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Good news about melanoma

Mark Elwood, Marius Rademaker

Deaths from melanoma, the most serious skin cancer, have reduced greatly in the last 10 years. Deaths have reduced by about one third. This is most likely due to earlier diagnosis and improved treatment. There has been increasing awareness of early disease and more effective diagnostic services. There have also been new drug treatments for advanced melanoma. However, melanoma remains a big problem, and the numbers of new cases of melanoma has not reduced.

Almost one in five emergency department presentations are by mental health clients: a secondary data analysis

Silke Kuehl, Abigail Freeland, James Stanley, Ruth Cunningham

In a 1-year period, nearly one in five presentations to the Wellington emergency department (ED) were by clients under mental health services. These people presented for mental health concerns, pain or following an injury. Many mental health clients waited a long time in ED. If these clients needed to stay in hospital, this was mostly done by the ED, not by mental health services.

Increasing access to cataract surgery in Counties Manukau by optimising the clinical pathway: a quality improvement report

Manlio Chiesa, Graham Reeves, Courtney Harper, Mary Seddon, Valerio Malez

Demand for cataract surgery is increasing and access has varied by health districts. Removing geographical inequity with a consistent clinical priority assessment criteria (CPAC) score for accessing cataract surgery is a national priority. Our paper describes a successful quality improvement initiative undertaken in Counties Manukau over 2020–2022 to address long waiting lists and a CPAC threshold for surgery that was higher than its regional counterparts. We introduced a combined first specialist assessment (FSA) and pre-admission clinic, telephone follow-up for low-risk patients and increased the use of our own surgeons in private theatres. These changes reduced the burden of clinic visits for patients and released senior medial officer time, enabling an increase in monthly surgeries from 192 to 215 (+12%), a decrease in the average time from FSA to surgery from 90 to 77 days (-13.5%) and reduction of the CPAC threshold from 55 to 50 points.

Consequences of cost barriers to prescriptions: cohort study in Aotearoa New Zealand

Mona Jeffreys, Megan Pledger, Fiona McKenzie, Lis Ellison-Loschmann, Maite Irurzun Lopez, Jacqueline Cumming

Our study linked two databases; one where people told us whether they had not collected a prescription due to cost in the last year, and one of all hospitalisations. We found that people who had a cost barrier to getting a prescription were more likely to go to hospital sooner than other people. We worked out the costs that the health system might save if these hospital costs did not have to happen; this was about NZ$32 million per year. We urge the Government that now is the time for the zero fees policy to be retained.

Guideline versus clinician recommended duration of dual anti-platelet therapy following acute coronary syndrome (ANZACS-QI 78)

Sophie J Rees, Andrew J Kerr

Dual antiplatelet therapy (DAPT) is a treatment prescribed following acute coronary syndrome (heart attack or unstable angina) in which two antiplatelet medications are taken to prevent blood clots forming. The recommended duration of these antiplatelets medications varies depending on the balance of risks of ischaemia (clotting) and major bleeding. Our study calculated the ischaemic (clotting) and bleeding risk scores for 100 patients and compared the clinician recommended duration of DAPT to the European Society of Cardiology guidelines. We found that up to four out of five patients could have been planned for a shorter duration of DAPT based on the ESC guideline recommendations.

Outcomes of asymptomatic common bile duct stones detected at intra-operative cholangiography

Xavier Field, Chelsea Tong, Sarah Cox, James Crichton, Bernadette Goodwin, Fraser Welsh, Ryan Cha

The aim of our paper was to retrospectively see how many people who had surgery to remove their gallbladder had a gallstone found incidentally in their bile duct at the time of surgery. We then checked to see how the surgeons had decided to manage these patients; either by referring them to have a second procedure to have the gallstone removed, obtaining an MRI scan to see whether the stone would pass itself or following them over time with blood tests. A significant number of patients had their gallstone pass without intervention.

Construction of the chronic temporomandibular disorder patients: the association between neural and psychological pathways

Ajith D Polonowita, Athula K Polonowita, Li Mei, Guangzhao Guan

Chronic temporomandibular disorder is a condition that causes long-term jaw pain and discomfort without a clear cause. Our genes could be part of why some people are more prone to pain in this condition. Different genes related to our nervous and musculoskeletal systems might play a role in causing it. Additionally, environmental triggers and changes in gene expression could contribute to the development of chronic temporomandibular disorder. To effectively address and manage chronic temporomandibular disorder, a comprehensive approach is needed, considering biological, psychological and social factors according to the biopsychosocial model.

Pae Ora (Disestablishment of Māori Health Authority) Amendment Act 2024: further Crown breaches of Te Tiriti o Waitangi

Heather Came, Clive Aspin, Nicole Coupe, Tim McCreanor

Te Aka Whai Ora was the pounamu or centre piece within Pae Ora (Healthy Futures) Act 2022 and enabled the development of research that encapsulated the key principles of the New Zealand Health Strategy. With strong community engagement, developing community leadership and workforce capacity-building, Te Aka Whai Ora was addressing disparities and contributing to enhanced health and wellbeing. Extensive consultation with community researchers strengthened the level of support from Māori communities and held significant potential for improved Māori health outcomes. The Disestablishment Act is likely to be profoundly damaging to Crown relationships with Māori. It failed to respect Māori tino rangatiratanga, Māori expertise and mātauranga Māori. The health reforms initiated by Pae Ora with Te Aka Whai Ora at the forefront were a once in a lifetime opportunity to address Māori health inequities and enhance health outcomes for everyone who calls Aotearoa home. Almost 200 years after the signing of Te Tiriti o Waitangi, both partners, and the Crown in particular, must commit to

the original intentions of the agreement and implement innovative measures such as a Māori-focussed entity to achieve equitable health outcomes for Māori and ensure social justice for all.

A case report of successful dual external defibrillation in cardiac arrest

Anna G Bergin, Chamé C Blackburn, Eric Chong, Ankur Gupta

This paper reports on a successful case in Aotearoa New Zealand of two defibrillators being used instead of one to save a 45-year-old male who presented to Whakatāne Hospital with a heart attack. In particular, this male had a heart attack caused by heart rhythm called ventricular fibrillation that was not responding to normal treatment (refractory ventricular fibrillation). After 30 minutes of cardiopulmonary resuscitation (CPR) and shocks using one defibrillator as per New Zealand guidelines, the decision was made to use a second defibrillator. This double shock resulted in the patient's heart returning to a normal rhythm and the patient surviving the heart attack.

An act of desperation: self-attempted gender-affirming mastectomy

Mairarangi Haimona, Sue Hui Ong, Scott Diamond

This paper describes a young transgender patient who attempted to remove his own breasts due to gender dysphoria. There was no suicidal intent or active psychiatric disorder. This patient was not able to pay for this surgery and had been waiting for years on the public waiting list. However, the public system is heavily limited by the lack of resources.

Management of early dysglycaemia in pregnancy varies by region in Aotearoa New Zealand with risks of widening inequities

Rosemary M Hall, Ruth CE Hughes, Elizabeth Lewis-Hills, Janet A Rowan

Pregnant people who have high blood glucose levels have a higher chance of having complications for them and their baby. Managing glucose levels during pregnancy has been shown to reduce these risks. Currently, all pregnant people are offered a blood test early in pregnancy to look at their glucose levels. However, different regions around the country approach these results differently. New guidelines are being developed, and these must ensure all people have equitable access to care for the best outcomes for them and their baby.

Good news about melanoma

Mark Elwood, Marius Rademaker

There is some good news about melanoma. Deaths from melanoma at all ages have fallen considerably; the table shows the reductions from 2012 to 2021 (see Table 1). In 2021 there were 333 deaths—if the 2012 rates had continued, there would have been 164 more.

Incidence rates of invasive melanoma since 2001 have been fairly stable.[1] However, death rates have declined sharply since about 2011. So, we seem to be managing melanoma better. The most important factor is probably earlier diagnosis. This has a downside of course; diagnoses of *in situ* melanoma and very thin melanoma are common and some of these might not progress if left alone.[2] A possible diagnostic drift in favour of a histologic diagnosis of *in situ* melanoma over a diagnosis of naevus with severe atypia has been debated. However, excisions of benign lesions to exclude melanoma are not reported nationally. The favourable mortality trends could be due to a reduced number of deeper, poor prognosis melanoma; however, the recorded thickness distribution has remained stable since 2001, although national data are incomplete.[1] The reduced mortality may also reflect increased survival rates of patients with advanced melanoma due to new treatments.

Improvements in the early diagnosis of melanoma started in 1985 with the development of the ABCD diagnostic criteria (Asymmetry, Border irregularity, Colour variegation, Diameter >6 mm).

These were later refined by adding E or Evolving, giving the ABCDE criteria, which were linked to practical advice on self-examination of the skin.[3] The development of diagnostic criteria for atypical naevi, followed by consensus agreement on the important dermoscopy features for diagnosis of early melanoma, further advanced early diagnosis. International studies, including from New Zealand, of the consistency of dermoscopy diagnosis have been encouraging.[4] The introduction of American Joint Committee on Cancer staging criteria[5] and the development of national and international treatment guidelines, such as the Australia/New Zealand Clinical Practice Guidelines for the Management of Melanoma[6] and the MelNet guidelines,[7] undoubtedly improved management of melanoma, in particular of deeper, poor prognosis melanoma. Prognostic indicators, firstly Clark level and then Breslow thickness, and now with the addition of sentinel lymph node biopsy, have been crucial. More recently, positron emission tomography (PET) scanning and ultrasound of regional lymph nodes have superseded sentinel lymph node biopsy as the more useful prognostic indicators, in addition to Breslow thickness.[8] Liquid biopsy for circulating melanoma genetic material may eventually supersede these.[9]

Table 1: Mortality rates from melanoma per 100,000 population, percent reduction over 10 years, and numbers of deaths averted in 2021, by age, New Zealand.

Age	2012–2014 rate	2019–2021 rate	% reduction over 10 years	Deaths averted
20–44	1.1	0.5	70	15
45–64	7.6	4.7	50	51
65–74	27.0	15.9	50	67
75+	57.6	51.2	15	30

Based on fitted trends on annual data, standardised for age within groups. Numbers averted based on applying 2012 rates to 2021 population numbers for each sex. Reductions significant at $p<0.05$ in all age groups except 75+. Data are from the New Zealand mortality web tool: https://tewhatuora.shinyapps.io/mortality-web-tool/. 2012 and 2021 are the earliest and latest years with detailed data available.

Further advances in early diagnosis are the result of the many public educational campaigns (e.g., Cancer Society's SunSmart: https://www.sunsmart.org.nz/), and the upskilling of primary care practitioners in the use of dermoscopy, which can increase diagnostic accuracy and reduce unnecessary excisions.[10] Many general practitioners, nurses and others have attended short courses on skin lesion recognition and management. Commercial organisations provide sequential digital dermoscopic and mole mapping services directly to patients. MoleMap New Zealand is a telemedicine service to detect melanoma, using expert review of photographic and dermoscopic images of suspicious lesions. Of 2,108 melanocytic lesions recommended for biopsy/excision in 2015–2016, 17% were melanoma, or one in six biopsies; most were thin or *in situ* lesions.[11]

Faster Cancer Treatment targets, first proposed in 2008 and again in 2014, set a 14-day target for a first specialist assessment and a 62-day target for treating lesions suspicious of melanoma in the public sector, but were not achieved nationwide when abandoned in June 2020. On 8 March 2024, the Government announced a health target for 1 July 2024 of 90% of patients receiving cancer management within 31 days of the decision to treat (https://www.health.govt.nz/new-zealand-health-system/health-targets). This applies to wide local excision (the definitive treatment for melanoma) after a diagnostic excision is undertaken in primary care.

The gradual introduction of eReferrals, often with attached images (macro and dermoscopic) has further reduced the delay in patients being seen by a specialist. The upskilling of surgical skills by general practitioners has meant that the primary excision of melanoma now often occurs in primary care—but good training is vital. However, audit studies have shown compliance with melanoma guidelines is often poor in secondary care,[12] although this has been compensated for by faster management through teledermatology.[13,14] Specific pigmented lesion clinics have been valuable in providing faster management with fewer unnecessary biopsies.[15]

For advanced melanoma, the biggest impact in reducing mortality has been the development of immune checkpoint inhibitors, in particular PD-1 inhibitors (e.g., pembrolizumab, nivolumab).[16] In overseas studies, median overall survival for advanced melanoma has increased from 6 months prior to the development of immune checkpoint inhibitors to 37 months (minimum follow-up 6.5 years) for patients on monotherapy with nivolumab, and to 72 months for the combination of ipilimumab and nivolumab.[17] The median survival following treatment with pembrolizmab is similar at 39 months.[18] Yet there is a significant cost, both monetary and in serious adverse effects. Overall survival in New Zealand is comparable to pivotal clinical trials and real-world data from other countries.[19,20] However, drugs for advanced melanoma are expensive, and funding is limited; they can also result in serious adverse effects.

What more could we do? Most of the improvements in services have been local initiatives, with no national approach to the optimum diagnosis and treatment of melanoma. This is despite it being a major burden; the cost for new patients with melanoma in 2021 was NZ$51.2 million, and for keratinocyte skin cancers NZ$129.4 million.[21] The creation of Health New Zealand – Te Whatu Ora provides the opportunity for a national approach to early diagnosis and to reduce inequities in providing services. Melanoma is much less common in our Māori, Pacific and Asian populations, but shows more nodular and acral lesions, more deeply invasive lesions and poorer prognoses.[22] More efficient skin lesion triage may benefit from adopting locally validated, secure artificial intelligence tools to support health professionals.[23] Prevention remains a priority: skin cancer prevention is cost-effective, with benefits being 2–4 times the costs.[24]

COMPETING INTERESTS
Dr Rademaker reports for MoleMap New Zealand, but has no financial or other interest in the company.

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Almost one in five emergency department presentations are by mental health clients: a secondary data analysis

Silke Kuehl, Abigail Freeland, James Stanley, Ruth Cunningham

ABSTRACT

AIM: Mental health-related emergency department (ED) presentations are steadily increasing, including presentations for both mental health and non-mental health concerns by existing clients of mental health services. The study aim was to examine and compare mental health clients and non-clients' ED presentations, identify data and clinical gaps and make recommendations for improvement.

METHOD: De-identified 2017/2018 ED data were used to describe presentations for current and recent (within last 5 years) clients of specialist public mental health and addiction services, compared to presentations of non-mental health clients.

RESULTS: Of 49,170 presentations, 18% were by clients of mental health services. Compared to other ED presenters, mental health clients were often younger, female and Māori, required more urgent care and waited longer. Mental health-related International Classification of Diseases (ICD) codes/referrals were most common for presentations by current mental health clients, whereas pain and trauma were often the reason for prior mental health clients' presentations. Discharge diagnoses rarely included self-harm behaviour, and admissions for these clients were more commonly by ED rather than mental health services.

CONCLUSION: Mental health clients are common in ED. Enhanced mental health data capture and improved systems and processes are needed to ensure that ED staff can better meet their often-complex needs.

World-wide and in Aotearoa New Zealand, the number of people trying to access mental health and/or addiction services has risen dramatically. Labelled an "epidemic of mental distress and addiction,"[1] Aotearoa New Zealand health services have been struggling to meet the increasing demand. Between 2009/2010 and 2018/2019, district health boards (DHBs) and non-government organisations had a 55% increase in mental health service contacts, while expenditure only increased by 24% in this timeframe.[2] Aotearoa New Zealand emergency departments (EDs) reported a doubling of the proportion of presentations related to mental health—defined as people who were referred to specialist mental health and addiction services—from 3.7% of all presentations in 2017 to 7.4% in 2018.[3] As a result, "mental health patients" were waiting dangerously long in the ED.[4]

The true extent of ED presentations with a mental health aspect is difficult to ascertain in the literature. Some studies included presentations coded with principal diagnoses from the International Classification of Diseases behavioural disorders chapter (ICD-10-AM: F00-F99), excluding self-harm and suicide-related ED presentations and including intellectual development disorders and organic causes.[5] Other studies excluded disorders such as dementia.[6] Randall et al.[7] used suicide assessment forms, completed by emergency psychiatrists for every consult, to identify ED presentations related to self-harm, as they regarded ICD-10 codes as limited in identifying eligible presentations.

Presentations by existing clients of mental health services are an important but often overlooked source of mental health-related ED interaction. When these clients present to ED, their health concerns are likely to include mental health aspects but may also include physical health aspects. Those presenting to ED for self-harm often present for other health issues within a short timeframe, potentially increasing their future serious-self-harm risk if mental health concerns remain undetected during "other" presentations.[8] Across the health system, mental health clients are vulnerable to unequal treatment, physical illness and adverse health outcomes,[9] including a more

than doubled premature mortality rate compared to the general population.[10] ED is likely a common provider of healthcare for mental health clients, since between 11% and 46% are likely to disengage with mental health services over 12 months,[11] and the combination of mental disorders and physical health concerns influences frequent ED use.[12] Understanding the characteristics of mental health clients in the context of their ED presentations provides a different lens for thinking about the role of emergency departments in providing care for mental health concerns by focussing on people with the most complex mental health needs in order to identify service and data gaps and improve service provision.

Objective

The objective was to examine and compare ED utilisation by clients of mental health and addiction services (current and prior) and non-clients, to identify possible clinical and data gaps that could lead to improved outcomes for this patient group.

Methods

Study design and setting

The study involved a retrospective descriptive analysis of routinely collected health service data. We examined mental health-related ED presentations over a 12-month period (1 July 2017 to 30 June 2018) from the ED at Wellington Regional Hospital, a 484-bed tertiary hospital in a major urban centre of Aotearoa New Zealand. An ED observation unit (EDOU) is attached to ED. The study received ethical approval from the Health and Disability Ethics Committee (20/NTB/64/AM03) and underwent Māori consultation (RAG-M #746).

Mental health clients can contact Te Haika, a 24-hour mental health and addiction telephone call centre, for mental health support. Te Haika staff screen whether people need to be seen urgently, or whether they need to be assessed in ED. During office hours, mental health clients are seen at the community mental health base. If they attend ED instead, ED staff liaises with Te Haika to determine if they can be assessed in the community instead. After hours, Te Haika refers clients to the Crisis Resolution Service (CRS). The CRS is based in the community to assess people in ED, the police station or in people's homes. The ED has one mental health liaison nurse who works from 7 am to 3:30 pm Sundays to Thursdays.

Data extraction and variables

Data were extracted from the Emergency Department Information System (EDIS) via Data Warehouse and the Mental Health, Addictions and Intellectual Disability Service (MHAIDS) Web Patient Administration System (webPAS). De-identified data were imported into SAS Enterprise Guide 7.1 for processing and analysis. ED presentations were included for people aged ≥10 years. Variables were age, gender, ethnicity, domicile code (area of residence) and mental health services' client status (current, prior in last 5 years, non-user).

Current mental health clients were those with open mental health referrals to a crisis or community mental health team, child and adolescent mental health services, or any MHAIDS specialist service, such as alcohol and drugs. Previous mental health clients had at least one mental health referral in the 5 years prior to the ED presentation but no current open referral. The remainder of ED presentations were classified as non-client presentations.

Clinical information was arrival date/time, triage priority following the Australasian Triage Scale (1–5),[13] mental health event (referral status "yes/no" to mental health services), ED discharge diagnosis (assigned by doctors from pre-defined drop-down menu), up to six ICD diagnoses (only available for ED presentations lasting at least 3 hours and/or resulting in an admission), assigned admission speciality (see Appendix 1) and discharge date/time.

Data cleaning and preparation

Age was grouped into age bands (10–14, 15–24, 25–34, 35–44, 45–54, 55–64, 65+). Gender had been recorded as female, male and unknown classifications; ethnicity had already been prioritised as Māori, Pacific peoples, Asian or Other. Place of residence (domicile code) was linked with the New Zealand Deprivation Index (NZDep2018)[14] to ascertain area level deprivation quintiles 1–5 (1 least deprived, 5 most deprived).

Length of stay was the difference between arrival date/time and discharge date/time, in minutes (for continuous summary) and grouped (<3 hours, 3–<6, 6–<9, 9+ hours).

ICD-10 codes and ED discharge diagnoses were categorised as "mental health-related", "potentially mental health-related" and "not mental health-related" (see Appendix 2 for categorisation process). Potentially mental health-related presentations were indicative of mental health or self-harm, such as "poisoning" or "wrist laceration", and are described elsewhere.[15]

Included mental health-related ICD-10 codes were F10–F19 (excluding intoxication and F17 Tobacco), F20–F50, F53–F55, F59–F69, F90–F99 (mental health and addiction diagnoses) and X60–X84 (self-harm). Excluded were F00–F09 and F70–F89 (dementia, Alzheimer's, delirium, intellectual disability).

ED discharge diagnoses for presentations by current and prior mental health clients were categorised following an adapted framework based on the systematic "airway, breathing, circulation, disability, exposure" (ABCDE) approach of assessing ED patients,[16] a presenting complaint categorisation structure[17] and a prior adaption of these categorisation tools (Appendix 3).[8]

Discharge diagnosis categories were respiratory, circulatory, neurological, medical, pain, trauma, function, poisoning and miscellaneous. Mental health-related discharge diagnoses were further categorised into "depression", "anxiety and self-harm" (combined because of low numbers), mental illness (e.g., manic disorder, psychosis, hallucinations, alcohol or drug addiction/withdrawal) and other (e.g., emotional crisis, stress reaction, psychosomatic symptoms, personality disorder, eating disorder). From the "not recorded" discharge diagnosis (classified as miscellaneous, detailed in Appendix 3), we used the disposition variable to code presentations that resulted in "did not wait", "absconded" or "self-discharge" into a "total did not wait" category.

Statistical analysis

Data were analysed using descriptive statistics and the Chi-squared test (X^2) for comparisons across groups. Percentages are reported with 95% confidence intervals (Wilson's method).

We calculated the median and interquartile range (IQR) for length of ED stays in the three groups, alongside the proportion of presentations departing ED by <3, 3–<6, 6–<9 and 9+ hours after presentation. We described and compared classified discharge diagnoses of current and prior mental health clients.

Results

In 2017/2018, the ED recorded 49,170 presentations by people aged 10 years and older. These ED presentations were made by 33,597 distinct people, of whom 1,145 were current mental health clients and 2,921 were prior mental health clients (Appendix 4). Current and prior (within 5 years) mental health clients made 8,874 presentations, almost one fifth of all presentations (18.1%: 6.7% by current clients, and 11.4% by prior clients) (Table 1). Nearly half of the presentations by mental health clients were by people between 15 and 34 years old (46.3% vs 33.2% of non-mental health clients), and few were in the 65 years and over category (6.3% vs 28.1% for non-mental health clients). Mental health service clients presenting to ED were more likely to be Māori, and less likely to be of Asian or Pacific peoples' ethnicity and lived in more deprived areas compared to non-mental health clients.

Presentations by current mental health clients were assigned more urgent triage codes (codes 1–3) compared to non-mental health clients (61.4% vs 56.6%) (Table 2). Approximately 30% of presentations by current mental health clients resulted in a referral to mental health services (mental health event). This compared to 9% of previous mental health clients referred to mental health services, and only 1% for ED clients not previously known to mental health services. A similar pattern was seen with recorded mental health diagnoses (relevant ICD code assigned), covering 30% of current clients, 12% of previous clients and 2% of those not known to services.

Clients of mental health services were most frequently recorded with "emergency department" specified as the admission speciality that provided care, substantially higher than for non-mental health clients (22.2% vs 10.3%).

Current mental health clients experienced longer ED stays (median 228 minutes, IQR 144–322: 13.9% 6+ hour wait) than prior (median 216, IQR 139–305) or non-mental health clients (median 210, IQR 137–297). Length of ED stay is summarised according to ED performance indicator categories[18] in Table 2.

Most presentations by current and prior clients of mental health services were assigned non-mental health discharge diagnoses. "Pain" and "trauma" diagnoses covered about two in five presentations for prior clients of mental health services (Table 3). Current clients had a greater proportion of mental health-related ED presentations compared to prior mental health clients (28.2% vs 9.0%), with depression the most common mental health-related discharge diagnosis. Poisoning discharge diagnoses were also more common for current compared to prior mental health service clients (8.8% vs 4.0%). Under miscellaneous, two thirds of presentations with "not recorded" discharge diagnoses for current (n=325) and prior (n=449) mental health clients had disposition classifications

Table 1: Number and proportion of ED presentations at Wellington Regional Hospital in 2017/2018 by age, gender, ethnicity and deprivation; by mental health client status.

Socio-demographic information	Detail	Total ED presentations *N*=49,170 (%)	Current MH client *n*=3,284 (%)	Prior[1] MH client *n*=5,590 (%)	Non-MH client *n*=40,296 (%)	P-value[3]
Age	10–14	2,054 (4.2)	110 (3.4)	145 (2.6)	1,799 (4.5)	<0.001
	15–24	9,214 (18.7)	738 (22.5)	1,380 (24.7)	7,096 (17.6)	
	25–34	8,179 (16.6)	782 (23.8)	1,124 (20.1)	6,273 (15.6)	
	35–44	5,861 (11.9)	608 (18.5)	817 (14.6)	4,436 (11.0)	
	45–54	5,991 (12.2)	457 (13.9)	816 (14.6)	4,718 (11.7)	
	55–64	5,591 (11.4)	382 (11.6)	582 (10.4)	4,627 (11.5)	
	65+	12,280 (25.0)	207 (6.3)	726 (13.0)	11,347 (28.1)	
Gender	Female	25,485 (51.8)	1,869 (56.9)	2,981 (53.3)	20,635 (51.2)	<0.001
	Male	23,676 (48.2)	1,413 (43.0)	2,608 (46.7)	19,655 (48.8)	
	Unknown	9 (0.0)	2 (0.1)	1 (0.0)	6 (0.0)	
Ethnicity (prioritised)	Māori	5,319 (10.8)	535 (16.3)	926 (16.6)	3,858 (9.6)	<0.001
	Pacific peoples	4,335 (8.8)	159 (4.8)	358 (6.4)	3,818 (9.5)	
	Asian	4,433 (9.0)	109 (3.3)	230 (4.1)	4,094 (10.2)	
	Other	35,083 (71.4)	2,481 (75.6)	4,076 (72.9)	28,526 (70.8)	
NZDep2018 deprivation quintile[2]	1	13,652 (27.8)	711 (21.7)	1,177 (21.1)	11,764 (29.2)	<0.001
	2	10,100 (20.5)	430 (13.1)	1,089 (19.5)	8,581 (21.3)	
	3	12,966 (26.4)	966 (29.4)	1,628 (29.1)	10,372 (25.7)	
	4	8,099 (16.5)	922 (28.1)	1,133 (20.3)	6,044 (15.0)	
	5	4,353 (8.9)	255 (7.8)	563 (10.1)	3,535 (8.8)	

[1]Within last 5 years; [2]1=lowest deprivation and 5=highest deprivation; [3]p-value from Chi-squared test comparing socio-demographic profile over the three client groups (current MH, prior MH, non-MH).

assigned that were related to leaving before being seen (did not wait, self-discharge, absconded). In total, 10.6% of presentations by current mental health clients and 8.8% of prior mental health service clients did not wait for or complete treatment.

