensed 80mg of atorvastatin at one year was being discharged on that dose. Other variables independently associated with receiving 80mg of atorvastatin at one year were younger age, male sex, smoking at admission, being previously on a statin, undergoing revascularisation and having an eGFR ≥30ml/min/1.73m^2.

Who discontinues statins? (Appendix Table 2)

After adjustment, the variables associated with statin discontinuation in those initially receiving any dose of statin at discharge were younger age, female sex, not having diabetes, prior CVD, no prior statin, initial low/medium dose, not receiving coronary revascularisation, eGFR<30ml/min/1.73m^2 and a high baseline LDL-C. The risk of discontinuation did not differ for those started on atorvastatin 80mg compared with "other high-dose" statins.

Figure 2: The distribution of statin dose prior to the index Acute Coronary Syndrome admission, early post-discharge and by one year post-discharge.



Low/medium dose = other doses of statins.

Discussion

This study captured over 95% of New Zealand patients discharged after ACS in New Zealand who were actively managed with coronary angiography with or without revascularisation. A key pillar of management for these patients is optimal LDL-C management. We found that 79% were discharged with a high-intensity statin, although only 36% received the maximum, guidelines recommended 80mg dose of atorvastatin. By one year only 14% of those initially on 80mg were down-titrated to a lower dose, and the most important predictor of being on the maximum dose was being discharged on that dose. One in five patients were not dispensed a statin in the last three months of the year post-discharge. After adjustment, an initial 80mg atorvastatin dose and "other high dose" were both associated with a similar and lower risk of discontinuation compared with a low/medium-dose statin. In contrast to the low down-titration rates observed, only a small proportion of patients (6%) initially dispensed a dose less than the maximum atorvastatin dose or no statin had it up-titrated or commenced. Although the range across DHBs in use of high-intensity statin was modest at 73% to 87%, there was a much greater variation, from 15% to 65%, in the use of the 80mg atorvastatin dose. This variation persisted after adjustment for covariates suggesting that much of the variation may be due to local clinical preference.

Comparison with prior studies

The New Zealand utilisation of high-intensity statins is higher than in older registry data for ACS patients in 2003 and 2009 from the US, but this gap is not as large in more recent international reports. Those papers do not report the proportion on 80mg atorvastatin separately. Using the similar definitions for high-intensity statins, the older studies reported rates of prescribing or dispensing of high-intensity statin post ACS of approximately between only 23% and 38%.[33–35] As in our study up-titration of doses post-discharge was also infrequent, with the discharge dose strongly associated with dose at one year.[34] In a large US study of MI patients in 2014, 72% of those <65y and 57% of those over 65y were discharged on high-intensity statin.[36] In a single centre in France in 2016, 61% of post-PCI patients were treated with high-intensity statin.[37] We were also able to investigate the relationship between initial dose and discontinuation rates, and found no difference in discontinuation between the highest and "other high" doses. The highest rate of discontinuation was in those patients discharged on low/medium doses. This may reflect the characteristics of patients likely to receive a lower dose.

Atorvastatin 80mg vs other high dose—does it matter?

In the CTT meta-analysis,[4] compared with less intensive regimens, more intensive regimens produced a highly significant 15% ($p<0.0001$) further reduction in major vascular events, consisting of significant reductions in coronary death or non-fatal myocardial infarction of 13% ($p<0.0001$), in coronary revascularisation of 19% ($p<0.0001$), and in ischaemic stroke of 16% ($p=0.005$). For each 1mmol/L reduction in LDL-C, total mortality was reduced by 10% over five years. Statins may also have anti-inflammatory and antioxidant effects beyond the effects of LDL-C lowering that may reduce events. There are no outcome studies, which randomised patients to receive atorvastatin 80mg vs 40mg. There are patients in whom it is clinically appropriate to use lower doses, particularly the very elderly (over 80 years), those with limited life expectancy and those on other medications known to interact with statins. Nevertheless the observed DHB variability after adjustment for covariates suggests that the choice of 40mg vs 80mg is strongly influenced by local clinical preference. By doubling the dose of a statin (atorvastatin 40mg to 80mg) it is expected that there will be a further 6% fall in LDL-C,[35,38,39] and based on the well-established relationship between LDL-C lowering and outcomes greater use of 80mg dose would be expected to further improve outcomes.[4]

There may be an additional benefit of 80mg atorvastatin in patients undergoing PCI. In the recent Statins Evaluation in Coronary Procedures and Revascularization (SECUREPCI) Trial randomised, placebo-controlled trial, which assessed the impact of peri-procedural loading with atorvastatin [two loading doses of 80mg, before and 24h after planned PCI] at 30 days in 4,191 patients with ACS.[40] All patients received atorvastatin 40mg per day starting 24h

after the second loading dose. The authors found no significant treatment benefit in the overall study population but there was a significant 28% relative risk reduction in a composite of all-cause mortality, myocardial infarction, stroke and unplanned coronary revascularisation in patients who underwent PCI. The benefit was even more pronounced in STEMI patients undergoing primary PCI. These benefits will be offset by even a small increase in side effects if used inappropriately in patients at increased risk for side effects.[14]

Are we choosing the right patients for atorvastatin 80mg?

In our study there was no evidence, after adjustment for covariates including DHB of domicile, that patients were more likely to require a dose reduction after commencing 80mg compared with 40mg atorvastatin. In addition the 80mg dose was less likely to be used in patients co-administered a drug with a risk of drug-drug interaction. However, we have no more specific information about adverse side effects. It is noteworthy that there were significant numbers of patients on high-intensity statins in this cohort in whom guidelines recommend caution. This includes the very elderly where side effects may occur from over medication, those with severe chronic kidney and hepatic disease and patients on essential concomitant medications with a risk of drug-drug interaction. In the current study, 15% of those over 80 years, and nearly a quarter of the 8% of patients on concomitant medications with risk of drug interactions received the atorvastatin 80mg dose. Conversely, there were many patients without obvious reasons for caution who could potentially have been treated with a higher dose of statin.

Clinical implications

In the year post-ACS discharge, patients usually continue on the dose of statin they were discharged with, but nearly one in five patients initially dispensed statin were not dispensed a statin in the last three months of the year post-discharge. Because the maximum dispensing period in New Zealand is three months, this finding suggests sub-optimal maintenance or discontinuation of medication for those patients. It is therefore important that both the optimal dose be chosen for each patient in-hospital, and that patients then be supported to continue with their medication.

Choice of the appropriate statin dose is usually made in-hospital by the medical team, although in some cases, such as prior intolerance or abnormal liver function tests, up-titration as tolerated, from an initial low dose, may be done by the secondary and/or primary care teams after discharge. Supporting longer-term maintenance in ACS patients begins during the hospital admission and is supported by in-hospital and early post-discharge cardiac rehabilitation. In New Zealand it then continues under the supervision of the primary care team. Commencement and continuation of statins and other medications of proven clinical benefit which have been started in hospital requires optimal performance across this care continuum. Important components include:

1. Medical staff having access to evidence-based recommendations for medications and doses. In New Zealand initial medications post-ACS are often prescribed by junior medical staff who rotate through the coronary care unit and are under the supervision of multiple different cardiologists, which can result in variation in practice. Each unit should have a locally endorsed medication guideline which is regularly updated according to evolving evidence, and which is used by all medical team members. It should be based on national and international guidelines and give specific advice regarding recommended medications and doses which takes into account patient-specific factors including age, liver and renal function and concomitant medications. Although for many units this will be a paper-based guideline there is an increasing opportunity to incorporate this content within electronic prescribing systems, which can include a default list of medications and doses for each condition and provide advice/alerts as appropriate.
2. Involvement of the medical, nursing, cardiac rehabilitation team and pharmacists while the patient is in hospital, to both educate and support patients and their families regarding the benefits and potential risk of medications. The non-medical health professionals, if enabled to do so, also provide an important cross-check on

medical prescribing decisions. Several New Zealand cardiology units now utilise the patient oriented "My Heart Recovery Plan". This was developed in 2019 by the Health Quality and Safety Commission and ANZACS-QI, and it is endorsed for use nationally by the National Cardiac Network. It is a checklist designed to be used interactively by patients and their in-hospital medical, nursing and pharmacy team to support their understanding of their medications, the importance of continuing them long-term, whether they have been invited to cardiac rehabilitation, whether a follow-up appointment is arranged with their general practitioner and whether they need psychological support post-discharge. More widespread uptake and systematic implementation of this tool should be considered.

3. Practical considerations include simplifying the dosing regimen and encouraging patients to have their medications blister packed or a similar process which has been shown to improve adherence.[41] Financial barriers do have an impact on adherence and these should be addressed.[41] The default change per item dispensed in New Zealand is $5. Although there are programmes to reduce this for high-needs populations, our experience is that there are still patients who defer medications because of cost considerations.
4. Facilitation of referral to culturally appropriate cardiac rehabilitation programs after discharge to continue to educate and support patients and facilitate the transition to primary care.[42]
5. Primary care based self-monitoring and self-management programmes have proven effectiveness to improve medication adherence use, and utilising technology to ensure re-prescription of medication continues after discharge is a promising approach.[41]
6. Publication of data is an important way to feedback to clinicians and modify prescribing behavior.[43] In particular, publication of the wide variation in use of the atorvastatin 80mg dose between DHBs is expected to prompt units to review their local guidelines. ANZACS-QI will continue to report back to the DHBs on high-intensity statin use as part of its annual Post-ACS Statin Adherence report to allow progress to be tracked.

Limitations

We used dispensing of statin as a marker of maintenance but not everyone who is dispensed a drug routinely is necessarily taking the medication. We had no other measures of adherence, nor did we have information about patient preferences. In some cases patients may be wary of high doses of a statin but accept a lower dose. Achieved LDL-C post discharge was not available for this study. The cohort comprised those patients who underwent invasive coronary angiography for whom we would expect intensive secondary prevention including high-intensity statins to be appropriate. The ANZACS-QI registry has very high rates of capture for these patients across New Zealand.[44] Approximately 40% of patients with ACS do not receive coronary angiography and are not included in this analysis.[45] However, of younger ACS patients between 60 and 70y, 70% receive an angiogram, and of those under 60y this increases to 80%.[46] It is likely that use of high-intensity statin would be lower in those not receiving an angiogram given the high burden of comorbidity previously documented in such patients.[45,47] No outcome data are presented and apart from medication discontinuation we cannot identify statin-specific side effects.

Conclusion

Only 36% of ACS patients who received invasive investigation post-ACS received guideline-recommended dose of 80mg atorvastatin. The dose of statin at hospital discharge is the most important determinant of dose by one year. The atorvastatin 80mg dose was not associated with higher discontinuation rates. Further optimisation of dosage at discharge by the secondary care team is feasible in New Zealand, for many clinically appropriate patients, and is an opportunity to achieve better long-term LDL-C reduction and thus improved clinical outcomes. GPs and primary care teams should have a greater role in medication up-titration, and systems need to be developed to ensure regular re-prescription of medication.

Appendix

Appendix Table 1: Multivariable regression analysis: variables associated with being dispensed 80mg atorvastatin at one year.

	Relative risk (95% CI)	P-value
Age (per 10 years increase)	0.94 (0.93–0.96)	<.01
Sex		
Male	1.11 (1.06–1.16)	<.01
Female	Reference	-
Ethnicity		
Māori	1.04 (0.99–1.10)	0.15
Pacific	1.00 (0.94–1.07)	0.97
Indian	0.96 (0.91–1.03)	0.24
Other Asian	0.86 (0.78–0.95)	<.01
Euro/Other	Reference	-
Current smoker		
Yes	1.07 (1.03–1.12)	<.01
No	Reference	-
Diabetes		
Yes	1.09 (1.04–1.13)	<.01
No	Reference	-
Prior CVD		
Yes	0.95 (0.91–0.99)	0.02
No	Reference	-
History of CHF		
Yes	1.05 (0.94–1.19)	0.38
No	Reference	-
Statin three months prior to admission		
Yes	1.32 (1.26–1.37)	<.01
No	Reference	-
Statin three months post D/C		
Atorvastatin 80mg	Reference	-
Atorvastatin 40mg and Simvastatin 80mg	0.11 (0.10–0.12)	<.01
Low/medium dose	0.04 (0.03–0.05)	<.01
No statin	0.06 (0.05–0.08)	<.01
LDL mmol/L		
<2	Reference	-
2–<3	1.01 (0.97–1.06)	0.58
3+	1.03 (0.98–1.08)	0.27

Appendix Table 1: Multivariable regression analysis: variables associated with being dispensed 80mg atorvastatin at one year (continued).

eGFR (ml/min/1.73m^2)		
0–<30	Reference	-
30–<60	1.15 (1.00–1.33)	0.05
60+	1.17 (1.02–1.35)	0.03
Revascularisation		
Yes	1.19 (1.13–1.24)	<.01
No	Reference	-
Common concomitant medications		
Yes	0.98 (0.91–1.06)	0.66
No	Reference	-

CHF = Congestive heart failure; CVD = Cardiovascular disease; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein-cholesterol.

Appendix Table 2: Multivariable regression analysis: variables associated with statin discontinuation by one year in patients initially dispensed statin.

	Relative risk (95% CI)	**P-value**
Age (per 10 years increase)	0.94 (0.90–0.97)	<.01
Sex		
Male	0.93 (0.86–1.00)	0.05
Female	Reference	-
Ethnicity		
Māori	1.09 (0.97–1.22)	0.13
Pacific	1.05 (0.88–1.26)	0.57
Indian	1.20 (1.00–1.44)	0.05
Other Asian	1.19 (0.98–1.46)	0.09
Euro/Other	Reference	-
Current smoker		
Yes	1.02 (0.93–1.11)	0.71
No	Reference	-
Diabetes		
Yes	0.84 (0.75–0.93)	<.01
No	Reference	-
Prior CVD		
Yes	1.31 (1.20–1.44)	<.01
No	Reference	-
History of CHF		
Yes	0.68 (0.52–0.90)	0.01
No	Reference	-
Statin 3 months prior to admission		
Yes	0.44 (0.39–0.49)	<.01
No	Reference	-

Appendix Table 2: Multivariable regression analysis: variables associated with statin discontinuation by one year in patients initially dispensed statin (continued).

Statin 3 months post D/C		
Atorvastatin 80mg	0.69 (0.62–0.77)	<.01
Other high dose	0.77 (0.69–0.85)	<.01
Low/Medium dose	Reference	-
LDL mmol/L		
<2	Reference	-
2–<3	1.22 (1.08–1.37)	<.01
3+	1.24 (1.10–1.40)	<.02
eGFR ml/min1.73²		
0–<30	Reference	-
30–<60	1.29 (0.95–1.75)	0.10
60+	1.24 (0.92–1.67)	0.15
Revascularisation		
Yes	0.69 (0.64–0.75)	<.01
No	Reference	-
Common concomitant medications		
Yes	0.90 (0.78–1.05)	0.17
No	Reference	-

CHF = Congestive heart failure; CVD = Cardiovascular disease; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein-cholesterol.

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The role and measurement of patient activation in the management of long-term conditions in New Zealand

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ABSTRACT

AIM: Patient activation represents people's knowledge, skills and confidence to manage their own health. We provide information regarding the nature of patient activation and use New Zealand data to consider its utility in New Zealand.

METHODS: Self-report data using the patient activation measure (PAM) and seven health and general practice experience measures were collected from 544 general practice patients in the MidCentral region. PAM scores were used to categorise respondents into four levels of activation. Mean scores were calculated by activation level, separately for Māori (14.9%) and non Māori (85.1%).

RESULTS: Patterns of activation similar to those reported in earlier research were found. More positive health and general practice experience scores were found for those at higher levels of activation for both ethnicities. The magnitudes of the differences by activation level were similar for both groups and overall differences were significant for all variables for non Māori and for three for Māori.

CONCLUSIONS: The PAM behaved as it has done in previous overseas studies with respect to score distribution, reliability and validity. We recommend its use for research and clinical practice in New Zealand to assist with designing appropriate levels of LTC education and self-management support aimed at increasing health engagement.

In this article we highlight the nature and utility of a construct called patient activation. This has been broadly defined as people's knowledge, skills and confidence to manage their own health and healthcare.[1] To date over 500 research studies of patient activation have been published, yet we were able to identify only one New Zealand study.[2] We believe that patient activation is an important construct that can be measured and used to advantage in New Zealand studies and clinical practice—particularly for patients with one or more long-term conditions (LTCs). After outlining why the notion of patient activation is highly relevant to the management of LTCs in New Zealand today, we focus on its measurement using the Patient Activation Measure (PAM). First we summarise some of the extensive international literature on the PAM's reliability and validity, and then report findings from our own study of 544 people with LTCs in the MidCentral DHB region. These findings include information on the PAM's reliability and validity for New Zealand respondents, including how their level of activation relates to their demographic attributes, their health, and to the quality of their experiences with general practice. Results are presented separately for Māori and non Māori participants to enable us to see how the measure works with different ethnicities in Aotearoa New Zealand.

Why is patient activation relevant to long-term condition management?

People living with long-term conditions (LTCs) are increasingly encouraged to improve their knowledge and understanding of their condition/s and engage in self-management activities in order to

maintain or improve their health status and quality of life. Self-management and the self-management support provided by health practitioners are gaining a higher profile due to the ageing population and increased prevalence of comorbidity.[3] Limited consultation time due to practitioner shortages and problems in access, particularly in rural locations, means that people themselves are required to step up. In a recent Listener interview,[4] Wellington GP Jeff Lowe was quoted as saying "the biggest part of the workforce who need to lift their game are patients themselves—we need them to be self-managing far better. We can equip them with the knowledge, data and advice they need, but we do need patients to take ownership of their own health".