Discussion

Results from our secondary data analysis show mental health clients make up a substantial proportion of ED presentations. About 7% of all ED presentations in our 1-year study period (2017/2018) were by current mental health clients (up to 18.1% of all presentations, if counting current or prior client in last 5 years). This is substantially higher than the 3.6%/4.0% of the Aotearoa New Zealand/CCDHB population who accessed specialist mental health and addiction services in 2017.[19,20]

The socio-demographic characteristics of mental health clients in ED aligned with similar studies

Table 2: Number and proportion of presentations by mental health client status by triage code, mental health event, ICD coding, admission speciality, length of stay.

Clinical information	Detail	Current MH client *n*=3,284 (%)	Prior[1] MH client *n*=5,590 (%)	Non-MH client *n*=40,296 (%)	P[5]-value
Triage code	1—Resuscitation	24 (0.7)	46 (0.8)	240 (0.6)	<0.001
	2—Emergency	544 (16.6)	803 (14.4)	5,418 (13.5)	
	3—Urgent	1,447 (44.1)	2,452 (43.9)	17,107 (42.5)	
	4—Semi Urgent	986 (30.0)	1,828 (32.7)	14,232 (35.3)	
	5—Non-Urgent	281 (8.6)	461 (8.3)	3,297 (8.2)	
Mental health event[2]	Yes	958 (29.2)	506 (9.1)	403 (1.0)	<0.001
	No	2,326 (70.8)	5,084 (91.0)	39,893 (99.0)	
ICD diagnosis related to mental health	Yes	995 (30.3)	654 (11.7)	833 (2.1)	<0.001
	No	720 (21.9)	2,153 (38.5)	19,624 (48.7)	
	Maybe	126 (3.8)	189 (3.4)	822 (2.0)	
	Not assigned[3]	1,443 (43.9)	2,594 (46.4)	19,017 (47.2)	
Admission speciality	Not recorded	1,957 (59.6)	3,453 (61.8)	24,703 (61.3)	<0.001
	Emergency	728 (22.2)	897 (16.1)	4,147 (10.3)	
	Medical	280 (8.5)	481 (8.6)	4,397 (10.9)	
	Other	231 (7.0)	711 (12.7)	6,807 (16.9)	
	Paediatric Medical	52 (1.6)	31 (0.6)	161 (0.4)	
	Intensive Care	15 (0.5)	15 (0.3)	78 (0.2)	
	Mental Health	21 (0.6)	2 (0.0)	3 (0.0)	
Length of stay[4]	<3 hours	1,184 (36.1)	2,141 (38.3)	15,925 (39.5)	<0.001
	3–<6 hours	1,645 (50.1)	2,863 (51.2)	20,563 (51.0)	
	6–<9 hours	302 (9.2)	434 (7.8)	2,902 (7.2)	
	9+ hours	153 (4.7)	152 (2.7)	906 (2.3)	

[1]Within last 5 years; [2]referral to mental health services; [3]includes presentations where a person was "fast tracked" to another ward/clinic from triage without being seen by a doctor in ED and presentations lasting less than 3 hours; [4]proportion of ED presentations departed from ED at this time point; [5]p-value from Chi-squared test comparing socio-demographic profile over the three client groups (current MH, prior MH, non-MH).

Table 3: Grouped discharge diagnoses by current and prior mental health clients.

Discharge diagnosis group	Subgroup examples	Current MH client *n*=3,284 % (95% CI)		Prior[1] MH client *n*=5,590 % (95% CI)	
Airway/ respiratory		127	3.9 (3.2–4.6)	294	5.3 (4.7–5.9)
Circulation		82	2.5 (2.0–3.1)	207	3.7 (3.2–4.2)
Neurological	Seizure, loss of consciousness, dementia, transient ischaemic attack, altered mental state	105	3.2 (2.6–3.9)	208	3.7 (3.2–4.3)
Medical	Allergic reaction, generally un-well, muscle weakness, diabetes (skin) infection	257	7.8 (6.9–8.8)	607	10.9 (10.1–11.7)
Pain	Abdominal, chest, other	402	12.2 (11.1–13.4)	1,093	19.6 (18.5–20.6)
Trauma	Head, upper limb, other	340	10.4 (9.3–11.4)	1,007	18.0 (17.0–19.0)
Function	Gastrointestinal, reproductive/ urinary/renal, other	205	6.2 (5.4–7.1)	660	11.8 (11.0–12.7)
Poisoning	Alcohol/drug/medication	290	8.8 (7.9–9.9)	223	4.0 (3.5–4.5)
Mental health	Depression	491	15.0 (13.7–16.2)	276	4.9 (4.4–5.5)
	Anxiety, self-harm[2]	89	2.7 (2.2–3.3)	65	1.2 (0.9–1.5)
	Illness, alcohol/drugs[3]	214	6.5 (5.7–7.4)	89	1.6 (1.3–2.0)
	Other	128	3.9 (3.3–4.6)	80	1.4 (1.1–1.8)
Miscellaneous	Tests	39	1.2 (0.8 – 1.6)	60	1.1 (0.8–1.4)
	Not recorded	493	15.0 (13.8–16.3)	680	12.2 (11.3–13.1)
	Did not wait for treatment	22	0.7 (0.4–1.0)	41	0.7 (0.5–1.0)

[1]Within last 5 years; [2]"Observation after suicide attempt" discharge diagnosis (n=21); [3]addiction or withdrawal.

using ICD coding to identify mental health-related ED presentations. Mental health presentations were often by younger adults and rarely by people over 65.[21] Prior research identified high levels of general mental health service contact among Māori.[22] This study adds important information on the socio-demographic make-up of mental health clients presenting to ED.

ED presentation patterns differed between current and prior mental health clients. Presentations by current mental health clients were more weighted towards mental health issues than was the case for prior clients. It makes sense that current mental health clients under mental health services experience high acuity mental health problems, sometimes needing additional after-hours support from ED. Prior mental health clients, who presented predominantly for pain or trauma, might have been at risk of overshadowing, whereby physical health conditions are attributed to their mental health.[23] However, we could not ascertain from the data how ED managed the physical and mental health complexity for people with mental illness[9] and so this concern requires further research.

The ED admitted one in five mental health clients

and one in 10 non-mental health clients into the EDOU. As ED admissions are driven by an attempt to not exceed patient's stay by 6 hours,[18] our findings suggest that mental health clients waited longer to be assessed, treated and discharged/admitted compared to other patients. While we do not have direct information on the reasons behind these findings, it may be that complex physical, mental or social concerns of some of these clients' presentations, such as following an overdose, might have required several speciality team involvements, lengthening their ED stay and increasing the need for an ED admission. Because of the low resourcing levels for mental health services in ED, the EDOU may have served as a "holding bay" for patients awaiting an assessment by the crisis team. With ED tasked to take on the interim care for a significant number of mental health clients presenting to ED, the clients are potentially missing out on timely mental health input and on a wrap-around care package by crisis mental health services. The extreme pressure on ED staff due to overcrowding[24] and an overall lack of mental health training for these staff[25] can lead to suboptimal ED care, at least for a subset of mental health clients. In line with earlier research,[26] nearly one in 10 mental health clients did not wait for assessment and/or treatment.

Examining ED presentations based on mental health client status is a novel person-centred approach to considering mental health-related ED utilisation. Usually, studies define mental health presentations by ICD codes recorded on presentations and estimate that this group make up between 4.1% and 8.1% of ED presentations.[21,27] Our approach of using mental health clients' status for considering mental health-related ED presentations acknowledges that mental health clients often have complex physical, mental and social needs[10] irrespective of their presentation or assigned discharge diagnosis. The monitoring of mental health clients' ED utilisation can guide future innovations.

The available EDIS data only broadly captured the clinical picture for mental health clients. While the "depression" discharge diagnosis was frequently assigned, self-harm behaviour was rarely recorded. As ED presentations for self-harm are common,[8] diagnoses such as overdose, poisoning, trauma or laceration might be related to self-harm, but these were difficult to capture from the available clinical data.[7] Such current mental health data gaps in ED indicate that opportunities for targeted intervention or improvement may be missed.[15]

The high number of presentations by mental health clients to ED indicates that mental health services were unable or not suitable to meet their often-complex needs. Given that mental health services are under-staffed, under-resourced and grappling with increasing caseloads,[2] it is likely that fewer resources are available for each client. Some current mental health clients might have chosen self-management because of being unhappy with their services[28] and presented to ED instead when needing help. The fact that 5,590 (11.4%) of all ED presentations were made by previous clients of mental health services indicates ongoing high need and could indicate some people had been prematurely discharged or found it difficult to re-enter mental health services.

EDs can better meet the needs of mental health clients. Several solutions have been suggested. Establishing and integrating an ED mental health team, preferably including a nurse practitioner and peer support workers,[4] would provide much needed assistance to ED staff and patients. Community innovations such as a co-response team[29] and alternative support locations could lower mental health clients' reliance on ED[3] and help reduce ED lengths of stay.[4] An equity lens is critical where enhancements of mental health and addiction support align with health and wellbeing outcomes for Māori.[30] Further research on ED presentation patterns/frequency/referral pathways of mental health clients and their EDOU utilisation is needed. An exploration on how to capture "self-harm" intent within the depression discharge diagnosis may be useful to guide service improvement. A major challenge will be how to improve data quality without additionally burdening ED staff. This could include improvements to current data capture systems, or eventually the use of artificial intelligence tools to aid classification for research purposes, though such tools would still require data of sufficient quality to meet needs.

The study had strengths and limitations. Utilising a complete regional dataset with linked data for mental health service and ED use, alongside systematic coding of ED-level discharge diagnoses into ED-specific categories, provided distinct presentation information. EDIS data was fit-for-purpose despite its shortcomings. As limited clinical information on self-harm presentations can be captured by EDIS, it did not always provide adequate information for the research questions. Relying solely on primary ED discharge diagnosis likely under-counted presentations

with mental health aspects, e.g., wrist lacerations. ED presentations with unassigned ICD codes—for presentations lasting less than 3 hours that did not result in an admission—could have resulted in under-counting of mental health-related presentations. Analysis was conducted using presentations as the denominator: this means that estimates (in all groups) will be slightly weighted towards the profile and experiences of people who frequently used ED in the study year (i.e., multiple presentations per person). However, the characteristics of ED presenters were also examined. We had no information on private psychiatric care. A substantial number of presentations by mental health clients resulted in "did not wait or did not complete treatment" discharge classifications. This information was unavailable for non-mental health clients, as time and resource constraints prevented us from grouping their discharge diagnoses (due to the substantially higher number of presentations by this group).

Mental health clients are a considerable ED sub-population. Many attend for mental health concerns, pain or trauma. Despite the often-urgent care needs of mental health clients, long stays in ED are common. Current ED data do not identify self-harm behaviour. While ED admitted a considerable proportion of mental health clients, it was rare that these decisions were made by mental health services (most admission decisions were taken by ED clinicians).

Optimal ED management requires a holistic approach and close links with mental health and community services. Improved ED mental health data capture and monitoring is needed to help guide service improvement initiatives. Assessing mental health clients' wellbeing and addressing their unmet needs is likely to reduce future ED presentations.

COMPETING INTERESTS
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Appendix 1

Health speciality

Cardiology
Cardiothoracic
Dental/Maxillofacial
Ears/Nose/Throat
Emergency
Eyes
Gastro
Gynaecology
Haematology
Intensive Care
Medical
Mental Health
Neurology
Neurosurgery
Not recorded
Obstetrics
Oncology—Haem
Oncology—Medical
Oncology—Radiation
Orthopaedic
Paediatric Medical
Paediatric Surgical
Renal
Respiratory
Stroke Team
Surgical
Urology
Vascular

Appendix 2

1. Discharge diagnoses/ICD codes related to mental health ("Yes")

Mental health-related discharge diagnoses/ICD codes were those related to a mental illness, self-harm or alcohol and drug addiction (Appendix Table 1). Further, we included disorders such as eating disorders and post-traumatic stress disorder, as well as clinical psychiatric management, as being related to mental health. ICD codes of relevant mental health related ED presentations are provided in Appendix Table 2.

Appendix Table 1: Mental health-related ED discharge diagnoses/ICD codes (Yes).

Discharge diagnoses/ICD codes related to mental health		
Group	**Subgroup**	**Description**
Mental illness	Anxiety	Also, mixed with depression, panic attack, neurosis, agoraphobia
	Depression	Mild to severe, acute vs recurrent, associated with psychosis or anxiety
	Schizophrenia	Unspecified, paranoid
	Schizoaffective disorder	Unspecified, depressive type, mixed type
	Delusional disorder	Paranoia
	Bipolar disorder	Mild to severe, persistent mood disorder, depression, hypomania, mania, bipolar affective disorder
	Psychotropic medication	Poisoning, causing adverse effects
Self-harm	Suicidal	Suicidal ideation
	Intentional self-harm	Trauma—razor blade, glass, knife, jumping, etc.
	Intentional self-poisoning	Medication, drugs, car exhaust, gas and vapours, petroleum gas etc.
Other disorders	Eating disorder	Unspecific, anorexia, bulimia, harmful use of laxatives etc.
	Behavioural disorders	Behavioural problems, conduct disorder, adjustment disorder, hysteria etc.
	Psychosomatic	Feigning of symptoms, non-epileptic seizures, conversion disorder, other somatoform disorders etc.
	Personality disorder	Unstable, impulsive, multiple, anxious, dependent, emotionally unstable etc.
	Mental state	Stress reaction, emotional crisis etc.
	Other	Other mental disorder, childhood emotional disorders, not specified, post-traumatic stress disorder etc.

Appendix Table 1 (continued): Mental health-related ED discharge diagnoses/ICD codes (YES).

Substance misuse	Alcohol	Dependence, withdrawal, physical symptoms because of addiction, e.g., cirrhosis, alcoholic psychosis, delirium
	Drugs	Harmful use, dependence, withdrawal, development of mental disorder, e.g., psychotic disorder
MH management		Observation for mental health disorder or post suicide attempt, psych examination etc.

Appendix Table 2: ICD codes and descriptions of included mental health-related ED presentations ("Yes").

Code	Description
F10.1–9	Mental and behavioural disorders due to use of alcohol, F10.1 harmful use, F10.2 dependence syndrome, F10.3 withdrawal state, F10.4 withdrawal state with delirium, F10.5 psychotic disorder, F10.6 amnesic syndrome, F10.7 psychotic disorder, F10.8 other mental and behavioural disorder, F10.9 unspecified mental and behavioural disorder
F11.1–9	Mental and behavioural disorders due to use of opioids, F11.1 harmful use, F11.2 dependence syndrome, F11.3 withdrawal state, F11.4 withdrawal state with delirium, F11.5 psychotic disorder, F11.6 amnesic syndrome, F11.7 psychotic disorder, F11.8 other mental and behavioural disorder, F11.9 unspecified mental and behavioural disorder
F12.1–9	Mental and behavioural disorders due to use of cannabinoids, F12.1 harmful use, F12.2 dependence syndrome, F12.3 withdrawal state, F12.4 withdrawal state with delirium, F12.5 psychotic disorder, F12.6 amnesic syndrome, F12.7 psychotic disorder, F12.8 other mental and behavioural disorder, F12.9 unspecified mental and behavioural disorder
F13.1–9	Mental and behavioural disorders due to use of sedatives or hypnotics, F13.1 harmful use, F13.2 dependence syndrome, F13.3 withdrawal state, F13.4 withdrawal state with delirium, F13.5 psychotic disorder, F13.6 amnesic syndrome, F13.7 psychotic disorder, F13.8 other mental and behavioural disorder, F13.9 unspecified mental and behavioural disorder
F14.1–9	Mental and behavioural disorders due to use of cocaine, F14.1 harmful use, F14.2 dependence syndrome, F14.3 withdrawal state, F14.4 withdrawal state with delirium, F14.5 psychotic disorder, F14.6 amnesic syndrome, F14.7 psychotic disorder, F14.8 other mental and behavioural disorder, F14.9 unspecified mental and behavioural disorder
F15.1–9	Mental and behavioural disorders due to use of other stimulants, including caffeine, F15.1 harmful use, F15.2 dependence syndrome, F15.3 withdrawal state, F15.4 withdrawal state with delirium, F15.5 psychotic disorder, F15.6 amnesic syndrome, F15.7 psychotic disorder, F15.8 other mental and behavioural disorder, F15.9 unspecified mental and behavioural disorder
F16.1–9	Mental and behavioural disorders due to use of hallucinogens, F16.1 harmful use, F16.2 dependence syndrome, F16.3 withdrawal state, F16.4 withdrawal state with delirium, F16.5 psychotic disorder, F16.6 amnesic syndrome, F16.7 psychotic disorder, F16.8 other mental and behavioural disorder, F16.9 unspecified mental and behavioural disorder

Appendix Table 2 (continued): ICD codes and descriptions of included mental health-related ED presentations ("Yes").

F18.0	Mental and behavioural disorders due to use of volatile solvents, F18.1 harmful use, F18.2 dependence syndrome, F18.3 withdrawal state, F18.4 withdrawal state with delirium, F18.5 psychotic disorder, F18.6 amnesic syndrome, F18.7 psychotic disorder, F18.8 other mental and behavioural disorder, F18.9 unspecified mental and behavioural disorder
F19.0	Mental and behavioural disorders due to multiple drug use and use of other psychoactive substances, F19.1 harmful use, F19.2 dependence syndrome, F19.3 withdrawal state, F19.4 withdrawal state with delirium, F19.5 psychotic disorder, F19.6 amnesic syndrome, F19.7 psychotic disorder, F19.8 other mental and behavioural disorder, F19.9 unspecified mental and behavioural disorder
F20–F29	Schizophrenia, schizotypal and delusional disorders
F30–F39	Mood (affective) disorders
F40–F48	Neurotic, stress-related and somatoform disorders
F50	Eating disorders
F53	Mental and behavioural disorders associated with the puerperium, not elsewhere classified
F54	Psychological and behavioural factors associated with disorders or diseases classified elsewhere
F55	Abuse of non-dependence-producing substances
F59	Unspecified behavioural syndromes associated with physiological disturbances and physical factors
F60–F69	Disorders of adult personality and behaviour
F90–F98	Behavioural and emotional disorders with onset usually occurring in childhood and adolescence
F99	Unspecified mental disorder
X60–X84	Intentional self-harm

2. Presentations not related to mental health ("No")

Discharge diagnoses and ICD codes not related to mental health were those depicting a physical health complaint rarely associated with mental health (Appendix Table 3, Appendix Table 4). However, we acknowledged that for some presentations, for example "abdominal pain", the person could have swallowed glass, and the intent was not documented. In view of the large numbers of common discharge diagnoses such as abdominal pain—most of them not related to self-harm—we coded it as "no". Similarly, chest pain is at times related with anxiety, but was coded "no". Hence, we are listing below only some of the diagnoses that could be closely related to mental health but were not included in our mental health definition for this study.

We coded "sleep disturbance, unspecified" as not related to mental health ("no"), whereas we included "sleep disorder" as part of insomnia into "maybe" related to mental health (see below). Our intention was to differentiate between not sleeping well at times, as opposed to poor sleep resulting in poor mental health.

Despite some evidence that adverse social circumstances can be related to poor mental health and self-harm, we restricted this study by not examining these further, coding them "no".

Appendix Table 3: Discharge diagnoses/ICD codes not related to mental health ("No").

Group	Description
Impairment of mentation or behaviour	Dementia, Alzheimer's, delirium, intellectual disability, malaise, fatigue, enduring personality change, unspecified, sleep disturbance, unspecified
Substance misuse	Smoking
Social aspects	Isolation, housing, economic circumstances, unemployment, partner violence, problems related to release from prison, maltreatment

Appendix Table 4: ICD codes and descriptions not related to mental health ("No").

Code	Description
F00–F09	Organic, including symptomatic, mental disorders
F10.0	Mental and behavioural disorders due to use of alcohol, acute intoxication
F11.0	Mental and behavioural disorders due to use of opioids, acute intoxication
F12.0	Mental and behavioural disorders due to use of cannabinoids, acute intoxication
F13.0	Mental and behavioural disorders due to use of sedatives or hypnotics, acute intoxication
F14.0	Mental and behavioural disorders due to use of cocaine, acute intoxication
F15.0	Mental and behavioural disorders due to use of other stimulants, including caffeine, acute intoxication
F16.0	Mental and behavioural disorders due to use of hallucinogens, acute intoxication
F17	Mental and behavioural disorders due to use of tobacco
F18.0	Mental and behavioural disorders due to use of volatile solvents, acute intoxication
F19.0	Mental and behavioural disorders due to multiple drug use and use of other psychoactive substances, acute intoxication
F70–F79	Mental retardation
F80–F89	Disorders of psychological development

3. Presentations maybe related to mental health ("Maybe")

For the "maybe" classification, we selected diagnoses/ICD codes that could possibly be related to self-harm or poor mental health. We included poisonings, foreign body ingestion and certain injuries, e.g., lower arm laceration as "maybe" related to mental health, as we were unable to determine the intent (Appendix Table 5). Some codes depicting the mental status, e.g., hallucinations, could be related to a one-off drug use experience or to a mental illness, hence we coded them as "maybe" related to mental health. We have not reported on "maybe" presentations as these are published elsewhere.

Appendix Table 5: Probable mental health-related ED discharge diagnoses/ICD codes ("Maybe").

Group	Subgroup	Description
Accidental harm (no information on intent)	Injuries	Common deliberate self-harm injuries, e.g., abrasions to the forearm/hip/thigh/upper limb, contact with knife, undetermined intent; asphyxiation by strangulation
	Medication poisoning	Overdose of medication used for physical health issues
	Substance poisoning	Gas and vapours, soaps and detergents, carbon monoxide, caustic alkali
	Drugs	GHB, methamphetamine, ecstasy, cannabinoids
	Alcohol	Alcohol poisoning, acute intoxication
	Mixed poisoning	Drugs, medication and biological substances
Mental status		Unable to determine if related to mental illness or substance dependence or withdrawal, e.g., hallucinations unspecified, mental status alterations
	Insomnia	Sleep disorder
	Non-compliance	With medication, e.g., diabetes treatment
	Psychosomatic	Somatoform disorder, unspecified (Note, "other somatoform disorders" is a "yes")
ED management		Observation after foreign body ingestions

Appendix 3

Discharge diagnosis coding framework

Code	Name	Subcategory
1	Respiratory (breathing)	Apnoeic Episode—Not Sleep Apnoea; Asthma, Acute; Asthma, Chronic; Breathlessness; Bronchiolitis, Acute; Bronchitis, Acute; Bronchitis, Chronic; Bronchopneumonia, Not Aspiration; Breathing Difficulty; COPD With Acute Asthma; Chronic Obstructive Airways Disease; COPD With Acute Asthma; Cough; Empyema; For Pleural Tap; Haemothorax Without Open Wound Into Thor; Lower Respiratory Tract Infection; Mixed Asthma; Pleural Effusion; Pleurisy; Pleuritic Pain; Pneumonia, Aspiration; Pneumonia, Atypical; Pneumonia, Bacterial; Pneumonia, Lobar; Pneumonia, Not Aspiration; Pneumonia, Viral; Shortness Of Breath; Wheezing, Viral Induced; Upper Respiratory Tract Infection; Respiratory Arrest; Respiratory Distress; Respiratory Distress Syndrome, Adult; Respiratory Failure; Pertussis; Pulmonary Oedema, Non-Cardiogenic; Pneumothorax, Spontaneous; Pneumothorax, Spontaneous Tension; Stridor; Throat Tightness; Lung Mass; Malignant Neoplasm Lung; Malignant Neoplasm Pharynx; Abscess, Lung.
2	Circulation (blood disorders, vascular, thrombosis, etc. heart)	Anaemia, Due To Chronic Blood Loss; Anaemia, Iron Deficiency; Anaemia, Unspecified; Bruising Tendency; Bleeding Tendency; Chronic Venous Insufficiency; Epistaxis; Gastrointestinal Haemorrhage; Haemarthrosis; Haemarthrosis, Ankle And Foot; Haemorrhage Post Tooth Extraction; Haemorrhage In Early Pregnancy, Unspecified; Haematemesis; Haematuria; Haemoptysis; Idiopathic Thrombocytopenic Purpura; Mallory-Weiss Syndrome; Melaena; Per Rectum Haemorrhage; Post Tonsillectomy Haemorrhage; Thrombocytopenia; Aortic Valve Stenosis, Other; Brachial Vein Thrombosis; Deep Venous Thrombosis Not Lower Limb; Deep Venous Thrombosis Of Lower Limb; Foot Ischaemia Due To Embolism Or Thromb; Ischaemia Of Lower Limb Unspecificed; Ischaemic Bowel; Leg Ischaemia Due To Embolism Or Thrombo; Lower Extremity Aneurysm; Superior Vena Cava Obstruction; Peripheral Vasc Comp. Of Surgical Or Med; Peripheral Vascular Disease; Pulmonary Embolism; Raynaud's Phenomenon; Superficial Venous Thrombophlebitis Of L; Vasculitis; Acute Coronary Syndrome; Angina Pectoris, Not Unstable; Angina Pectoris, Unstable; Arrhythmia, Not Arrest Or Heart Block; Atrial Fibrillation; Atrial Flutter; Beats, Premature; Beats, Premature, Ventricular; Benign Intracranial Hypertension; Bradycardia; Cardiac Arrest; Cardiac Failure; Congestive Heart Failure; Congestive Heart Failure, Hypertensive; Fluid Overload; Heart Block, Complete; Heart Block, Second Degree Av; Hypertension, Essential; Hypertensive Crisis; Hypertensive Encephalopathy, Hypertensiv; Hypertensive Heart And Renal Disease; Hypotension; Hypotension Postural; Left Ventricular Failure; Myocardial Infarction, Acute; Myocarditis, Acute; Observation For Suspected Cardiac Diseas; Other Circulatory System Disorder; Sinus Bradycardia; Sinus Tachycardia; Paroxysmal Atrial Tachycardia; Paroxysmal Supraventricular Tachycardia; Ventricular Tachycardia; Tachycardia; Supraventricular Tachycardia; Palpitations; Pericardial Effusion; Pericarditis; Pulmonary Oedema, Cardiogenic; Pneumopericardium, Atraumatic.