However, there is ambiguity regarding what self-management actually means, and Van de Velde and colleagues[5] conducted a concept analysis in an effort to provide an operational definition and to delineate self-management more clearly. They concluded that "self-management is the intrinsically controlled ability of an active, responsible, informed and autonomous individual to live with the medical, role and emotional consequences of his chronic condition(s) in partnership with his social network and the healthcare provider(s)" (p.10). They further propose that self-management incorporates 10 attributes. These include person-oriented—incorporating active involvement, taking responsibility for the care process and coping under adversity, person-environment-oriented—relating to having correct information, an individual approach, reciprocal relationships with providers and openness to social support, and summarising attributes—describing self-management as a lifelong task requiring personal skills such as problem-solving and decision-making.[5]

Patient activation encompasses the person's self-management capabilities and engagement with their own health and healthcare.[6] According to Hibbard and Gilburt, patient activation is similar to but different from other constructs that may be related to self-management behaviours, including self-efficacy and readiness to change.[7] They describe it as a general concept with a broader application than related concepts that tend to focus on a single behavioural outcome such as smoking cessation. This broader approach is appropriate when considering the varied knowledge, skills and tasks involved in self-management of a range of long-term conditions, especially in different combinations. The relevance of patient activation to LTC management should be evident but its application in research and clinical practice requires that it be measurable. This has been achieved through the Patient Activation Measure (PAM) to which we now turn.

The PAM and international evidence

The PAM is a multi-item, self-report measure completed by an individual. The 22-item PAM was initially developed by Hibbard and colleagues using Rasch analysis.[1] This was then abbreviated to the 13-item version now in common use.[8] It is a Guttman style measure with statements representing four hierarchical stages: (1) belief in the importance of taking an active role (eg, 'I am the person responsible for taking care of my health'); (2) confidence and knowledge to act (eg, 'I know what each of my prescribed medications do'); taking action (eg, 'I am confident that I can carry out medical treatments I may need to do at home'); and (4) staying the course (eg, 'I am confident that I can maintain lifestyle changes, like healthy eating and exercising, even during times of stress'). For each statement respondents choose one of five Likert-type response options ranging from 'disagree strongly' (1) to 'agree strongly' (4), or 'not applicable'. Insignia Health, the licencing body, provides a spreadsheet to weight responses and convert them into scores on a 0–100 point scale where a higher score represents greater activation. Mean scores in the 50s,[9–12] 60s[13–18] and 70s[19] have been reported. Individuals' scores are used to assign them to one of four activation levels: Level 1 (0–47); Level 2 (47.1–55.1); Level 3 (55.2–72.4) and Level 4 (72.5–100). In nine studies involving primary care patients with one or more LTCs, the percentage of people at Level 1 ranged from 6.8 to 18.5%, at Level 2 from 13.0 to 29.1%, at Level 3 from 22.0 to 39.8% and at Level 4 from 17.2 to 51.0%.[12–15,17,18,20–22]

Reliability

Internal consistency analyses have generated Cronbach's alphas ranging from .81 to .88.[11,15,18,19,23,24] These alphas are interpretable, as PAM responses are uni-dimensional,[8] and are of reassuring magnitude. Test-retest reliability of r=.85 using a one-week test interval has been reported using a sample of 65 people prior to undergoing elective spine surgery.[25] This appears acceptable given the presumed stability of activation over short time periods. However, the sample was small and the short timeframe is potentially problematic as earlier responses may have been remembered. A few studies have identified changes in PAM scores within the same sample, over longer time periods and without an intervention,[12,14,18] suggesting activation is not a fixed attribute.

Validity

Evidence of construct validity should be supported by finding moderate strength relationships between PAM scores and scores on measures of similar health-related constructs. In this section we consider the relationship between patient activation and two other constructs considered to be similar;[7] self-efficacy and readiness-to-change. Research has identified patient activation to be correlated with various types of self-efficacy in people with: heart-failure (r=.71);[26] cardiac issues (r=.39);[11] multiple sclerosis (r=.50);[15] depression (r-.51)[27] and spinal problems (r=.65).[25] While this is a fairly broad range of correlations, all are in the expected direction and of moderate magnitude. Readiness-to-change with respect to living healthily was measured along with patient activation in a sample of 625 healthcare organisation and airline employees, two-thirds of whom had one or more chronic conditions. Higher PAM scores were found in those who had already made behavioural changes (*M*=73.9) compared to those with no intention to change their behaviour (*M*=62.4).[16] To assist with interpretation of this finding, a 5 point or greater difference in scores is considered to be of clinical significance with respect to changing outcomes.[28]

Evidence of validity has also been found in relation to behaviour, with PAM scores used to identify people engaging in specific health behaviours such as seeking and using health information[9] and self-care.[29] Links have also been made to experiences with health practitioners. Higher PAM scores were associated with: more positive ratings of overall care;[21] better communication with providers, more contact beyond office appointments and fewer care coordination problems;[30] greater satisfaction with care;[13,23] increased involvement in treatment plans;[13,23] and perceptions that providers have a good interpersonal style and spend enough time with patients.[23]

With respect to health, PAM scores are positively associated with self-ratings of general health;[9,11] mental health;[9,16] lower levels of depression;[10,11,17,19,31] better physical health/functioning;[10,16,19,26] and better quality of life.[17,31]

As most studies have been cross-sectional, the relationships between patient activation and health or behavioural variables cannot be interpreted causally. Therefore we cannot be sure whether, for example, activation generates better health or vice versa. However, the predictive validity of patient activation has also been explored and PAM scores have been linked to future changes in ratings of healthcare provision,[21] improved medication adherence,[27] ambulatory care service utilisation and odds of developing another chronic condition.[32] Greene et al[6] found that PAM scores predicted healthy behaviours, clinical outcomes and costs two years later, and changes in activation level were associated with health outcomes and costs, in expected directions.

Intervention studies have found that it is possible to modify patient activation and provide information on how to do so. For example Greene and colleagues identified five LTC management strategies that may enhance activation: emphasising patient rather than clinician ownership of health; partnering with patients to create goals and strategies to solve problems; collaborative decision-making about small, realistic steps towards healthier behaviours; provision of frequent follow-up support; and showing that they care about their patients' well-being.[28] Alegria and colleagues demonstrated an improvement in activation, as well as efficacy in patient-physician interactions, following a relatively brief intervention.[33] In another intervention study PAM scores were compared before and after

attendance at a six-session, condition-specific self-management programme. Mean scores increased from 52.2 to 60.2 with 53.9% improving by ≥4 points.[34]

There is growing evidence that PAM scores are associated with certain demographic characteristics. Although the findings are mixed with respect to sex and age, higher scores are consistently found for people with more education[9–11,14–16,21,26,32] and higher income.[9,10,14,16,24]

Overall the PAM appears to be a widely used instrument, with international evidence of reliability and validity. However, it is not known how well the measure works in New Zealand, particularly with respect to our indigenous population, and that is what we consider next.

The PAM in New Zealand

The rest of this paper focuses on measuring patient activation in 'Talking about Health', a study of people with LTCs based in MidCentral DHB.[35] The study has a longitudinal design but only data from the first of three assessments are used in the analyses reported here.

Method

Following ethics approval from the Health & Disability Ethics Committee (ref. 16/NTA/32) study invitations were sent to all people in the DHB aged 18+ years who had a comprehensive health assessment (CHA) documented during the previous three (Māori/Pasifika) or two (other ethnicities) years (N=2,730). The CHA was part of a LTC package of care meaning that potential participants had at least one LTC and were enrolled with a general practice. Questionnaires were sent out (or made available through SurveyMonkey) on receipt of consent forms. Of the 569 (20.8%) people returning questionnaires, 25 did not have sufficient patient activation data to compute scores and levels, therefore the sample consisted of 544 people with LTCs.

Materials

The questionnaire included measures of patient activation (PAM), overall health, physical and mental health, the effect of LTCs on quality of life, general practice experiences and support provided, and demographics.

Health was measured by a single item general rating of overall health from 1 'poor' to 5 'excellent', and by the physical (GPH) and mental health (GMH) scales of the short form of the Patient-Reported Outcomes Measurement Information System global health questionnaire.[36] Each scale consists of four items, some rescaled and reverse coded according to the scale instructions. The scale developers reported good internal consistency (GPH α=.81; GMH α=.86), and construct validity; correlations between the PROMIS and the EQ-5D, a widely used health-related quality of life measure, were r=.82 (GPH) and r=.61 (GMH).[36]

Effect of LTCs on quality of life was measured with a single rating of 'how much does having one or more long-term conditions affect your quality of life?' using a scale ranging from 0 'no effect' to 10 'very large effect'. This was reverse coded for analysis so that a higher score represents a more positive result in line with the other variables.

General practice experiences (GPE) were assessed in relation to doctors and nurses separately using nine questions from the New Zealand version of the General Practice Assessment Questionnaire.[37] Minor wording changes were made and five additional questions were developed by the study team. The item stem was "when you see the doctor/nurse at your practice, how good are they at …" and items covered various aspects of the consultation including listening, making you feel comfortable during a physical examination, involving you in decisions about your care and spending enough time. Response options ranged from 1 'very poor' to 6 'excellent'. Two of the 14 questions (relating to involvement of family/whānau/fanau in decision making and practitioners learning about social support needs) were rated as not applicable by a number of respondents and the total General Practitioner (GPE:GP) and Nurse (GPE:N) scales were consequently calculated allowing two missing responses.

General practice team support was rated with the question "how good is the care and support you get for managing your long term conditions from the doctors and nurses at your general practice?" measured on a 0 'not at all good' to 10 'extremely good' scale.

Demographics included sex, age (measured in 10 year increments), ethnicity (more than one was allowed and anyone identifying as Māori or Māori and one or more ethnicities was coded as Māori) and income adequacy ("how well does your total household income meet your everyday needs for such things as accommodation, food, clothing and other necessities" with response options of 'not enough', 'just enough', 'enough' and 'more than enough').

SPSS Statistics 20 was used for analysis. Descriptive statistics were used to describe means and percentages and analysis of variance (ANOVA) was used to test the significance of observed mean differences according to level of activation.

Results

Participant details are provided in Table 1.

A similar distribution of the sexes and educational level was found for Māori and non Māori. However, Māori participants were younger, in line with the demographic profile for the Māori population and rates of mortality, and reported their income to be less adequate overall.

Descriptive information about PAM scores and levels is provided in Table 2. The score ranges used to define the four levels of activation are reiterated in the table.

The mean scores were similar for Māori and non Māori with the main difference being apparent in the percentage of participants at levels 2 and 3. Cronbach's alphas for PAM scores were .91 for Māori and .90 for non Māori.

To assess construct validity of the PAM with this sample we compared scores on a range of variables considered relevant

Table 1: Demographics for Māori (n=81) and non Māori (n=463) participants.

Demographic	Māori N (%)	Non Māori N (%)
Sex		
Male	35 (43.2)	202 (43.8)
Female	46 (56.8)	259 (56.2)
Age (years)		
<65	45 (57.7)	112 (24.4)
65–74	26 (33.3)	156 (34.0)
75+	7 (9.0)	191 (41.6)
Education		
No school qualifications	27 (35.1)	166 (36.8)
School qualifications	15 (19.5)	95 (21.1)
Trade/Polytechnic qualification	24 (31.2)	121 (26.8)
University qualification	11 (14.3)	69 (15.3)
Income adequacy		
Not enough	22 (27.5)	67 (14.7)
Just enough	27 (33.8)	163 (35.8)
Enough	22 (27.5)	170 (37.4)
More than enough	9 (11.3)	55 (12.1)

Table 2: PAM scores and levels for Māori, non Māori and the total sample.

PAM score	Māori		Non Māori		Total	
Range	36.8–100		33.0–100		33.0–100	
Mean (SD)	64.2 (15.7)		63.1 (15.7)		63.3 (15.7)	
PAM Level (score)	**Māori N (%)**	**Mean**	**Non Māori N (%)**	**Mean**	**Total N (%)**	**Mean**
1 (0–47)	11 (13.6)	43.1	60 (13.0)	42.9	71 (13.1)	42.9
2 (47.1–55.1)	8 (9.9)	51.3	76 (16.4)	51.1	84 (15.4)	51.1
3 (55.2–72.4)	40 (49.4)	61.0	208 (44.9)	60.8	248 (45.6)	60.8
4 (72.5–100)	22 (27.2)	85.4	119 (25.7)	85.0	141 (25.9)	85.1

to people with LTCs within the context of primary LTC care. Mean health and general practice experience scores by patient activation level are presented in Table 3.

The pattern of means in Table 3 is consistent for non Māori and more or less consistent for Māori across these measures; the higher the level of activation, the more positive the ratings of health and general practice experiences and the smaller the perceived effect of long-term conditions on quality of life. Given the large difference in Māori/non Māori sample sizes, the magnitude and pattern of means was of greater interest than the statistical significance of any differences. However, ANOVAs were run to look at the differences in means across levels of activation for Māori and non Māori separately, using Bonferroni adjusted alpha levels of .004 (.05/14). All were significant for non Māori and three were significant for Maori; general health, mental health and GPT support.

Table 3: Mean ratings of health and general practice experiences for Māori and non Māori across different levels of patient activation.

	PAM levels	Māori means	Non Māori means
General health	1	1.73	2.23
	2	2.57	2.69
	3	2.78	2.83
	4	3.14	3.15
	Total	**2.71**	**2.81**
		F=7.22 p<.001	F=17.74 p<.001
Effect of LTCs on QoL*	1	2.82	2.78
	2	4.50	3.95
	3	3.78	4.62
	4	4.27	4.76
	Total	**3.88**	**4.30**
		F=0.82 p=.486	F=9.15 p<.001

Table 3: Mean ratings of health and general practice experiences for Māori and non Māori across different levels of patient activation (continued).

Physical health	1	35.66	37.14
	2	39.21	40.31
	3	41.98	43.10
	4	45.22	45.82
	Total	**41.75**	**42.60**
		F=4.58 p=.005	F=18.21 p<.001
Mental health	1	37.58	40.75
	2	43.47	44.35
	3	44.70	47.36
	4	47.51	49.77
	Total	**44.38**	**46.64**
		F=5.87 p=.001	F=20.15 p<.001
GP experiences	1	3.90	4.16
	2	4.14	4.35
	3	4.70	4.70
	4	4.81	5.06
	Total	**4.57**	**4.67**
		F=2.28 p=.088	F=14.68 p<.001
Nurse experiences	1	3.86	4.19
	2	4.90	4.53
	3	4.71	4.77
	4	4.88	5.17
	Total	**4.65**	**4.77**
		F=2.27 p=.088	F=17.72 p<.001
General practice team support	1	5.64	6.63
	2	7.71	7.59
	3	8.22	7.97
	4	8.65	8.67
	Total	**7.92**	**7.92**
		F=6.88 p<.001	F=15.06 p<.001

*Reverse coded as described earlier.

Discussion

Self-management, with support from health professionals, whānau and community groups/organisations, is expected of people with long-term conditions. It follows that the more engaged people are in understanding their own conditions, in the decisions made about treatment and in setting realistic goals, the better able they are to have the knowledge and confidence to self-manage on a daily basis. Patient activation appears to represent a set of key attributes of patient

engagement and considerable evidence exists to suggest that the measure of patient activation developed by Hibbard and colleagues,[8] and comprehensively tested by Hibbard and independent researchers, is reliable, valid and practical.

The patient activation data generated by the Talking about Health study suggests that the measure works similarly for New Zealanders with LTCs as it has in other populations. Overall, mean scores were similar to those found in previous research and the distribution of participants across activation levels was also comparable. The largest proportion of both Māori and non Māori participants were categorised as Level 3 as found in previous studies.[9,12,14,15,17,18,20–22,32] With respect to reliability, the level of internal consistency was good for both Māori and non Māori samples with Cronbach's alphas of .91 and .90 respectively. The pattern of mean scores for health and general practice experiences by activation level indicated that a higher level of activation was associated with more positive ratings of health as well as with higher ratings of experiences with doctors/nurses in the general practice setting and support from general practice teams. This provides evidence of the validity of the measure as well as supporting the results of previous studies described earlier.

Although fewer of the ANOVA results were significant for Māori than non Māori, the difference in sample size may account for this since the range and pattern of means across activation levels was generally similar for both groups. Across the health and general practice measures, the most notable differences in scores were between those at activation Levels 1 and 2. This suggests that the people with the lowest ratings of health and experiences with general practice are also the least activated and are consequently less likely to be able to self-manage well or to adopt behaviours that are challenging or require sustained effort.[38] Consequently it is particularly important for these individuals to be identified and receive education and self-management support that is tailored to their level of understanding, current preparedness to engage in a health partnership, and their specific health and social needs. A 'one size fits all' approach is clearly not an appropriate way to partner with people with LTCs[38] and using a measure such as the PAM is a relatively quick and easy way to identify people's activation level and undertake risk stratification in order to guide individualised care planning and self-management support. For example, while people at the lower levels may need encouragement to take ownership of their health and contribute more to care planning and identification of personal goals, people at the higher levels can be supported to further their expertise, or to maintain self-management skills in times of major life stress. In this paper we have focused on level of activation, and there is evidence demonstrating changes in health outcomes and costs concomitant with changes in level.[6] However the individuals' PAM scores are also useful for tracking changes over time and for evaluating the impact of interventions as even a small change in the score can be meaningful.[28] Regardless of level of activation, a collaborative approach that considers 'what matters to' rather than 'what is the matter with' the person should be adopted.[39]

Our study was limited by the small number of Māori participants and further research regarding how well the PAM reflects the Māori worldview would add to our findings and ensure that it is truly has an indigenous application. Other limitations were the convenience sampling, which affected the generalisability of findings, and the use of self-reported data which was consequently prey to the usual biases. However, given how little patient activation research in New Zealand could be found, the current study makes a new contribution to the literature by presenting New Zealand data for Māori and non Māori with LTCs.

More effective long-term condition management is a global priority and given the increasing number of people with long-term conditions, and the rising costs and pressures faced by the health workforce, ways to improve care quality and efficiency are worthy of consideration. The PAM has been adopted by the UK's National Health System as part of its plan for Universal Personalised Care partly as a risk stratification tool.[40] Enhancing patient activation can reduce health inequalities,[7] particularly as people at levels 1 and 2 have been shown to be amenable to increases in activation.

Consequently, although there are licensing costs associated with using the PAM, we encourage health services in Aotearoa New Zealand to consider this practical way to identify people's level of engagement with their own health. Services can then be designed, in a culturally appropriate way, to meet the requirements of people at each level of activation with the aim of increasing or maintaining their self-management skills, knowledge and confidence—essential steps to reach the goal for all New Zealanders to "live well, stay well, and get well".[3]

Competing interests:
Nil.

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Incidence of venous thromboembolism after total hip, total knee and hip fracture surgery at Waitemata District Health Board following a peer-reviewed audit

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ABSTRACT

AIM: The incidence of venous thromboembolism (VTE) following arthroplasty and hip fracture surgery remains an important metric for quality and financial reasons. An audit at our institution between 2006–2010 showed a higher VTE rate than international data did at the time. This study aims to determine rates of DVT and PE in patients undergoing hip and knee arthroplasty and hip fracture surgery at Waitemata District Health Board (Waitemata DHB) between 1 January 2013 and 31 December 2016.

METHODS: This study is a retrospective review of all VTE within three months of elective hip or knee replacement or hip fracture surgery. Data were identified for the period between 2013 and 2016 from Waitemata DHB patient databases, including a dedicated VTE database.

RESULTS: The current rates of deep vein thrombosis (DVT) and pulmonary embolism (PE) at our institution following hip or knee arthroplasty or hip fracture surgery are 1.5% and 0.6% respectively, a lower rate than 2.3% and 0.9% respectively in 2006–2010. DVTs were significantly more prevalent after hip fracture surgery than after elective hip or knee arthroplasty, and 71% of DVTs were confined to the distal veins. Of the patients undergoing surgery, 93% received post-operative chemoprophylaxis, mainly aspirin or low molecular-weight heparin (LMWH).

CONCLUSION: There has been a significant reduction in VTE rates following elective hip and knee joint replacement and hip fracture surgery between the time periods. This occurred over a period when Waitemata DHB introduced a multi-modal, interdisciplinary team approach to VTE prophylaxis utilising enhanced recovery after surgery (ERAS) pathways. These measures may therefore have contributed to the reduction in VTEs.

Venous thromboembolism (VTE) remains a significant cause of potentially preventable morbidity and mortality following total hip arthroplasty (THA), total knee arthroplasty (TKA) and hip fracture surgery. The decision for chemoprophylaxis is balanced with the consequences of an increased risk of bleeding. Additional measures, such as mechanical prophylaxis also reduce the risk of post-operative VTEs.[1] Current multimodal techniques involve a multidisciplinary approach towards reducing total VTE load.

The rates of VTE without chemical or mechanical prophylaxis are high; DVTs develop in 39–74% of patients undergoing THA,[2] with the majority of these being asymptomatic. Half of all proximal DVTs, that is, those occurring in the popliteal vein or more proximally, are associated with PE,[3] and up to half of patients with a proximal DVT will develop post-thrombotic syndrome.[4] Fatal PEs occur after 0.4% of hip and knee arthroplasties in the absence of prophylaxis.[5] Age and medical comorbidities, including a history of thrombosis, previous and/or family history of VTE, congestive heart failure, varicose veins, obesity and hypertension, add to the risk of VTE .[6] This risk is estimated at 1–3% a year.[7]

The American Association of Orthopaedic Surgeons (AAOS),[8] American College of Chest Physicians,[9] National Institute for Health and Care Excellence (NICE)[10] and The New Zealand National Policy Framework: VTE Prevention[11] have guidelines promoting the routine use of VTE thromboprophylaxis, although the chemoprophylactic agent of choice remains debatable. Guidelines recommend using one of several agents including heparin products, vitamin K antagonists, antiplatelet drugs and novel oral anticoagulant agents such as rivaroxaban, with the choice of agent left to the clinician. Mechanical prophylaxis such as intermittent compression devices and graduated stockings are now routinely used in combination with chemoprophylaxis.[12]

International literature suggests that the incidence of symptomatic VTE following lower limb arthroplasty or hip fracture surgery in patients taking prophylaxis ranges from 1.0%[13] to 2.7%.[14] Although most DVTs and PEs occur within the first 10 days of operation,[15] due to shorter average lengths of stay, most events occur after discharge from hospital.[16]

This study examines the incidence of DVT and PE in patients undergoing hip arthroplasty, knee arthroplasty and hip fracture surgery at Waitemata DHB between 1 January 2013 and 31 December 2016. This audit is a follow-up to an audit performed over the time period 2006–2010 in our institutions, as previously published.[15]

Materials and methods

In 2004, a VTE database was established at Waitemata DHB by consultant haematologists to collect data on all VTE events at time of occurrence covering the entire DHB population, which now totals over 600,000 people. This is linked to the Waitemata DHB electronic database, which includes all inpatient data and was used for data collection and analysis.

We included all patients with radiologically diagnosed symptomatic DVT and/or PE occurring within three months of lower-limb arthroplasty and hip fracture surgery between 1 January 2013 and 31 December 2016. Only symptomatic patients are scanned, as Waitemata DHB does not routinely screen asymptomatic postoperative patients. Waitemata DHB uses whole-limb ultrasound to diagnose DVTs, and computed tomography pulmonary angiography (CTPA) or V/Q scan to diagnose PEs—with some, but not all PE diagnoses being followed with subsequent lower-limb ultrasound. Lower-limb DVT included distal, proximal and bilateral DVT. As virtually all VTE events within Waitemata DHB are referred to haematology thrombosis services, this would capture nearly all inpatient and outpatient VTEs occurring.

We excluded portal vein thromboses, upper limb thromboses, subsegmental PE and all VTEs following surgery at other institutions in order to provide comparable data to 2006–2010.[15] In addition, all VTE event data were reviewed to ensure there were no duplicate patient entries in the database. The focus on THA, TKA and hip fracture surgery enabled comparison with our earlier audit performed at Waitemata DHB.[15] Chemoprophylaxis data was extracted from the Pyxis® automated drug dispensing cabinet (Becton Dickinson, New Jersey, US).

Statistical analysis was performed using SAS version 9.4. DVT and PE rates were compared between procedure groups, and between the study period (2013–2016) and a previous audit period at Waitemata DHB (2006–2010).[15] DVT and PE outcomes were analysed using logistic regression modelling

Table 1: Comparison of VTE rates after type of surgery 2013–2016.

Procedure	*n*	Male (% of total)	Female (% of total)	PE	Distal DVT	Proximal DVT	Total DVT
THA	2,280	1,031 (45.2%)	1,249 (54.8%)	11 (0.5%)	9 (0.4%)	8 (0.4%)	17 (0.7%)
TKA	2,442	1,114 (45.6%)	1,328 (54.4%)	18 (0.7%)	37 (1.5%)	3 (0.1%)	40 (1.6%)
Hip fracture surgery	1,657	536 (32.3%)	1,121 (67.7%)	8 (0.5%)	24 (1.5%)	17 (1.0%)	41 (2.5%)
Total	6,379	2,681 (42.0%)	3,698 (58.0%)	37 (0.6%)	70 (1.1%)	28 (0.4%)	98 (1.5%)

THA, total hip arthroplasty; TKA, total knee arthroplasty; n, number; DVT, deep vein thrombosis; PE, pulmonary embolism.

and included procedure group in the model. Analyses were adjusted for sex, age and American Society of Anaesthesiologists' (ASA) score.

Results

There were 6,379 THA, TKA and hip fracture surgical procedures performed at Waitemata DHB between 2013 and 2016. Of the patients, 3,698 were female (58%) and 2,681 male (42%), with average ages of 73.8 and 72.4 years respectively. There were fewer males than females in the hip fracture group (32.3%) compared to THA (45.2%) and TKA (45.6%). There was no significant difference in age between men and women. Patients in the hip fracture group were on average a decade older (80.7) than the knee arthroplasty (69.2) or hip arthroplasty (69.8) groups.

A total of 135 VTEs were identified as occurring within three months of surgery. These included 98 lower-limb DVTs and 37 PEs (see Table 1). Most DVTs were confined to the distal veins (71.4%). Proximal DVTs occurred more frequently after THA and hip fracture surgery (47.1% and 41.5% of all DVTs respectively) than after TKA (7.5% of all DVTs).

The overall DVT rate in this audit was 1.5%, a statistically significant reduction from 2.3% in 2006–2010[15] (OR 0.68, 95% CI 0.51–0.89; *p=0.006*) when adjusted for sex, age and ASA score (see Table 2).

The reduction in DVT rate was significant for THA (OR 0.49, 95% CI 0.26–0.92; *p=0.025*) and TKA (OR 0.44, 95% CI 0.29–0.67; *p=0.001*). The increase in DVT rate after hip fracture surgery from 2.0% to 2.5% was not statistically significant (see Table 2). There were significantly more DVTs occurring in both hip fracture (OR 2.94, 95% CI 1.53–5.65; *p=0.001*) and TKA patients (OR 2.13, 95% CI 1.20–3.78: *p=0.01*) compared to THA patients, calculated with adjusted logistical regression.

The overall PE rate in this audit was 0.6%. There was a statistically significant reduction in the adjusted rate of overall PE from 0.9% in 2006–2010 to 0.6% in 2013–2016 (OR 0.63, 95% CI 0.40–0.98: *p=0.041*), although the procedure-specific rates for THA, TKA and hip fracture surgery did not reach statistical significance at the *p<0.05* level (see Table 3).

Table 2: Comparison of DVT rates between 2006–2010 and 2013–2016 by procedure.

Surgery type	2006–2010	2013–2016	OR	95% CI	*p value*
THA	1.5%	0.7%	0.49	0.26–0.92	0.025
TKA	3.6%	1.6%	0.44	0.29–0.67	0.001
Hip fracture surgery	2.0%	2.5%	1.20	0.76–1.87	0.436
Overall	2.3%	1.5%	0.68	0.51–0.89	0.006

THA, total hip arthroplasty; TKA, total knee arthroplasty, OR, odds ratio; CI, confidence interval.

Table 3: Comparison of PE rates between the 2006–2010 and 2013–2016 periods by procedure.

Surgery type	2006–2010	2013–2016	OR	95% CI	*p value*
THA	0.8%	0.5%	0.68	0.30–1.53	0.354
TKA	1.0%	0.7%	0.62	0.30–1.26	0.183
Hip fracture surgery	1.0%	0.5%	0.55	0.23–1.29	0.167
Overall	0.9%	0.6%	0.63	0.40–0.98	0.041

THA, total hip arthroplasty; TKA, total knee arthroplasty, OR, odds ratio; CI, confidence interval.

DVT rates following hip fracture surgery were higher than DVT rates following THA or TKA, but there were no differences in PE rates between the three surgical groups.

The chemoprophylaxis data available were limited to a binary outcome of whether prophylactic agents were prescribed while patients were in hospital, and did not allow analysis of specific regimens, doses and duration of treatment. Overall, 92.6% of all patients (5,909 out of 6,379) and 92.6% of those who developed a post-operative VTE (125 out of 135) received inpatient chemoprophylaxis. This included 94.7% of THA patients, 86.7% of TKA patients and 94.7% of hip fracture surgery patients, with stable year-to-year trends across 2013 to 2016. Patients receiving more than one agent were included in each drug category, so were counted more than once overall. The agent used was most commonly LMWH, then aspirin, with smaller numbers of patients receiving direct oral anticoagulants, warfarin or unfractioned heparin. LMWH was prescribed in 60.7% of hip arthroplasty, 50.9% of knee arthroplasty and 57.0% of hip/femur fracture surgeries. Aspirin was used in 41.9% of THAs, 53.0% of TKAs and 49.5% of hip fracture surgeries. In total, 470 patients (7.6%) received no chemoprophylaxis, and 10 of these went on to develop a VTE (2.1%), which is the same VTE rate as the total surgical sample, and those who did not receive chemoprophylaxis. Case numbers are too small to allow any further analyses. In comparing the use of LMWH and aspirin in patients with VTE, LMWH use increased from 35% to 73%, while aspirin use slightly decreased from 59% to 57% from 2006–2010 to 2013–2016.

One patient died within three months of surgery, giving an all-cause mortality of 0.74%—compared with three patients and mortality rate 1.8% in the previous audit.[15] Unfortunately, data are unavailable regarding the cause of death; however, this occurred after discharge from hospital following surgery.

Discussion

VTE rates following hip and knee surgery vary over time and between different institutions. Published rates range from 2.1–2.8%[14,16] in older studies, to 0.64–1.2% in more contemporary research.[13,17,18] Confounding variables occurring between institutions, population demographics and changes in practice can make comparisons difficult.

There was a significant fall in VTE rates between audits, with reductions in DVT from 2.3% to 1.5% and PE from 0.9% to 0.6%. The authors of the 2006–2010 audit[15] concluded that their VTE rate was higher than that found in literature at the time, attributed to suboptimal chemical and mechanical prophylaxis. Direct comparison of thromboprophylaxis rates for all surgical patients is not possible as the earlier audit only presented this data for VTE cases. However, in this subgroup we note that in 2006–2010, 14% of postoperative VTE patients had received no chemoprophylaxis, compared to 7.4% in the current audit. This limited data may suggest increased rates of chemoprophylaxis over time.

This audit included a variety of surgical procedures, performed for different indications in a heterogeneous population. This heterogeneity can lead to a variation in VTE rate between types of procedure or indication for surgery. Bjørnaå et al found comparable rates of DVTs after hip fracture surgery to that of TKA and THA in the same era as our initial audit.[14] In comparison, our current adjusted DVT

rates were higher in the acute hip fracture surgery group compared to the elective THA and TKA groups. Compared to the previous audit, the current hip fracture DVT rate did not decrease and remained above 2%. However, DVT rates in the elective THA and TKA groups fell (*p=0.025* and *p=0.001* respectively).

The DVT rates include both proximal and distal DVT, and the relative proportion of these varied within procedure groups with higher proportions of distal DVTs after TKA compared to THA. Compared to the 2006–2010 audit,[15] there was a reduction in the overall rate of distal DVTs for THA patients (0.9% to 0.4%), but a relatively stable rate of proximal DVTs (0.3% to 0.4%). This had the effect of reducing the relative proportion of distal DVTs following THA between audits (from 77.8% to 52.9%). In contrast, in the TKA group, it was the rate of proximal DVTs that fell between audits (from 0.6% to 0.1%), resulting in an increase in the relative proportion of distal DVTs (from 86% to 92.5%). This is also consistent with the established association of TKA with more distal DVTs.[19] In the acute hip fracture surgery group, the proportion of proximal to distal DVT did not change between the two audits. With the three groups pooled, overall proximal DVT rate declined from 0.6% to 0.4% between the current period and 2006–2010.[15] PE rates declined across all three groups in the subsequent audit when pooled (0.9% to 0.6% *p=0.041*) but not as individual treatment groups, likely due to the small number of these events occurring. Given that not all PE patients receive subsequent ultrasound to assess clot burden (but some do), we acknowledge the possibility that some pulmonary emboli may not be thrombotic in origin but may be from other sources such as bone or debris produced perioperatively.

The reasons for the unchanged DVT rate in hip fracture patients are likely multifactorial, such as older age, poor nutritional and hydration status pre-operatively, slower post-operative mobilisation and rehabilitation, poor pre-operative function and mobility, and delays in rehabilitation due to medical issues and investigations. Additionally, despite reductions in time to theatre as discussed below, hip fracture patients remain preoperatively immobilised for longer periods than elective THA and TKA patients. This group had significantly longer inpatient stays, with an average length of stay of 21 versus 4 days. We did not have sufficient data to include these factors in multivariate analysis. The appointment of an orthogeriatrician at the start of 2018, with a dedicated focus on the pre-operative and post-operative management of neck of femur fracture patients will medically optimise these patients and may help reduce their rates of VTE.

Considerable changes have occurred at Waitemata DHB between the time periods of the two audits, which we believe may have contributed to reductions in VTE rates in hip and knee arthroplasty patients. They are improved collaboration, enhanced recovery after surgery (ERAS) and VTE risk assessment.

Clinical leads have been set up in the orthopaedic surgery and haematology departments to facilitate liaison and provide feedback, along with input and analysis from a public health physician. Case study evaluation occurs between the disciplines on a regular basis, with an increase in dialogue between surgeons and physicians regarding more complex individual cases. There is heightened awareness of the implications of therapeutic anti-coagulation of a proven VTE soon after surgery, with haematology clinical nurse specialists providing a consistent point of contact for the investigation and management of these patients. Regular discussion of all proven VTE cases and their subsequent management occurs within the orthopaedic department every three months, and regular VTE meetings have been scheduled into standard teaching commitments to provide emphasis on the importance of VTE prophylaxis to junior staff.

ERAS protocols[21] are a bundle of evidence-based multimodal interventions aimed at improving post-operative recovery and reducing complications by aggregating marginal gains in care before, during and after surgery. ERAS protocols were introduced during the current audit. Adherence to these protocols was easier in THA and TKA patients, taking longer to implement for hip fracture surgery and for this group, only being in effect towards the end of this audit. Hip fracture protocols focused

particularly on reducing time to surgery, improving peri-operative analgesia enabling earlier postoperative mobilisation (within 24 hours), and cohorting patients in dedicated wards. Other data from Waitemata DHB indicates that there has been a reduction in average hours to theatre for hip fracture patients from 39 hours in 2010 to 29 hours in 2016, a decrease in the median hours to mobilisation from 44 to 24 hours, and an associated reduction in crude 30-day mortality rate from 10.2% to 5.7%.[22]

Although ERAS protocols do not make specific recommendations regarding the use of chemoprophylaxis, other ERAS measures such as early post-operative mobilisation and early hospital discharge have been linked to reduced VTE rates after orthopaedic surgery.[23] A recent cohort study showed a significant reduction in median length of stay after elective hip and knee arthroplasty after implementing ERAS protocols without an increase in complications.[24] However, the link between ERAS and reduced VTE rates is controversial, with other authors reporting no link between ERAS protocols and VTE rates after arthroplasty despite reductions in overall death rates, rates of myocardial infarctions and strokes.[21,25]The National Policy Framework *"DVT/PE Prevention in Hospitalised Patients in New Zealand guidelines"*[11] recommends the routine use of chemoprophylaxis after hip arthroplasty, knee arthroplasty and hip fracture surgery. The rate of chemoprophylaxis use was 92.6% and was identical between both the denominator and the numerator groups for diagnosed VTE. This is an improvement from the 86% numerator rate in 2006–2010.[15] No VTE risk stratification tools were used in Waitemata DHB during the audit period, although VTE risk stratification is done for all orthopaedic patients since 2018. Chemoprophylactic agents were more widely prescribed after THA and hip fracture surgery (both 94.7%) than in TKA (86.7%), with LMWH and aspirin the most commonly used agents.