Appendix 3 (continued): Discharge diagnosis coding framework

3	Neurological (disability)	Seizures/tremors, dizziness, LOC1/collapse/faint, impairment (TIA2, CVA3): Acute Confusional State With Dementia; Acute Confusional State Without Dementia; Acute Delirium With Dementia; Acute Delirium Without Dementia; Altered Mental State (Not Alcohol/ Drug); Alzheimer's Disease; Amnesic Syndrome Due To Use Of Solvents; Ataxic Gait; Bell's Palsy; Brain Damage, Hypoxic; Cerebrovascular Accident With Deficit, O; Cerebrovascular Accident, Cause Unknown; Cerebral Embolism; Cerebral Infarction; Confusion; Coma; Convulsion, Afebrile; Convulsion, Status; Delirium; Encephalitis; Encephalopathy; Drowsiness; Dizziness; Faint; Facial Droop; Facial Nerve Disorder; Hydrocephalus, Acquired; Intracerebral Haemorrhage, Atraumatic; Intracranial Haemorrhage, Atraumatic; Loss Of Consciousness Of Brief Duration (Less Than 30 Minutes); Loss Of Consciousness Of Moderate Duration (30 Minutes To 24 Hours); Mental Status Alteration; Multiple Sclerosis; Neuropathy, Peripheral; Neurosurgical Shunt Infected; Neurosurgical Shunt Problem; Organic Brain Syndrome With Dementia; Other Central Nervous System Disorder; Other Peripheral Nerve Injury; Post-Ictal; Parkinson's Disease; Parkinsonism; Post-Concussion Syndrome; Paraesthesias; Palsy; Seizure; Senile Dementia Uncomplicated; Senile Dementia With Delirium; Subarachnoid Haemorrhage, Atraumatic; Tremor; Transient Ischaemic Attack.
4	Infection (general)	Chills, Fever; Fever And Chills; Flu-Like Symptoms; Glandular Fever; Gout; Glandular Swelling; Herpangina; Influenza With Other Respiratory Manifestations, Other Influenza Virus Identified; Influenza-Like Syndrome; Malaria; Meningitis; Meningitis, Viral; Nasal Congestion; Septicemia; Sepsis Due To Unspecified Staphylococcus; Sepsis Following A Procedure; Viral Infection; Viral Infective Agent; Severe Sepsis; Other Specified Sepsis; Peritonitis; Scarlet Fever; Unspecified Infection Following A Procedure; Urosepsis.
5	Skin Infection (e.g., abscess)	Acute Dacrocystitis; Atopic Eczema; Bee Sting; Candidiasis, Skin/Nails; Cellulitis, Arm Any Part Above Wrist; Cellulitis, Axilla; Cellulitis, Breast; Cellulitis, Buttock; Cellulitis, Chest Wall; Cellulitis, Ear (External); Cellulitis, External Cheek; Cellulitis, Eyelid; Cellulitis, Face Any Part Except Eye; Cellulitis, Flank; Cellulitis, Foot Except Toe; Cellulitis, Forearm; Cellulitis, Groin; Cellulitis, Hand Except Finger Or Thumb; Cellulitis, Heel; Cellulitis, Hip; Cellulitis, Knee Skin; Cellulitis, Leg Except Foot; Cellulitis, Multiple Sites; Cellulitis, Neck; Cellulitis, Periorbital; Cellulitis, Skin Site Unspecified; Cellulitis, Toe; Cellulitis, Trunk; Central Line Infected; Dermatitis; Dermatitis, Purulent; Dermatitis, Seborrheic; Decubitus Ulcer; Eczema; Erythema; Erythema Multiforme; Folliculitis; Fungal Foot Infection; Fungal Infection; Genital Rash, Non-Vesicular; Gout, Unspecified, Lower Leg; Herpes Zoster Infection; Impetigo; Necrotising Fasciitis, Lower Leg; Olecranon Bursitis; Other Bursitis Of Elbow; Other Bursitis Of Hip; Bursitis Of Shoulder; Other Non-venomous Spider Bite Infected; Toe Paronychia; Skin Infection; Rash Non-Vesicular, Not Nappy Or Urticaria; Rash, Non Specific; Rash, Urticarial; Psoriasis; Pruritis, Unspecified; Other Specified Open Wounds; Posttraumatic Wound Infection; Prepatellar Bursitis; Scabies; Scalp Blister Not Infected; Shingles; Skin Lump Not Breast; Skin Ulcer, Chronic Not Decubitus; Sunburn; Tinea Cruris; Viral Warts; Malignant Melanoma; Sebaceous Cyst; Perianal Abscess; Carbuncle, Trunk; Buttock Wound—Uncomplicated; Abscess, Trunk; Abscess, Pilonidal; Abscess, Neck; Abscess, Head Except Face; Abscess, Groin; Abscess, Gluteal Region; Abscess, Face Any Part Except Eye; Abscess, External Cheek; Abscess, Chest Wall; Abscess, Breast; Abscess, Back; Abscess, Axilla; Abscess, Anal.

Appendix 3 (continued): Discharge diagnosis coding framework

6	Diabetes	Diabetes For Stabilisation No Dka Or Com; Diabetic Coma Hyperosmolar Nonketotic (Diabetic Ketoacidosis W'Out Coma (Nid); Hyperglycaemia; Hypoglycaemic Coma Due To Insulin; Hypoglycaemia Without Coma; Type 2 Diabetes Mellitus With Proliferative Retinopathy.
7	General Medical, (e.g., allergic reaction)	Generally unwell/multiple medical complaints: Acidosis, Metabolic; Ascites; Extremity Swelling; Fatigue; Facial Swelling; Fluid, Electrolyte Or Acid-Base Disorder; Hyperkalaemia; Hypocalcaemia; Hypokalaemia; Hypomagnesaemia; Hyponatraemia; Hypothermia; Hypothyroidism; Ketoacidosis; Kidney Polycystic Disease; Lethargy; Malnutrition; Lymphadenopathy; Lymphadenitis, Acute; Lymphadenitis; Lymph Node Enlargement; Multiple Open Wounds, Unspecified; Multiple, Uncomplicated; Muscle Spasm; Muscle Weakness; Myositis; Myositis In Sarcoidosis, Hand (D868+); Numbness; Neutropenia; Other Haematological Disorder; Other Complication Of Medical Care; Other Medication Side-Effect; Other Porphyria; Physical Exhaustion; Other Postoperative Wound Complication; Pneumoperitoneum, Atraumatic; Portacath Site Haemorrhage; Post Procedural Haematoma; Syncope Not Heat; Tingling; Unconscious ? Cause; Wound Infection Following A Procedure; Weakness, Generalised; Weakness, Unilateral; Vertigo; Vasovagal Syncope; Vasovagal Attack; Collapse; Debility; Gait, Ataxic; Myeloid Sarcoma, Without Mention Of Remission; Allergic Reaction, Nos; Anaphylactic Shock Due To Serum; Anaphylaxis Due To Drug, Not Shock; Anaphylaxis Not Due To Serum, Not Shock; Angioneurotic Oedema—Angioedema; Dystonic Reaction; Lip Swelling; Mouth Swelling; Rhinitis, Allergic; Vaccination Complicated By Allergic Reac; Urticaria, Allergic; Breast Swelling; Observation In The Ed Or Short Stay Ward.
8	Pain general (including extremities)	General/multiple sites, arthritis, Muscular Pain, Atraumatic; Myalgia, Multiple Sites; Neuralgia; Osteoarthritis; Rheumatoid Arthritis; Pain In A Joint, Other Site; Other Soft Tissue Disorder; Shoulder Capsulitis; Arthralgia; Arthritis, Crystal; Arthritis, Unspecified, Site Unspecified; Fibromyalgia, Multiple Sites; Fibromyalgia, Other; Malignant Neoplasm Bone, Cartilage; Multiple Metastases; Lower/upper extremities: Achilles Tendinitis; Chondromalacea Patellae; Effusion, Elbow Joint; Effusion, Knee Joint; Ingrowing Nail Of Finger/Toe; Myalgia, Lower Leg; Myalgia, Upper Arm; Pain In Limb, Ankle And Foot; Pain In Limb, Forearm; Pain In Limb, Lower Leg; Pain In Limb, Multiple Sites; Pain In Limb, Shoulder Region; Pain In Limb, Upper Arm; Plantar Fasciitis; Radial Styloid Tenosynovitis (De Quervain); Septic Joint, Ankle And Foot; Joint Effusion; Limb Cramps; Limb Pain; Limb Swelling; Lower Extremity Skin Ulcer; Peripheral Oedema; Other Specified Soft Tissue Disorders, Lower Leg; Other Specified Disorders Of Bone, Hand; Swelling, Hand Joint; Swelling, Lower Leg Joint; Fistula Of Joint, Forearm; Chronic Osteomyelitis With Draining Sinus, Ankle And Foot; Baker's Cyst; Abscess, Leg Except Foot; Abscess, Knee Joint; Abscess, Hand Except Finger Or Thumb; Abscess, Forearm; Abscess, Arm Any Part Above Wrist; Shoulder And Upper Arm; Other Part, Closed.
9	Pain ear, nose, throat, dental (non-trauma)	Dental Infection; Dental Caries; Ear, Impacted Wax In Cerumen; Earache; Impacted Cerumen; Impacted Tooth; Other Ear Disorder; Otitis Externa; Otitis Media; Pharyngitis; Pharyngitis, Acute Streptococcal; Quinsy; Salivary Gland/Duct Calculus; Sinusitis; Sinusitis, Acute; Sore Throat; Stomatitis, Ulcerative; Throat Pain; Temporomandibular Joint Disorder; Tonsillitis; Toothache; Tympanic Membrane Perforation, Atraumati; Tympanum Rupture With Acute Otitis Media; Uvulitis; Mouth Ulcer; Abscess, Dental; Abscess, Cheek (Internal); Nose, Uncomplicated.

Appendix 3 (continued): Discharge diagnosis coding framework

10	Pain head and neck	Head; Cervical Lymphadenitis; Cervical Radiculitis; Cervical Stenosis (Cervix Uteri); Cervical Disc Disorder, Unspecified; Headache, ? Cause; Headache, Tension; Migraine Headache; Neck Pain; Neck Stiff; Trigeminal Neuralgia; Torticollis.
11	Pain chest	Chest Pain; Chest Pain—Non Trauma; Chest Tightness; Chest Wall Pain—Non Trauma; Chest Wall Pain; Costochondral Junction Syndrome; Costochondritis.
12	Pain back	Back, hip, neck, shoulder, Back Pain; Back Pain Due To Displacement Of Iv Disc; Back Sprain/Strain; Coccyx Pain; Effusion, Hip Joint; Hip, Irritable; Low Back Pain (Not An Injury); Pelvic And Perineal Pain; Radiculitis; Radiculopathy, Cervicothoracic Region; Sciatica; Rotator Cuff Syndrome; Sacroiliac Pain; Septic Hip Joint; Septic Joint, Shoulder; Spinal Cord Compression; Spinal Instabilities, Site Unspecified; Supraspinatus Tendonitis; Thoracic Back Pain.
13	Pain abdominal	Abdominal, anal, Abdomen, Acute; Abdominal Cramps; Abdominal Pain; Abdominal Pain Unspecific; Abdominal Pain, Recurrent; Abdominal Wall Anterior, Uncomplicated; Anal Pain; Biliary Colic; Biliary Tract Disease; Cholangitis, Acute; Cholecystitis, Acute; Cholelithiasis; Epigastric Pain; Flank Pain.
14	Trauma multiple	Dislocation; Dog Bite With Skin Puncture; Flash Burns; Human Or Animal Bite; Multiple Burn Sites—Erythema (First Degree); Multiple Burn Sites—Unspecified Degree; Multiple Fractures, Unspecified, Closed; Multiple Injuries Of Shoulder And Upper Arm; Muscle Strain, Site Unspecified; Observation After Foreign Body Ingestion; No Injury Found; No Injury Found Following Mva; Other Dislocation, Closed; Other Hip And Thigh Injury; Other Or Multiple Abrasions Infected; Other Or Multiple Superficial Injuries N; Other Shoulder And Upper Arm Injury; Other Site Sprain/Strain; Shoulder & Upper Arm Abrasion Not Infect; Tendonitis.
15	Trauma head (head/face, dental, throat, eye)	Acquired Deformity Of Nose; Asphyxiation By Strangulation; Auditory Canal Foreign Body; Broken Tooth/Teeth, Complicated; Broken Tooth/Teeth, Uncomplicated; Burn Of Oesophagus; Cheek Bruise Injury; Cervical, Closed #; Cervical Sprain/Strain; Concussion, ? Loss Of Consciousness; Concussion, Loss Of Consciousness ? Dura; Concussion, No Loss Of Consciousness; Face Burn—Unspecified Degree; Face Crushing Injury; Face, Abrasion Not Infected; Face, Incld Forehead, Eyebrow, Cheek; Face, Other Superficial Inj; Fracture Of Angle Of Jaw; Fracture Of Mandible, Multiple Sites; Fracture Of Mandible, Part Unspecified; Fracture Of Nasal Bones; Head Injury, Close & 1–24hrs Loss Of Con; Head Injury, Close And; Head Injury, Close And ? Duration Loss Of; Head Injury, Closed & ? Loss Of Consciou; Head Injury, Closed & No Loss Of Conscio; Head Injury, Closed And ? Loss Of Consciousness; Head Wound Or Non Venomous Bite; Injury Of Eye And Orbit, Unspecified; Jaw Dislocation, Closed; Jaw Sprain/Strain; Intracranial Haemorrhage, Traumatic; Laceration Of Eye; Multiple Superficial Injuries Of Head; Observation For Concussion; Neck Wound—Complicated; Oesophagus Foreign Body; Open Wound To Other Part Of Head Incl. Chin And Forehead; Open Wound Of Scalp, Uncomplicated; Open Wound Of Lip; Open Wound Of Mouth, Part Unspecified; Open Wound Of Tongue And Floor Of Mouth; Orbital Floor Blowout, Closed #; Other Eye Foreign Body; Other Facial Bones, Closed #; Other Open Wound Of Head, Complicated; Other Open Wound Of Head, Uncomplicated; Superficial Injury Of Scalp, Contusion; Superficial Injury To The Head. Non Specific; Superficial Injury Of Nose, Contusion; Superficial Injury Of Other Parts Of Head, Contusion; Superficial Injury Of Other Parts Of Head, Unspecified;

Appendix 3 (continued): Discharge diagnosis coding framework

		Subdural Haemorrhage, Traumatic; Superficial Injury Of Lip And Oral Cavity, Abrasion; Superficial Injury Of Lip And Oral Cavity, Contusion; Superficial Injury Of Lip And Oral Cavity, Unspecified; Superficial Injury Of Nose, Contusion; Scalp Abrasion Not Infected; Superficial Injury Of Ear; Unspecified Contusion Of Eye.
16	Trauma trunk, including neck	Abdominal Wall Sprain/Strain; Acetabulum, Closed #; Acromioclavicular Joint Dislocation, Clo; Acromioclavicular Joint Sprain/Strain; Cauda Equina Injury Without Fracture; Chest Wall Contusion; Coccyx Sprain/Strain; Closed Clavical Fracture; Contusion Of Lower Back And Pelvis; Contusion Of Shoulder And Upper Arm; Fracture Of Acromial Process; Fracture Of Body Of Scapula; Fracture Of Lumbar Vertebra, L3 Level; Fracture Of Lumbar Vertebra, L5 Level; Fracture Of Neck Of Femur, Part Unspecified; Gastrointestinal Tract Foreign Body Nec; Hip & Thigh Abrasion Not Infected; Hip & Thigh Splinter Not Infected; Hip Contusion; Hip Dislocation, Closed; Hip Dislocation, Open; Hip Sprain/Strain; Intertrochanteric, Closed #; Injury Of Spleen, Unspecified; Lumbar Sprain/Strain; Lumbar, Closed #; Major Laceration Of Liver; Muscle Strain, Shoulder Region; Neck Sprain/Strain; Open Wound Of Hip And Thigh; Other Abdominal Wall Injury; Other Neck Injury; Rib Sprain/Strain; Ribs, Closed #; Shoulder Dislocation, Closed; Shoulder Dislocation, Open; Shoulder Sprain/Strain; Pubis, Closed #; Pelvis Sprain/Strain; Other Trunk Injury; Other Vertebral, Closed #; Recurrent Dislocation And Subluxation Of Joint, Shoulder Region; Pneumothorax With Open Wound Into Thorax; Sacrum Sprain/Strain; Rotator Cuff Sprain/Strain; Superficial Injury Of Other Parts Of Neck, Contusion; Subcapital Nof Fracture; Sprain And Strain Of Other Specified Sites Of Hip; Thoracic Sprain/Strain; Whiplash Injury To Neck; Unspecified Open Wound Of Other And Unspecified Parts Of Shoulder Region.
17	Trauma upper extremity	# Shaft Radius, Closed; Amputation Of Fingers Excluding Thumb—Uncomplicated; Closed Finger Fracture; Closed Metacarpal Fracture; Colle's Fracture; Contusion Of Elbow; Contusion Of Finger(s) Without Damage To Nail; Contusion Of Other And Unspecified Parts Of Forearm; Contusion Of Other Parts Of Wrist And Hand; Elbow Dislocation, Closed; Elbow Other Superficial Injury Not Infected; Elbow Sprain/Strain; Elbow Wound, Complicated; Elbow Wound, Uncomplicated; Dislocation Of Interphalangeal (Joint), Hand; Finger Abrasion Infected; Finger Abrasion Not Infected; Finger Blister Infected; Finger Dislocation, Closed; Finger Dislocation, Open; Finger Hand Wrist Tenosynovitis; Finger Other Superficial Injury Not Infected; Finger Paronychia; Finger Sprain; Finger Superfic'l Foreign Body Not Infec; Finger Wound Complicated; Finger Wound Uncomplicated; Forearm Abrasion Not Infected; Forearm Burn; Forearm Burn—Partial Thickness; Forearm Nonvenom's Insect Bite Infected; Forearm Nonvenom's Insect Bite Not Infec; Forearm Other Superficial Injur Not Infe; Forearm Other Superficial Injury Infecte; Forearm Sprain/Strain; Forearm Superficial Injury; Forearm Wound Or Non Venomous Bite; Forearm Wound, Complicated; Forearm Wound, Uncomplicated; Fracture Of Distal Phalanx (Finger); Fracture Of Distal Phalanx Of Thumb; Fracture Of Forearm, Part Unspecified; Fracture Of Head Of Radius; Fracture Of Lower End Of Radius, Unspecified; Fracture Of Phalanx (Fingers), Part Unspecified; Fracture Of Proximal Phalanx (Finger); Fracture Of Shaft Of Radius, Part Unspecified; Fracture Of Shafts Of Both Ulna And Radius; Fracture Of Upper End Of Humerus, Part Unspecified; Gamekeeper's Thumb; Hand Abrasion Infected; Hand Abrasion Not Infected; Hand Or Finger Burn—Partial Thickness; Hand Or Finger Burn—Unspecified Degree; Hand Other Superficial Injury Not Infect; Hand & Finger Wounds Or Non Venomous Bit; Hand (Excl Finger Only) Crushing Injur; Lower End Radius, Closed #; Median Nerve Injury; Metacarpal Fracture/S;

Appendix 3 (continued): Discharge diagnosis coding framework

		Multiple Fractures Of Fingers; Open Wound Of Other Parts Of Wrist And Hand; Open Wound Of Other Parts Of Forearm; Other Hand Injury, Except Finger; Other Elbow, Forearm And Wrist Injury; Wrist Abrasion Not Infected; Wrist Crushing Injury; Wrist Or Hand Wound Complicated; Wrist Or Hand Wound Uncomplicated; Wrist Other Superficial Injur Not Infect; Wrist Sprain/Strain; Wrist Wound, Complicated; Wrist Wound, Uncomplicated; Wrist Wound, With Tendon Involvement; Upper Arm Sprain/Strain; Upper Arm Wound, Complicated; Upper Arm Wound, Uncomplicated; Upper End Humerus #, Closed; Thumb Crushing Injury; Sprain And Strain Of Finger(s), Part Unspecified; Scaphoid Fracture Wrist; Proximal Radius Fracture; Ring Unable To Be Removed From Finger; Shaft Or Other Part Humerus #, Closed; Subungual Haematoma—Finger; Spontaneous Rupture Of Other Tendons, Upper Arm;
18	Trauma lower extremity	Abrasion Of Lower Limb, Level Unspecified; Achilles Tendon Sprain/Strain; Ankle Burn, Partial Thickness; Ankle Sprain/Strain; Ankle Wound—Uncomplicated; Ankle And Foot, Excluding Toes; Bimalleolar, Closed; Bimalleolar, Open; Calcaneus, Closed #; Calcaneus, Open #; Closed Patella #; Contusion Of Knee; Contusion Of Ankle; Contusion Of Other And Unspecified Parts Of Lower Leg; Contusion Of Thigh; Contusion Of Toe(S) With Damage To Nail; Contusion Of Toe(S) Without Damage To Nail; Foot & Toe Abrasion Infected; Foot & Toe Abrasion Not Infected; Foot & Toe Other Superficial Injury Not Infected; Foot & Toe Superficial Fb Not Infected; Foot & Toe Wound—Complicated; Foot & Toe Wound—Uncomplicated; Foot & Toes Wound—Uncomplicated; Foot (Excl Toe Only) Crushing Injury; Femur Distal Part, Closed #; Foot Sprain/Strain; Fracture Of Cuboid, Foot; Fracture Of Fibula, Part Unspecified; Fracture Of Intracapsular Section Of Femur; Fracture Of Other Toe; Fracture Of Shaft Of Fibula; Fracture Of Shaft Of Tibia With Fracture Of Fibula (Any Part); Fracture Of Talus; Fracture Of Upper End Of Fibula; Fracture Of Upper End Of Tibia With Fracture Of Fibula (Any Part); Great Toe, Closed #; Great Toe, Open #; Hand Splinter Infected; Hand Splinter Not Infected; Hand Sprain/Strain; Hand Superficial Foreign Body Infected; Hand Superficial Foreign Body Not Infect; Injury Of Unspecified Muscle And Tendon At Ankle And Foot Level; Injury Of Other Muscle(S) And Tendon(S) At Lower Leg Level; Injury Of Achilles Tendon; Knee Cartilage Injury; Knee Ligament Injury; Knee Ligament Injury, Old; Knee Meniscus Injury; Knee Wound—Complicated; Knee Wound—Uncomplicated; Knee, Leg & Ankle Abrasion Infected; Knee, Leg & Ankle Abrasion Not Infected; Knee, Leg & Ankle Nonvenom's Insect Bite; Leg Burn—Unspecified Degree; Lateral Malleolus, Closed #; Leg Superficial Injury; Lower Leg Crushing Injury; Lower Leg Muscle Sprain; Lower Leg Sprain/Strain; Medial Malleolus, Closed; Metatarsal Fracture; Multiple Open Wounds Of Lower Leg; Open Patella #; Open Wound Of Thigh; Open Wound Of Other Parts Of Lower Leg; Open Wound Of Lower Leg, Part Unspecified; Other Fracture Of Lower End Of Tibia; Other Fracture Of Lower End Of Tibia With Fracture Of Fibula (Any Part); Other Knee, Leg, Ankle, And Foot Injury; Toe Crushing Injury; Toe Dislocation, Closed; Toe Sprain/Strain; Thigh Sprain/Strain; Thigh Wound—Uncomplicated; Unspecified Knee Sprain/Strain; Patella Dislocation, Closed; Rupture Of Anterior Cruciate Ligament; Sprain/Strain Lateral Collateral Ligament; Sprain/Strain Of Medial Collateral Ligament; Trimalleolar, Closed;

Appendix 3 (continued): Discharge diagnosis coding framework

19	Function eye	Blurred Vision; Cataract, Nec; Cataract, Traumatic; Conjunctival Oedema; Conjunctivitis Viral; Conjunctivitis, Acute; Conjunctivitis, Allergic; Cornea And Conjunctival Sac; Corneal Abrasion; Corneal Foreign Body; Corneal Ulcer, Atraumatic; Dry Eyes; Diplopia; Eye Injury Excluding Foreign Body In Ext; Eye Review; Eye, Discharging; Eye, Inflammation; Eye, Loss Of Vision In One; Eye, Painful; Eye, Red; Eye, Swelling; Eyeball; Eyelid And Periocular Area; Eyelid Foreign Body; Eyelid Inflammation; Eyes, Loss Of Vision In Both; Foreign Body In External Eye; Iritis; Labyrinthitis; Other Medical Eye Disorder; Vitreous Haemorrhage; Retinal Tear Without Detachment; Subconjunctival Haemorrhage, Atraumatic; Abscess, Eyelid;
20	Function gas-trointestinal	Constipation/PR4 bleed, nausea/diarrhea/ vomiting, diverticulitis; Abdominal Distension, Feeling Of; Abdominal Pain Gi Symptoms; Abdominal Swelling; Acute Pancreatitis, Unspecified; Anal/Rectal Haemorrhage (Not Pr Bleed; Anal Fissure; Anal Fistula; Appendicitis, Acute; Bowel Obstruction; Colitis, Ulcerative; Colitis, Unspecified; Colostomy Complication; Constipation; Constipation With Overflow Incontinence; Choking Episode; Crohn's Disease; Diarrhoea; Diarrhoea, Bloody; Dehydration; Enteritis Bacterial; Diverticulitis; Diverticulosis; Feeding Problems In Elderly Or Infant; Food Poisoning; Gastritis; Gastritis Acute; Gastro-Oesophageal Reflux; Gastroenteritis And Colitis Of Unspecified Origin; Gastroenteritis Viral; Gastroenteritis, Infectious; Gastroenteritis, Noninfectious; Gastrostomy Feeding Tube Blockage; Groin Pain; Haemorrhoids; Haemorrhoids External Thrombosed; Hepatic Coma; Hepatitis; Hepatitis, Acute; Intestinal Colic; Inguinal Hernia; Inguinal Hernia, Bilateral; Irritable Bowel Syndrome; Jaundice; Liver Disease, Chronic; Nausea; Oesophageal Stricture; Oesophagitis; Oesophagitis, Reflux; Other Gastroenteritis And Colitis Of Infectious Origin; Other Hernia; Other Liver Disorder; Rectal Foreign Body; Peptic Ulcer; Pancreatic Pseudocyst; Stomach Foreign Body; Swallowed Foreign Body; Volvulus; Vomiting, Not Blood; Ischaemic Bowel; Melaena;
21	Function urine	Cystitis; Cystitis, Acute Haemorrhagic; Dysuria; Indwelling Urinary Catheter Change; Loin Pain; Nephrotic Syndrome; Ureteric Calculus (Stone); Ureteric Colic; Urethral Catheter Blocked; Urethral Catheter Leaking; Urethral Colic; Urinary Incontinence; Urinary Retention; Urinary Tract Infection; Pyelonephritis; Pyelonephritis, Acute; Suprapubic Catheter Blocked; Renal Calculus; Renal Colic; Renal Disease; Other Urinary Catheter Device Complications; Proteinuria; Renal Failure, Acute; Renal Failure, Chronic;
22	Function reproductive	Abdominal Pregnancy; Endometriosis; Endometritis; Ectopic Tubal Pregnancy Ruptured; Disorder Of Penis, Unspecified; Dysmenorrhoea; Epididymitis; Epididymo-Orchitis; External Genitalia—Other Parts, Complicated; Hyperemesis Gravidarum; Hyperemesis Gravidarum With Metabolic Disturbance; Labour, Premature; Menorrhagia; Miscarriage, Complete; Miscarriage, Incomplete; Miscarriage, Inevitable; Miscarriage, Missed; Miscarriage, Threatened; Normal Period; Orchitis; Other Menstrual Problem; Pregnancy, Ectopic; Vomiting During Pregnancy; Vaginal Discharge; Vaginal Foreign Body; Vaginal Haemorrhage (Not Pv Bleeding); Vaginitis, Trichomonal; Vagina, Uncomplicated; Per Vaginal Bleeding ? Cause; Ovarian Cyst; Ovarian Cyst Haemorrhage; Ovarian Dysfunction; Ovarian Torsion; Testicular Dysfunction; Testicular Torsion; Sexually Transmitted Disease; Scrotal Swelling; Scrotum And Testis, Uncomplicated; Period Pain; Penile Oedema; Penis, Uncomplicated; Pelvic Inflammatory Disease; Pelvic Inflammatory Disease, Acute; Postpartum Endometritis; Postmenopausal Haemorrhage; Paraphimosis; Sexual Assault Examination; Mastitis; Bartholin's Gland; Breast Mass;

Appendix 3 (continued): Discharge diagnosis coding framework

23	Miscella-neous, tests	Review/tests, medication requests, social; Blood Test Only—Not Bal; Dressing Change; Inadequate Housing; Laboratory Test Only; Lack Of Care Of Specified Person; Medical Certificate Only; Medication Only; No Disease Found With No Symptoms; Operative Wound Dehiscence; Results Only; Suture Removal; Social Admission; Social Admission While Awaiting Nursing; Social Problem; Prescription Only; Plaster Cast Aftercare; Plaster Of Paris Check; Plaster Of Paris Removal Only; Person Consulting For Explanation Of Investigation Findings; Parental Anxiety Only; Reassurance; Other Specified Complication Of Surgical And Medical Care Nec; Prophylactic Antibiotic For Dental Treat; Rhinitis, Unspecified; Wound Review;
24	Exposure poisoning	Alcohol Intoxication; Antiallergic Drug Poisoning; Anticholinergic Poisoning; Anticonvulsant Poisoning; Antidepressants Poisoning; Antiemetic Poisoning; Antihistamine Poisoning; Benzodiazepines Poisoning; Beta-Blocker Poisoning; Carbon Monoxide Poisoning; Caustic Alkali Poisoning; Caustic Substance Poisoning; Codeine Poisoning; Ethyl Alcohol Poisoning; Diazepam Poisoning; Insulin Poisoning; Iron Poisoning; Marijuana Poisoning; Mental And Behavioural Disorders Due To Use Of Alcohol; Mental And Behavioural Disorders Due To Use Of Cannabinoids, Acute Intoxication; Mental And Behavioural Disorders Due To Use Of Opioids, Acute Intoxication; Methanol; Mushroom Poisoning; Nonsteroidal Anti-inflammatory Poisoning; Opiate Poisoning; Other Alcohol Poisoning; Other Analgesic Or Antipyretics Poisoning; Other Antibiotic Poisoning; Other Antihypertensive Poisoning; Other Drug Affecting Cvs Poisoning; Other Drug Affecting Git Poisoning; Other Autonomic System Drug Poisoning; Other Cns Stimulant Poisoning; Other Poisoning; Other Metal Poisoning; Sympathomimetic Poisoning; Poisoning By Narcotics And Hallucinogens; Poisoning By Psychostimulant—Ecstasy; Other Psychotropic Agent Poisoning; Other Sedative Or Hypnotic Poisoning; Salicylate Poisoning; Paracetamol Poisoning; Other Vasodilator Poisoning; Other Systemic Agent Poisoning; Ment & Behave Disorders D/T Multiple Drug Use & Use Of Psychoactive Substances, Acute Intoxication; Smoke Inhalation; Soap And Detergent Poisoning;
25	Mental health, illness	Bipolar Affective Disorder, Unspecified; Hallucinations; Manic Disorder; Schizophrenia, Unspecified; Other Specified Mental Disorder Usually Onset Adolescent; Psychotic Episode;
26	Mental health, depression	Depression; Depression With Anxiety; Depression, Neurotic; Depressive Disorder; Depressive Episode, Unspecified; Severe Depressive Episode Without Psychotic Symptoms;
27	Mental health, anxiety	Anxiety; Anxiety State/Panic Attack/Neurotic; Anxiety, In Acute Stress Reaction; Neurosis—Generalised Anxiety; Neurosis, Impulsive/Obsessional;
28	Mental health, self-harm	Observation After Suicide Attempt;
29	Mental health, other	Antisocial Behaviour; Anorexia; Anorexia Nervosa; Behavioural Problems, Adolescent; Behavioural Problems, Adult; Behavioural Problems, Child; Bulimia; Emotional Crisis—Acute Reaction To Str; Emotional Crisis—Adjustment Reaction; Emotional Crisis, Nec; Emotional Disorder; Feared Complaint Unfounded; Grief Reaction; Hyperventilation, Psychogenic; Hysteria; Mental Disorder, Not Otherwise Specified; Multiple Somatic Symptoms For Investigation; Stress Reaction, Acute; Personality Disorder; Psychosomatic Disorder; Other Psychotropic Drugs, Not Elsewhere Classified; Paranoid State Unspecified; Respite Care; Psychogenic Non-Epileptic Seizure; Violent Behaviour;

Appendix 3 (continued): Discharge diagnosis coding framework

30	Mental health, alcohol	Alcohol Intoxication In Alcoholic; Alcohol Withdrawal Syndrome; Alcoholic Hallucinosis; Alcoholic Psychosis; Cirrhosis With Alcoholism; Gastritis, Alcoholic;
31	Mental health, drug	Drug Addiction; Drug Induced Mental Disorder; Drug Withdrawal Syndrome; Intravenous Drug User; Mental And Behavioural Disorders D/T Use Of Cannabinoids, Residual And Late-Onset Psychotic Disorder; Mental And Behavioural Disorders Due To Use Of Cannabinoids, Withdrawal State; Opioid Withdrawal Syndrome; Opium Addiction; Psychoactive Substance Withdrawal; Psychotic Disorder Due To Use Of Solvents; Psychotic Disorder Due To Use Of Hallucinogens; Psychotic Disorder Due To Use Of Solvents; Unspecified Mental Disorders Due To Solvent Use;
32	Miscella-neous, did not wait	Did Not Wait For Treatment;
33	Miscella-neous, not recorded	Not recorded

Appendix 4

Appendix Table 6: Number and prevalence of ED patients' first mental health-related ED presentation within 12 months by age, gender, ethnicity and deprivation.

Socio-demographic information	Detail	First ED presentations *N*=33,597 (%)	Current MH client *n*=1,145 (%)	Prior[1] MH client *n*=2,921 (%)	Non-MH client *N*=29,531 (%)
Age	10–14	1,682 (5.0)	69 (6.0)	103 (3.5)	1,510 (5.1)
	15–24	6,656 (19.8)	272 (23.8)	818 (28.0)	5,566 (18.9)
	25–34	5,722 (17.0)	223 (19.5)	640 (21.9)	4,859 (16.5)
	35–44	4,020 (12.0)	191 (16.7)	409 (14.0)	3,420 (11.6)
	45–54	41,251 (2.3)	177 (15.5)	397 (13.6)	3,551 (12.0)
	55–64	3,648 (10.9)	127 (11.1)	232 (7.9)	3,289 (11.1)
	65+	7,744 (23.0)	86 (7.5)	322 (11.0)	7,336 (24.8)
Gender	Female	17,208 (51.2)	623 (54.4)	1,593 (54.5)	14,992 (50.8)
	Male	16,382 (48.8)	521 (45.5)	1,328 (45.5)	14,533 (49.2)
	Unknown	7 (0.0)	1 (0.1)	0 (0.0)	6 (0.0)
Ethnicity (prioritised)	Māori	3,362 (10.0)	207 (18.1)	481 (16.5)	2,674 (9.1)
	Pacific	2,830 (8.4)	64 (5.6)	178 (6.1)	2,588 (8.8)
	Asian	3,289 (9.8)	40 (3.5)	125 (4.3)	3,124 (10.6)
	Other	24,116 (71.8)	834 (72.8)	2,137 (73.2)	2,1145 (71.6)
NZDep2018 deprivation quintile[2]	1	9,696 (28.9)	260 (22.7)	663 (22.7)	8,773 (29.7)
	2	7,196 (21.4)	184 (16.1)	578 (19.8)	6,434 (21.8)
	3	8,802 (26.2)	334 (29.2)	849 (29.1)	7,619 (25.8)
	4	5,068 (15.1)	263 (23.0)	533 (18.3)	4,272 (14.5)
	5	2,835 (8.4)	104 (9.1)	298 (10.2)	2,433 (8.2)

[1]Within last 5 years; [2]1=lowest deprivation and 5=highest deprivation.

Increasing access to cataract surgery in Counties Manukau by optimising the clinical pathway: a quality improvement report

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ABSTRACT

AIM: To streamline the cataract surgery pathway to improve the time from first specialist assessment (FSA) to surgery, while reducing the clinical priority assessment criteria (CPAC) score from 55 to 50.

METHOD: A quality improvement project using Lean Six Sigma tools and the Model for Improvement. Most data were collected from the i.Patient Manager (iPM) system and analysed using statistical process control charts. Change interventions included combining FSA and pre-admission clinics (PAC); post-operative telephone review by non senior medical officers (SMO); and using our own surgeons in private theatres.

RESULTS: The standard cataract pathway was reduced from 5 to 3 appointments. This removed 1,514 hours of appointments, released 113 SMO hours and saved patients NZ$156,000 in indirect costs over a year. The average waiting time from FSA to surgery decreased from 90 to 77 days (-13.5%). The number of overdue patients reduced from 127 to 44 (-35%). The average number of patients on the FSA waiting list dropped from 322 to 205 (-40%). There was no change to the proportions of surgeries or appointment attendance rates by ethnicity. Average monthly cataract surgeries increased from 192 to 215 (+12%), and the CPAC score threshold was decreased to 50 in February 2021.

CONCLUSION: Despite significant demand pressures, and the disruptions of COVID-19, we were able to reduce the CPAC score for accessing cataract surgery by optimising the clinical pathway to better utilise staff capacity and maximise value for patients.

Cataracts are a common, highly treatable cause of vision impairment in New Zealand. While surgical intervention is cost effective and offers numerous benefits to patients, cataract services are under pressure from rising demand that is forecast to continue with the growing and ageing population.[1] Patients can experience long waits and delays, with potential clinical implications from sight loss, falls and reduced quality of life.[2] Access to cataract surgery is determined by the clinical priority assessment criteria (CPAC) score, yet these scores vary by the district a person lives in.[1,3] Reducing these geographical inequities is now a national focus, with the Minister of Health announcing in July 2023 a CPAC score of 46 for cataract surgeries to be phased in across all Health New Zealand – Te Whatu Ora health districts.[4] Standardising the threshold will further increase demand in some districts, necessitating innovative service delivery models to release capacity to undertake more procedures.

In 2020, the Counties Manukau District identified increasing access to cataract surgery as a priority for service improvement. From January to December 2019, Counties Manukau had 2,940 patients referred to the cataract pathway—the majority (75%) referred from ophthalmologic sub-specialities. The overall surgical conversion rate was 85%. On average, 410 patients were on the waiting list for a first specialist assessment (FSA) at any given time, of which 171 were overdue (42%) and with an average waiting time of 154 days. The average time from FSA to surgery was 89 days. On top of this demand, Counties Manukau had a significant access barrier, requiring a CPAC score of 55 for cataract surgery, higher than its regional counterparts Auckland (45) and Waitematā (48). The Ophthalmology Department partnered with Ko Awatea, the centre for improvement and innovation at Counties Manukau, to embark on a quality improvement initiative to address these issues.

In this article we report on this initiative to reduce the CPAC threshold for cataract procedures in Counties Manukau while managing existing demand by optimising the outpatient surgery

pathway. The project aim was to maintain or improve time from FSA to surgery while reducing the CPAC score from 55 to 50 for new cataract patients by December 2022. We describe our approach and lessons learned, offering insights for other districts now preparing for projected increases in cataract procedures.

Method

A literature review was conducted to answer the questions: 1) what are best-practice models of cataract surgery services and improving access to cataract surgery, and 2) what makes these models work well in practice? A rapid scoping methodology was used with the aim of achieving a balance of breadth and depth, and confidence that best practice and evidence-informed innovation were identified. The project team used the review findings alongside an analysis of local data to identify priority areas for process improvement, testing and implementation.

A combination of Lean Six Sigma tools and the Model for Improvement[5] were employed to guide the improvement approach. As a high-volume, low-complexity procedure, the cataract pathway is an ideal candidate for process refinement using these techniques.[6] Lean aims to systematically improve efficiency by reducing processes and procedures that do not add value, while Six Sigma focusses on process control and unwarranted variation reduction. The Model for Improvement uses small tests of change: Plan-Do-Study-Act (PDSA) cycles to rapidly trial different ways of working.

Critical to these improvement methods was participation from all staff groups involved in the outpatient cataract surgery pathway. Regular meetings were held with senior medical officers (SMO), nurses, optometrists, technicians, administration and surgical booking staff, both by role and collectively to develop the improvement framework. Specific activities with staff groups included process mapping, root cause analysis, generating change ideas and developing action plans. This initiative was part of a wider improvement programme in ophthalmology subspecialities including glaucoma and intravitreal injections, which allowed cross-pollination of insights, ideas and staff engagement.

Measurement strategy

The main outcome measures for the cataract pathway project were time from FSA to surgery (the average waiting time a patient experienced between FSA and surgery, expressed in days, and measured monthly) and total number of cataract surgeries.

To understand changes in demand and capacity, we measured the total number of patients on the FSA waiting list, additions to the FSA waiting list, average waiting days to FSA and overdue FSA, and number of appointments per patient.

We assessed resource allocation by measuring aspects of appointment delivery, including proportions delivered by different health professional roles (SMO/optometrist/nurse/technician), delivery modes (phone/in person) and facility utilisation (clinic spaces, internal/external theatres).

To support equity monitoring, surgery volumes and appointment "did not attend" (DNA) rates were stratified by ethnicity.

Data collection and analysis

Data collection and analysis was informed by Provost and Murray's best practice guidance for healthcare quality improvement.[7] Most data, including volumes and types of appointments, surgeries, and waiting list information were collected from the i.Patient Manager system (iPM). This was supplemented with manual data collection of the times taken to complete activities within appointments and structured observations, and collection of staff and patient feedback within PDSA cycle reviews.

Key measures were analysed with statistical process control charts throughout the improvement initiative to assess process variation over time. We present these charts to evaluate the impact of change package implementation from the baseline period (January to December 2019) to the project close in November 2022. All charts were produced in Microsoft Excel using QiMacro.

Change package

The cataract surgery pathway process at Counties Manukau in 2019 involved a minimum of five in-person appointments (FSA), pre-admission clinic (PAC), surgery, 1-day post-operative follow-up (FU) and 30-day post-surgery FU. Our literature review demonstrated that this number of appointments could be reduced without negatively impacting clinical outcomes, with a model of care based on three appointments for most patients, which is increasingly common in other centres overseas.[6,8–10]

While some centres have focussed on

streamlining the community to specialist interface to achieve appointment reduction through devolution of FSA to community-based optometrist assessment,[8] our team decided to begin with combining the FSA and PAC into one appointment. This reflected our project scope within secondary services only, and our referrals mostly come from other ophthalmology sub-specialties (75% compared to 25% from primary care). Counties Manukau also has one of the lowest per capita number of optometrists in the country, which decreased access from this group.

The combined FSA and PAC required administrative changes and scheduling coordination, which were tested in PDSA cycles. These changes included booking under a new clinical code for the appointment type and reviewing and aligning all activities (processing tests, technical tests, time with doctor, time with pre-admission nurse) to maximise staff use of time and minimise unnecessary testing. This meant as soon as a patient was deemed to not qualify or want surgery they could then exit without further tests. A patient information leaflet was also created and provided at this appointment to support patients with understanding and preparing for the cataract procedure and recovery.

Additionally, the team saw an opportunity to remodel the 1-day post-operative FU. The literature indicates that nurse-led telephone FU after uncomplicated surgery, utilising a structured protocol, is safe and acceptable to patients.[12–13] The team were already successfully utilising non-SMO FU prior to the project initiation but began substituting in-person with telephone consultation from April 2020. The FU model for an individual patient was determined by the surgeon at conclusion of the cataract procedure, with low-risk patients being assigned to telephone FU by a nurse, optometrist or technician. Surgeons continued to see patients who they considered higher risk (e.g., due to a complication in surgery, risk of raised post-operative intra-ocular pressure or who had a toric intra-ocular lens implanted) or those who were determined at the time of telephone FU to require surgeon review.

The change to FU occurred during the first COVID-19 Alert Level 4 response, alongside the wider adoption of telephone appointments in outpatient services at Counties Manukau. While this meant implementation proceeded rapidly, there were challenges with technology access, which were resolved by the Department acquiring three additional phones with conference call capability that allowed connection to interpreters.

Besides the appointment model of care, the other major driver impacting access to cataract surgery was theatre space. Capacity was assessed as 100 surgeries per month based on theatre space in Counties Manukau facilities, a surgery duration of 15 minutes and the sessions that the service could organise and perform. The service relied on outsourcing to private providers to deliver additional surgeries at NZ$2,800 per surgery. From June 2020 the team increased "wet leasing" (using our Counties Manukau surgeons in private theatres) to accommodate the additional demand. Wet leasing cost $2,400 per surgery. Greater use of wet leasing became possible due to the reduction in face-to-face appointments in the pathway that freed up SMO capacity, as well as recruitment of another SMO to cover an existing vacancy. In February 2021, the CPAC score was dropped from 55 to 50.

Results

Surgery volumes and time to surgery

With the CPAC score reduced to 50, the average monthly volume of cataract surgeries increased 12% (n=192 to n=215). The average percentage of wet leased surgeries increased from 28% (54/192) to 53% (114/215) and outsourced surgeries decreased from 22% (42/192) to 7% (15/215). Overall, 61% (130/215) of cataract procedures were performed outside of Counties Manukau facilities (see Figure 1). There were 426 outsourced surgeries avoided through wet leasing, which led to a cost saving for the public health system of $170,400 (based on fundamental costs provided by the Health Economics team at Counties Manukau Health of a wet leased surgery at $2,400 and an outsourced surgery at $2,800).

The volume of in-house surgeries was stable at around 100 surgeries per month until August 2021, when it decreased to around 66 per month because of the high volume of elective surgery cancellations due to COVID-19 in the community; surgical session utilisation dropped from 95% (January 2019 to July 2021) to 85% (August 2021 onwards). This influenced the overall outcome measure of average time from FSA to surgery, which initially decreased from 89 to 77 days (-13.5%), but then increased to 101 days from August 2021 (see Figure 2).

Managing demand

The number of patients on the FSA waiting list dropped from 322 in the period from January

Figure 1: Statistical process control charts of total cataract surgeries—the proportion outsourced or wet leased.

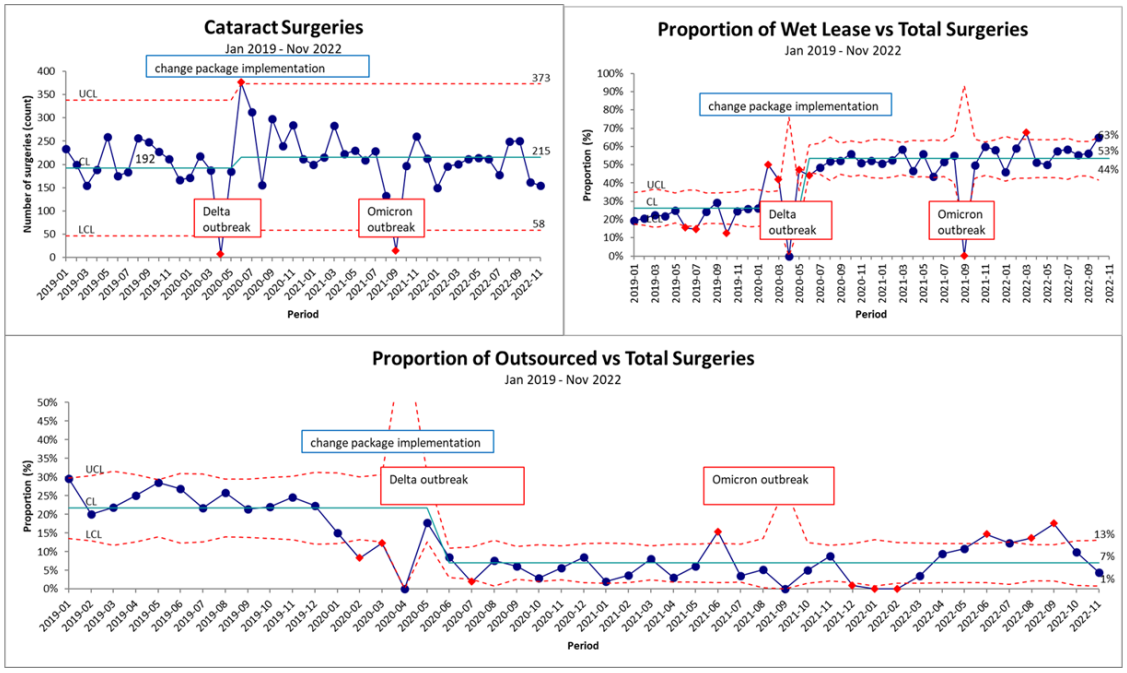
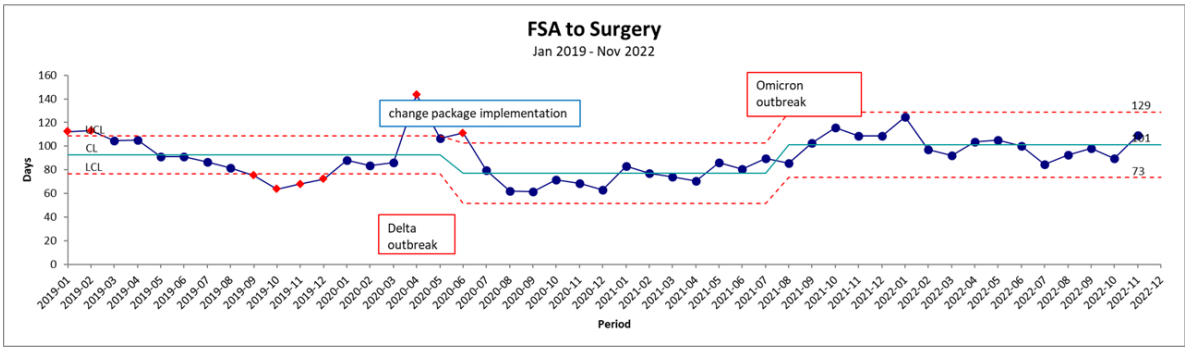


Figure 2: Impact of COVID-19 outbreaks on time to surgery.



2019 to change package implementation in June 2020, down to 205 (-40%; the 2019 baseline was impacted by special cause variation, whereby an SMO took extended leave during the first part of the year and *ad hoc* recovery efforts were evident in the last quarter of the year). The average waiting days decreased from 139 to 79 (-43%) and the number of overdue patients reduced from 127 to 44 (-35%). The improvement was even higher until April 2022 when the removal of COVID-19 protections initiated a significant demand rise (red circle, +89% Figure 3).

Resource reallocation

For most patients, the cataract surgery clinical pathway was reduced from five to three in-person appointments. This removed 1,514 hours of appointments, which is estimated to have saved patients more than $156,000 in indirect costs (e.g., loss of productivity, transportation, and caregiver costs) on an annual basis.[14]

Over the 12 months following the implementation of combined FSA and PAC (July 2020 to June 2021), 452 SMO consultations—or 113 SMO hours—could be reallocated to other activities including surgery and seeing patients with more complex needs.

Similarly, since the re-designed 1-day post-operative FU began in May 2020, there was an improved allocation of SMO resources to higher clinical need. Sixty-four percent (96/150 per month) of FUs were delivered to patients via telephone by non-SMO staff. Phone appointments were undertaken in Busy Pods (sound-proofed office booths), which also allowed the redeployment of clinic rooms for other in-person clinics. Over the year

Figure 3: Rebound increase in demand post-COVID.

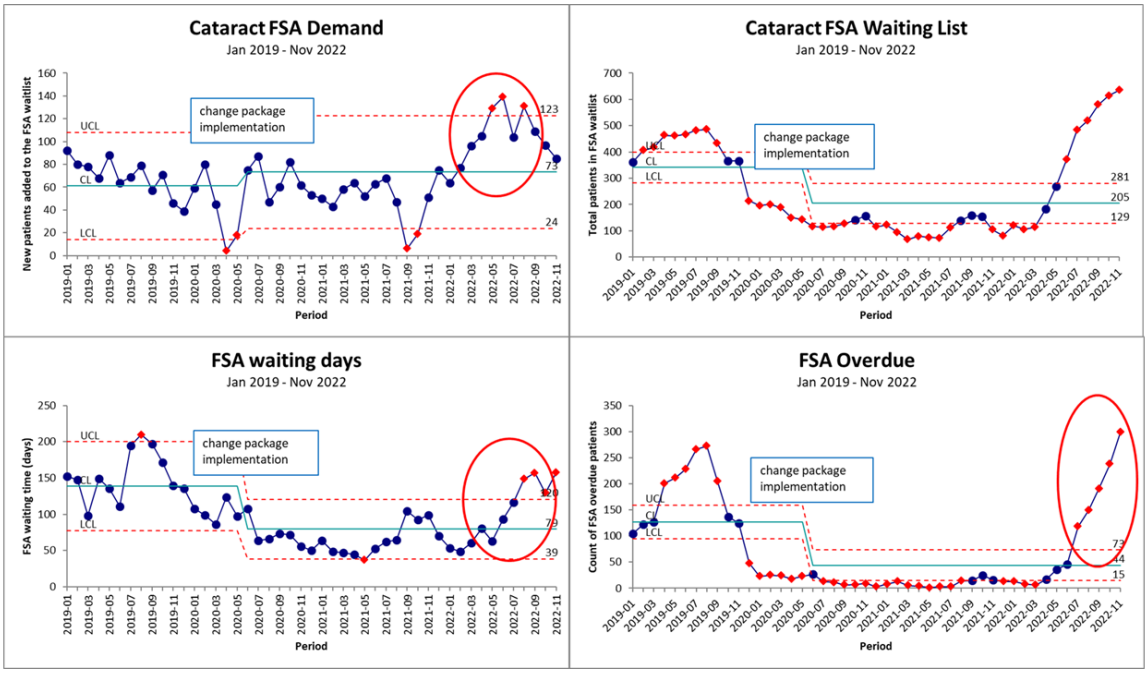


Figure 4: Surgeries by ethnicity and proportion of population in Counties Manukau over 60-years-old, 2019–2022.