Some studies show aspirin to be as efficacious for VTE prophylaxis after hip and knee arthroplasty as LMWH,[13,26–29] but this remains contentious.[9] The AAOS and ACCP have now endorsed aspirin as a viable chemoprophylaxis agent in low-risk groups undergoing arthroplasty.[30] The use of novel anticoagulants such as rivaroxaban and dabigatran is increasing, both within the general population and as prophylaxis following surgery, particularly after TKA. This, along with an increased use of aspirin, appears to coincide with less use of LMWH in TKA after 2013 in our audit.

Warfarin, dabigatran and rivaroxaban were utilised more in acute hip fracture patients compared to THA and TKA patients in this audit. This was most likely a reflection of these patients being more comorbid and being on these medications prior to admission and surgery.

The strengths of this study were that there were no exclusions of any patient and the numbers audited are large for a public hospital in New Zealand. The limitations with this audit relate mainly to data collection. In addition, there may be additional VTEs in this post-operative population that cause death prior to radiographical diagnosis. By their nature, these would not be included in our data. The chemoprophylaxis data was collated from PYXIS. It collates the rate of inpatient prescription, but not the timing (first or subsequent doses), the dosage or the duration of prophylaxis. There are no data about the patients that were not given chemoprophylaxis, either the reasons for this or alternative medications or measures taken.

Patients who received more than one agent were included in each drug category, so were counted more than once. We are unable to say how many dual therapy patients were in each group. Data are unavailable regarding the type and duration of mechanical prophylaxis. A mixture of calf compression devices, foot pumps and TED stockings were used, both intra-operatively and post-operatively. Documentation of mechanical prophylaxis is poor compared to chemoprophylaxis. Electronic prescribing was in place at Waitemata DHB by the end of 2016, so future audits can collect accurate and detailed data regarding inpatient dosing and timing of VTE chemoprophylaxis and TED stockings. Similarly, electronic discharge summaries and electronic records of community dispensing can record chemoprophylaxis after discharge.

Conclusion

There has been a significant reduction in post-operative VTE rates at our institution following hip and knee arthroplasty and hip fracture surgery, from 3.2% (2006–2010)[15] to 2.1% (2013–2016). We believe that this has been contributed to by a multidisciplinary approach with improved collaboration, the introduction of ERAS protocols and VTE risk assessment tools, and an increase in chemo-prophylaxis rates.

Competing interests:
Dr Farrington reports personal fees from Stryker, personal fees from LIMA Orthopaedics, outside the submitted work.

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Vitamin D concentrations in New Zealanders with and without inflammatory bowel disease: do they differ?

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ABSTRACT

AIM: Patients with inflammatory bowel disease (IBD), Crohn's disease (CD) or ulcerative colitis (UC) are at risk of low vitamin D owing to reduced absorption, medication-associated sunlight exposure restrictions and/or increased requirements due to inflammation. This study aimed to determine if the serum vitamin D concentration of New Zealand IBD patients relates to disease activity and differs from controls. **METHOD:** Data concerning demographics, sunlight exposure, vitamin D supplementation and disease activity were collected using a retrospective questionnaire. Serum vitamin D concentrations were measured in dried blood spots and validated against blood samples in a participant sub-group. **RESULTS:** Vitamin D concentration was significantly increased by supplementation (82.8 v 66.4nmol/L, $p<0.001$) and sunlight exposure while on holiday (75.2 v 63.7nmol/L, $p<0.001$). Patients with CD who reported active disease in the last year had significantly lower vitamin D concentrations (68.6 v 84.6nmol/L, $p=0.008$) than those who reported remaining in remission. **CONCLUSION:** In this cohort of New Zealand residents, mean vitamin D of patients with IBD was not different from controls. In patients with CD, recent disease activity was significantly associated with lower vitamin D. The use of vitamin D supplementation may have implications for reducing disease activity occurrence in patients with CD.

Vitamin D (25(OH)D or calcidiol) is a fat soluble steroid that plays a major role in bone health through the regulation of serum calcium and phosphate concentrations.[1] While the importance of vitamin D to bone health is well established, clear evidence of this hormone's involvement in immune function has come to light following the discovery of vitamin D receptor expression by a number of immune cell types,[2–4] and, similarly, local production of the active vitamin D metabolite calcitriol by certain immune cells.[5] Adequate vitamin D concentrations also appear to be important in the prevention of diseases such as cancer, heart disease and immune system mediated diseases.[6,7]

Two such diseases are Crohn's disease (CD) and ulcerative colitis (UC), collectively termed inflammatory bowel disease (IBD).[8] A link between IBD and vitamin D was proposed in response to the geographical distribution of IBD with the greatest incidences observed in regions furthest North and South of the equator,[99] namely New Zealand,[10] Canada,[11] South Australia[12] Iceland[13] and Sweden.[14] This latitudinal trend is mirrored in other risk factors implicated in IBD including greater industrialisation, extent of development and European ethnicity.[9] Moreover, in regions of these latitudes, the combined effect of a greater solar zenith angle, colder temperatures and a conscious reduction in sunlight exposure to reduce skin cancer risk,[1,15–17] led to significantly lower vitamin D concentrations than those observed closer to the equator.

New Zealand has one of the highest reported rates of IBD worldwide, and unlike many Western countries where the number of new diagnoses has plateaued, the incidence and prevalence continue to rise.[10,18] In

the last two decades researchers have identified a number of genes strongly associated with IBD, especially CD.[19] However, heritability does not account for all instances and the current consensus is that environmental and immune factors are also involved. New Zealand's high IBD incidence, elongated shape and southern geographical position make it an ideal location to explore the possible relationship between vitamin D levels and IBD. The aim of this study was to determine the vitamin D concentration of New Zealand patients with IBD and to establish whether they differ from those of controls and are related to disease activity.

Methods

Questionnaire

Participants aged 16 years and above, diagnosed with IBD, or controls with no family history of gastrointestinal disorders, responded to advertisements placed in gastroenterology clinics and at community IBD support organisations located throughout New Zealand. Participants were asked to complete a self-administered retrospective questionnaire developed expressly for this study (see Appendix), concerning demographic data, residence location, holiday history in the previous 12 months, sunlight exposure habits, time spent outdoors per week, vitamin D testing in the previous 12 months and vitamin D supplementation in the previous six months. Residence location was coded by island (North or South), then by latitude (<37.5°S, 37.5–40°S, 40–42.5°S, 42.5–45°S, and >45°S). Sunlight exposure habits comprised sunblock use, protective clothing worn, shade seeking behaviour, sunbathing and sunburn. Patients with IBD were asked additional questions about disease history. Questionnaires were provided in hardcopy or via a secure online survey platform (surveymonkey.com).

Sample collection

Participants residing in or close to 10 major cities and towns throughout the length of New Zealand were invited to take part in the second part of the study by providing a small blood spot sample for serum vitamin D (25(OH)D) measurement. Exclusion criteria included individuals with a blood borne disease or not living predominantly in New Zealand in the previous 24 months. To obtain the sample, a finger lance was used and a minimum of three blood spots were collected from the second or third finger of the non-dominant hand. The blood spot collection cards were air-dried and stored according to the manufacturer's instructions (ZRT Laboratory, Oregon, US). Samples were collected by the same researcher over two calendar months to reduce potential sampling method disparities and to minimise seasonal differences in vitamin D concentration. Participants consenting to a blood spot sample, and residing in one of two cities, were also invited to take part in a sub-study where a 5ml blood sample was taken by venepuncture to measure serum vitamin D in order to validate the blood spot method.

Blood spot validation

Serum vitamin D levels were measured in dried blood spots using liquid chromatography tandem mass spectrophotometry (LC-MS/MS), and in venous blood samples using high-performance liquid chromatography (HPLC) tandem mass spectrometry (Canterbury Health Laboratories, Christchurch, New Zealand).

Ethics

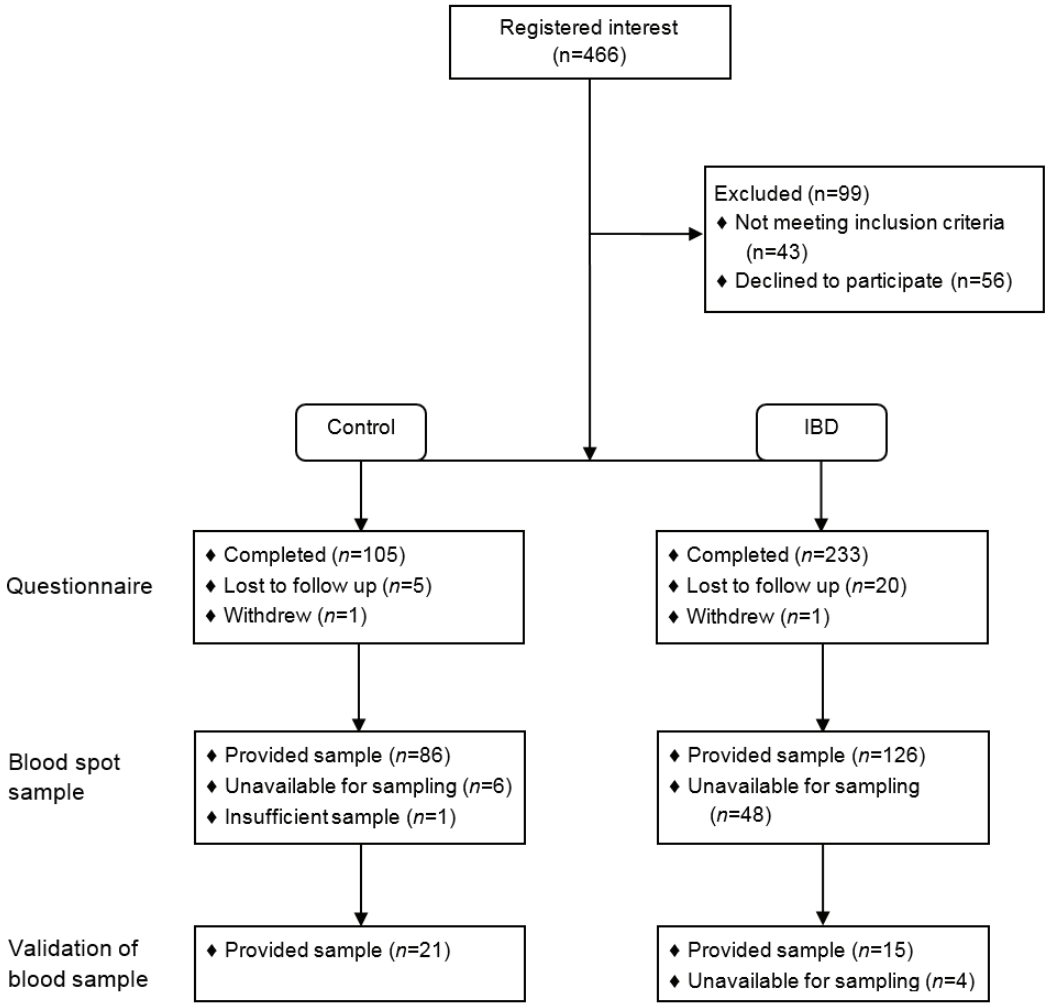
The protocol for this study was approved by the Massey University Human Ethics Committee: Southern A, Palmerston North, New Zealand (MUHEC Reference 13/58). All study participants provided informed consent.

Statistical analysis

Differences in vitamin D concentration between patients with IBD and controls, and between patients with CD and UC, were analysed using ANOVA TEST (SAS 9.2, North Carolina, US). A p-value of <0.05 was considered statistically significant for all data analysis.

For analysis of vitamin D status, the ranges recommended by the Working Group of the Australian and New Zealand Bone and Mineral Society, Endocrine Society of Australia and Osteoporosis Australia, were applied; severe deficiency <12.5nmol/L, moderate deficiency 12.5–25nmol/L, mild deficiency 25–50nmol/L, and adequacy >50nmol/L.[20] Pearson's Chi-square test was used to test for association between vitamin D status and participant groups; patients

Figure 1: Inclusion and completion of controls and patients with IBD.



with IBD and controls, and between IBD subgroups (CD and UC) (Minitab 17.3.1, Pennsylvania, US).

A linear regression model was applied to vitamin D values from venous blood samples and their corresponding blood spot values (Minitab 17.3.1, Pennsylvania, US). A correction factor was derived from the regression equation (y=10.36+1.1444x) and applied to all blood spot values.

Results

Demographic and participant characteristics

A total of 198 participants completed the questionnaire and provided a blood spot sample, 81 controls, 79 patients with CD, 31 with UC and seven with IBDU.

Vitamin D

No significant difference was observed between the mean vitamin D concentration of patients with IBD and controls, or between IBD subgroups (CD and UC). More than three quarters of patients with IBD and controls had vitamin D concentrations considered adequate (>50nmol/L) (Table 2). Mild vitamin D deficiency (25–50nmol/L) percentages were comparable between patients with IBD and controls, however a greater difference was observed between IBD subgroups. Moderate vitamin D deficiency (12–25nmol/L) was only observed in patients with IBD, and no participants were considered severely vitamin D deficient (<12.5nmol/L). The difference in vitamin D status between patients with IBD and controls, and between IBD subgroups, was not significant.

Table 1: Baseline characteristics of the 198 participants.

	Control ***n=81***	**IBD** (CD, UC, IBDU) ***n=117***	**CD** ***n=79***	**UC** ***n=31***
Mean age, years	42.4±15.1	40.9±14.1	39.0±13.5	42.9±14.5
Female, no. (%)	60, (74.1)	85, (72.6)	57, (72.2)	23, (74.2)
Mean body mass index (kg/m^2)	25.2±3.9 *n=79*	26.1±7.0 *n=111*	26.5±7.8 *n=76*	24.3±4.3 *n=29*
Location, North Island, no. (%)	54, (66.7)	72, (61.5)	49, (62.0)	19, (61.3)
Mean time since diagnosis, years	n/a	9.9±0.9	9.4±0.9	10.9±2.1
Mean vitamin D (25(OH)D), nmol/L	70.1±2.6	69.8±2.1	72.5±2.7	64.9±3.8
Supplementation				
• Current, no. (%)	5, (6.2)	22, (18.8)	16, (20.3)	3, (9.7)
• In last six months, no. (%)	5, (6.2)	10, (8.5)	6, (7.6)	4, (12.9)
• None, no. (%)	71, (87.7)	85, (72.6)	57, (72.2)	24, (77.4)
Vitamin D measurement in previous 12 months, no. (%)	3, (3.7)	12, (10.3)	6, (7.6)	5, (16.1)
Active disease in previous 12 months, no. (%)	n/a	88, (75.2)	60, (75.9)	22, (71.0)

Vitamin D concentration was significantly higher in patients with IBD, but not controls, who reported increased sunlight exposure on holiday in the previous 12 months (62.0 v 76.3, p=0.001), current supplementation (63.9 v 86.9, p<0.001) or supplementation within the previous six months (63.9 v 81.8, p<0.018). When the effect of increased sunlight exposure on holiday in the previous 12 months was analysed by IBD subgroup, the difference only remained significant in patients with CD (65.0 v 78.4, p=0.011). When the effect of supplementation was analysed by IBD subgroup, the difference associated with current supplementation remained significant in patients with CD (66.8 v 87.9, p<0.001) and in patients with UC (59.7 v 91.5, p=0.018), while the difference associated with previous supplementation only remained significant in patients with CD (66.8 v 85.5, p=0.046). No differences were observed between vitamin D concentration and time spent in sunlight per week, sunlight exposure habits, location or latitude.

Disease activity

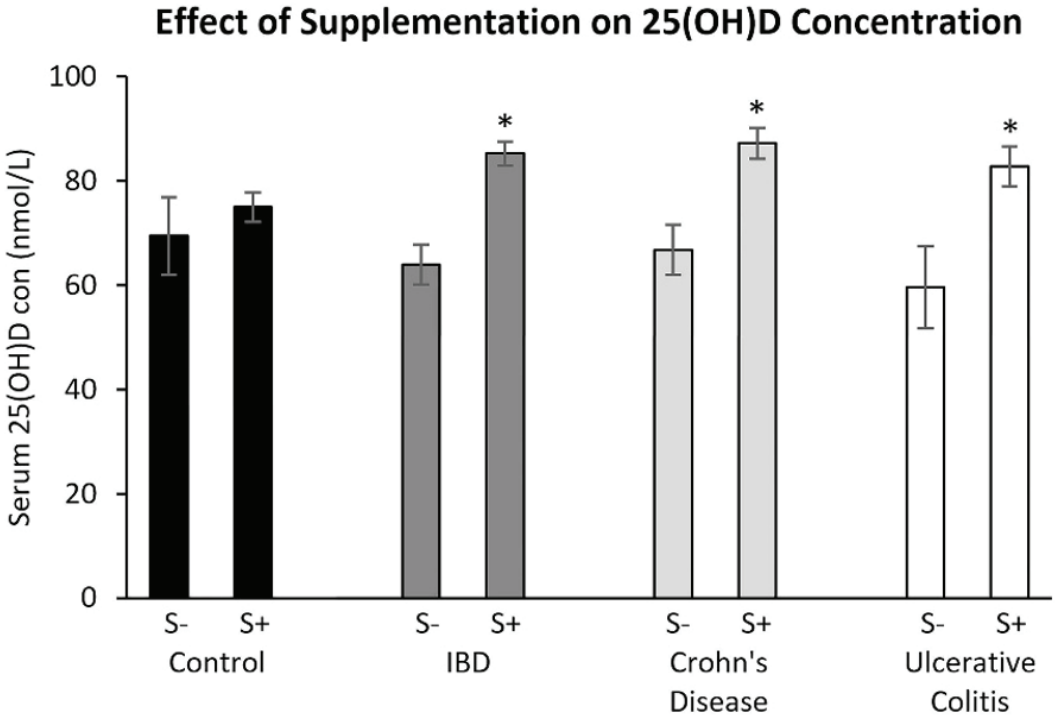
A significant difference in vitamin D concentration was determined between patients with IBD that reported disease activity in the previous 12 months and those that reported remaining in remission only when supplementation was take into account (Table 3).

Table 2: Vitamin D status of controls, patients with IBD, CD and UC.

	Control ***n=81***	**IBD** (CD, UC, IBDU) ***n=117***	**CD** ***n=79***	**UC** ***n=31***
Moderate deficiency[1], no. (%)	0	1, (0.9)	1, (1.3)	0
Mild deficiency[2], no. (%)	15, (18.5)	26, (22.2)	14, (17.7)	10, (32.3)
Adequacy[3], no. (%)	66, (81.5)	90, (76.9)	64, (81.0)	21, (67.7)

[1] 12.5-25nmol/L, [2] 25–50nmol/L, [3] >50nmol/L.