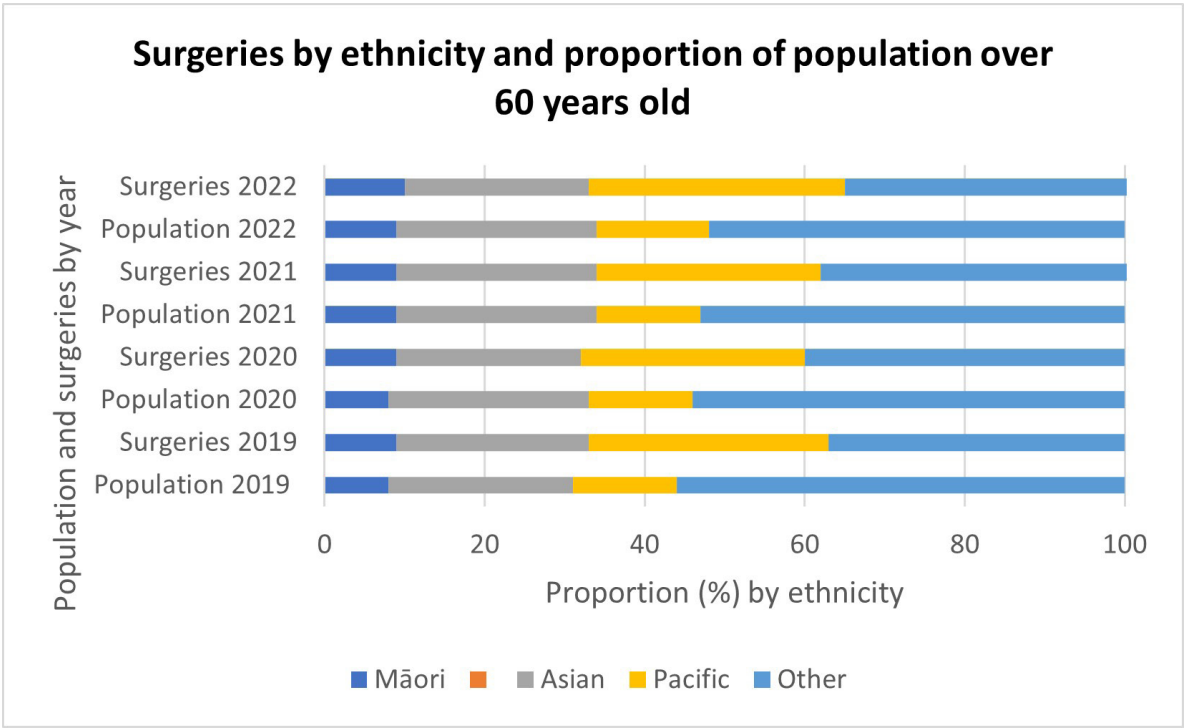
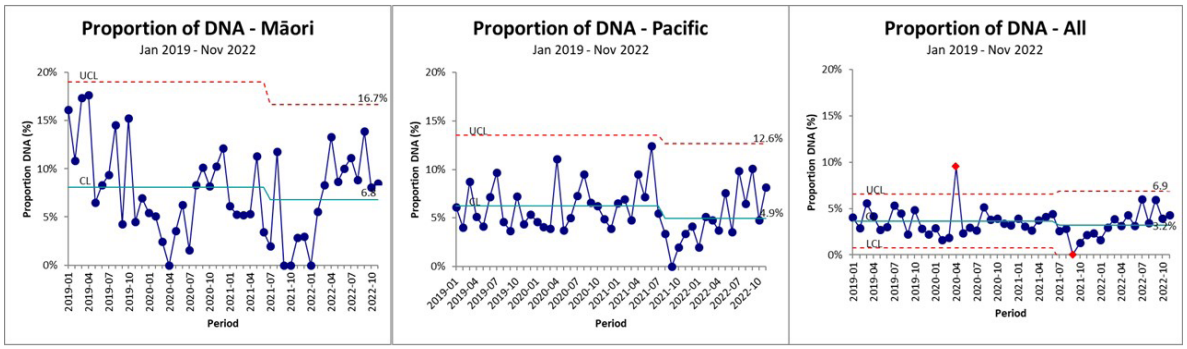


Figure 5: "Did not attend" (DNA) rates, January 2019–November 2022.



July 2021 to June 2022, 531 hours of clinic room utilisation was available for reallocation.

Equity

There was no change to the proportions of surgeries by ethnicities over the course of the initiative, with surgeries for Māori and Asian people in line with the population over 60 years old for these ethnicities in Counties Manukau. Pacific people were over-represented and "Other" under-represented across the project duration (see Figure 4).

There was no change in total, Māori or Pacific DNA rates across appointments over the course of the initiative (see Figure 5).

Discussion

The new CPAC threshold of 46 to be implemented across Health New Zealand – Te Whatu Ora districts will require the adoption of innovative models of care to release capacity needed to increase access and achieve geographical equity in cataract surgery. We have demonstrated that evidence-informed changes to the surgical pathway, accomplished using quality improvement methods, can increase service capacity to deliver more cataract procedures, improve patient access and system efficiency.

Reconfiguring how, when and where cataract care is delivered enables better matching of capacity and demand while reducing the burden of low-value appointments. Traditionally, our appointment scheduling has placed significant disruptions on the work and caring responsibilities of patients and whānau and disregarded the costs of getting to clinics.[15] In our new pathway, the combined FSA/PAC and telephone FU by nurses, optometrists or technicians means most patients avoid such costs and disruptions, and we are able to redirect SMO resources to other tasks. This aligns with benefits seen overseas from streamlining the cataract pathway, whereby clinical capacity is shifted to more complex care and, importantly, that patients favour the convenience of reduced appointments.[12,16]

Further reduction in appointments may be possible by reconsidering FU models of care. By international comparison, we acted conservatively in introducing a 1-day post-operative telephone review. Literature recommends a low threshold for FU for patients that experience intra-operative complications or have comorbid eye conditions;[9] however, there is no consensus on routine FU timing for most patients—some jurisdictions have eliminated routine appointments,[10,11] offered patient-initiated review[11] or transferred FU to community-based optometrists.[16] Experience in England suggests if private or community providers are used in shared care pathways, effective and equitable implementation requires consideration of funding structures, training and accreditation, and data sharing for quality and outcome monitoring.[8,16,17]

In our study, monitoring equity for Māori and Pacific patients by proportions of completed surgeries suggests these were broadly reflective of the Counties Manukau population, although we do not have a clear understanding of unmet need. Research indicates that, on average, Māori and Pacific people present with cataracts at an earlier age than European ethnic groups (69.5, 68.0 and 77 years of age respectively), with significantly worse visual acuity, and that Māori may have higher rates of intra-operative complications.[3,18] Several studies highlight that there are greater barriers to timely referral and prioritisation for Māori and Pacific cataract patients.[18–20]

Pacific peoples were over-represented in our surgical numbers due to the high proportion of referrals from ophthalmologic sub-specialties for patients with diabetes, many of whom are Pacific. The proportionately low numbers of community optometrists in Counties Manukau limits access to the cataract pathway outside of secondary care. However (out of scope for our project), quality improvements at the primary/secondary interface could support timelier access.

Our changes neither improved nor reduced inequities in DNA rates for Māori and Pacific patients. There is an absence of qualitative literature with New Zealand cataract patients to understand their experience with care delivery. Overseas, it has been identified that patients can feel a lack of reassurance, emotional support and information throughout their cataract journey, which can be reflected in appointment outcomes.[21] A Kaupapa Māori study investigating Māori community perspectives on eye health more generally identified that the quality of clinician–patient communication, impact of historical health experiences, consideration of the holistic view of health and the cultural safety of providers may influence access and engagement in eye healthcare.[22] While our team created a patient information leaflet, any future refinement to the pathway would benefit from co-design, particularly with Māori and Pacific patients, to ensure information, psychosocial and cultural needs are met. This is critical for equity of access and our ability to generate further capacity in the pathway.

A unique feature of capacity management in our change package was rethinking our use of private providers through wet leasing. Our ability to reduce reliance on outsourcing to increase access to surgery led to direct cost savings for the public health system. Additionally, the wet leasing option created a virtuous cycle for workforce capacity; the service was able to recruit another SMO because it was able to offer a greater proportion of surgical work within the public system, which is often a reason private practice is attractive in ophthalmology. This is an excellent achievement within the constraints of our current system; however, there is potentially much more that could be done to improve surgical efficiency and sustainability. Creating high throughput cataract units, sourcing less expensive equipment at an agreed national standard and, where clinically appropriate, same-day bilateral cataract surgery have been identified as evidence-based strategies to increase surgeries in New Zealand.[1,16]

Although we successfully met our aim to increase surgeries and maintain or improve FSA to surgery time for most of the project, the COVID-19 pandemic had a disruptive influence. Our change package took over 12 months to fully implement, and our results were impacted by COVID-19 in several ways, including surgery cancellations, reduced theatre session utilisation and FSA demand increases at the end of the project. These shifts in the external context emphasise the importance of using quality improvement tools to continually identify and implement strategies that support patient flow. By gaining widespread clinical agreement and front-line collaboration in design and implementation, our approach encouraged the development of a continuous improvement culture, seen in ongoing efforts to reduce wait times between activities in the FSA/PAC post-project. This also enabled a further reduction of the CPAC score to 48 and has instilled staff confidence in our ability to transition to the new national threshold.

There are some limitations with this study. As a quality improvement initiative focussed on iterative learning in practice, and responsiveness to contextual changes, it is not possible to definitively attribute specific change ideas within the change package to improvements made, as might be the case with a randomised control trial. We did not apply a single improvement methodology, so it is also difficult to identify with certainty the specific methods that had the biggest impact. While this means results may not be generalisable to other settings, our key learning regarding the importance of thoroughly interrogating capacity and demand assumptions, and planning for implementation success through collaborative engagement, will be highly relevant and replicable in other districts in New Zealand.

Conclusion

This quality improvement report describes the approach taken at Counties Manukau to increase access to cataract surgery over 2020–2022. Faced with significant demand pressures and the disruptions of the COVID-19 pandemic we were able to reduce the CPAC score by targeting areas of the surgical pathway to better utilise staff capacity and maximise value for patients. Future work should focus on optimising the end-to-end pathway in the community to further improve equity of access.

COMPETING INTERESTS
Nil.

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Consequences of cost barriers to prescriptions: cohort study in Aotearoa New Zealand

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ABSTRACT

AIMS: A NZ$5 co-payment prescription charge was removed in July 2023 but may be reinstated. Here we quantify the health impact and cost of not being able to afford this charge.

METHODS: We linked New Zealand Health Surveys (2013/2014–2018/2019) to hospitalisation data using data available in Integrated Data Infrastructure (IDI). Cox proportional-hazards models compared time to hospitalisation between those who had faced a cost barrier to collecting a prescription and those who had not.

RESULTS: Of the 81,626 total survey respondents, 72,243 were available for analysis in IDI. A further 516 were excluded to give an analysis dataset of 71,502. Of these, 5,889 (8.2%) reported not collecting a prescription due to cost in the previous year. Among people who faced a cost barrier, 60.0% (95% confidence interval [CI] 58.7–61.2%) were admitted to hospital during the study period, compared to 43.9% (95% CI 43.6–44.3%) of those who did not. Having adjusted for socio-demographic variables, people who faced a cost barrier were 34% (hazard ratio 1.34; 95% CI 1.29–1.39) more likely to be admitted to hospital than those who did not. Annual avoidable hospitalisation costs—were prescription co-payments to remain free—are estimated at $32.4 million per year based on the assumption of a causal relationship between unmet need for prescription medicines and subsequent hospitalisation.

CONCLUSIONS: The revenue to the health system from co-payments may be offset by the costs associated with avoidable hospitalisations.

KEY MESSAGES:

- Facing a cost barrier to collecting a prescription is associated with a 34% higher rate of hospitalisations.
- Hospitalisations that are potentially avoidable are estimated to cost about $32.4 million per year.
- Reinstating prescription co-payments may have detrimental effects on health, health equity and health system costs.

Universal health coverage (UHC) is defined by the World Health Organization as all people having access to quality health services as needed, without financial hardship.[1] Although UHC is often described in terms of accessing health professionals, being able to afford prescribed medications is an essential component of care, as stated explicitly in Sustainable Development Goal 3.8.[2]

In Aotearoa New Zealand (Aotearoa), the Pharmaceutical Management Agency (Pharmac) subsidises many medications, and until recently (1 July 2023) people aged over 14 years paid a NZ$5 charge per item dispensed from a community pharmacy. There were no exceptions based on income or (in)ability to pay, other than an annual household cap of $100. The co-payment presented a financial barrier to healthcare; the proportion of adults who reported being unable to collect a prescription due to cost in 2022/2023 was 4%, but there are significantly higher rates for some population groups (see below).[3]

The cost of outpatient medicines is a source of financial hardship in many European countries, particularly among the poorest people.[4] International evidence suggests that user co-payments for medications undermine health equity.[5] In Aotearoa, Māori are over twice as likely to face a cost barrier to collecting a prescription than non-Māori.[3] These inequities are more evident among poorer people; 26% of Māori in low-income households reported not being able to pay a prescription charge at least once in the previous year, compared to 9% of non-Māori.[6] A study using the Survey of Family, Income and Employment found that people who could not afford to collect a prescription had poorer self-reported physical and mental health,[7] and subsequent declines in health.[8]

The few studies that have directly investigated the effect of co-payments on health outcomes have found that small changes in co-payments

can directly affect health. A study in Italy found that abolishing a €1.50 co-payment improved patient anti-hypertensive compliance; this was in turn associated with a reduced risk of hospitalisation and mortality.[9] A study comparing adherence to asthma medications in England (where patients pay charges) with Scotland (where no charges are payable) found that co-payments were associated with twice the risk of severe asthma exacerbations.[10]

In summary, the prescription co-payment was dropped in July 2023, with plans by the new Government to reinstate this, although it will remain free for some population groups. The objective of this study was to establish whether facing a cost barrier to obtaining a prescription medicine in Aotearoa was associated with time to an inpatient hospitalisation, so as to inform policy regarding the re-instating of prescription charges.

Methods

The study cohort comprised respondents to the New Zealand Health Survey (NZHS) linked to hospitalisation and mortality databases using a Ministry of Health unique identifier. These databases were linked and analysed within the Integrated Data Infrastructure (IDI),[11] and were accessed in the secure environment of the Datalab at Statistics New Zealand. All output is checked by Statistics New Zealand specialists to make sure it has been suitably confidentialised before being released.

The NZHS is an annual, cross-sectional, face-to-face national survey that samples people aged 15+ from across Aotearoa. Respondents were included in the IDI if they agreed that their data could be used for further research and they could be matched to a National Health Index number based on their name, date of birth and address. On average, 400 respondents per survey requested that their responses not be linked and 1,105 respondents per survey could not be matched (in email, Ministry of Health, 2023). The IDI contains data from surveys that were run between 2011/2012 and 2018/2019, but the data analysed for this report are from 2013/2014 to 2018/2019 to match the constraints of the hospitalisation database.

From July 2013, the hospitalisation database contains data on all inpatient discharges resulting from any treatment of over 3 hours from public hospitals in New Zealand, including events that occur in the emergency department or start as outpatient appointments, and it records information on the start and end dates of the hospital stay. At the time of analysis, the database included hospital events up until 30 June 2021. The mortality dataset contains records of all deaths, and at the time of analysis this was complete to the end of 2018.

The main exposure variable was a self-report of facing a barrier to obtaining a prescription due to cost. Respondents to the NZHS were asked if during the previous year they had been given a prescription but did not collect one or more items because of cost. Respondents were classified as "unmet need" or "no unmet need" depending on whether they answered "yes" or "no" to this question. Forty-five people who answered "don't know" or refused to answer were analysed with the "no unmet need" group. Outcome data were: whether or not a hospitalisation occurred during the study period; a count of the number of hospitalisations during the study period; length of (first) hospital stay; and the time in days from the start of the study period to the first hospitalisation for those hospitalised.

Potential confounding variables included were **gender** (male or female); **age group** (10-year age bands from 15–24 to 75+ years); **self-reported ethnicity**,[12] prioritised into four mutually exclusive groups (Māori, Pacific peoples, Asian and European New Zealanders/Others); **area-level deprivation** (New Zealand Index of Deprivation [NZDep]. For surveys in 2013/2014 and 2014/2015 we used NZDep06, and for the later surveys NZDep13); **self-reported health**, measured on a five-point scale from excellent to very poor; 87 respondents (0.1%) who did not answer this question were assigned to the most commonly reported category, "very good"; **education**, based on highest completed qualification—missing data for 762 (1.1%) respondents were completed based on answers from a related question on highest secondary school qualification, or otherwise analysed in the largest category, post-secondary education; **household income**, reported in 16 categories in early surveys and eight in the later survey, with the latter used here. There was a large amount (n=12,270, 17%) of missing data in this question. Respondents who did not report household income, but did report personal income, had their household income imputed. This was done by filling the missing category with the most commonly reported household income category for each category of personal income. Those with no household or personal income reported (n=6,477, 9%) were analysed in a separate category.

Statistical analysis

Statistics New Zealand require that the data output is confidentialised. For the results presented here this means that: counts are rounded to the nearest multiple of 3 with probability 2/3 or the next closest with probability 1/3 (counts are checked so the rounding is consistent across outputs); and percentages and means are calculated using the randomly rounded base 3 counts. These methods were used to produce statistics on the demographic and health profile of the respondents and their hospitalisation characteristics. Given the large size of the cohort, focussing on statistical significance can be misleading; focussing on the magnitude of the differences is more important. We define conventional levels of statistical significance as $p<0.05$.

Kaplan–Meier survival curves that graphically represent the number of people who have not been admitted to hospital against follow-up time were inspected visually to assess potential violations of the proportional-hazards assumption. The time to hospitalisation was modelled using Cox proportional-hazards regression. Follow-up started at the date of the end of the survey a respondent was in and lasted until either the respondent was hospitalised, was known to have died or 30 June 2021, whichever came first. Mortality data are not available after 1 January 2019, which led to some respondents being censored at the end of the study period rather than at their unknown date of death. There were 156 recorded deaths without a hospitalisation between the start of the study period and 31 December 2018. From observing when deaths fell, it was estimated that 147 deaths without a hospitalisation (0.2%) would have occurred between 1 January 2019 and 30 June 2021.

Results

In total there were 72,243 respondents available for analysis in the IDI dataset across the six surveys. Some respondents took part in more than one survey; for these people, one observation was chosen at random to be kept (516, 0.7%, observations deleted). A further 225 people died before the study period started (0.3% deleted). Thus, 71,502 respondents were included in the analysis, ranging from 10,932 in the 2014/2015 survey (out of 13,497 respondents in the total survey, 81%) to 12,579 in the 2018/2019 survey (out of 13,572, 93%). Of these, 5,889 (8.2%) reported not being able to collect a prescription due to cost in the previous year. The average follow-up time for those with unmet need was 730 days and those with met need was 1,095 days.

The demographic and health profile of the two groups is shown in Table 1. Those who faced a cost barrier were more likely to be female and be younger. Māori and Pacific peoples were more likely to face these barriers than Asian or NZ European/Other ethnicities. Those in the unmet need group were more likely than those in the no unmet need group to live in the most deprived quintile of NZDep and report low incomes and lower education levels. Respondents in the unmet need group were more likely to be in the lowest two categories of self-rated health than the no unmet need group (33% vs 12%). They were more likely to have been told by a doctor that they have a chronic illnesses, with the greatest differences evident for depression, asthma, an anxiety disorder and diabetes.

Table 2 shows hospitalisation characteristics according to unmet prescription need. Overall, 60% of people who faced a cost barrier to obtaining a prescription had a hospitalisation during the study period compared to 44% of the no unmet need group. The same pattern was seen for both males and females, all age groups except for the oldest category (75+, $p=0.29$), all ethnic groups, all deprivation, education and income groups and all self-rated health groups, other than among those who reported very poor health, who had similar levels of hospitalisations (69% vs 67%, $p=0.38$).

Among those who had a hospitalisation during the study period, those in the unmet need group had an average of 3.8 hospitalisations compared to 3.1 in the no unmet need group. Across all socio-demographic categories, the unmet need group had more hospitalisations than the no unmet need group. An exception to this was among Asian peoples, where the difference did not meet conventional levels of statistical significance ($p=0.15$), and among people living in the most deprived areas or with the highest education levels, where the difference was small in magnitude. Although the unmet need group had more hospitalisations than the no unmet need group across all levels of self-rated health, the only group that reached conventional levels of statistical significance ($p<0.05$) was those with good health.

The mean length of the first hospital stay during the study period was 2.7 days for the unmet need group compared to 3.2 days for the no unmet need group. When stratified by socio-demographic categories, in most groups the stay was shorter for

Table 1: Demographic and health profile of 71,502 people in Aotearoa, according to unmet need in paying for prescriptions.

	Unmet need for prescriptions due to cost			
	Unmet need		No unmet need	
	N=5,889		N=65,613	
	%	95% CI	%	95% CI
Sex				
Female	71.5	(70.3–72.6)	56.0	(55.6–56.4)
Male	28.5	(27.4–29.7)	44.0	(43.6–44.4)
Age group				
15–24	12.3	(11.4–13.1)	11.1	(10.8–11.3)
25–34	22.1	(21.0–23.1)	15.1	(14.9–15.4)
35–44	19.5	(18.4–20.5)	16.2	(15.9–16.5)
45–54	19.7	(18.6–20.7)	15.8	(15.6–16.1)
55–64	15.6	(14.7–16.6)	16.2	(15.9–16.5)
65–74	7.5	(6.9–8.2)	14.4	(14.1–14.6)
75+	3.4	(2.9–3.8)	11.1	(10.9–11.4)
Prioritised ethnicity				
Māori	39.1	(37.9–40.4)	19.0	(18.7–19.3)
Pacific people	11.9	(11.1–12.7)	4.6	(4.5–4.8)
Asian	4.5	(4.0–5.0)	8.4	(8.2–8.6)
NZ European/Other	44.5	(43.2–45.7)	67.9	(67.6–68.3)
NZDep quintiles				
1 (least deprived)	5.0	(4.4–5.5)	14.6	(14.3–14.9)
2	8.8	(8.1–9.5)	17.5	(17.2–17.8)
3	15.2	(14.3–16.1)	20.3	(20.0–20.6)
4	22.3	(21.2–23.4)	22.8	(22.5–23.1)
5 (most deprived)	48.7	(47.4–50.0)	24.8	(24.5–25.1)
Highest educational qualification				
None	37.0	(35.8–38.2)	29.9	(29.5–30.2)
Secondary	20.5	(19.4–21.5)	14.7	(14.4–15.0)
Post-secondary	32.5	(31.3–33.7)	34.2	(33.8–34.6)

Table 1 (continued): Demographic and health profile of 71,502 people in Aotearoa, according to unmet need in paying for prescriptions.

Undergraduate	6.3	(5.7–6.9)	11.7	(11.5–12.0)
Postgraduate	3.7	(3.2–4.2)	9.5	(9.3–9.7)
Household income (NZ$)				
Loss or up to 20,000	24.6	(23.5–25.7)	9.3	(9.1–9.5)
20,001–30,000	17.1	(16.1–18.0)	11.5	(11.3–11.8)
30,001–50,000	19.0	(18.0–20.0)	16.3	(16.1–16.6)
50,001–70,000	12.8	(12.0–13.7)	15.4	(15.1–15.6)
70,001–100,000	7.7	(7.1–8.4)	14.6	(14.3–14.9)
100,001+	6.6	(6.0–7.3)	24.1	(23.8–24.4)
Missing	12.1	(11.3–13.0)	8.8	(8.6–9.0)
Self-rated health				
Excellent	5.8	(5.2–6.4)	13.9	(13.6–14.1)
Very good	22.3	(21.2–23.3)	40.5	(40.1–40.9)
Good	38.9	(37.6–40.1)	33.6	(33.2–34.0)
Poor	22.4	(21.3–23.5)	9.8	(9.5–10.0)
Very poor	10.6	(9.9–11.4)	2.3	(2.1–2.4)
Have you been told by a doctor that you have				
had a heart attack	5.2	(4.6–5.8)	4.1	(4.0–4.3)
angina	6.4	(5.8–7.0)	3.9	(3.8–4.1)
heart failure	4.4	(3.9–4.9)	2.4	(2.3–2.6)
other heart disease	10.7	(10.0–11.5)	8.5	(8.3–8.7)
had a stroke[a]	3.1	(2.6–3.5)	2.1	(2.0–2.2)
diabetes[b]	12.2	(11.3–13.0)	6.9	(6.7–7.1)
asthma	35.9	(34.6–37.1)	19.7	(19.4–20.0)
arthritis[c]	23.9	(22.9–25.0)	20.9	(20.5–21.2)
depression[d]	39.1	(37.8–40.3)	16.8	(16.5–17.1)
bipolar disorder[d]	4.3	(3.8–4.8)	1.1	(1.0–1.1)
anxiety disorder[d,e]	27.0	(25.9–28.1)	9.9	(9.7–10.2)

Note: a) does not include transient ischaemic attacks; b) does not include diabetes during pregnancy; c) includes gout, lupus and psoriatic arthritis d) conditions that lasted or expected to last more than 6 months; e) includes panic attacks, post-traumatic stress disorder, phobias and obsessive-compulsive disorders.

Table 2: Inpatient hospitalisations and length of hospital stay according to unmet need for paying for prescriptions, by demographic variables.

	Percentage hospitalised during the study period				Number of hospitalisations during the study period*				Length of first hospital stay during the study period*			
	Unmet need		No unmet need		Unmet need		No unmet need		Unmet need		No unmet need	
	n=5,889		n=65,613		n=3,531		n=28,827		n=3,531		n=28,827	
	%	95% CI	%	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
All	60.0	(58.7–61.2)	43.9	(43.6–44.3)	3.8	(3.6–4.0)	3.1	(3.1–3.1)	2.7	(2.6–2.8)	3.2	(3.0–3.4)
Sex												
Female	62.2	(60.8–63.7)	47.0	(46.5–47.5)	3.7	(3.5–3.9)	3.0	(3.0–3.1)	2.6	(2.4–2.7)	3.1	(2.9–3.3)
Male	54.5	(52.1–56.8)	40.0	(39.5–40.6)	4.1	(3.6–4.5)	3.2	(3.1–3.3)	3.1	(2.8–3.4)	3.4	(3.1–3.8)
Age group												
15–24	58.5	(54.9–62.1)	34.7	(33.6–35.8)	3.8	(3.3–4.3)	2.4	(2.3–2.5)	2.2	(2.0–2.3)	2.8	(2.2–3.3)
25–34	59.9	(57.2–62.6)	40.5	(39.5–41.4)	3.4	(3.1–3.7)	2.5	(2.3–2.6)	2.3	(2.1–2.5)	2.5	(2.4–2.7)
35–44	54.7	(51.8–57.6)	31.3	(30.4–32.1)	3.2	(2.9–3.5)	2.3	(2.2–2.4)	2.8	(2.5–3.2)	2.7	(2.4–3.0)
45–54	57.1	(54.3–60.0)	35.2	(34.3–36.1)	3.6	(3.3–4.0)	2.7	(2.6–2.8)	2.7	(2.4–3.0)	2.7	(2.5–2.9)
55–64	60.6	(57.4–63.7)	43.1	(42.2–44.1)	4.5	(3.8–5.1)	3.0	(2.9–3.1)	2.9	(2.6–3.2)	2.9	(2.6–3.2)
65–74	75.2	(71.2–79.2)	56.5	(55.5–57.5)	4.5	(4.0–5.0)	3.6	(3.4–3.7)	3.4	(2.8–4.0)	3.1	(3.0–3.3)
75+	76.9	(71.0–82.8)	73.5	(72.5–74.5)	5.2	(4.3–6.2)	4.3	(4.2–4.4)	3.9	(3.3–4.5)	5.1	(4.0–6.1)
Prioritised ethnicity												
Māori	63.0	(61.0–65.0)	45.9	(45.0–46.7)	3.8	(3.6–4.1)	3.1	(3.0–3.2)	2.6	(2.4–2.8)	3.1	(2.8–3.5)
Pacific people	59.8	(56.2–63.5)	42.6	(40.8–44.3)	4.2	(3.4–5.0)	3.0	(2.8–3.2)	2.9	(2.6–3.2)	3.1	(2.8–3.4)
Asian	40.4	(34.6–46.3)	28.5	(27.3–29.6)	2.8	(2.2–3.4)	2.4	(2.2–2.5)	2.8	(2.1–3.5)	2.6	(2.4–2.8)

Table 2 (continued): Inpatient hospitalisations and length of hospital stay according to unmet need for paying for prescriptions, by demographic variables.

	Percentage hospitalised during the study period				Number of hospitalisations during the study period*				Length of first hospital stay during the study period*			
	Unmet need		No unmet need		Unmet need		No unmet need		Unmet need		No unmet need	
	n=5,889		n=65,613		n=3,531		n=28,827		n=3,531		n=28,827	
	%	95% CI	%	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
NZ European/ Other	59.3	(57.4–61.2)	45.4	(44.9–45.9)	3.8	(3.5–4.0)	3.2	(3.1–3.2)	2.8	(2.6–3.0)	3.3	(3.0–3.6)
NZDep quintiles												
1 (least deprived)	53.1	(47.4–58.8)	36.8	(35.8–37.8)	4.0	(3.2–4.7)	2.7	(2.6–2.8)	2.7	(2.2–3.1)	2.9	(2.7–3.0)
2	58.4	(54.1–62.6)	40.5	(39.6–41.4)	4.0	(3.4–4.5)	3.0	(2.9–3.1)	2.8	(2.4–3.1)	3.1	(2.6–3.6)
3	58.2	(55.0–61.5)	43.8	(43.0–44.7)	3.6	(3.2–4.1)	3.1	(3.0–3.2)	2.5	(2.2–2.8)	2.9	(2.7–3.1)
4	58.5	(55.9–61.2)	46.0	(45.2–46.8)	4.0	(3.6–4.3)	3.1	(3.0–3.2)	2.7	(2.4–3.0)	3.5	(2.8–4.1)
5 (most deprived)	62.2	(60.5–64.0)	48.7	(48.0–49.5)	3.8	(3.5–4.0)	3.4	(3.3–3.5)	2.8	(2.6–2.9)	3.5	(3.1–3.9)
Highest educational qualification												
None	66.4	(64.4–68.4)	53.4	(52.7–54.1)	4.2	(3.8–4.5)	3.6	(3.5–3.7)	2.7	(2.5–2.9)	3.7	(3.2–4.2)
Secondary	55.5	(52.7–58.3)	39.0	(38.0–39.9)	3.3	(3.0–3.6)	2.7	(2.6–2.8)	2.6	(2.3–3.0)	3.1	(2.7–3.5)
Post-secondary	59.9	(57.7–62.1)	44.3	(43.7–45.0)	3.9	(3.6–4.2)	3.0	(2.9–3.1)	2.8	(2.6–3.1)	3.1	(2.8–3.4)
Undergraduate	45.2	(40.1–50.2)	33.9	(32.9–35.0)	3.1	(2.4–3.7)	2.6	(2.4–2.7)	2.6	(2.1–3.1)	2.6	(2.5–2.8)
Postgraduate	46.6	(40.0–53.2)	32.8	(31.7–34.0)	2.8	(2.1–3.6)	2.6	(2.4–2.8)	2.7	(2.2–3.2)	2.5	(2.4–2.7)
Household income ($)												
Loss, 0–20,000	66.0	(63.6–68.5)	57.6	(56.3–58.8)	4.5	(4.0–4.9)	4.0	(3.8–4.2)	3.2	(2.9–3.5)	3.9	(3.4–4.4)

Table 2 (continued): Inpatient hospitalisations and length of hospital stay according to unmet need for paying for prescriptions, by demographic variables.

	Percentage hospitalised during the study period				Number of hospitalisations during the study period*				Length of first hospital stay during the study period*			
	Unmet need		No unmet need		Unmet need		No unmet need		Unmet need		No unmet need	
	n=5,889		n=65,613		n=3,531		n=28,827		n=3,531		n=28,827	
	%	95% CI	%	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI	Mean	95% CI
20,001–30,000	64.5	(61.5–67.4)	59.5	(58.4–60.6)	3.9	(3.5–4.3)	3.8	(3.7–3.9)	2.5	(2.3–2.7)	3.9	(3.2–4.6)
30,001–50,000	59.5	(56.6–62.4)	48.9	(47.9–49.8)	3.2	(2.9–3.4)	3.2	(3.1–3.3)	2.7	(2.4–3.0)	3.2	(2.9–3.5)
50,001–70,000	50.4	(46.8–54.0)	40.9	(39.9–41.8)	3.3	(2.9–3.6)	2.7	(2.6–2.8)	2.4	(2.2–2.7)	2.8	(2.2–3.4)
70,001–100,000	53.3	(48.7–57.9)	36.9	(35.9–37.8)	3.1	(2.6–3.7)	2.5	(2.4–2.6)	2.5	(2.1–2.9)	2.5	(2.4–2.7)
100,001+	46.9	(42.0–51.9)	31.7	(30.9–32.4)	3.1	(2.4–3.7)	2.3	(2.2–2.4)	2.0	(1.8–2.2)	2.6	(2.4–2.7)
Missing	63.9	(60.3–67.4)	50.8	(49.5–52.0)	4.4	(3.9–4.9)	3.4	(3.2–3.5)	2.8	(2.5–3.1)	4.1	(2.8–5.5)
Self-rated health												
Excellent	53.5	(48.2–58.8)	36.7	(35.7–37.7)	3.0	(2.5–3.4)	2.6	(2.5–2.7)	2.6	(2.2–3.0)	2.9	(2.6–3.2)
Very good	53.7	(51.0–56.4)	40.7	(40.1–41.3)	3.0	(2.8–3.3)	2.8	(2.7–2.8)	2.6	(2.3–2.8)	3.1	(2.8–3.4)
Good	60.2	(58.2–62.2)	46.1	(45.5–46.8)	3.8	(3.4–4.1)	3.2	(3.1–3.2)	2.7	(2.5–2.9)	3.1	(2.8–3.5)
Poor	63.0	(60.4–65.6)	54.4	(53.2–55.6)	3.9	(3.7–4.2)	4.0	(3.8–4.1)	2.8	(2.5–3.1)	4.0	(3.1–4.8)
Very poor	69.4	(65.8–73.0)	67.4	(65.0–69.8)	5.4	(4.7–6.0)	4.8	(4.4–5.3)	3.0	(2.6–3.3)	4.7	(3.4–6.1)

*For those people with at least one inpatient hospitalisation.

Table 3: Time to first inpatient hospitalisation according to unmet need for paying for prescriptions, by demographic variables, among 32,358 people who were hospitalised.

	Days till first inpatient hospitalisation*			
	Unmet need		No unmet need	
	n=3,531		n=28,827	
	Mean	95% CI	Mean	95% CI
All	582	(564–599)	650	(644–657)
Sex				
Female	576	(556–596)	643	(635–651)
Male	596	(561–630)	662	(652–672)
Age group				
15–24	579	(531–627)	743	(720–765)
25–34	567	(530–603)	650	(633–666)
35–44	611	(568–654)	693	(674–712)
45–54	617	(575–660)	729	(710–749)
55–64	579	(536–621)	702	(685–719)
65–74	534	(482–587)	624	(609–638)
75+	497	(413–580)	510	(498–523)
Prioritised ethnicity				
Māori	573	(546–601)	660	(645–675)
Pacific people	567	(517–617)	642	(611–672)
Asian	630	(524–737)	671	(644–698)
NZ European/ Other	590	(564–616)	647	(639–654)
NZDep quintiles				
1 (least deprived)	604	(529–680)	687	(669–706)
2	638	(576–700)	683	(667–700)
3	575	(530–620)	674	(659–688)
4	563	(526–599)	625	(612–637)
5 (most deprived)	580	(555–604)	620	(608–632)
Highest educational qualification				
None	613	(585–641)	666	(655–677)
Secondary	554	(515–592)	600	(583–616)
Post-secondary	552	(523–582)	648	(637–659)

Table 3 (continued): Time to first inpatient hospitalisation according to unmet need for paying for prescriptions, by demographic variables, among 32,358 people who were hospitalised.

Undergraduate	616	(536–697)	662	(641–683)
Postgraduate	594	(483–704)	663	(639, –686)
Household income				
Loss, 0–20,000	553	(520–587)	600	(582–618)
20,001–30,000	589	(549–629)	557	(543–572)
30,001–50,000	581	(543–620)	641	(626–656)
50,001–70,000	609	(556–662)	689	(671–706)
70,001–100,000	610	(543–676)	687	(669–706)
100,001+	584	(505–662)	691	(676–706)
Missing	589	(538–641)	703	(681–725)
Self-rated health				
Excellent	713	(632–795)	734	(714–753)
Very good	656	(615–696)	696	(685–706)
Good	593	(565–621)	633	(623–644)
Poor	538	(503–572)	544	(528–561)
Very poor	455	(412–499)	423	(396–450)

*For those people with at least one inpatient hospitalisation

the unmet need group compared to the no unmet need group, but in many instances the difference did not reach conventional levels of statistical significance.

During the study period, 32,358 people were hospitalised. Table 3 shows the time taken to the first hospitalisation. This was 582 days for the unmet need group and 650 days for the no unmet need group. For both males and females, the difference between groups was just over 2 months, with the unmet need group attending earlier. For all ethnicities other than among Asian peoples, the unmet need group were quicker to be hospitalised than the no unmet need groups. Similarly, across NZDep, household income and education groups, the unmet need group were quicker to be hospitalised than the no unmet need group, other than those in quintile 2 of NZDep and the second lowest income category. In the unmet need group, people with good health arrived at hospital 40 days earlier than the no unmet need group; no differences were seen for other categories of self-rated health.

In univariate analyses, people who reported unmet need had a 58% higher risk of hospitalisation during follow-up than those who reported no unmet need (hazard ratio [HR] 1.58, 95% confidence interval [CI] 1.52–1.63). Adjusting for socio-demographic variables and self-rated health did not explain this association, with the HR in the fully adjusted model being 1.34 (95% CI 1.29–1.39).

Based on visual inspection of Kaplan–Meier survival curves, the 25–34-year age group were more likely to be hospitalised earlier and less likely to be hospitalised later relative to other age groups. To see if the failure in the assumption of proportional hazards affected the HR for the unmet need compared to no unmet need group, the fully adjusted model was refitted with this age group removed. Doing so had no material impact

Table 4: Fully adjusted model of time to first hospitalisation.

	Hazard ratio	95% CI	P-value
Need groups			
Unmet need	1.34	(1.29–1.39)	<0.001
Met need	1		
Sex			
Female	1.19	(1.17–1.22)	<0.001
Male	1		
Age group			
15–24	1.08	(1.03–1.13)	0.002
25–34	1.37	(1.32–1.43)	<0.001
35–44	1		
45–54	1.09	(1.05–1.14)	<0.001
55–64	1.40	(1.34–1.46)	<0.001
65–74	2.08	(1.99–2.17)	<0.001
75+	3.27	(3.13–3.43)	<0.001
Prioritised ethnicity			
Māori	1.49	(1.41–1.57)	<0.001
Pacific people	1.43	(1.34–1.54)	<0.001
Asian	1		
NZ European/Other	1.42	(1.35–1.50)	<0.001
NZDep quintiles			
1 (least deprived)	1		
2	1.07	(1.02–1.12)	0.002
3	1.15	(1.10–1.19)	<0.001
4	1.21	(1.17–1.26)	<0.001
5 (most deprived)	1.27	(1.21–1.32)	<0.001
Highest educational qualification			
None	1.16	(1.10–1.22)	<0.001
Secondary	1.10	(1.04–1.16)	<0.001
Post-secondary	1.15	(1.09–1.20)	<0.001

Table 4 (continued): Fully adjusted model of time to first hospitalisation.

Undergraduate	1.03	(0.98–1.09)	0.27
Postgraduate	1		
Household income			
Loss, 0–20,000	1		
20,001–30,000	1.28	(1.23–1.34)	<0.001
30,001–50,000	1.24	(1.19–1.30)	<0.001
50,001–70,000	1.16	(1.11–1.20)	<0.001
70,001–100,000	1.08	(1.04–1.13)	<0.001
100,001+	1.06	(1.02–1.11)	0.004
Missing	1.16	(1.11–1.21)	<0.001
Self-rated health			
Excellent	1		
Very good	1.14	(1.10–1.18)	<0.001
Good	1.41	(1.36–1.47)	<0.001
Poor	1.80	(1.72–1.88)	<0.001
Very poor	2.45	(2.30–2.61)	<0.001

Note: Hazard ratios are adjusted for all other variables in the table.

on the HR, meaning we can be confident that the result is robust to this failure in the proportional-hazards assumption.

Finally, we modelled the estimated cost savings to the health system of removing prescription cost charges. Data from NZHS 2022/2023, applied to the national population, indicate that about 168,000 adults reported not being able to afford a prescription.[3] If the hospitalisation rates for these people with an unmet need were reduced to the levels of those with no unmet need, 27,000 hospitalisations could be avoided over the median follow-up time of 3 years (1,095 days). Given the cost of one night in hospital is estimated at $1,200,[13] and with an average of three nights in one stay, we conservatively estimate that $32.4 million in hospitalisation costs could potentially be saved each year.

Discussion

We have demonstrated higher rates of hospitalisations among people who have previously faced an inability to afford a prescription. This effect was independent of the socio-demographic variables that we measured, and only partly explained by the confounding effect of underlying health status, as measured using self-reported health.

A small randomised controlled trial of the provision of free prescriptions in Aotearoa found similar results to ours; participants who were provided with free medications had a lower rate of hospitalisations (all cause, and for selected conditions), although the primary outcomes of hospital length of stay did not meet conventional levels of statistical significance.[14] This experimental design—albeit on a relatively small sample—coupled with benefits seen in an international

trial[15] and the larger observational data that we present strongly support not re-introducing prescription charges in Aotearoa.

Differential access to healthcare is a key contributor to ethnic inequities in health,[16] which significantly impacts Māori.[17] Analyses of the implementation of the 2001 Primary Health Care Strategy (PHCS)[18] demonstrated that the Strategy is not compliant with the articles of Te Tiriti o Waitangi.[19,20] Re-introduction of prescription charges would have significant impact for Māori, who experience a significant inequity in this indicator of accessing care.[3]

The most significant limitation of our study is the potential for residual confounding by unmeasured or poorly measured confounding variables. In particular, although we included three measures of socio-economic hardship (area-level deprivation, household income and education levels), each of these may be measured with some degree of imprecision and are unlikely to capture all dimensions of individual level socio-economic position. People with lower incomes are more likely to face a cost barrier to obtaining a prescription, and are more likely to be hospitalised for reasons unrelated to this barrier, due to high levels of, for example, smoking and other social determinants of health. Thus, we suggest that the results should be interpreted with a degree of caution.

A further possible limitation of the analysis is the possibility of selection bias arising from the exclusion of those people who could not be matched in the IDI and those who requested their responses not be linked. Approximately 8% of the sample could not be matched and this is more likely to be due to unmatched address information rather than from the other matching variables, i.e., age, sex. Previous research has shown that around 5.5% of the population experiences transience—defined as relocating more than three times within a 3-year period—with 4.3% classified as vulnerable transients, i.e., having had at least one housing incident in a socio-economically deprived area.[21] It seems plausible that the unmatched subset may exhibit a higher likelihood of relocation, including multiple relocations, indicating higher levels of socio-economic deprivation that would put pressure on accessing continuous healthcare and affording prescription medicines. Consequently, the identified differences in hazard rates comparing people with an unmet need and no unmet need in this study are likely to be conservative.

The prevalence of facing a cost barrier to collecting a prescription was lower in the years 2020/2021 (3.1%) to 2022/2023 (4.0%) than previous years (2019/2020, 5%), meaning that our results are based on a higher prevalence of facing this barrier than is currently reported.[3] It is likely that disruptors to the health system due to the COVID-19 pandemic could explain this, e.g., the lower rate of primary healthcare consultations during the lockdowns is likely to have resulted in lower rates of prescribing. It is also not clear whether our results still apply to the smaller proportion of the population that reports facing these barriers, as compared to the higher proportion in our study years. To err on the side of caution, we used the prevalence of unmet need as reported in 2022/2023 in the calculation of potential cost savings.

We did not include a formal economic evaluation as part of the work that we report. However, we estimated significant savings to the health system due to potentially avoided hospitalisations, were prescriptions to be fully funded. This calculation is based on the assumption of causality, which may not be the case. However, the concurrence of our results with those from experimental[14,15] and other observational studies of different designs[22,23] reinforces the likelihood of there being a causal link between lack of access to medication and increased hospitalisations.

Improving access to primary healthcare was a key aim of the PHCS.[18] Many of the measures that were implemented as part of the Strategy related to access to seeing a general practitioner or other member of the primary healthcare team; funding to remove prescription co-payments was not addressed in the Strategy. There are significant health reforms currently underway in Aotearoa, and many community pharmacists are working in expanded roles, such as provision of some medications without a prescription.[24] These moves increase access to the wider primary healthcare team and may be reflected in reduced cost barriers to primary healthcare in the future, although they do not directly address prescription co-payments.

Since prescriptions require a prior GP visit, our results need to be interpreted in the context of co-payments, which are payable to see a GP in Aotearoa, and represent another important cost barrier to UHC.[6] For example, a GP consultation at a Very Low Cost Access practice is currently $19.50, equivalent in cost to a four-item prescription ($20). A further facet of cost barriers to obtaining a prescription is how frequently this occurs, and

how people behave in the face of a barrier. We were not able to analyse the former as the NZHS only asks about the presence or absence of a cost barrier in the previous 12 months. Qualitative work has explored the impacts of these barriers on individuals and their families. Being unable to afford all items on a prescription means that people make decisions regarding which treatments to prioritise, cut back on doses to make a prescription last longer or go without food in order to pay for prescriptions.[25]

Some large pharmacy chains began covering the cost of co-payments for those items that they dispensed prior to the July 2023 removal of all co-payments and are likely to continue this practice if prescription charges are re-introduced. However, there remain problems with this move; these large chains are generally in urban centres and do not allow access for people who may already be struggling with access to care, for example through living rurally. Furthermore, they may not offer the range of extended services that many community pharmacies are offering, thus reducing access to pharmacist-led care. It has been suggested that the presence of these chains could result in the closure of some independent community pharmacies,[26] further reducing access to care for some people, and potentially increasing health inequities.

A review of 24 European countries regarding the use of user co-payments for healthcare recommended that an annual cap on co-payments be used.[4] Such a cap was applied at the family level in Aotearoa for prescription co-payments prior to their removal in July 2023. This applied to an individual, their partner and dependent children aged 14–18 years (as no charges were payable for younger children). However, this policy can fail when different family members, or the same member on different occasions, obtain(s) their prescriptions from different pharmacists. In addition, the $100 household cap was not widely known about,[25] meaning that not everyone benefitted from this in the absence of improved systems. Improved IT systems across pharmacies in Aotearoa could improve access to the annual household cap on payments, were this to be re-introduced.

In summary, our analysis provides evidence of the potential risks of reinstating prescription charges in Aotearoa, as this may have detrimental effects on health, health equity and health system costs. The revenue to the health system from co-payments may be offset by the costs associated with avoidable hospitalisations. Given the current health reforms in Aotearoa, with the increased focus on community health, accessing primary healthcare and addressing inequities, now is the time for the zero fees policy to be retained.

COMPETING INTERESTS
Nil.

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STATEMENT BY STATISTICS NEW ZEALAND
Access to the data used in this study was provided by Stats NZ under conditions designed to give effect to the security and confidentiality provisions of the *Statistics Act 1975*. The results presented in this study are the work of the author, not Stats NZ or individual data suppliers. These results are not official statistics. They have been created for research purposes from the Integrated Data Infrastructure (IDI), which is carefully managed by Stats NZ. For more information about the IDI please visit https://www.stats.govt.nz/integrated-data/.

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Guideline versus clinician recommended duration of dual anti-platelet therapy following acute coronary syndrome (ANZACS-QI 78)

Sophie J Rees, Andrew J Kerr

ABSTRACT

AIM: The recommended duration of dual anti-platelet therapy (DAPT) following acute coronary syndrome (ACS) for patients without atrial fibrillation varies from 1 month to 1 year depending on the balance of risks of ischaemia and major bleeding. Patients on DAPT with a high risk of gastrointestinal bleeding are also recommended to receive a proton pump inhibitor (PPI). Our aim was to audit current practice against the 2020 European Society of Cardiology (ESC) guideline recommendations.

METHODS: One hundred consecutive ACS patients treated with percutaneous coronary intervention discharged from Middlemore Hospital and without atrial fibrillation in the first quarter of 2023 were studied. ANZACS-QI ischaemic (I) and bleeding (B) risk scores were calculated, with patients categorised in four groups based on ESC recommendations—low I/low B risk, low I/high B, high I/low B and high I/high B. Guideline and clinician recommended duration of DAPT and prescription of PPI were compared.

RESULTS: All patients were planned for DAPT at discharge and 91% a PPI. Up to four out of five ACS patients could have been planned for shorter DAPT durations based on the ESC guideline recommendations. Over half of included patients (53%) had a high bleeding risk, yet 85% of these patients received 12 months of DAPT despite ESC recommendations of 1–3 months.

CONCLUSIONS: There was a divergence between clinical practice and the recommendations of the 2020 ESC guidelines. We discuss these results in relation to the updated August 2023 ESC guidelines, which have reaffirmed a 12-month duration of DAPT as the default position.

The recommended duration of dual anti-platelet therapy (DAPT—aspirin and a $P2Y_{12}$ inhibitor) following acute coronary syndrome (ACS) for patients without atrial fibrillation varies from 1 month to 1 year depending on the balance of risks of ischaemia and major bleeding. In addition, patients on DAPT who have a high risk of gastrointestinal bleeding are recommended to receive a proton pump inhibitor (PPI).[1]

Over the last 10 years, national and international guidelines have progressively revised recommendations regarding DAPT duration according to estimated ischaemic and bleeding risk. The 2012 European Society of Cardiology (ESC) guidelines recommended 12 months of DAPT unless there was an excessive risk of bleeding.[2] The New Zealand guidelines at the time had a similar 12 months of DAPT default recommendation.[3] By 2018 the ESC, in response to new clinical trial data, recommended reducing the DAPT duration to 6 months in those with high bleeding risk, defined by a Precise-DAPT score ≥25, which is equivalent to a 1-year risk of major bleeding of more than 2%.[4] Following this, the 2020 ESC guidelines for the management of ACS in patients presenting without persistent ST-segment elevation, in force at the time of this study, took an even more nuanced approach.[5] Patients with high bleeding risk were recommended to have up to 3 months of DAPT regardless of ischaemic risk. Those with low ischaemic risk were also recommended to have only 3 months of DAPT, whereas those with high ischaemic but low bleeding risk were recommended 12 months of DAPT. For some patients with high bleeding risk, the use of clopidogrel, a less potent $P2Y_{12}$ inhibitor, may be preferred over the more potent ticagrelor as the second anti-platelet agent. However, these recommendations have been difficult to implement in practice because there have been no risk scores available that accurately estimate bleeding and recurrent ischaemic risks over the relevant 28-day to 1-year post-ACS period. The recently published Aotearoa New Zealand All Cardiology Services Quality Improvement (ANZACS-QI) ischaemic and bleeding risk scores were specifically designed for

this purpose.[6] There is now an opportunity to audit current practice against the 2020 ESC guideline recommendations using these risk scores and to identify opportunities for improvement.

Methods

New Zealand patients with ACS investigated with coronary angiography are routinely recorded in the ANZACS-QI registry. Consecutive ACS patients (n=100) treated with percutaneous coronary intervention (PCI) discharged from Middlemore Hospital were selected from the ANZACS-QI registry from 1 January 2023 to 1 May 2023. Patients with atrial fibrillation were excluded, as the risk scores were developed for those without atrial fibrillation. For each patient, the electronic clinical notes were reviewed to confirm the ACS diagnosis and PCI procedure. The clinician-recommended DAPT duration at the time of hospital discharge was taken from the electronic clinical record. The ANZACS-QI 28-day to 1-year ischaemic (I) and bleeding (B) risk scores were calculated for each patient using the published algorithms using the variables shown in Table 1.[6] Patients were initially categorised in four groups based on ESC-recommended risk cut-points (≤2% vs >2%)—low I/low B risk, low I/high B, high I/low B and high I/high B.[5] The guideline recommendations are the same for patients with high bleeding risk irrespective of the ischaemic risk, so for reporting purposes the low I/high B and high I/high B groups were combined. Guideline recommended versus clinician recommended DAPT duration for each of the three groups was compared. The prescription of proton pump inhibitors (PPI), another guideline recommended medication, was also recorded. This audit has received Counties Manukau locality approval (application #1803).

Results

Of the 100 patients included, the mean age at index presentation was 63.4 years (SD 12.3) and 73% were male (Table 1). Thirty-nine percent were European, 7% New Zealand Māori and 25% Pacific peoples. The mean New Zealand Index of Deprivation (NZDep) quintile was 3.5 (SD 1.5).

Table 1: Baseline demographics, clinical features and relevant investigations.

Demographic	**Frequency (n=100)**
Age (SD), year	63.4 (12.3)
Male	73
Ethnicity	
European	39
New Zealand Māori	7
Pacific peoples	25
Indian	15
Chinese/Other Asian	14
New Zealand Index of Deprivation (SD), (quintile)	3.5 (1.5)
Quintile 1	16
Quintile 2	18
Quintile 3	15
Quintile 4	17
Quintile 5	34

Table 1 (continued): Baseline demographics, clinical features and relevant investigations.

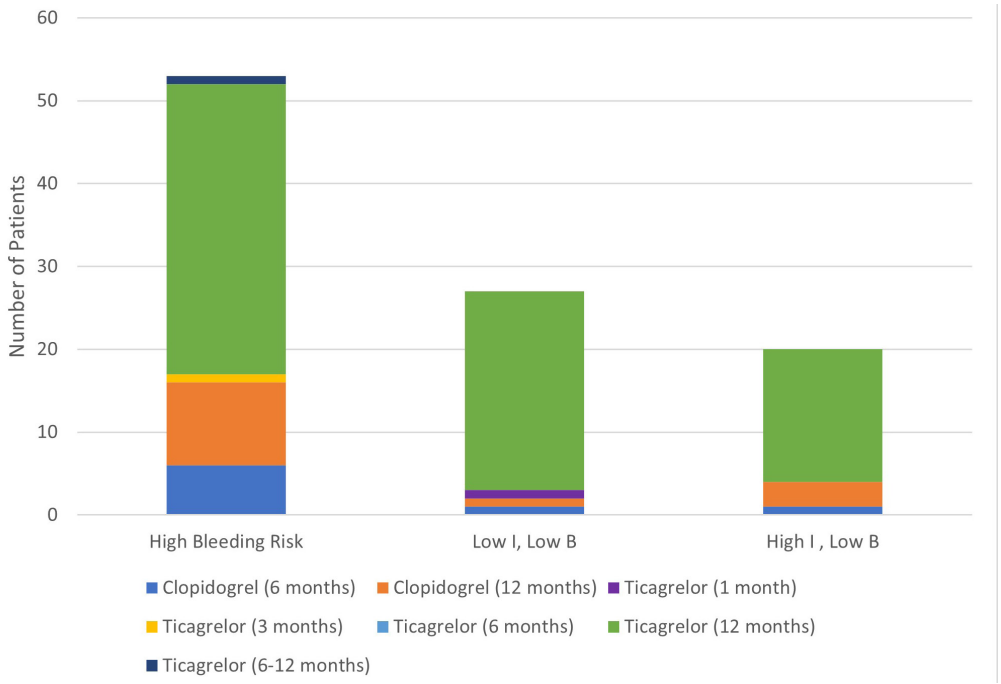
Heart rate (bpm)	91 (15)
Estimated GFR[1] (SD), (mL/min/1.73m2)	69.4 (24.4)
Haemoglobin level (SD), units	157 (15.3)
Low Hb[2]	17
Coronary artery disease severity	
Single vessel disease	74
Double vessel disease	23
Triple vessel disease or LMS	3
History of CVD[3]	
No prior CVD	73
Prior MI	26
Other prior CVD	1
Diabetes mellitus	33
With insulin	13
Current smoker	18
Type of ACS[4]	
NSTEMI[5]	57
STEMI[6]	29
Unstable angina	14
Worst Killip class in hospital	1 (0.5)
I	92
II–IV	8
Left ventricular ejection fraction	
Normal (≥50%)	61
Mid-range (40–49%)	18
Reduced (<40%)	17
Prior hospitalisation for bleeding	2
Index admission bleeding	1
Total: HDL cholesterol ratio	4.5 (1.8)

[1]Glomerular filtration rate
[2]Low haemoglobin: Hb <115g/L for women, <130g/L for men
[3]Cardiovascular disease
[4]Acute Coronary Syndrome
[5]Non-ST Elevation Myocardial Infarction
[6]ST Elevation Myocardial Infarction

Table 2: Duration of planned dual anti-platelet therapy by risk group.