Figure 2: Effect of supplementation (current or previous six months) on serum vitamin D concentration by participant group.



S- no supplementation, S+ supplementation
Bars marked with * are significantly (p<0.05) different to no supplementation within the same participant group.
NB Vitamin D for patients diagnosed with both CD and UC, or IBDU were not analysed individually due to insufficient numbers, they were however included in IBD cohort analyses.

When analysed by IBD subgroup, the mean vitamin D concentration remained significantly lower in patients with CD that had reported disease activity in the previous 12 months (68.6 v 84.6, *p*=0.008) compared to those that reported remaining in remission, irrespective of supplementation. There was no difference in patients with UC.

Blood spot method validation

A subset of participants with and without IBD provided a venous blood sample for vitamin D measurement in addition to their blood spot sample (*n=36*). The mean vitamin D concentration in this subset was 69.9±23.4nmol/L measured in blood spots and 75.0±25.1nmol/L measured in venous blood samples. The blood spot values were lower than their corresponding venous blood sample values and were closely correlated (r^2=0.805) (Figure 3).

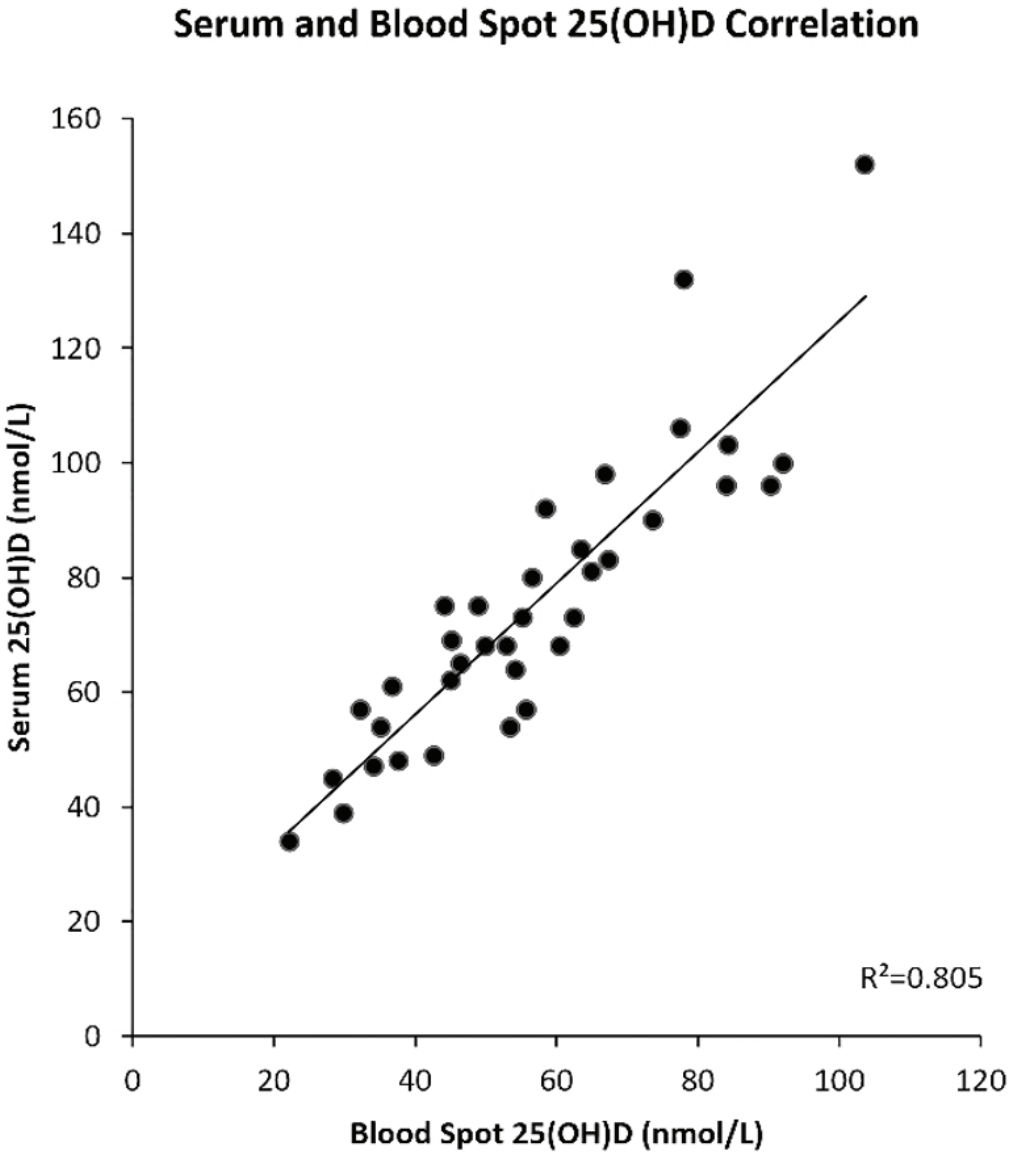
Discussion

The mean vitamin D concentration in this cohort, measured in blood spot samples collected over eight weeks (April–June), was 69.9nmol/L, a level that correlates well with the month standardised national mean of 63.0nmol/L as reported in the New Zealand Adult Nutrition Survey 2008/09.[21] This value also falls between the mean concentrations reported in two other New Zealand studies; 53 and 63nmol/L for female and

Table 3: Difference between vitamin D (25(OH)D) concentration and disease activity in the previous 12 months by vitamin D supplementation.

	No supplementation (nmol/L)	**Supplementation (nmol/L) (current or previous six months)**	**P value**
Active disease, *n=88*	62.2±2.6	81.8±4.1	0.001
Remission, *n=29*	68.5±4.4	100.5±8.6	<0.001

Figure 3: Correlation between vitamin D measure in blood spot and venous blood samples.



male participants aged 15 years and over, from samples collected nationwide during Autumn (March–May) 1997,[22] and 85nmol/L in South Island participants aged 18 years and over in samples collected during late summer (February) 2005.[23] Overall, there was no significant difference between the mean vitamin D concentration of controls and patients with IBD, or between IBD subgroups. Accordingly, the percentage of participants considered to have adequate, mildly deficient or moderately deficient vitamin D concentrations was not significantly different between participant groups or subgroups. As discrepancies exist between vitamin D status terminology and the 25(OH)D parameters associated with each status, it is difficult to compare these findings with similar research. However, a meta-analysis of observational studies demonstrated that the odds ratio of being vitamin D deficient (≤50nmol/L) compared to controls is 1.63 in CD and 2.28 in UC.[24] In line with this data, a greater proportion of mild vitamin D deficiency was observed in patients with UC than those with CD, 32.3% and 17.7% respectively.

The percentage of patients with IBD who reported taking vitamin D supplementation was three-fold greater than controls, and two-fold greater in patients with CD than in patients with UC. Vitamin D concentrations

were significantly higher in patients with IBD, but not controls, who reported supplementation currently or in the previous six months. This is unsurprising given the daily dose of 100–1,000IU reported by the controls, compared to 284–7,143IU reported by patients with IBD. While the total number of individuals reporting supplementation was too small to allow further statistical analysis, based on the variance in supplement use and dose, we might have expected a greater difference between the mean vitamin D concentrations. Comparison of the mean vitamin D concentration in patients with IBD, and in controls, reporting no supplementation revealed no significant difference, suggesting a considerable imbalance between oral intakes and ensuing serum concentration likely attributable to impaired nutrient absorption associated with inflammation, notably fat malabsorption,[25,26] or having undergone small intestinal resection.[27] Researchers have also demonstrated impaired absorption of supplemental vitamin D in clinically quiescent patients with CD compared to controls, a 30% reduction across all patients, and 20% in those without a resection, thus another mechanism may be involved.[28] Genetic factors could influence vitamin D concentrations as polymorphisms of the genes that encode the vitamin D binding protein (VDBP) are reported to influence vitamin D concentrations,[29] and expression of VDBP genetic variants has been demonstrated to differ between patients with IBD and controls.[30]

Once adjusted for supplementation, a significant difference in vitamin D concentration was observed between patients with IBD who reported disease activity in the previous 12 months and those that reported remaining in remission. When analysed by IBD subgroup, the difference remained significant only in patients with CD irrespective of supplementation. This may reflect a stronger association between vitamin D concentration and CD, a relationship proposed in the Nurses' Health Study.[31] Alternatively, the present study may not have been adequately powered to investigate a difference in vitamin D concentration between patients with UC reporting active disease and patients with UC reporting remission. Differences in supplementation could potentially explain this observation, as of the patients with UC in remission, 0% reported current supplementation and 25% reported supplementation in the previous six months, compared to 19% and 33% respectively of the equivalent patients with CD.

While the leading source of vitamin D is skin exposure to UV B radiation,[5] the only UV exposure factor that had an effect on vitamin D concentration was seen in patients with IBD who reported increased sunlight exposure on holiday in the previous 12 months. Unlike other studies, no effect of latitude on vitamin D concentrations was observed,[21,22,32] though a review of global vitamin D status demonstrates this relationship is less marked now than in earlier studies.[15] A discernible effect of latitude may also have been obscured by supplement use and dose. No effect was observed between vitamin D concentration and sunlight exposure score, and patients with IBD and controls produced similar scores. This is surprising, as some routinely prescribed IBD medications are associated with increased skin cancer risk, namely azathioprine and mercaptopurine, thus patients prescribed these medications are advised to limit UV exposure.[33] Closer inspection revealed that while the patients with IBD reported greater sunscreen reapplication compliance and shade seeking, this was counteracted by lower sunhat use and a higher incidence of sunburn.

Finally, in the present study a close correlation was observed between the two measures of serum vitamin D; blood spot assay using LC-MS/MS and venous blood samples using HPLC. In agreement with other work, the vitamin D concentrations measured in blood spot LC-MS/MS assay were lower than their corresponding HPLC assay values.[34,35] This difference may in part be explained by variation between the assay methods, a recognised obstacle that led to the development of the Vitamin D External Quality Assessment Scheme (DEQAS), a scheme formed in 1989 to appraise vitamin D assay reliability.[36,37] It has also been suggested that assay variability may be attributable to either one or a combination of three factors; blood spot volume less than 50µl, differences in blood volume absorption due to variations in filter paper weight and

the location of the punched spot on the paper.[38] While obtaining adequate blood spot volume can be improved by collecting additional spots, the latter two factors may be more difficult to control.

Conclusion

Vitamin D concentrations were not different between New Zealand patients with IBD compared to controls irrespective of marked differences in supplement use and dose. This likely presents a challenge for reaching and maintaining adequate vitamin D concentrations in patients with IBD who have limited UV exposure or are not receiving regular supplementation. In patients with CD, a history of active disease in the previous 12 months was significantly associated with lower vitamin D concentration compared to patients who reported remaining in remission. Although this difference was not observed in patients with UC, vitamin D supplementation is safe, effective and inexpensive[39] and may have implications for reducing disease activity recurrence. Vitamin D supplementation should be recommended to patients with IBD by their healthcare providers, especially to patients with a recent history of active disease and those being treated with sun-sensitising medications.

Study limitations

The present study had several limitations. Samples were collected from April through June when vitamin D concentrations are expected to be moderately high following summer and thus do not indicate nadir concentrations. Obtaining this information would have been useful for demonstrating the extent of both vitamin D inadequacy and deficiency in this at-risk population group. A retrospective questionnaire was used to collect data about sunlight exposure, vitamin D supplementation and disease activity. The sunlight exposure habits assessed were assumed to be of equal contribution to UV exposure due to the lack of a suitable validated questionnaire. The collection of disease activity history over a period of 12 months prevented the use of a validated disease activity index; however, using an index would have been time consuming and may have reduced the number of responses. Lastly, the sample size, particularly the number of patients with UC, may have limited the findings.

Competing interests:
Dr Coad reports grants from Massey University School of Food and Advanced Technology, grants from Graduate Women New Zealand, awarded to Hannah Morton (PhD student) during the conduct of the study. Ms Morton reports grants from Massey University School of Food and Advanced Technology, grants from Graduate Women New Zealand, during the conduct of the study.

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URL:
www.nzma.org.nz/journal-articles/vitamin-d-concentrations-in-new-zealanders-with-and-without-inflammatory-bowel-disease-do-they-differ

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Food taxes and subsidies to protect health: relevance to Aotearoa New Zealand

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ABSTRACT

The hazardous and obesogenic food environment are major contributors to health loss in Aotearoa New Zealand. Here we consider the potential use of food taxes and subsidies to protect health in this country. We find that each one of the 14 recent systematic reviews on the tax and/or subsidy topic since 2015 in the scientific literature report that such interventions have favourable impacts from a health perspective. The New Zealand evidence we considered (n=12 studies since January 2010) is less definitive, but the pattern of results is consistent with the international evidence. Given this overall picture, the New Zealand Government should seriously consider such tax/subsidy interventions, potentially starting with a UK-style sugary drinks industry levy.

The hazardous and obesogenic food environment are very major contributors to health loss in Aotearoa New Zealand. In this country 32% of adults are obese (Māori: 47%; Pacific: 65%)[1] and ischaemic heart disease (for which diet is a key risk factor) is the most common cause of death.[2] Indeed, dietary risk factors and high body mass index are both in the top three leading causes of health loss in the country, when considering death and disability combined.[2]

Nutrition-related diseases such as diabetes, cardiovascular disease and obesity-related cancers are a particular burden for Māori and Pasifika and so are a major driver of ethnic inequalities in health in New Zealand. In particular, obesity appears to increasingly be a key contributor to ethnic inequalities in the cancer burden in this country.[3]

To give an indication of the huge size of the preventable burden in the nutrition domain, a single intervention to reduce salt (sodium) in the processed food supply in New Zealand could save an estimated 294,000 years of life (more precisely quality-adjusted life-years [QALYs]), over the remaining lifetime of the New Zealand population.[4] This salt reduction intervention was also estimated to produce net cost-savings to the health system of NZ$ 1.5 billion. (Examples of other health generating and cost-saving dietary interventions for New Zealand and Australia are detailed in a recently developed online league table[5]). More specifically, the health risks of some food products are increasingly being clarified eg, consumption of sugar-sweetened beverages (SSBs) is associated with increased mortality primarily through cardiovascular mortality, with a graded association with dose.[6] Furthermore, there is evidence that sugary drinks may provide greater health risks relative to sugar-containing foods.[7]

There are some regulations around food safety in New Zealand, but processed food is largely unregulated for nutritional content, eg, manufacturers have no limits on the amount of potentially hazardous ingredients (ie, sugar, salt and saturated fat) they can add to processed foods (albeit a diverse category that includes ultra-processed foods[8]). None of the damage to health of New Zealanders and the public health system costs associated with processed food are specifically paid for by the food industry (ie, it is largely paid by the taxpayer-funded health system and also by individuals and

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their families). This means a major market failure exists[9] and negative externalities are imposed on society. Such a market failure provides one rationale for taxes on unhealthy foods and beverages; or at least some type of regulation. But a potentially more important rationale is that taxes can be used as an effective instrument to achieve a societal goal, such as reducing child obesity.

Under Te Tiriti o Waitangi (the Treaty of Waitangi), the New Zealand Government has a duty to protect the health and wellbeing of Māori. In addition to the well-established ethical arguments for reducing health inequities in society, the Treaty provides a strong basis for Government action to reduce the harm imposed on Māori by the processed food industry. The Treaty is also relevant to the protection of land, the environment and the food sources cultivated and harvested by Māori. Treaty settlements also need to address the confiscation of Māori land in the 1800s, with some of this land being a source of food for Māori and part of economic prosperity.[10]

Given this background, this article briefly considers recent international and New Zealand evidence concerning food/beverages taxes and subsidies. It then identifies those issues of potential relevance to the New Zealand Government around making progress in these domains.

International evidence

Following a literature search, 14 systematic reviews on food or beverage interventions involving taxes or subsidies were identified (search in PubMed from 1 January 2015 to 15 June 2019). The study designs used within these reviews varied and included experimental, cross-sectional, simulations and 'real world' quasi-experimental studies. Each systematic review consistently showed changes in food or beverage consumption in directions favouring health (Appendix Table 1). There is some evidence that such interventions can benefit all socio-economic groups (reviews by Olstad et al[11] and Backholer et al[12] in Appendix Table 1) and may reduce inequities.[11] However, one systematic review found unclear income-related impacts (Mizdrak et al[13]). The evidence around substitution behaviours is somewhat limited. Nevertheless, one meta-analysis reported a suggestive pattern of increased bottled water use with SSB taxes (Teng et al,[14] in Appendix Table 1).

Given that most of the evidence in these 14 systematic reviews comes from high-income country studies, it probably has fairly high generalisability to the New Zealand setting. Even the systematic review of sugary drink taxes in middle-income countries[15] may have relevance—especially for low-income New Zealand populations who may also be experiencing financial hardship. The overall international evidence is also consistent with the evidence for behaviour changes and health benefits from other health protecting taxes, including tobacco tax for tobacco control[16] and alcohol taxes for alcohol control.[17–19]

Evidence from New Zealand

The recent New Zealand evidence around pricing interventions and food costs is summarised in Appendix Table 2 (covering 12 studies published between 1 January 2010 and 31 March 2019; albeit 13 articles). In general, this evidence is consistent with the international evidence detailed above, by showing that tax and subsidy interventions can potentially improve health. The studies provide some indication that food pricing interventions may have pro-equity impacts (as in Appendix Table 2: Ni Mhurchu et al 2013[20]; Ni Mhurchu et al 2014[21]). In some of this work a modelled 20% tax on all carbonated drinks was estimated to reduce daily energy intakes, avert or postpone 0.2% of all deaths in New Zealand per year, and reduce diabetes and obesity. The impact was estimated to be larger in Māori and Pacific populations compared to non-Māori and non-Pacific populations due to greater responsiveness to food price changes, and among children and young people compared to other ages due to their higher consumption of SSBs.[21] A 20% tax on SSBs was estimated to generate NZ$ 40 million in revenue per year (even allowing for reduced consumption), which could be used for health promotion and healthy food subsidies.