Second anti-platelet	Duration (months)	Low I, low B	Low I, high B	High I, low B	High I, high B
Clopidogrel	6	1	3	1	3
	12	1	0	3	10
Ticagrelor	1	1	0	0	0
	3	0	0	0	1
	6	0	0	0	0
	6–12	0	0	0	1
	12	24	6	16	29
Total		27	9	20	44

Figure 1: Clinician decision on duration and type of dual anti-platelet therapy by risk group.



All patients were planned for DAPT at discharge and 91% a PPI. All patients received aspirin. For the second anti-platelet agent, 78 were planned for ticagrelor and 22 clopidogrel (Table 2). The majority of patients (89%) received a recommendation for 12 months of DAPT.

High ischaemic/low bleeding risk (20% of patients): The 2020 ESC guidelines recommend 12 months of DAPT. Of the 20 patients in this category, 19 were consistent with the guidelines and were planned for 12 months of DAPT (Table 1). Sixteen of these patients were planned for ticagrelor and three for clopidogrel alongside aspirin. The one patient who did not receive 12 months of DAPT was planned for 6 months of aspirin and clopidogrel.

Low ischaemic/low bleeding risk (27% of patients): The 2020 ESC guidelines recommend 3 months of DAPT. Of the 27 patients in this category, 24 (89%) were planned for 12 months of DAPT

Figure 2: Percentage of patients planned for guideline recommended duration of dual anti-platelet therapy according to ischaemic and bleeding risk categories.

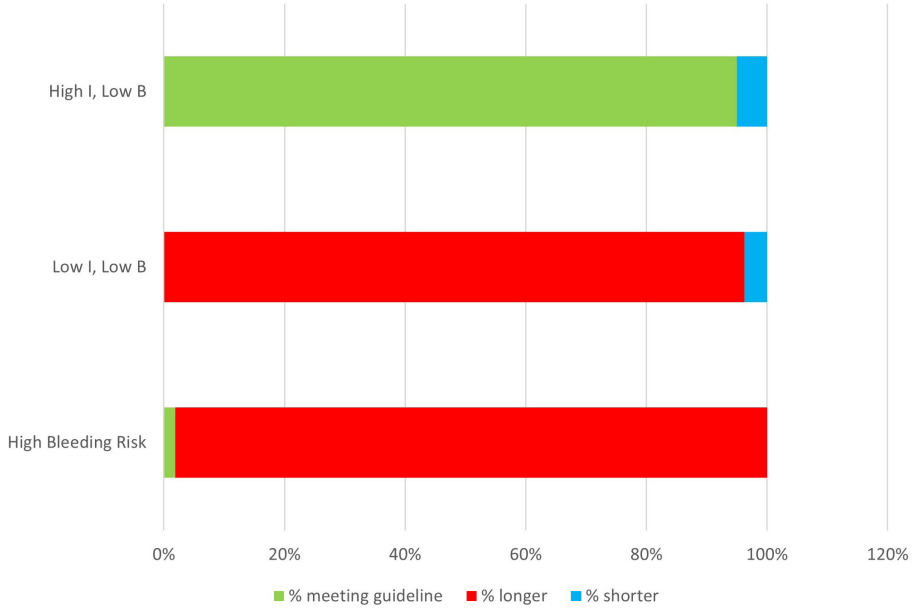
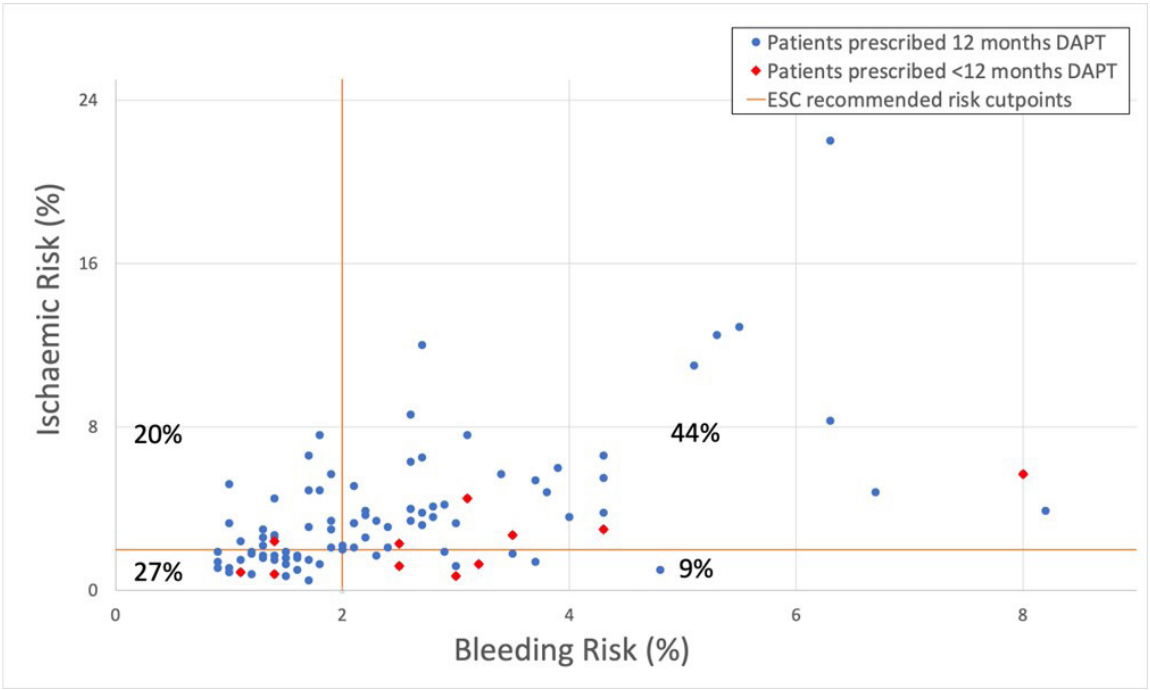


Figure 3: Bleeding versus ischaemic risk.



with aspirin and ticagrelor. The three other patients were planned for DAPT with clopidogrel for 6 months, clopidogrel for 12 months and ticagrelor for 1 month, respectively.

High bleeding risk (53% of patients): The 2020 ESC recommendation for high bleeding risk is 1–3 months of DAPT. Fifty-three patients had a high bleeding risk. Of these, 44 (83%) had a high ischaemic risk. Forty-five patients (85%) were planned for DAPT for 12 months—35 with ticagrelor (78%) and 10 with clopidogrel (22%). Alongside aspirin, one patient was planned for ticagrelor for 3 months, six for clopidogrel for 6 months and one for ticagrelor for 6–12 months. Fourteen patients had a bleeding risk greater than 4% and 12 of these patients received a recommendation for 12 months of DAPT.

Ninety-one of the 100 patients received a concurrent PPI (Table 3). Of the 53 with a high bleeding risk, 48 (91%) received a PPI. Of the 48, 31 (65%) were planned for omeprazole, 16 (33%) pantoprazole and one patient received lansoprazole.

Discussion

At discharge, post-ACS patients were appropriately planned for both DAPT and PPIs. There was, however, a divergence between clinical practice and the recommendations of the 2020 ESC guidelines that prevailed during the time course of this study regarding the duration of DAPT. Clinicians appear to have been adhering more to the older guidelines,[2,4] which recommended 12 months of DAPT as the default position. Since this study was performed, the ESC, after further consideration of the evidence, have modified their recommendations in the 2023 guidelines for the management of ACS.[1] Twelve months of DAPT is again recommended as the default approach, although alternative approaches of reducing DAPT duration or de-escalation of therapy intensity can be considered, particularly with the aim of reducing bleeding events in high bleeding risk patients.

In our real-world cohort, over 50% of patients were at high bleeding risk, for which the 2020 ESC guideline recommended ≤3 months of DAPT, and the current 2023 guideline suggests a reduced duration can be considered. Although 85% of the high bleeding risk patients in this study were planned for 12 months of DAPT, there are indications that clinicians are modifying DAPT therapy in response to bleeding risk. In particular, a higher proportion of high bleeding risk patients were planned for clopidogrel than those at lower risk. There were also more high bleeding risk patients planned for a reduced, 6-month course of DAPT.

A meta-analysis of coronary stenting trials assessing short versus longer duration DAPT found that ischaemic events were reduced by longer DAPT for patients at low bleeding risk, but in those at high bleeding risk, defined using the Precise-DAPT score, longer DAPT duration was associated with similar ischaemic event rates but higher bleeding rates.[7] In the subgroup with acute

Table 3: Choice of proton pump inhibitor.

	Choice of PPI	Number of patients
Clopidogrel	Lansoprazole	1
	Omeprazole	3
	Pantoprazole	15
	No PPI	3
	Total	22
Ticagrelor	Lansoprazole	0
	Omeprazole	60
	Pantoprazole	12
	No PPI	6
	Total	78

coronary syndromes they reported a similar result, albeit with relatively small numbers of events. Two subsequent clinical trials in patients at high bleeding risk treated with third generation stents have reported similar findings.[8,9] Despite these studies supporting a shortened period of DAPT, concern has been expressed that the clinical trials for reducing DAPT intensity have excluded the highest risk ACS patients, and that the trials were non-inferiority trials and were therefore not powered to detect differences in ischaemic outcomes.[1]

Most patients in the high ischaemic, low bleeding risk group (95%) received treatment consistent with the guidelines (Figure 2). However, no patients in the low ischaemic, low bleeding risk group were planned for a shorter course of DAPT. Although the 2020 ESC guideline recommended a shorter duration of DAPT in this low ischaemic/low bleeding risk group, the updated guideline does not make this recommendation. We are unaware of specific clinical trial data to guide clinicians for these patients. The availability of the ANZACS-QI risk scores would theoretically make it possible to investigate the benefits of 12-month versus 3-month DAPT in this sub-group, but the low event rates in these patients may make this challenging to do.

There are likely to be a number of reasons for the divergence between clinical practice and guideline recommendations. The ESC guidelines do not provide a clear risk stratification implementation process. In particular, they do not recommend a specific ischaemic risk score to guide the decision regarding DAPT duration, and while the Precise-DAPT score is discussed as a bleeding score developed to guide DAPT duration decision, its use is not strongly endorsed. This leaves clinicians uncertain regarding how to implement the guideline in practice. During the period when these patients were admitted, clinicians did not routinely use multivariable risk scores to assess bleeding or ischaemic risk. Translation of the guideline into clinical practice requires relevant, readily accessible and easy to calculate risk scores. A further reason is likely to be that there is no randomised clinical trial evidence that applying a risk stratification guided DAPT duration decision making improves outcomes. In the absence of accessible multivariable risk stratification tools and clinical trial guidance, clinicians are more likely to follow a one-size-fits-all approach for all but those with very obvious single risk factors for bleeding, such as the very elderly and those with chronic renal disease. There may also be a time lag for clinical practice to catch up with changes in guideline recommendations. Cardiology clinicians may give greater weight to ischaemic complications than bleeding complications and perceive using a longer DAPT duration as "veering on the side of caution", despite the clinical trial evidence that a shorter course of DAPT may be of greater overall benefit for many patients. In clinical practice there are also other factors not accounted for by the risk scores that might also influence the decision regarding DAPT duration. These include procedural variables such as stent type, lesion location and length, and vessel size, and specific clinical situations such as the need for non-cardiac surgery. Other factors include clinicians being slow to adapt to changes in guidelines and risk scores not used or available to implement the guideline recommendations. During the period when these patients were admitted, clinicians did not routinely use multivariable risk scores to assess bleeding or ischaemic risk. There is an opportunity to improve care by making these scores a part of routine practice. Integration of these risk scores into routine clinical practice will require clear guideline guidance together with making the risk scores readily available. The risk scores are currently available via a web-based calculator (https://www.vareanz.auckland.ac.nz/anzacs-qi-calculator/). They will shortly be available within the ANZACS-QI registry, and the risk scores will be automatically generated at the time the registry forms are completed and made available to clinicians for use at discharge and at the first post-discharge visit.

The evidence around DAPT duration post-ACS continues to evolve. Key limitations have been difficulties in standardising ischaemic and bleeding risk assessment and concerns around selective clinical trial enrolment. The use of the ANZACS-QI equations embedded in the real-world comprehensive ANZACS-QI cohort is an opportunity to design clinical trials to help answer important questions in post-ACS management.

Limitations

This study is retrospective and from a single centre, and thus is subject to the usual limitations of this design. However, it is likely that practice in most other cardiology units in New Zealand would be broadly similar. This study focussed on clinician decision for DAPT duration and is not powered to assess the impact on ischaemic or bleeding events, and is based on the duration

planned at discharge, not on how long DAPT was actually continued. This study also did not consider interventional factors that may require a longer duration of DAPT. The study did not audit practice in ACS patients who did not receive PCI.

Conclusion

During the period when these patients were admitted, clinicians did not routinely use multivariable risk scores to assess bleeding or ischaemic risk. However, by applying the new ANZACS-QI risk scores to the cohort, we have found that up to four out of five ACS patients could have been planned for shorter DAPT durations based on the 2020 ESC guideline recommendations. Although the more recent 2023 ESC guideline has swung back towards a default 12-month DAPT approach, it still endorses shorter durations in high bleeding risk patients. There may therefore be an opportunity to improve care by making the ANZACS-QI scores a part of routine practice. The ANZACS-QI registry is a real-world clinical trial platform. It could be utilised to study whether treating the nearly half of patients with high bleeding risk for shorter DAPT courses can reduce bleeding complications without increasing ischemic complications, and whether in the one third of low I and B risk patients shorter courses can minimise use of expensive anti-platelet agents without increasing risk.

COMPETING INTERESTS
No relevant disclosures.

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Outcomes of asymptomatic common bile duct stones detected at intra-operative cholangiography

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ABSTRACT

AIMS: The aim of this study was to investigate the outcome of common bile duct stones (CBDS) in asymptomatic patients at laparoscopic cholecystectomy (LC) and intra-operative cholangiogram (IOC).

METHODS: All patients undergoing LC and IOC at Te Whatu Ora – Health New Zealand Waikato between January 2017 and January 2022 were retrospectively reviewed. Electronic records were screened for asymptomatic CBDS. Exclusion criteria were hyperbilirubinaemia, gallstone pancreatitis, cholangitis and imaging-detected CBDS. IOC reports were reviewed to determine presence of CBDS. A second blinded review was undertaken by a radiologist. Outcomes were use of endoscopic retrograde pancreatography (ERCP), complications and readmission with retained CBDS.

RESULTS: Included were 1,297 patients undergoing LC and IOC. Of these, 150 (24.1%) patients had a positive IOC, of which 58 (38.7%) were asymptomatic. Attempted flushing of CBDS was employed in 49 cases, 10 successfully. Common duct exploration was successful in a further six out of seven cases. Of the remaining 42 patients, 18 were offered ERCP. Seven had no stone at endoscopy. Sixteen had imaging, revealing clear ducts in 14. The remaining two then had ERCP confirming choledocholithiasis. Eight patients were managed expectantly, of whom none required readmission with retained stones.

CONCLUSION: Rates of retained asymptomatic stones after positive IOC were low. Acknowledging risks associated with intervention and low rates of readmission with retained CBDS, an expectant approach could be more readily considered.

The natural history of incidentally discovered asymptomatic common bile duct stones (CBDS) at laparoscopic cholecystectomy (LC) remains poorly understood. Despite international guidelines recommending that all CBDS be removed,[1–4] several studies have suggested that a majority of CBDS will pass spontaneously. Encountering a previously undetected CBDS intra-operatively leaves the surgeon with a dilemma. Should laparoscopic transcystic or transcholedochal common bile duct exploration (CBDE) be undertaken? Should the patient undergo early endoscopic retrograde pancreatography (ERCP)? Is it safe to observe the clinical course of the CBDS? Each option has its own merits and drawbacks. Laparoscopic CBDE requires specific surgical expertise and there is a risk of post-operative bile leak, which may require further intervention. ERCP carries a risk of complications including pancreatitis, bleeding and duodenal perforation. Furthermore, studies have suggested an increased risk of post-ERCP pancreatitis with asymptomatic CBDS.[5,6]

Published evidence to guide management of incidental CBDS is relatively sparse and there is a lack of data specific to the New Zealand health system. Most published series have relatively low numbers and have not clearly defined when CBDS were considered asymptomatic.

Asymptomatic CBDS have been defined as having no clinical symptoms such as pain, fever or jaundice.[7] The possibility of pain or fever arising from stones in the gallbladder rather than CBDS leaves room for ambiguity in this definition. The National Institute for Health and Care Excellence (NICE) defines asymptomatic CBDS as stones found incidentally due to imaging investigations unrelated to gallstone disease in people who have been symptom-free 12 months prior to diagnosis.[8] This definition is difficult to apply to patients at the time of cholecystectomy who again may have symptoms relating to their gallbladder but not necessarily their CBDS. For the purposes of this study a pragmatic definition of asymptomatic, excluding all patients with high serum bilirubin, pre-operative imaging demonstrating CBDS or a history of cholangitis or gallstone pancreatitis, was used.

The aim of this study was to investigate the natural history of CBDS in our institution for patients with asymptomatic CBDS discovered at intra-operative cholangiography (IOC) during LC.

Methods

This was a retrospective review of all patients undergoing LC and IOC at Waikato Hospital in New Zealand between January 2017 and January 2022. Electronic records were screened to detect patients who were asymptomatic for CBDS at the time of LC and IOC. Exclusion criteria were patients deemed symptomatic for CBDS, and this was taken as the presence of hyperbilirubinaemia, gallstone pancreatitis, cholangitis and positive pre-operative imaging findings of CBDS. Written IOC reports were reviewed to determine presence of CBDS. A blinded retrospective review of the IOC films deemed positive was undertaken by a radiologist. Where there was uncertainty, a blinded read by a second radiologist was sought. The patients' electronic records were then followed to determine the treatment of CBDS. The utilisation of post-operative imaging, use of ERCP and its complications, as well as the rate of readmission with retained CBDS were interrogated.

Results

A total of 1,297 patients underwent LC and IOC between the study dates. One hundred and fifty (24.1%) patients had a positive IOC, and of these 58/150 (38.7%) were classified as asymptomatic. In the asymptomatic cohort 46/58 (79.3%) were female. The median age was 49 years (22–76). Median length of hospital stay was 1 day (0–96). Thirty-seven out of 58 (63.8%) were elective cases. The outcomes in both the acute and elective cohorts are detailed in Table 1.

Attempted flushing of CBDS via cholangiogram catheter was employed in 49/58 cases (83.1%). Ten were successful (20.4%). A further six ducts were cleared with transcystic manipulation with a Nathanson catheter, leaving 42 patients whose ducts were not cleared leaving the operating theatre. Sixteen of 42 (38.1%) had further diagnostic imaging. Eleven patients had an acute inpatient magnetic resonance cholangiopancreatography (MRCP); four an urgent outpatient MRCP. One had an outpatient endoscopic ultrasound (EUS) that was not followed by ERCP. Fourteen out of 16 (87.5%) had imaging showing clear ducts. Two out of 16 (12.5%) proceeded to ERCP, which confirmed choledocholithiasis.

Eight out of 42 (19.0%) patients were managed expectantly. None (0/8) of these patients managed expectantly had readmission with a retained stone; however, one patient who had a clear IOC after flushing subsequently represented with retained stone and required an ERCP.

Eighteen out of 42 (42.9%) were offered ERCP after discovery of an asymptomatic CBDS. Seven out of 18 (38.9%) of these had no stone at endoscopy. One patient had an ERCP due to a retained stone after a clear IOC post-flushing. An additional patient had an ERCP due to possible bile leak.

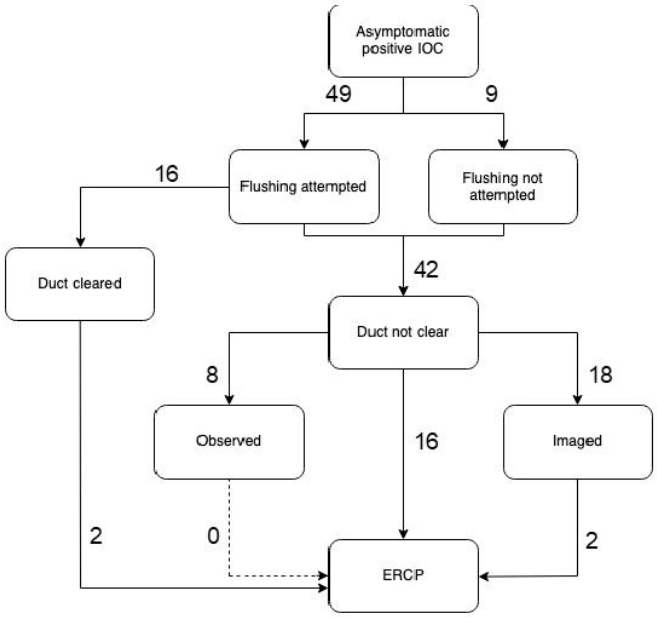
In total, 22/58 (37.2%) patients in this asymptomatic cohort underwent ERCP. As described above, 18 had been offered direct ERCP post-operatively and two patients had post-operative imaging showing CBDS.

Table 1: Outcomes of asymptomatic CBDS stratified by acute and elective patient setting.

Admission type	Acute (n=21)	Elective (n=37)
Flushing of duct successful	3/21	7/37
Manipulation with Nathanson successful	1/21	5/37
Imaged (positive)	8/17 (2/8)	7/25 (0/7)
ERCP (positive)	7 (5/7)	13 (8/13)

ERCP = endoscopic retrograde pancreatography.

Figure 1: Flow diagram of asymptomatic patients with CBDS.



Combining flushing, CBDE and ERCP, 29 patients had a therapeutic intervention that removed an asymptomatic stone, whereas the other 29 did not need this. Only 14 (24.1%) had a therapeutic ERCP after discovery of incidental CBDS at the time of operation.

There were no cases (0/22) of post-ERCP pancreatitis. However, of note is one patient who had a duodenal perforation after ERCP resulting in a large infected retroperitoneal collection, which necessitated multiple debridements and a 94-day hospital stay.

Of note, 27 of the 92 symptomatic patients did not have an ERCP after positive IOC. There were 17 (63%) who had an MRCP after the IOC showing clear ducts. The remaining 10 were monitored clinically and there were no readmissions in this group. The median days from LC to ERCP in the symptomatic group was 5 days and for the asymptomatic group it was 7 days.

In a blinded radiology review of the 58 asymptomatic patients whose IOC runs were deemed to be positive intra-operatively, 6/58 did not have their IOC images available for review and were excluded from this second review. Of the remaining 52, 27/52 (52.0%) were deemed to be a truly positive result. In all, 3/27 were flushed successfully and 3/27 were removed with transcystic manipulation with a Nathanson catheter. Of the remaining 21, 5 had an MRCP which was positive in 2/5. There was 1 patient who had an EUS, which was negative. There were 14/21 who underwent an ERCP, which was positive in 9/14 (64.3%). A further 4 out of 21 were observed and none had readmission with a retained stone.

For those patients in whom radiology review deemed the IOC to be a false positive (25/52), 13/25 (52.0%) had a duct that was deemed cleared with flushing or by manipulation with a Nathanson basket. Of the remaining 12, eight had imaging confirming a clear bile duct. One patient had an ERCP and this was negative.

Discussion

This large retrospective review of patients undergoing LC and IOC at a tertiary hospital in New Zealand captured a modest number of patients with asymptomatic CBDS. In our series, the rates of retained CBDS after detection of asymptomatic CBDS at IOC are low. None of the eight patients who were treated expectantly represented with a retained stone. Additionally, 14 patients had post-operative imaging demonstrating clear ducts. Finally, seven of 18 patients (38.9%) who proceeded directly to ERCP after surgery did not have a CBDS. There are a number of possible reasons for this phenomenon.

Some cholangiograms may have been false positives. In a series by Hublet, 5/31 (16%) IOCs were considered false positives when comparing IOC with laparoscopic ultrasound.[9] Ampullary spasm or stenosis, or air bubbles during cholangiogram, may mimic the presence of CBDS. Indeed, a blinded review of the IOCs included in our study by a radiologist showed that 25/52 (48.1%) were possible false positives. It is noteworthy that of these 25 false positive IOCs, slightly more than half had been reported to be cleared with flushing or transcystic manipulation by the operating surgeon. Lack of real-time assessment may have made interpretation of these sequences difficult for the retrospective blinded reviewer. Also, it is possible that the initial positive IOC runs were not saved and only the post-clearance IOCs were saved. We have used the operator's interpretation of the IOC as the basis of our analysis, given that in practice, IOCs would not be routinely reviewed by a radiologist and clinical decisions are made on the basis of the operator's interpretation of the IOC findings.

Where false positives have been excluded, the discrepancy between IOC and negative post-operative imaging or ERCP is likely to be accounted for by spontaneous passage of CBDS. A recent systematic review and meta-analysis of patients who were asymptomatic for choledocholithiasis but had a positive IOC demonstrated that 20.9% of patients treated expectantly for CBDS subsequently had retained stones, while 50.6% had a stone at a planned post-operative ERCP.[10] The findings of this study are in line with the systematic review and the discrepancy between rates of retained stones in those treated expectantly, 0% vs 20.9%, and those who had CBDS at ERCP, 61.1% vs 50.6%, are likely to be due to low numbers in our cohort. Our observations here suggest that a reasonable number of patients with asymptomatic CBDS can be successfully managed expectantly and support a selective approach to intervention for CBDS.

Overall, rates of complications were low. Of note, there were no cases of post-ERCP pancreatitis (PEP) in our cohort. This is in contrast with studies that have shown that asymptomatic CBDS are a risk factor for PEP.[5,6] The incidence of PEP in those undergoing ERCP for asymptomatic CBDS ranges between 12.5% to 20.8%, compared to 3.0% to 6.9% in those who have symptomatic CBDS.[11,12] One possible reason why this was not seen was due to the low absolute number of ERCP in our study population.

One patient who had common duct exploration via a transcystic approach with a Nathanson catheter proceeded to have an ERCP for a suspected bile leak. The ERCP did not demonstrate a bile leak, but the patient did have a biloma that was percutaneously drained. One patient from the cohort of patients who had an ERCP had a major complication by way of a duodenal perforation. This led to the patient having an admission to the high dependency unit and necessitated multiple retroperitoneoscopic debridements.