The evidence from randomised controlled trials (RCTs) in New Zealand is more limited. One of these was only a pilot study and involved free fruit in schools.[22] It indicated an increase in fruit intake, albeit in the short-term. Another RCT indicated some

increased purchasing of healthier foods with price discounts in supermarkets.[23]

More generally in terms of health protecting taxes, New Zealand research provides evidence that tobacco taxes are effective for tobacco control,[24,25] and that alcohol taxes contribute to alcohol control.[26,27]

Issues that the New Zealand Government should consider

The above evidence suggests that the New Zealand Government should give serious consideration to food and beverage pricing interventions. This approach would certainly be favoured by many New Zealand-based experts. For example, expert panels have described this country's use of "food fiscal policies" as involving "no action" in both 2014[28] and in 2017.[29] Furthermore, this work found that over 50 expert panel members rated the implementation of a tax on SSBs as a high priority for the Government. Below we consider some of the more specific issues that the Government should consider.

A strategic shift towards health protecting taxes

The Tax Working Group recommended in their 2019 Report,[30] and the Government accepted, the need to apply "corrective taxes" to reduce externalities and mitigate environmental damage caused by industry. We agree with aspects of this approach, but suggest that the prime reason for such taxes should be to achieve societal goals (ie, reducing child obesity), as opposed to merely "correcting" for negative externalities which are often hard to quantify. In Appendix Table 3 we detail the key issues around adopting a new health protecting tax (ie, a sugary drink industry levy) and make comparisons with an existing health protecting tax: tobacco tax.

Introduction of a sugary drink industry levy

Such a tax is increasingly being adopted in other jurisdictions.[31] For New Zealand, it could potentially be modelled on the UK "soft drink industry levy" which triggered substantial reformulation by industry,[32] and which modelling studies have suggested there will be significant health benefits.[33] Such a levy would be in line with current Government's priority on improving wellbeing and protecting child health and would be consistent with New Zealand's approach to taxing tobacco and alcohol. Substitution concerns may be ameliorated by improving access to water (eg, provision of more drinking fountains in public places—which are deficient at present in New Zealand[34,35]). The literature is also suggestive concerning SSB taxes being associated with increased water consumption (eg, in Berkeley, California[36]).

In Australia, the discourse around SSB taxes has been informed by using a citizens' jury,[37] and this methodology could be used in New Zealand. However, public support in New Zealand is already relatively high (eg, 67% in one poll in 2017,[38] up from 52% in an earlier poll in 2015[39]), if the tax revenue is used to fund childhood obesity prevention programmes. Support might be further enhanced, at least among parents of adolescents, if the SSB tax also encompassed sugary alcohol drinks (ie, ready-to-drink beverages).

A Mexican-style junk food tax

There is real-world evidence for such a tax having an impact from Mexico.[40,41] Chile also has such a tax, with modelling work suggesting a likely favourable impact.[42] These type of taxes have the particular advantage of covering a wide range of potentially hazardous foods (ie, processed foods high in sodium, sugar and saturated fat, which are also low in dietary fibre). As such, this broad type of tax may lower the risk of adverse substitution effects (eg, people switching from junk food high in sugar to junk food high in sodium). Including targeted food subsidies and tax revenue recycling could be considered as part of the same policy package (as detailed further below).

Tax revenue recycling to the community

To help address concerns around regressivity of food and beverage taxes, it is ideal that the tax revenue is recycled to the community. This could be by funding free (fully-subsidised) healthy breakfasts and lunches in all low-income schools and early childhood education centres, and ensuring adequate drinking water fountains in all public settings. But as per the experience in Philadelphia with a new SSB

tax, the community might favour directing the tax revenue to non-health areas such as improved childcare services or parental leave.[43] Reductions in GST and income tax are other recycling options. This type of tax revenue recycling can increase community support for raising specific taxes, as seen with British Columbia's successful carbon tax.[44,45]

Further research

We consider that there is clearly enough evidence around food/beverage pricing interventions for policy-makers in New Zealand to seriously consider such approaches, eg, adopting a UK-style sugary drinks industry levy. But while doing so, health authorities should also keep systematically evaluating ongoing research (eg, a RCT in New Zealand suggesting benefits of food tax/subsidy interventions that was published just after the review period used in this article[46] and as part BODE^3 modelling work, as per these publications on dietary interventions[47,48]). Also further research may help fine-tune any New Zealand-adopted interventions so as to maximise the health gain, the cost-effectiveness of intervention application and the impact on reducing health inequalities in the New Zealand setting (ie, especially for Māori, Pasifika and low-income New Zealanders).

Conclusions

In this article we briefly consider recent literature (14 recent systematic reviews and 12 relevant New Zealand studies) on food/beverage taxes and subsidies. This evidence clearly indicates that tax and subsidy interventions have favourable impacts from a health perspective and would seem likely to work in the New Zealand setting. Given this overall picture, such tax/subsidy interventions would be an important evidence-based policy as part of a wider strategy to improve the nutritional health of New Zealanders. A UK-style sugary drinks industry levy should be considered as an initial step. The findings of this review forms the basis of the position of the Health Coalition Aotearoa, a new non-governmental organisation which includes New Zealand health workers and researchers with expertise in nutrition.

Appendix

Appendix Table 1: Systematic reviews (n=14) published on food and beverage taxes and subsidies (ordered by ascending publication year for the period January 2015 to June 2019).*

Main findings of the systematic review	Review citation
Impact of taxes and subsidies dietary behaviours: This systematic review reported that: "there was consistent evidence that taxation and subsidy intervention influenced dietary behaviors." … "To maximise success and effect, this review suggests that food taxes and subsidies should be a minimum of 10 to 15% and preferably used in tandem. Implementation of population-wide polices for taxation and subsides with ongoing evaluation of intended and unintended effects are supported by this review."	Niebylski et al 2015[49]
Tax and subsidy interventions: This systematic review included studies from a range of country types, but the analysis was still dominated by high-income country studies. It reported that fiscal interventions: "on foods can influence consumption of taxed and subsidised foods and consequently have the potential to improve health."	Alagiyawanna et al 2015[50]

Appendix Table 1: Systematic reviews (n=14) published on food and beverage taxes and subsidies (ordered by ascending publication year for the period January 2015 to June 2019) (continued).*

Main findings of the systematic review	Review citation
Inequities impact: This systematic review reported on a range of interventions to promote healthy eating. For price interventions it reported that: "'Price' interventions were most effective in groups with lower SEP [socioeconomic position], and may therefore appear likely to reduce inequalities. All interventions that combined taxes and subsidies consistently decreased inequalities." The authors concluded that: "'Upstream' interventions categorised as 'Price' appeared to decrease inequalities, and 'downstream' 'Person' interventions, especially dietary counselling, seemed to increase inequalities."	McGill et al 2015[51]
Responsiveness by personal characteristics: This systematic review of food and beverage experimental studies reported that: "the difference in price elasticity varied from 0.02 to 2.43 between groups within the same study." Income-related factors were considered: "but the direction of this effect was not clear." The review concluded that: "Patterns in price sensitivity by personal characteristics are complex. General conclusions pertaining to the effects of personal characteristics on price sensitivity are not supported by the evidence, which shows heterogeneity between studies and populations."	Mizdrak et al 2015[13]
Inequities impact: This systematic review concluded that: "Fiscal measures had consistently neutral or positive impacts on inequities."	Olstad et al 2016[11]
Inequities and SSB tax: This systematic review concluded that: "Based on the available evidence, a tax on SSB will deliver similar population weight benefits across socioeconomic strata or greater benefits for lower SEP [socioeconomic position] groups. An SSB tax is shown to be consistently financially regressive, but to a small degree."	Backholer et al 2016[12]
Taxes on SSBs: This systematic review was on SSB taxation in middle-income countries (nine studies). It reported: "estimates for own-price elasticity ranged from -0.6 to -1.2, and decreases in SSB consumption ranged from 5 to 39 kilojoules per person per day given a 10% increase in SSB prices. The review found that milk is a likely substitute…". The review concluded that: "taxing SSBs will increase the prices of SSBs" and that "taxing SSBs will also reduce net energy intake by enough to prevent further growth in obesity prevalence, but not to reduce population weight permanently."	Nakhimovsky et al 2016[15]
Salt tax: This systematic review concluded that: "Tax and community based counselling could, each typically reduce salt intake by 0.3g/day." But this tax impact was considered likely to have less impact than: "comprehensive strategies involving multiple components (reformulation, food labelling and media campaigns)."	Hyseni et al 2017[52]
Impact of taxes and subsidies on consumption: This systematic review and meta-analysis reported that: "In pooled analyses, a 10% decrease in price (ie, subsidy) increased consumption of healthful foods by 12% (95%CI=10–15%; N=22 studies/intervention arms) whereas a 10% increase price (ie, tax) decreased consumption of unhealthful foods by 6% (95%CI=4–8%; N=15)." … "Each 10% price increase reduced sugar-sweetened beverage intake by 7% (95%CI=3–10%; N=5)"…"These prospective results, largely from interventional studies, support efficacy of subsidies to increase consumption of healthful foods; and taxation to reduce intake of unhealthful beverages and foods. Use of subsidies and combined multicomponent interventions appear most effective."	Afshin et al 2017[53]

Appendix Table 1: Systematic reviews (n=14) published on food and beverage taxes and subsidies (ordered by ascending publication year for the period January 2015 to June 2019) (continued).*

Main findings of the systematic review	Review citation
Impact of SSB taxes: Although this was a systematic review of health-related taxes in general, it did also consider specific ones, eg, SSB taxes. It reported that: "Findings demonstrate that high tax rates on sugar-sweetened beverages are likely to have a positive impact on health behaviours and outcomes…". The review concluded that: "If the primary policy goal of a health tax is to reduce consumption of unhealthy products, then evidence supports the implementation of taxes that increase the price of products by 20% or more. However, where taxes are effective in changing health behaviours, the predictability of the revenue stream is reduced." Additionally, "…earmarking health taxes for health spending tends to increase public support so long as policymakers follow through on specified spending commitments."	Wright et al 2017[54]
Price promotions: This systematic review covered pricing interventions with these mainly around promotion of foods (primarily fruit and vegetables). It reported that: "Pricing interventions generally increased stocking, sales, purchasing, and consumption of promoted foods and beverages."	Gittelsohn et al 2017[55]
Impact of SSB taxes: This systematic review reported that: "Findings indicated that purchases or sales of SSBs decreased significantly with taxation amounts of 8% (Berkeley, CA) and 10% (Mexico)." The review found one study that "found no effect on sales of SSBs" and 12 studies "resulting in a decrease in either purchasing behavior or sales or intent behaviour". The review concluded: "Taxation significantly influences planned purchases and increases the probability of the purchase of healthy beverages. SSB taxes have the potential to reduce calorie and sugar intake, but further research is needed to evaluate effects on diet quality."	Redondo et al 2018[56]
Impact of SSB taxes: This systematic review and meta-analysis reported that: "The equivalent of a 10% SSB tax was associated with an average decline in beverage purchases and dietary intake of 10.0% (95%CI: -5.0% to -14.7%, n=17 studies, 6 jurisdictions) with considerable heterogeneity between results (I^2=97%). The equivalent of a 10% SSB tax was also associated with an average 1.9% increase in total untaxed beverage purchases and dietary intake (eg, for bottled water), but this was not statistically significant (95%CI: -2.1% to 6.1%, n=6 studies, 4 jurisdictions)."… The review concluded that: "Based on real-world evaluations, SSB taxes introduced in jurisdictions around the world appear to have been effective in reducing SSB purchases and dietary intake."	Teng et al 2019[14]
Sugar reduction interventions—economic tools: This Cochrane systematic review included a section on economic tools (seven studies). The categories of interventions included were: (i) Price increases on SSBs; (ii) Financial incentives to purchase low-calorie beverages implemented through supermarket loyalty cards; and (iii) Price discounts on low-calorie beverages in community stores. The overall results were: "we found moderate-certainty evidence that price increases on SSBs are associated with decreasing SSB sales. For price discounts on low-calorie beverages reported effects on SSB sales varied." One New Zealand study was included in this review, ie, Ni Mhurchu et al 2010[23] (see Table A2). This review noted that there is to be a forthcoming Cochrane Review on taxation of SSBs.	von Philipsborn et al 2019[57]

Note: *The search was conducted in PubMed for the period 1 January 2015 to 15 June 2019 with a range of search terms: eg, "systematic review" and food/beverage, price/subsidy. But the Teng et al study was identified via co-author involvement in this particular study and the Cochrane review was identified via media reporting. We excluded the systematic review by Sisnowski et al 2015[58] as it did not cover impacts (but rather the popularity of taxation relative to other interventions).

Appendix Table 2: New Zealand studies (n=12) relating to food pricing and food costs, published in the peer-review literature (for the period January 2010 to March 2019; ordered by publication year).*

Main findings of the New Zealand study	Study citation/s
RCT of price changes: The SHOP randomised controlled trial in eight New Zealand supermarkets reported the impact of price discounts of 12.5% (the same as a removal of GST at this time). At six months there was no impact on saturated fat purchased. "However, those subjects who were randomly assigned to receive price discounts bought significantly more predefined healthier foods at 6 mo (11% more; mean difference: 0.79 kg/wk; 95% CI: 0.43, 1.16; P<0.001) and 12 mo (5% more; mean difference: 0.38 kg/wk; 95% CI: 0.01, 0.76; P=0.045)." The authors concluded: "the significant and sustained effect of discounts on food purchases suggests that pricing strategies hold promise as a means to improve population diets."[23] In terms of the impact by ethnicity, the authors wrote in a separate paper: "While a statistically significant variation by ethnicity in the effect of price discounts on food purchasing was found, the authors caution against a causal interpretation due to likely biases (eg, attrition) that differentially affected Māori and Pacific people."[59]	Ni Mhurchu et al 2010[23] Blakely et al.2012[59]
Price sensitivity: This study of New Zealand food expenditure data reported that: "Own-PE [price elasticities] estimates (with two exceptions) ranged from -0.44 to -1.78. Cross-PE estimates were generally small; only 31% of absolute values were greater than 0.10. Excluding the outlier 'energy drinks', nine of 23 food groups had significantly stronger own-PEs for the lowest versus highest income quintiles (average regression-based difference across food groups -0.30 (95% CI -0.62 to 0.02)). Six own-PEs were significantly stronger among Māori; the average difference for Māori: non-Māori across food groups was -0.26 (95% CI -0.52 to 0.00)." The authors concluded that: "Food pricing policies have potential to improve population diets. The greater sensitivity of low-income households and Māori to price changes suggests the beneficial effects of such policies on health would be greatest for these groups."	Ni Mhurchu et al 2013[20]
Low cost and sustainable diets: This modelling study: "identified daily dietary patterns that met key nutrient requirements for as little as a median of NZ$ 3.17 per day (US$ 2.41/d) (95% simulation interval [SI]=NZ$ 2.86 to 3.50/d). Diets that included 'more familiar meals' for New Zealanders, increased the cost. The optimised diets also had low GHG [greenhouse gas] emission profiles compared with the estimate for the 'typical New Zealand diet' …" … "All of the optimised low-cost and low-GHG dietary patterns had likely health advantages over the current New Zealand dietary pattern, ie, lower cardiovascular disease and cancer risk."…"These results could help guide central and local government decisions around which foods to focus policies on. That is which foods are most suitable for: food taxes (additions and exemptions); healthy food vouchers and subsidies;…"	Wilson et al 2013[60]
Cost of low salt diets: In this modelling study that constrained daily food cost to <NZ$ 9/day [d], it was possible to have a diet with the sodium intake levels below the 2,300 mg/d (5.8 g salt/d) recommended maximum. The authors concluded that: "These results provide some reassurance for the feasibility of substantially reducing population sodium intake given currently available low-cost foods and while maintaining some level of familiar meals."	Wilson et al 2013[61]

Appendix Table 2: New Zealand studies (n=12) relating to food pricing and food costs, published in the peer-review literature (for the period January 2010 to March 2019; ordered by publication year) (continued).*

Main findings of the New Zealand study	Study citation/s
Access to lower cost food at fresh food markets: This modelling study on access to markets reported that: "farmers' markets provided fairly poor access for the total population: 7% within 12.5 km (15 min driving time); 5% within 5km; and 3% within 2km. Modelling the optimal distribution of the 48 markets substantially improved access for the most deprived groups…"…"Access for Māori also improved: 22% (vs 7%) within 12.5km…" The authors concluded that: "These results highlight the potential for improving farmers' market locations to increase accessibility for groups with low FV [fruit and vegetable] consumption. Given that such markets are easily established and relocated, local governments could consider these results to inform decisions, including subsidies for using government land and facilities."	Pearson & Wilson 2013[62]
Fruit and vegetable prices in fresh food markets: This study of prices in markets (including farmers markets) reported that: "In these locations general markets appear to be providing some substantially lower prices for fruit and vegetables than supermarkets. They also appear to be depressing prices in neighboring supermarkets. These results, when supplemented by other needed research, may help inform the case for interventions to improve access to fruit and vegetables, particularly for low-income populations."	Pearson et al 2014[63]
SSB tax modelling: This modelling study considered the impact of a 20% tax on all carbonated drinks. It reported that this intervention would reduce obesity and diabetes and avert or postpone 0.2% of all deaths in New Zealand a year. The impact was estimated to be larger in Māori and Pacific populations (due to greater price sensitivity to food price changes, and among children and young people due to their higher consumption of SSB). It was estimated that this 20% tax would generate NZ$ 40 million in revenue per year in revenue.	Ni Mhurchu et al 2014[21]
Salt tax modelling: A modelled salt tax for New Zealand was estimated to generate large health gains and cost savings. However, the benefits were not as much as a sinking lid on the sodium supply down to recommended levels, or when compared to a subsequently studied reformulation intervention (using potassium salts) for sodium reduction.[4] "Also the salt tax would raise revenue (up to NZ$ 452 million/year)".	Nghiem et al 2015[64]
Tax and subsidy modelling: This modelling study estimated that: "a 20% subsidy on fruit and vegetables would result in 560 (95% uncertainty interval, 400 to 700) DPP [deaths prevented or postponed] each year (1.9% annual all-cause mortality). A 20% tax on major dietary sources of saturated fat would result in 1,500 (950 to 2,100) DPP (5.0%), and a 20% tax on major dietary sources of sodium would result in 2,000 (1300 to 2,700) DPP (6.8%). Combining taxes on saturated fat and sodium with a fruit and vegetable subsidy would result in 2,400 (1,800 to 3,000) DPP (8.1% mortality annually). A tax on major dietary sources of greenhouse gas emissions would generate 1,200 (750 to 1,700) DPP annually (4.0%). Effects were similar or greater for Maori and low-income households in relative terms." The authors concluded that: "Health-related food taxes and subsidies could improve diets and reduce mortality from diet-related disease in New Zealand. Our study adds to the growing evidence base suggesting food pricing policies should improve population health and reduce inequalities, but there is still much work to be done to improve estimation of health impacts."	Ni Mhurchu et al 2015[65]

Appendix Table 2: New Zealand studies (n=12) relating to food pricing and food costs, published in the peer-review literature (for the period January 2010 to March 2019; ordered by publication year) (continued).*

Main findings of the New Zealand study	Study citation/s
Stakeholder views (taxes/subsidies): Based on 20 New Zealand stakeholder interviews, this study found that: "According to key stakeholders there appears to be little appetite for taxes on foods high in saturated fat or salt in New Zealand. Stakeholders largely agreed that a tax on sugar-sweetened beverages (SSBs) and a subsidy on fruit and vegetables were both feasible and likely acceptable. There was strong support for starting with a SSBs tax, possibly framed around protecting children and dental health."…"A tax on SSBs and a subsidy on fruit and vegetables, possibly in tandem, could be part of the solution in New Zealand."	Signal et al 2018[66]
Cost of New Zealand diets/GST exemption: "The average cost of healthy household diets was NZ$27 more expensive than the average cost of current diets, but 25.8% of healthy diets were cheaper than the average cost of current diets. This cost differential could be reduced if fruits and vegetables became exempt from Goods and Services Tax. Healthy diets were cheaper with an allowance for discretionary foods and more expensive when including takeaway meals. For Māori and Pacific households, healthy diets were on average $40 and $60 cheaper than current diets due to large energy intakes." (That is, a healthier diet with a lower energy intake would be cost-lowering in these populations).	Vandevijvere et al 2018[67]
Cost of New Zealand diets/GST removal: "The modelled healthy diet was cheaper than the current diet for the total population (3.5% difference) and Pacific households (4.5% difference) and similar in cost for Māori households (0.57% difference). When the diets were equivalent in energy, the healthy diet was more expensive than the current diet for all population groups (by 8.5% to 15.6%). For households on the minimum wage, the diets required 27% to 34% of household income, and if receiving income support, required 41% to 52% of household income."…"Both the modelled healthy and current diets are unaffordable for some households as a considerable portion of income was required to purchase either diet. Policies are required to improve food security by lowering the cost of healthy food or improving household income." Gifting and gathering of kai was estimated to reduce the cost of a healthy diet by 10%. Removing GST from fruit and vegetables was estimated to reduce the cost of a healthy diet by 5%, and removing GST off core foods would reduce the cost by 13%.	Mackay et al 2018[68]

Note: * The search was conducted in PubMed for the period 1 January 2010 to 31 March 2019. Search terms included: "Zealand" and food/beverage and price/subsidy/cost.

Appendix Table 3: Primary and supportive issues when considering a new health protecting tax (ie, in this case on sugary drinks) and a comparison with an established tax in New Zealand on tobacco.

Key issue	Tobacco tax (in place in New Zealand and regularly increased)	Sugary drinks industry levy (a potential policy tool for New Zealand)
Primary issue: Does the tax work to help achieve a societal/health goal?	**Yes:** Tobacco tax is a very effective and well-established tobacco control tool.[16] It is probably the most important single mechanism used in New Zealand to make progress towards the New Zealand Government's goal[69] of a Smokefree Aotearoa by 2025.	**Yes:** As per a meta-analysis, SSB taxes appear to be effective in reducing purchases and dietary intake.[14] This approach would therefore help achieve the New Zealand Government's "Childhood obesity plan"[70] that includes the aim of preventing obesity in children and young people. The New Zealand Government's budget of 2019 was titled a "Wellbeing budget" which also included the relevant goals of "Improving child wellbeing" and "Supporting Māori and Pasifika aspirations".[71]
Supportive issue: Does the tax help to address negative externalities including harm to others?	**Yes:** Part of the justification for a tobacco tax is the harm to non-smokers (and fetuses) from second-hand smoke exposure. Sometimes other negative externalities are detailed in the New Zealand setting eg, litter from tobacco packaging[72] and nuisance impacts.[73]	**Yes:** In the case of sugary drinks, the extra health costs generated by the users of SSBs that are imposed on the public health system are appropriately considered as a "fiscal externality" (albeit also described as a "moral hazard cost").[74]
Supportive issue: Is the cost of administering the tax relatively low?	**Yes:** This is such a well-established tax with well-refined collection mechanisms that administration costs appear to be very low.	**Yes:** Other countries with SSB taxes have not reported administration costs as a major concern and New Zealand has a very long experience of taxing a wide range of alcoholic beverages.
Supportive issue: Can any downsides of the tax be ameliorated?	**Yes:** While there is the potential to increase financial harm to smokers who don't quit or cut down, this can be ameliorated via: welfare benefits; permitting access to very much cheaper e-cigarettes (currently the case in New Zealand); providing free quitting services (eg, the New Zealand Quitline); and potentially by subsidising e-cigarettes (not yet in place in New Zealand).	**Yes:** There is a slight potential for increased financial hardship for those in poverty who don't reduce their SSB intake after the tax. But this can be ameliorated by: (i) promoting water as a substitute (eg, done in many New Zealand schools); (ii) improving access to drinking water fountains in public places (being done in parts of New Zealand); and (iii) requiring warning labels on SSBs so that people are informed of the hazard (not yet in place in New Zealand).

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Hypothyroidism causing bilateral lower-limb compartment syndrome

Cameron Schauer, Young E Koo, Stephen McBride, Alpesh Patel

Case report

A 17-year-old Indian man was referred to hospital with bilateral anterior leg pain and difficulty walking after a forty-minute session of recreational basketball 36 hours prior. There was no history of trauma. He took no regular medications. His family history was significant for maternal Grave's disease and two grandparents having type 2 diabetes mellitus.

Physical examination revealed diffuse erythema and non-pitting oedema over the anterior tibial compartments bilaterally, minimal tenderness on palpation, restricted passive dorsiflexion of the ankles and a high stepping gait with bilateral foot drop. Power was grade 1/5 in the ankle dorsiflexors with paraesthesiae of the dorsum of both feet. Ankle and toe plantarflexion were normal with symmetrical deep tendon reflexes in both lower extremities. Peripheral pulses were intact. His hair, skin and nails were normal. There were no palpable neck masses or thyromegaly. The remainder of the systemic examination was also normal, including cardiorespiratory examination and clinical euvolemia.

Laboratory investigations revealed a CK >22,000IU/L (normal 60–220), thyroid-stimulating hormone (TSH) >100mIU/ml (0.3–4.0) and free thyroxine (T4) <3.0pmol/L (10–20). Urinanalysis showed myoglobin 1.9mg/l (<1) and urine haemoglobin pigments. Serum creatinine was normal, and anti-thyroid peroxidase (TPO) antibodies were negative. He was provisionally diagnosed with hypothyroidism-induced myositis with rhabdomyolysis. He commenced thyroxine replacement therapy, and was treated aggressively with intravenous fluids.

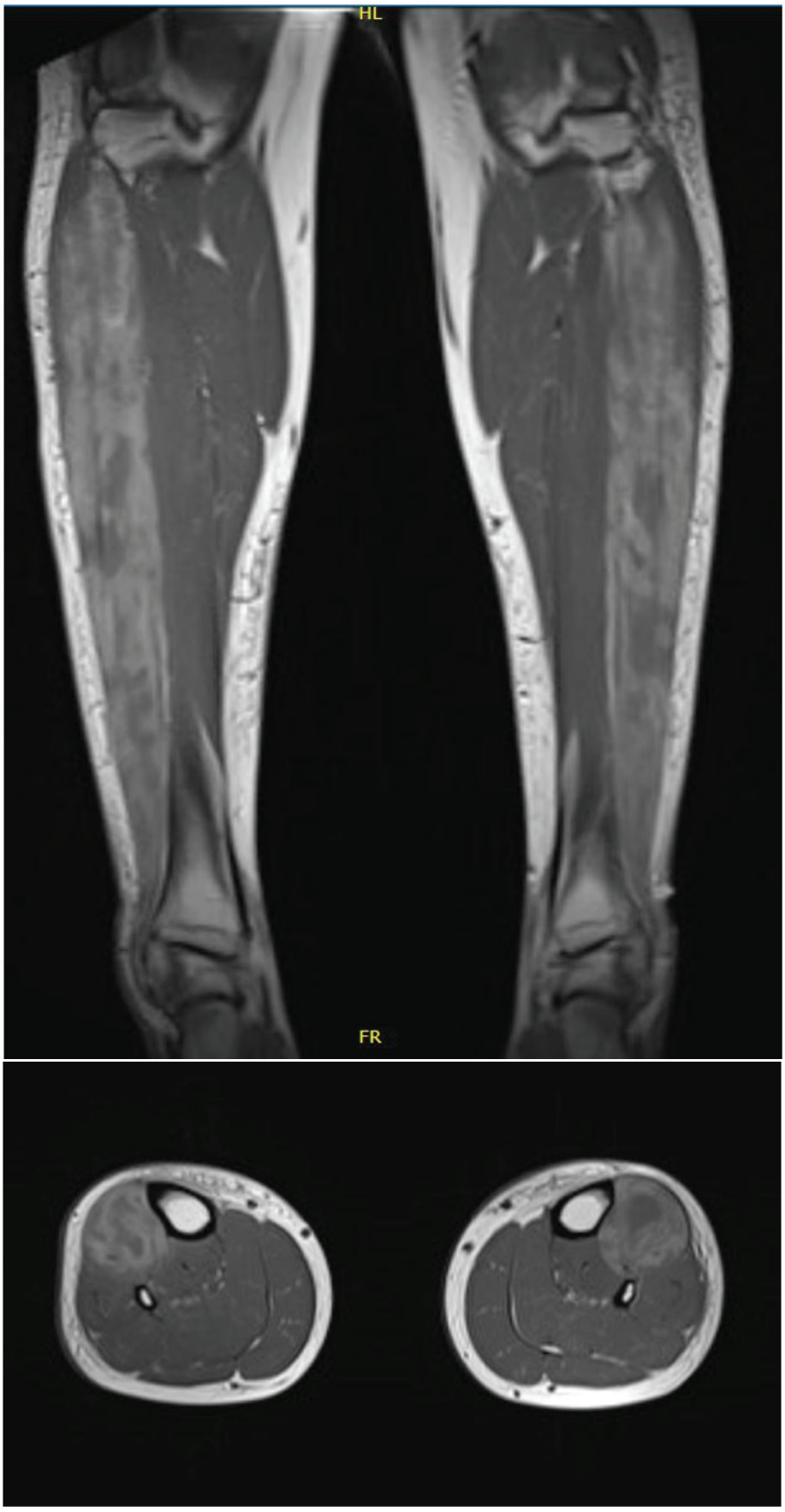
The following day pain worsened with increasing analgesia requirement. Subsequent magnetic resonance imaging (MRI) of the lower limbs confirmed high signal within the muscles of the anterior compartment without any fluid collection, and bulging of the anterior deep fascia (Figure 1). Due to the prolonged duration of symptoms, high likelihood of underlying necrotic tissue with little chance of recovery and risk of post-operative infection, orthopaedic consult advised against performing a fasciotomy. He was discharged for outpatient follow-up with bilateral ankle-foot orthoses and ongoing thyroxine replacement. At six-week follow-up, the anterior compartment discomfort had largely resolved. Thyroid function tests normalised within eight weeks. At six-month follow up, the patient had ongoing bilateral foot drop and is awaiting bilateral tibialis anterior tendon transfers.

Discussion

Although abnormality in thyroid function is a common endocrine disorder, compartment syndrome associated with severe hypothyroidism is rare.[1–6] Notably all but one case[3] was managed operatively; however, functional outcome was comparable among the other six patients described in the literature. Our case is the youngest adult presentation reported.

The pathophysiology of hypothyroidism-induced myopathy and associated compartment syndrome is incompletely understood. Thyroid hormone is known to exert influence on muscle metabolism, with low levels inducing a mitochondrial driven metabolic myopathy.[7] Additional effects include alteration of the actin-myosin unit

Figure 1: T1 sequence magnetic resonance imaging of the lower limbs demonstrating areas of high signal within the muscles in the anterior compartments, consistent with haemorrhage. There is bulging of the anterior deep fascia overlying the anterior compartment and bowing of the interosseous membrane, consistent with increased pressure within the anterior compartment.



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with changes in regeneration, proliferation and contractility. Further differences in glycosaminoglycan metabolism may lead to abnormal deposition, accumulation and damage. We hypothesise that the combination of abnormal metabolism and function of the muscle cell, coupled with impaired vascularity, lymphatic drainage and oedema due to stimulated fibroblasts predisposed our patient to compartment syndrome, which was precipitated by exercise. Furthermore, increased extracellular fluid accumulation associated with hypothyroidism may be more substantial in the lower limbs due to gravitational effect accounting for presentation in the legs alone.

In contrast to an acute compartment syndrome, our patient had a subacute course over a period greater than 72 hours. Severe pain is often considered a hallmark of compartment syndrome; however, was absent in our patient at the time of presentation.

Additional history obtained from the patient in retrospect did reveal he had suffered from non-specific fatigue for "some years". It is unclear when he became so grossly hypothyroid, given his normal pubertal development and height. He did not appear to suffer from any additional systemic features of profound hypothyroidism, specifically reduced cardiac function, reduced respiratory or exercise capacity, dermatological or neurological abnormality. The cause for his hypothyroidism is thought most likely to be chronic autoimmune (Hashimoto's) thyroiditis, despite negative anti-TPO antibodies. He denied any recent flu-like illnesses, fevers or neck discomfort that would suggest acute post viral thyroiditis as the potential aetiology.

This rare case illustrates two points that are generally applicable. Firstly, patients presenting with myopathic symptoms, even in the absence of other typical hypothyroid manifestations should have their thyroid function checked to ensure that occult underlying hypothyroidism is not present. Secondly, early recognition of compartment syndrome that may present in patients without classic risk factors or symptoms is crucial. Symptoms such as an acute foot drop may denote the presence of compartment syndrome and prompt rapid surgical assessment may influence outcomes if recognised early.

Competing interests:
Nil.

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Incidence of Takotsubo syndrome vs acute myocardial infarction in New Zealand (ANZACS-QI 45)

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Takotsubo syndrome (TS) (also known as apical ballooning syndrome) is characterised by acute but usually rapidly reversible left ventricular (LV) dysfunction with distinct wall motion abnormalities, predominantly affecting postmenopausal women.[1,2] At presentation TS typically mimics acute myocardial infarction (MI). The reported frequency of TS is 1.0–2.5% in patients presenting with suspected acute MI, and 12% in women presenting with anterior ST-elevation myocardial infarction (STEMI).[1] The incidence of TS has been increasing internationally, likely a consequence of an improved awareness of the existence of this syndrome and easier access to early echocardiography and coronary angiography.[2–4] Recent international reports suggest that 1–3% of patients (up to 6–9% if only women are considered) with suspected acute coronary syndrome (ACS) have TS.[3,5]

This study describes the incidence of TS in New Zealand in comparison to MI. Two hundred and fifty-nine TS patients presented to five of the major tertiary hospitals in New Zealand (Middlemore Hospital, Auckland City Hospital, North Shore Hospital, Christchurch Hospital and Dunedin Hospital) between January 2015 and June 2018 were included. Only patients who underwent angiography (either cardiac CT angiography [n=27] or invasive coronary angiography [n=232]) were included. TS was defined using the International Takotsubo Diagnostic Criteria (InterTAK Diagnostic Criteria).[6] TS cases were subdivided according to the presentation ECG—ST-elevation TS (STE-TS) or non-ST-elevation TS (NSTE-TS).[7]

The MI cohort comprised of all patients who presented to the participating hospitals with acute MI between over the same time period. A total of 11,900 of patients (8,405 men and 3,495 women) with STEMI and non-ST-myocardial infarction (NSTEMI) were identified. The data was extracted from the All New Zealand Acute Coronary Syndrome Quality Improvement (ANZACS-QI) registry.[8] This registry captured almost all New Zealand public hospital coronary angiography procedures, including those associated with an acute coronary syndrome (ACS) diagnosis.[9] Only patients who had coronary angiography were included to ensure comparability with the TS cohort.

The demographic characteristics of the TS and MI populations are summarised in Table 1; 96.1% (n=249) of the TS patients were women (n=249, 96.1%) with median age of 66 years (IQR 57–73 years). They were slightly younger than MI females. Majority of both cohorts were European. Two-thirds of female TS cohort presented without ST-segment elevation and they had less conventional cardiovascular risk factors compared to the female MI cohort. A stressful trigger (defined as an unusual emotional or physical stress occurring before symptom onset) was identified in 190 patients (73.4%); 117 had an emotional stressor and 73 reported a physical stressor (defined as medical conditions that trigger TS).

A reduced left ventricular ejection fraction (LVEF), defined as LVEF<50% was noted in 84.3% of TS women on admission but in only 28.5% of women with MI. More than half of women with TS had LVEF<40% on admission. Thirty-one female TS patients had coronary artery disease documented on either invasive coronary angiography (n=6) or CT coronary angiography (n=25). Of note, 19.9%

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Table 1: Clinical characteristics of patients with Takotsubo syndrome versus patients with myocardial infarction.

	Female		**P-value**	**Male†**	
	TS (n=249)	MI (n=3,495)		TS (n=10)	MI (n=8,405)
Age			<.001		
Median (IQR)	66 (57–73)	69 (59–77)		55 (53–73)	64 (55–72)
Ethnicity			<.001		
European	196 (78.7)	2,492 (71.3)		8 (80.0)	6,038 (71.8)
Māori	33 (13.3)	330 (9.4)		1 (10.0)	531 (6.3)
Pacific Islanders	10 (4.0)	337 (9.6)		1 (10.0)	640 (7.6)
Asian	10 (4.0)	336 (9.6)		0 (0)	1,196 (14.2)
Hypertension			<.001		
Yes	130 (52.2)	2,233 (63.9)		3 (30.0)	4,637 (55.2)
No	119 (47.8)	1,262 (36.1)		7 (70.0)	3,768 (44.8)
Diabetes			<.001		
Yes	32 (12.9)	960 (27.5)		1 (10.0)	1,983 (23.6)
No	217 (87.1)	2,535 (72.5)		9 (90.0)	6,422 (76.4)
Dyslipidaemia*			<.001		
Yes	96 (61.4)	1,294 (37.0)		4 (40.0)	3,667 (43.6)
No	153 (38.6)	708 (20.3)		6 (60.0)	1,862 (22.2)
Missing data	-	1,493 (42.7)		-	2,876 (34.2)
Current smoker			<.001		
Yes	26 (10.4)	748 (21.4)		4 (40.0)	2,090 (24.9)
No	223 (89.6)	2,747 (78.6)		6 (60.0)	6,315 (75.1)
Prior CVD			<.001		
Yes	22 (8.8)	955 (27.3)		1 (10.0)	2,478 (29.5)
No	227 (91.2)	2,540 (72.7)		9 (90.0)	5,927 (70.5)
Type of ACS/TS			0.196		
STEMI/ STE-TS	82 (32.9)	1,016 (29.1)		4 (40.0)	2,816 (33.5)
NSTEMI/ NSTE-TS	167 (67.1)	2,479 (70.9)		6 (60.0)	5,589 (66.5)
Stressor on admission		-	-		-
Yes	181 (72.7)			9 (90.0)	
No	68 (27.3)			1 (10.0)	
Type of stressor		-	-		-
Emotional	113 (45.4)			4 (40.0)	
Physical	68 (27.3)			5 (50.0)	
No stressor	68 (27.3)			1 (10.0)	
Angio findings			<.001		
Normal/mild disease	218 (87.6)	696 (19.9)		9 (90.0)	659 (7.8)
1–2 vessel disease	29 (11.6)	1,986 (56.8)		1 (10.0)	5,045 (60.0)
3VD/LMS	2 (0.8)	813 (23.3)		0 (0)	2,701 (32.1)
LVEF assessment*			<.001		
Normal (EF>50%)	39 (15.7)	1,793 (51.3)		2 (20.0)	3,881 (46.2)
Mild (40–49%)	68 (27.3)	526 (15.1)		2 (20.0)	1,590 (18.9)
Moderate/severe (<40%)	142 (57.0)	471 (13.5)		6 (60.0)	1,479 (17.6)
No EF available	-	705 (20.2)		-	1,555 (18.5)

*The missing data has been excluded in the comparison.
†P values for comparison between men with TS and men with MI are not given due to the small number of men with TS.
ACS, acute coronary syndrome; TS Takotsubo syndrome; CVD, cardiovascular disease; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST elevation myocardial infarction; STE-TS, ST elevation Takotsubo syndrome; NSTE-TS, Non ST elevation Takotsubo syndrome; 3VD; 3 vessels disease; LMS, left main stem disease, LVEF, left ventricular ejection fraction.

of women with a diagnosis of MI had no obstructive coronary artery disease (defined as <50% epicardial coronary stenosis in all major epicardial coronary arteries).

The numbers of women with TS and MI in five of the major tertiary hospitals in New Zealand are presented in Table 2. Between 1 January 2015 and 30 June 2018, 259 patients (249 females and 10 men) had TS. Over this period, 3,495 women and 8,405 men presented with MI. The proportions with TS, of those with MI or TS, were therefore 6.6% for women and 0.1% for men. Two-thirds of the female TS patients (n=167) presented without ST-segment elevation. The proportion of TS in women with NSTEMI/NSTEACS was 6.3%, and in women with STEMI/STE-TS was 7.5%. Overall, 2.2% of the MI/TS cohort had TS. The InterTAK investigators reported an overall prevalence of 4.1% in an ACS registry in Zurich hospitals[10] and the incidence of TS in women with suspected MI has been found to be 5.9–7.5%.[11] Even in this contemporary New Zealand MI cohort, women were three times more likely than men to have non-obstructive coronary artery disease. At least some of those patients may have been misdiagnosed as ACS, particularly if echocardiography was delayed. TS diagnosis requires identification of typical LV wall motion abnormalities, which are relatively transient. If imaging is delayed and they have resolved, the default diagnosis is likely to be MI. If a third of these 696 women were reclassified as TS it would nearly double our estimated number with TS.

Table 2: Numbers of MI and TS cases and the proportion with TS—female sub-cohort and overall cohort (female and male).

Hospitals	**Female**			**All**		
	TS (n=249)	MI (n=3,495)	% TS	TS (n=259)	MI (n=11,900)	% TS
Auckland						
ST-segment elevation	14	160	8.0%	14	691	2.0%
Non-ST-segment elevation	28	361	7.2%	29	1,211	2.3%
All	42	521	7.5%	43	1,902	2.2%
Middlemore						
ST-segment elevation	21	185	10.2%	23	728	3.1%
Non-ST-segment elevation	52	471	9.9%	55	1,644	3.2%
All	73	656	10.0%	78	2,372	3.2%
North Shore						
ST-segment elevation	10	229	4.2%	11	890	1.2%
Non-ST-segment elevation	11	677	1.6%	12	2,138	0.6%
All	21	906	2.3%	23	3,028	0.8%
Christchurch						
ST-segment elevation	27	258	9.5%	27	929	2.8%
Non-ST-segment elevation	58	598	7.5%	59	1,943	2.9%
All	85	856	7.8%	86	2,872	2.9%
Dunedin						
ST-segment elevation	10	184	5.2%	11	594	1.8%
Non-ST-segment elevation	18	372	4.6%	18	1,132	1.6%
All	28	556	4.8%	39	1,726	2.2%

TS, Takotsubo syndrome; MI, myocardial infarction.

The proportion with TS varies among the five hospitals, ranging between 2.3–10.0%. The proportion of women with STEMI/STE-TS who had TS was 4.2–10.2%, and of those with NSTEMI/NSTE-ACS it was 1.6–9.9%. Of note, the proportion of women at Middlemore Hospital with TS was 10.0%. This seems high compared to the other four hospitals. Because Middlemore Hospital has had an active interest in TS for many years and maintains a prospective registry, it is likely that very few TS diagnoses are missed, which may in part account for the higher proportions. The proportion of TS/MI patients with TS in Christchurch hospital was 7.8%. Following the major Christchurch and Kaikoura earthquakes, Christchurch Hospital has seen unprecedented case clusters of TS, which accounts for the higher level observed.[12]

Two extensive overviews of American TS cohorts found that anxiety and chronic stress were both associated with significantly higher odds of developing TS.[13,14] Depression has also been reported to be associated with higher odds of developing TS. The increased prevalence of premorbid psychiatric diagnoses, particularly anxiety disorders and depression in TS patients suggests a potential link between neuropsychiatric disorders and TS. In particular, the Canterbury earthquakes had significant adverse impact on mental health, which could account for the high incidence observed.

It is difficult to estimate the frequency of TS in the general population. Indeed, the true incidence of TS may still be underestimated because TS may be misdiagnosed as ACS. It is still unclear if the extent to which the growing numbers increase in incidence compared with prior reports reflects an increasing awareness of this syndrome by clinicians or if it is in fact a true rise in its incidence. Nevertheless, it is worth remembering that TS is not rare and heightened awareness of this condition will likely lead to a higher reported incidence.

Competing interests:
Nil.

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Pharmaceutical opioid changes risk overdose increase in New Zealand

Rhys Ponton, Jason George

In late 2019, a change was initiated to limit long acting (slow-release) morphine preparations available in New Zealand to one product, m-Eslon capsules. The previously funded slow-release tablets, Arrow Morphine LA, will no longer be available once existing supplies are exhausted (as of the beginning of March, both the 10mg and 100mg are no longer available). As a bioequivalent product, this should have minimal impact on patients, and indeed brings some benefits for patients, notably those with swallowing difficulties.

New Zealand has a unique and peculiar illicit drug market in that it lacks true illicit opioids; heroin is sporadically available, but is generally uncommon and not widespread. The opioid market is largely filled with diverted pharmaceutical opioids (opioid agonist treatments and analgesics) including morphine tablets. People who misuse opioids often seek to reproduce or simulate the euphoriant effects of heroin, in New Zealand this is achieved by converting morphine from these pharmaceutical preparations into a crude form of heroin by heating a crushed morphine tablet with acetic anhydride, which converts the morphine to mono- or diacetyl-morphine (heroin), depending on the skill of the 'cook'.

m-Eslon capsules have been approved for the market in New Zealand since 2002. During this period, supplies of this formulation inevitably have reached the illicit market, but they are not quite as straightforward to convert to heroin as the tablet formulations. As a consequence, the illicit market has continued to favour the tablet product. The move to the slow-release capsule form is likely to have unintended consequences for people who misuse these drugs.

The conversion of a tablet, by the user, ensures a product of consistent quantity and dosage (it is assumed a person using the same process each time will produce a similar yield of drug consistently). As the tablet formulation becomes unavailable for the illicit market, anecdotal reports suggest that in some parts of New Zealand the content of the morphine capsules is now reaching the illicit market in the form of pre-manufactured heroin powder, in contrast to the standard pharmaceutical formulations previously traded. The conversion to heroin is being undertaken by those dealing in the market. Powdered heroin is of concern as it means that it will be harder for users to measure or gauge doses. There may be unintended consequences if batches of these products are made to varying standards, and there is a significant risk if the product is diluted, or cut for profit by unscrupulous individuals.

A shift in the availability of sought after products also lends itself to changes in drug user preference. A significant concern is that a shift to illicit powdered drugs replacing pharmaceutical formulations leaves the potential of powdered fentanyl being added to the supply chain as is commonly seen in many other Western nations with a market for heroin. Anecdotal reports suggest illicit fentanyl has recently been sold as heroin in at least one major New Zealand city. The change in the available morphine formulations could have the unintended consequence of opening the illicit opioid market for widespread fentanyl use to develop in New Zealand. The major risk posed is that of opioid overdose.

Ironically, until now, the presence of morphine tablets on the market has ensured that people are able to accurately measure their opioid intake and this is likely to have contributed to New Zealand's relatively low overdose death rate (approximated to be between 35 and 47 deaths a year,[1] although

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thought to be higher[2]). In comparison, the overdose rate in Australia, where the illicit opioid market includes powdered heroin, is significant: 438 heroin-induced deaths in 2018[3] with pharmaceutical opioids compounding these.

New Zealand is woefully underprepared for the arrival of illicit fentanyl or a shift to an illicit powdered opioid market. Naloxone availability is poor, despite legislative change in 2016 to allow its supply to those at risk.

This letter is not an argument for a return to the use of the tablet form or promote the misuse of pharmaceutical products. It intends to highlight potentially significant overdose concerns and raise awareness within the health community.

Competing interests:
Nil.

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Nutrition guidelines for dental care vs the evidence: is there a disconnect?—a response

Harriette Carr

The Ministry of Health considers that parts of the summary provided by the authors[1] includes an incorrect interpretation of New Zealand's dietary guideline advice, and a misrepresentation of the overall recommended eating pattern for New Zealanders.

The Ministry of Health's core dietary guidelines, the Eating and Activity Guidelines (EAGs) recommend a healthy eating pattern that is high in vegetables and fruit; includes whole grain cereals; low-fat milk products; legumes and nuts; fish and other seafood; and unsaturated oils. This eating pattern is low in processed meats, saturated fat, sodium (salt) and sugar-sweetened foods and drinks. A large body of evidence shows that this way of eating is associated with a lower risk of heart disease, stroke and other health conditions. The EAGs recommend choosing mainly whole and less processed foods to support the adoption of the healthy eating pattern described above.

The EAGs were published in 2015 and primarily focus on adult New Zealanders between 19–64 years. The eating pattern described above is generally considered the Ministry's basis for healthy eating advice for all population groups from two years of age and beyond. The Ministry is now going through a process to transition its population-specific dietary guidelines into the EAG series.

The Ministry continues to recommend that those over two years of age choose low-fat dairy options. There is evidence that some forms of saturated fat, from some dairy sources in particular, may be less harmful than previously thought. When this is considered within the total body of evidence on saturated fat though, healthy eating advice remains to choose unsaturated fats over saturated fats where possible, along with the other aspects of a healthy eating pattern as identified above.

The influence of diet on oral health is always considered during the Ministry's guidelines development process, and the Ministry's Oral Health Team is involved in the review process.

Prevention of tooth decay involves a combination of the following:

- brushing your child's teeth twice a day with regular-strength fluoride toothpaste
- ensuring that your child has a healthy diet that is low in sugar
- ensuring that your child has regular dental check-ups from an early age.

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The Discipline of Practice

February 1920

Latent in the desire for a State Medical Service is the hope among many of the removal of the irksome restraints and annoyances of private practice. No doubt, in some ways National Medical Service would mean the fulfilment of this desire and be the antidote for old ills, but it would inevitably create new ones. The British Prime Minister has recently observed that we cannot have nationalisation without bureaucracy, and in every bureaucracy promotion does not often go by merit, and a graded series of officials may be less just than our taskmasters, the public, under whom we now travail in service.

When the medical student leaves the sheltering care of his *Alma Mater* he is still cloistered for a time in a junior hospital appointment. At this stage of his development, it may be observed in passing if he thinks his knowledge is great, it may yet be possible for him to succeed commercially in his profession, but in no other way, unless, indeed, his knowledge has been gained in a previous incarnation, if we can adopt that ancient and modern out strange belief. Next comes the discipline of beginning practice in hope and fear, but this is common to all professions, and the race is not always to the swift or the battle to the strong.

Stephen Paget thinks that the peculiar discipline of medical practice lies in the fear of making mistakes. People, he writes, "talk of the Fine Arts: but what art is so fine as Medicine, which works in lives and cannot correct its proofs, or begin with a sketch, or waste its fabrics, or rehearse its effects, or use a model; and, by a mistake, injures not an image of life, but life? Why, that is just why Medicine is not fine. It is not the art, but the stuff which is fine." The fear of doing harm, with all respect to the accomplished essayist whom we quote, is not, in our opinion, the chief strain of practice, and to one who has been well taught and is reasonably careful it should not be a haunting fear, but belongs chiefly to a morbid state of mind. Doctors are mainly burdened with the terrible responsibility they carry both in youth and in age, and which they can never lay aside night or day until they voluntarily retire or are summoned hence to sleep with their fathers. There may be more heroism buried in a quiet corner of a country churchyard than in a cathedral aisle.

It is sharp discipline, too, to have cases fail through no fault of the doctor; he may save the father and lose the son, and hear the heart-break of the cry of David over Absolom. Day in and day out, from morn to night, the doctor's ears are the repository of complaints and sorrows as he treads the *via dolorosa* from one scene of suffering to the next. His words, his manner, even expression of his face are noted, and he is ever under the discipline of restraint. He may not say too much or too little. If he is successful, he must scorn delights and live laborious days, and however tired or sleepy he may be, he is expected to be cheerfully responsive to every call. This is a strain which flesh and blood can scarce endure.

The public expect great things of the medical profession, and this is to our honour. Medical science has made triumphal progress, but the rank and file, the flesh and blood of the movement, have harsh discipline and forced marches. By better generalship and tactics the strain and the wastage may be stayed before the breaking-point is reached. It is a hard discipline, but a great service, and there are many compensations.

In the development and evolution of the medical profession, the doctors themselves must see to it that while not putting aside the essential responsibility, discipline, and restraint inseparable from the calling, their nature shall not be subdued to what it works in, like the dyer's hand. Then it cannot be said of a doctor that he knows nothing but his own profession.

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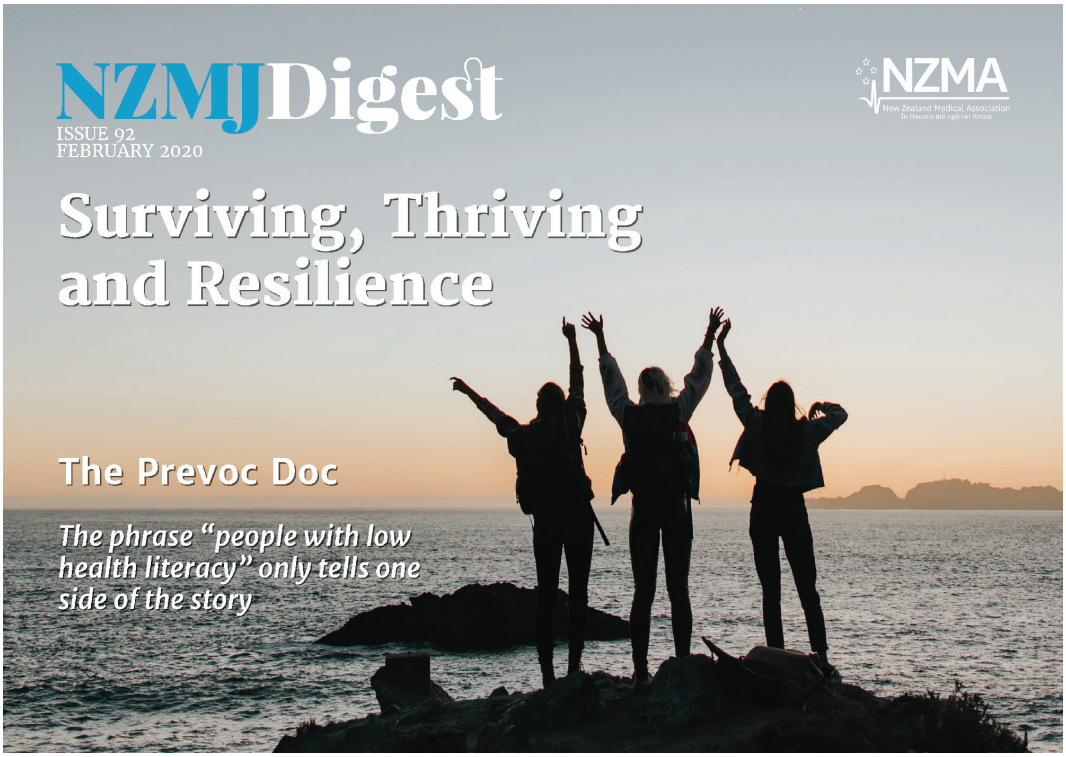

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