Previous single-centre studies have encountered a similar problem of low numbers.[13–18] Our study differs from most in that we have attempted to clearly categorise patients with asymptomatic stones and in this regard our study remains one of the largest to report data specific to asymptomatic stones from a single centre. There has also been a lack of data specific to New Zealand. Chen et al. reported on the outcomes of 75 patients with incidental CBDS (defined by normal serum bilirubin and gamma glutamyl transferase) at IOC over a 5-year period in an Australian tertiary hospital and found that 43% had no residual choledocholithiasis within 30 days of LC and IOC.[19] It is uncertain if any of this group had presented with pancreatitis or had pre-operative imaging suggesting CBDS. While published data examining the outcomes of expectant management for asymptomatic stones are limited, the largest available series are from the multi-centre GallRiks database in Sweden.[20,21] A sub-analysis of 2,168 patients from this nation-wide database found that for patients stated to be asymptomatic with stones <4mm in size, only 5.6% of patients directed to expectant management represented with evidence of retained stones during follow-up.

The merits of routine versus selective cholangiography in cholecystectomy have been debated extensively. The arguments are complex, with advocates for routine cholangiography suggesting reduced rates of bile duct injury with routine cholangiography, although this is a rare event.[22] Our results suggest high rates of spontaneous passage of filling defects detected at IOC in patients considered to be asymptomatic. This could lend weight to the argument for omitting IOC for patients with no symptoms to suggest CBDS, given that positive findings might be managed conservatively in any case.

There were several weaknesses of this study. The study design was retrospective in nature and included only a single institution. The study was observational and as such there was no blinding

or randomisation. Furthermore, despite the large number of patients undergoing LC and IOC at our institution, the incidence of asymptomatic CBDS led to low numbers of patients in the study: 58/1,297 (4.47%).

Our study did not investigate factors that may be associated with stone passage, such as the size of the stone or the passage of contrast into the duodenum on IOC. Some authors[23] suggested stones <4.5mm were more likely to pass spontaneously while others[21] did not find a difference in outcome between small and large CBDS. Further studies would be helpful to investigate these features in more detail.

Decision making around the management of asymptomatic CBDS remains a vexing clinical problem with potential risk attached to both over-treatment and under-treatment. The relative merits of a blanket approach to treating all CBDS versus a selective approach may be influenced by the dynamics of local healthcare systems. Evidence to guide management of asymptomatic CBDS specific to New Zealand is currently limited. Given the relatively low incidence of truly incidental CBDS and the low incidence of adverse events, pooled data from multiple centres may provide more robust data.

Conclusion

In this study focussing on CBDS defined as asymptomatic, rates of retained stones after positive IOC were low. Acknowledging risks associated with intervention (ERCP) and the low rates of readmission with retained CBDS, a strategy of watchful waiting in this patient group could be more readily considered.

COMPETING INTERESTS
Nil.

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Construction of the chronic temporomandibular disorder patients: the association between neural and psychological pathways

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ABSTRACT

Chronic temporomandibular disorder (cTMD) as a term based on the diagnostic criteria for temporomandibular disorders (DC/TMD) classification refers, in this paper, to the condition listed that has a non-mechanical association without any obvious organic cause. Specifically, this is the condition that falls under the International Classification of Diseases 11th revision (ICD-11) classification of chronic primary and chronic secondary pains. This implies that there is increased responsiveness of nociceptive neurons in the central nervous system, a phenomenon known as central sensitisation. cTMD patients may have their beginning with genetic susceptibility to pain. Although no single gene is exclusively linked to cTMD, various genes associated with nervous and musculoskeletal systems are believed to play a role. Environmental triggers and epigenetic changes are also thought to contribute to cTMD development. The biopsychosocial model emphasises the need to comprehensively address biological, psychological and social factors in cTMD assessment and management. In this study, we leverage the cyclic causation framework within the biopsychosocial model to illuminate the intricate interplay between biological and psychosocial factors in the context of cTMD. The conceptualisation of cTMD involves the dynamic evolution of genetic predispositions, influenced by life events and other biological susceptibilities. These factors collectively contribute to the emergence of nociplastic changes, ultimately manifesting as the distinctive features observed in individuals afflicted with cTMD.

Pain was described by the International Association for the Study of Pain (IASP) as "*An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage*".[1] While there is no precise boundary defining the transition from acute to chronic pain, the International Classification of Diseases (ICD) and International Classification of Orofacial Pain (ICOP) indicates that pain persisting beyond the anticipated healing period (lasting more than 3 months and occurring on at least 15 days per month) is considered pathological.[2,3]

Expanding upon this concept, we specifically designate chronic temporomandibular disorder (cTMD) in this article to encompass conditions outlined in the diagnostic criteria for temporomandibular disorders (DC/TMD) classification. This condition is characterised by a lack of mechanical origins and without clear organic causes. Temporomandibular disorder (TMD) is a comprehensive term encompassing various conditions causing pain and dysfunction in the orofacial region. In the decade since its inception, the DC/TMD classification has outlined various conditions, including mechanical aberrations tied to the temporomandibular joint such as disc displacement, disc perforations, condylar hyperplasia and certain tumours. Additionally, it addresses conditions linked to chronic pain, encompassing myogenous and arthrogenous sources, along with headaches associated with cTMD, among others collectively denoted as cTMD.[4] This second group encompasses the majority of patient cohorts frequently encountered in pain management clinics. cTMD specifically denotes individuals grappling with pain persisting for more than 3 months, a definition set forth by IASP. In contrast, the International Classification of Diseases 11th Revision (ICD-11), introduced by the World Health Organization in 2018, presents a more comprehensive framework that categorises this prevalent, non-mechanical cohort under the umbrella of chronic primary pain. This system integrates specific codes and classifications for a diverse range of diseases and conditions, including chronic pain disorders like cTMD. Recognising chronic pain, including cTMD, as a pathology, the

ICD-11 provides a clinically relevant perspective. The National Academy of Medicine (NAM) has underscored a critical gap in the assessment, diagnosis and management of both acute and chronic conditions, with a particular focus on cTMD.[5] It highlights the intricate, multisystem nature of cTMDs, necessitating a comprehensive, multidisciplinary approach to treatment. Unlike conventional medicine, which predominantly addresses diseases with discernible physiological mechanisms, the ICD-11's definition of chronic primary pain recognises the absence of pathology at the perceived pain site. This perspective challenges clinicians to adapt their approach to managing conditions where the pathology may not be immediately evident. In this context, the term TMD is deemed misleading, as it implies an association with the structures housing the pain and utilises the term "Disorder," which lacks a specific pathology for diagnosis.

Addressing the intertwined challenges of pain and psychological distress, common components of many diseases, proves to be a formidable task. Unlike conditions where pathology primarily affects peripheral organs, the origins of pain and distress are deeply embedded in intricate brain functions.[6] Remarkably, alterations in nerve function could manifest in individuals genetically predisposed to pain sensitivity and psychological distress.[7] When examining conditions like ICD-11 chronic primary pain associated with chronic temporomandibular disorder (CPP cTMD), it becomes crucial to unravel the intricate factors contributing to these patients' experiences. This deeper understanding is essential for the effective management of chronic pain sufferers, enabling healthcare professionals to devise more targeted and empathic interventions. Furthermore, cTMD has been integrated into the established biopsychosocial model employed for studying and managing various chronic pain conditions.[8] The findings from *Orofacial Pain: Prospective Evaluation and Risk Assessment (OPPERA)* bolster the complexity of cTMD, emphasising the need for a nuanced approach. The alignment between OPPERA findings and the biopsychosocial framework affirms a non-local aetiology for cTMD, emphasising the multifaceted nature of this condition.[9]

A plausible model for understanding the development of cTMD involves commencing with a consideration of genetic predisposition to pain, for example the catechol-o-methyltransferase (COMT) gene in the case of cTMD. This genetic susceptibility may contribute to an individual possessing a distinct neuroanatomical makeup, rendering them more susceptible to experiencing pain. Variations among individuals could mean that some are more predisposed to undergoing "temporary" neuroplastic changes, eventually leading them to become chronic pain sufferers. This correlation has been demonstrated in conditions like fibromyalgia, where genetic factors potentially account for up to 50% of disease susceptibility.[10] Under the ICD-11 classification, cTMD and fibromyalgia share a similar pathogenesis.[2]

This intricate construction, rooted in the individual's genetic makeup, is further complicated by the influence of psychosocial factors, which also harbour genetic components and may be influenced by factors such as childhood trauma and other biological intervening variables (Figure 1).[11–13]

Biopsychosocial model

George Engel conceptualised the biopsychosocial model as a dynamic and interactive perspective on human experience, acknowledging the intricate interplay between the mind and body.[14] This model emphasises the mutual influence and interconnectedness of psychological, biological and social factors in shaping the human condition.[15] The notion of various system levels and the emergence of a whole that possesses information greater than the sum of its parts is the foundational concept of this model. In this framework, neural network activity serves as a foundational level from which psychological functions arise. Furthermore, biological, psychological and social factors relate in a cyclic manner, meaning that each can influence and affect the others in a continuous loop, which is a fundamental concept in the biopsychosocial model and systems theory. This recognition of bidirectional influences is an essential aspect of understanding the complexity of human experiences and interactions.[16,17] Such cyclic interaction between environment and genes have been also demonstrated.[18]

It is of interest then to consider causative paths between the three levels (biology, psychology and social). Both biological and social factors appear to play a role in shaping psychological aspects. For instance, neural networks, starting from membrane function and extending upwards, could serve as a model for understanding the parallel between biological and psychological modules. Additionally, interventions such as family and group therapy have demonstrated their impact on individual mental health. Numerous research studies highlight

potential continuous links from the cellular membrane (ion channels) level to the neuronal and network levels. An illustrative example is the cyclic nucleotide-gated channel, present in both the prefrontal cortex and the hippocampus. This channel is involved in recurrent information processing within individual neurons, providing valuable insights into the intricate connections between biological and psychological processes.[19–21] Significantly, the same regions of the brain play a crucial role in attention, memory and affective functions. Specifically, well-established association networks such as the central executive network, dorsal attentional network, salience network and default mode network (DMN) are pivotal in influencing cognitions associated with affect.[22] Hence, there is the potential to construct a nuanced comprehension of information processing, tracing it from the biological level (beginning with membrane function) to the generation of cognitive and affective functions by biological networks (akin to hardware), to the way learning acts as software or a form of self-programming. How psychological phenomena could affect social activity could be observed in how memes affect group social behaviour. However, the reverse—purely psychological phenomena directly influencing biological function—raises intriguing questions. While the observations of alterations in neural pathways due to psychotherapy are compelling, it's important to acknowledge potential objections rooted in social factors, particularly the dynamics between therapists and patients.[23] These interactions could introduce complexities, making it difficult to purely isolate the psychological impact. One potential avenue for exploring this connection lies in long-term, introspective practices devoid of social interference. For instance, individuals engaging in silent meditation over extended periods may offer valuable insights. Through practices like insight meditation, where individuals delve deeply into their own psyche, devoid of external social influences, we may gain a clearer understanding of how purely psychological experiences intricately influence biological functions. Such evidence has been demonstrated in scientific studies.[24] Mindfulness practice is known to assist in pain management.[25]

Engaging in mindfulness practice has been linked to the augmentation of grey matter in key brain regions, including the anterior cingulate, prefrontal cortex and hippocampus, while concurrently structural changing in the amygdala.[26,27] Additionally, mindfulness has been observed to improve the functioning of the DMN.[28] Notably, the DMN plays a role in directing attention towards pure sensations, effectively dampening the activity of other networks. This inhibition contributes to a reduction in cognitive and affective functions, potentially influencing distress and pain experiences.

In the realm of causation, the proximity of the temporal connection between cause and effect often determines the consideration of a more proximal cause. Nevertheless, cascades of causation can unfold, commencing with an early factor such as childhood trauma, which may engender vulnerabilities, thereby heightening the probability of succumbing to additional causes in the future. For instance, the experience of childhood trauma may lead to both psychological and biological impairments, rendering the individual more susceptible to subsequent traumas, whether they be of environmental, social or biological origin (Figure 1). These interconnected causes can set in motion a divergent cascade of events that eventually converge to influence a singular disorder, such as cTMD. A construct can be formulated to illustrate how the biopsychosocial model might alter the initial interplay between afferent and descending pathways of nociception. While the placebo and nocebo concepts offer a simplified explanation of the impact on the descending pathway through learning and other neural mechanisms, our proposition is to integrate these concepts within a broader biopsychosocial model (Figure 2).[29]

Chronic temporomandibular disorders

The concept of disease, defined as a bodily disruption or abnormality, is just a linguistic construct. Disease and illness share similar semantics, both pointing to human suffering and dissatisfaction with one's condition. Whether this suffering arises from purely biological factors within the brain or body, psychological factors rooted in one's thoughts and feelings or social factors related to interactions with the external world, all of these sources are valid and significant considerations in understanding the human experience of health challenges. The use of the term disorder is often an apt compromise when pathophysiology is not completely established.

Regarding cTMD, the journey starts with an individual's genetic makeup. Chronic pain sufferers often commence life with a genetic

predisposition to pain sensitivity, setting the stage for their unique pain experiences. While there have been studies exploring potential genetic links to cTMD, the field is complex, and no specific "TMD genes" have been universally identified. While there isn't a single gene that is exclusively associated with cTMD, several genes that code for receptors and proteins, such as serotonin and sensory neuron receptors, cytokines, matrix metalloproteinases, oestrogen receptor and calcitonin gene-related peptide in the nervous and musculoskeletal systems have been studied in relation to cTMD.[30–35] A systematic review has revealed a genetic overlap involving three specific genes—ESR1, MTHFR and COMT—in the genetic profiles of patients diagnosed with both primary headaches and cTMD.[36] The COMT gene is additionally linked to susceptibility to mood and anxiety disorders.[37,38]

However, cTMD is a multifactorial condition, meaning it is influenced by a combination of genetic, environmental and lifestyle factors. The triplet code of DNA, once thought to be entirely deterministic of the phenotype, does not exclusively dictate an organism's traits. Environmental influences could induce changes in DNA structure, driving evolutionary shifts. This dynamic interaction implies that lower-level factors (e.g., DNA) may shape outcomes at higher levels (complex network, e.g., Organism), creating a feedback loop of circular causation.[17] Furthermore, genetic information flows from DNA to RNA, but it's important to note that RNA could also influence DNA (Figure 3).[39] The field of epigenetics has unveiled a fascinating phenomenon: environmental factors, through processes like histone methylation, could modify gene expression.[40] Frequently, this modification may involve the recombination and relocation of genetic domains, resulting in the transmission of new traits down the germ line.

Environmental triggers and epigenetic changes

Genetic susceptibility plays a role in gastrointestinal tract sensitivity.[41] A gastrointestinal infection could trigger an immune response, not only against the infection itself but also against the gastrointestinal tract lining. This primed immune reaction could lead to peripheral nociception. This process, in turn, causes peripheral and central sensitisation through sacral, lumbar and thoracic pathways, contributing to conditions like irritable bowel syndrome.[42–44] These individuals may become predisposed to other chronic pains such as cTMD due to this priming, resulting in the development of chronic overlapping pain conditions.[45] Moreover, lifestyle factors like smoking may downregulate stress responses and inhibit serotonin synthesis, while alcohol use may increase dopamine and endogenous opioids synthesis (Figure 4).[46,47] Additionally, a high-fat diet may enhance reward-related circuitry.[48,49] These factors further contribute to the complexity of pain conditions, highlighting the intricate interplay between genetics, environmental triggers and lifestyle choices in the development of chronic pain conditions (Figure 4).

Pain perception is intricately influenced by alterations in modulators and receptors within the body. For instance, the Homer genes may have the potential to inhibit nociceptive signal transmission in the posterior horn of the spinal cord, modulating pain signals.[50] Prodynorphin (PDYN) has links to conditions such as depression, stress and substance addiction.[51–53] Stress triggers the release of norepinephrine and cortisol while simultaneously decreasing testosterone levels, potentially contributing to the higher prevalence of pain in females.[54,55] The impact of stress and its associated humoral response has the potential to induce changes in immune response, potentially affecting gut function. This cascade effect may contribute to sensitisation processes, ultimately predisposing individuals to chronic pain. Additionally, histone modification may lead to central sensitisation, amplifying pain signals.[56] Vitamin D deficiency has been associated with decreased pain threshold and tolerance.[57,58] Pronociceptive factors act as amplifiers, intensifying sensitisation processes.[59] These factors involve reactive oxygen species, proinflammatory cells and signals, adaptations in neuroimmune synapses, negative affective states, structural volume changes (hippocampus), loss of control systems, reduced positive interactions and decreased environmental exposure.[60–63] Peripheral injuries could activate both the innate and adaptive branches of the immune system to resolve tissue damage, but prolonged immune activation may contribute to the chronicity of pain.[64]

Pain processing and central sensitisation

The sensory-discriminative aspects of pain originating from trigeminal nociceptive neurons are processed in specific brain regions. These include the primary somatosensory cortex, posterior insular cortex and thalamus, all of which receive direct projections from these neurons in

the orofacial region (Figure 4).[65] In the thalamus, trigeminal nociceptive inputs undergo modulation before being transmitted to both cortical and subcortical structures, highlighting the intricate processing and integration of pain signals in the central nervous system.[66] The primary somatosensory cortex and insular cortex play crucial roles in encoding the intensity of painful stimuli. These regions exhibit graded increases in activity, directly corresponding to the intensity of the stimulus presented. This precise modulation reflects the brain's sophisticated processing of pain perception.[67,68] The prefrontal cortex, anterior cingulate cortex and secondary somatosensory cortex are integral to the comprehensive processing of pain signals, encompassing emotional, cognitive, sensory and spatial aspects of pain perception and modulation. The prefrontal cortex is involved in the emotional aspect of pain processing and is essential for cognitive evaluation of pain-related stimuli.[69,70] The anterior cingulate cortex is considered to be involved in a variety of cognitive and emotional processes such as pain and coping mechanisms, especially affective pain.[71] The secondary somatosensory cortex integrates and processes nociceptive information, enhancing the brain's ability to perceive and respond to pain signals, and contributes to the sensory-discriminative dimension of pain.[72,73]

In central sensitisation, the central nervous system becomes hyperresponsive to pain stimuli, involving complex changes in various brain regions. The exact pattern of activation or deactivation varies based on the individual or condition; however, several important brain regions are thought to be associated with central sensitisation. Chronic pain has the potential to alter the brain's structure, indicating that prolonged pain experiences might lead to observable changes in the brain's anatomy.[74] For example, research reveals chronic back pain correlates with reduced grey matter density in both the prefrontal cortex and thalamus.[69,75] This finding indicates structural changes in these brain regions, suggesting a link between persistent back pain and alterations in specific areas of the brain.[75] A study also showed a decrease in grey matter within regions, including the cingulate cortex, insula and prefrontal cortex, associated with pain processing in individuals experiencing chronic pain.[76] However, another study argued that structural brain changes observed in chronic pain patients likely do not indicate damage or atrophy. This suggests that alterations in the brain's structure related to chronic pain might be different in nature from traditional damage or degeneration.[77,78] cTMD has been associated with central sensitisation. For example, experimental pain studies showed individuals experiencing painful cTMD exhibit higher sensitivity to experimental pain stimuli. They demonstrate lower thermal and ischemic pain thresholds, along with reduced tolerance values, compared to individuals without cTMD symptoms.[79,80] Quantitative sensory testing in patients with painful cTMD have revealed lower pain thresholds and increased pain responses to various stimuli, supporting the presence of central sensitisation.[79,81] Functional magnetic resonance imaging studies have shown abnormal altered brain activity patterns in response to both innocuous and painful stimuli and altered connectivity in pain-related brain regions in individuals with cTMD, suggesting central nervous system involvement in cTMD-related pain.[82] Studies have demonstrated enhanced temporal summation of pain in cTMD patients, indicating an increased response to repeated noxious stimuli, which is a characteristic feature of central sensitisation.[83] Clinically, cTMD patients often report widespread pain and increased sensitivity to pressure, heat or cold, symptoms consistent with central sensitisation.[84–86] Treatments focussing on central sensitisation, such as cognitive behavioural therapy and medications acting on the central nervous system, have shown effectiveness in managing cTMD-related pain.[87] This offers indirect evidence supporting the involvement of central sensitisation in cTMD. In considering the previous section, it is apparent that a vicious cycle can emerge where physical and mental suffering reinforce each other.

A biopsychosocial model of cTMD

In the context of the biopsychosocial model, the Al-Khotani et al. study highlights significant connections between psychosocial, somatic and behavioural coexisting conditions and cTMD-related pain among children and adolescents.[88] Biological factors, including genetics, hormonal influences and anatomical variations, have been implicated in cTMD. Certain genetic markers and hormonal changes have been specifically associated with the development of cTMD symptoms, underscoring the disorder's biological basis.[89] Psychological factors such as stress, anxiety and depression, along with individual coping mechanisms, significantly influence the onset and

worsening of cTMD symptoms. Extensive research demonstrates a strong association between psychological distress and the severity of cTMD symptoms. Implementing stress management and relaxation techniques has proven effective in alleviating cTMD-related pain.[90] Social factors, encompassing socio-economic status, social support and cultural influences profoundly shape how individuals perceive and manage pain. Strong social support systems enhance coping mechanisms, especially in chronic pain conditions such as cTMD. Furthermore, cultural beliefs and attitudes toward pain significantly influence how cTMD symptoms are experienced and communicated.[91–95] Evidence from cognitive behavioural therapies, which address maladaptive pain beliefs and coping strategies, have been successful in managing cTMD-related pain, which is indirect evidence supporting the biopsychosocial model.[96] Treatment strategies for cTMD typically integrate dental, medical, psychological and physical therapies. Multidisciplinary programmes, rooted in the biopsychosocial model, have demonstrated significant success in enhancing pain management and overall quality of life for individuals dealing with cTMD.[97] The evidence supporting the biopsychosocial model in the context of cTMD is strong. It highlights the importance of addressing biological, psychological and social factors comprehensively in the assessment and management of cTMD.

Childhood trauma and sleep disorder in cTMD

Childhood trauma (emotional/physical) may be a contributing factor to the development of cTMD; however, it's important to note that the relationship between childhood trauma and cTMD is complex and multifaceted. Childhood trauma may lead to chronic stress, altered pain processing pathways and changes in the way the brain perceives and responds to pain.[98,99] Childhood trauma is a common antecedent of mood and anxiety disorders, which are co-morbid with pain disorders and could generate a vicious cycle.[100–102] These factors might contribute to the onset or exacerbation of cTMD symptoms. Moreover, individuals who have experienced childhood trauma may be at increased risk of parafunction habits such as bruxism.[103] This, in turn, could trigger epigenetic changes, further predisposing these individuals to the process of chronification. The influence of trauma, whether experienced in childhood or adulthood, has been minimally explored in relation to cTMD. Nevertheless, it is crucial to acknowledge the potential link between trauma, especially childhood trauma, and the establishment of chronic vulnerability to a cascade of future traumas, both social and biological.

Sleep plays a crucial role in chronic cTMD.[104] Conditions like obstructive sleep apnea (OSA) and insomnia, whether individually or in combination (comorbid insomnia and obstructive sleep apnea [COIMSA]), may worsen cTMD symptoms.[104–106] A healthy sleep routine of 7–9 hours is recommended, and chronic insomnia often involves underlying psychosocial and behavioural factors.[107] Managing sleep disorders involves various approaches. Continuous positive airway pressure and mandibular advancement appliances may be used for OSA, while cognitive behavioural therapy for insomnia is effective for insomnia.[108,109] A combination of these methods is often employed for COIMSA cases.[110] Studies suggest that more than one third of Australian adults experience nocturnal symptoms.[111] Notably, sleep disorders have genetic links and are associated with an increased risk of anxiety and depression.[112,113] Assessments, including detailed sleep history and diaries (digital, visual and text-based), provide valuable insights into cTMD.

Points of direction

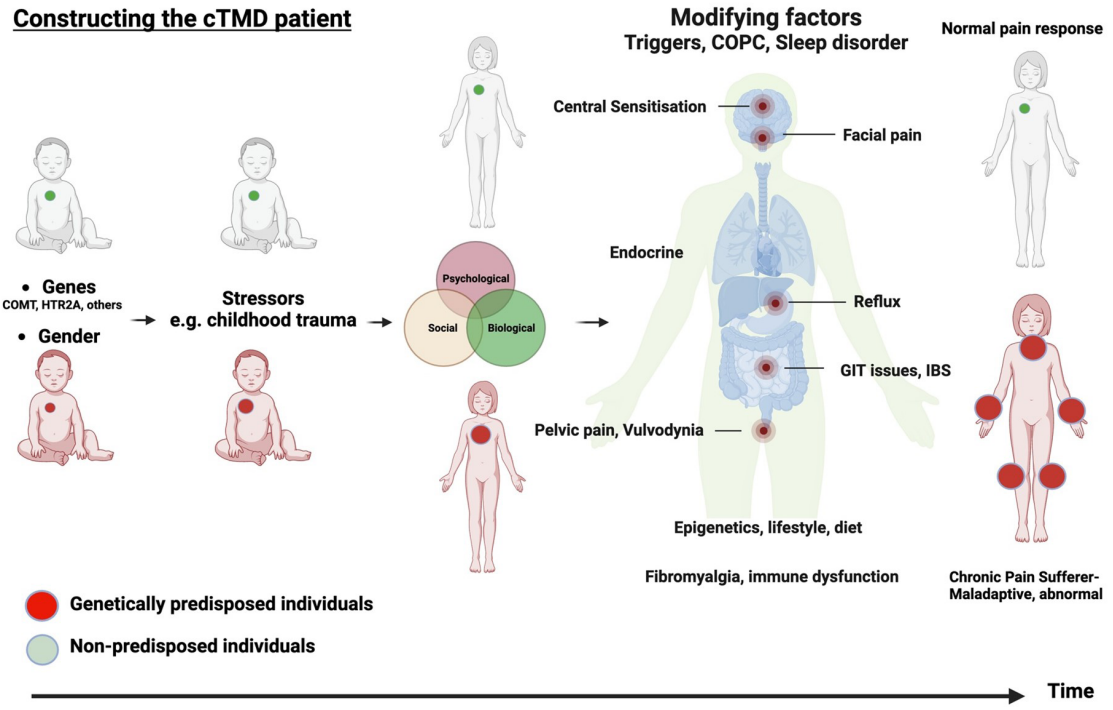
In this paper, we employ the biopsychosocial model to formulate a framework for understanding the pathological evolution of cTMD. Although the model is not exhaustive, it serves as an initial template to guide the construction of more intricate models for comprehending cTMD as a disorder. Our conceptualisation defines a disorder as any condition leading to a homeostatic imbalance from a biopsychosocial perspective. We assert the validity of the biopsychosocial model for both understanding the causation and managing the manifestations of diseases. Specific environmental and psychological factors play significant roles in triggering the persistence of neural mechanisms associated with cTMD. Consequently, we propose that this model could serve as a catalyst for further research in the field. Furthermore, our understanding and focus on the underlying process shows promising research into management of chronic pain in the future through fields like chemogenetics. Chemogenetics has been shown as a possible means to suppress the hyperexcitability and maladaptive changes seen in chronic pain.[114] We advocate for the integration of advanced

biological techniques with psychosocial interventions as an ongoing strategy for advancing research in cTMD. While biological research in cTMD is advancing rapidly, investigations into psychosocial causes and interventions lag behind. Recognising this gap, we emphasise the need for further research into childhood trauma and its cascading susceptibility to trauma as an area requiring more attention compared to general research in chronic pain. Furthermore, despite the longstanding use of mindfulness-based cognitive therapy as a treatment strategy in chronic pain for nearly 5 decades, its application in the field of cTMD warrants continued development. This highlights the importance of concurrently advancing both biological and psychosocial dimensions in cTMD research to comprehensively address the complexities of this disorder.

Conclusion

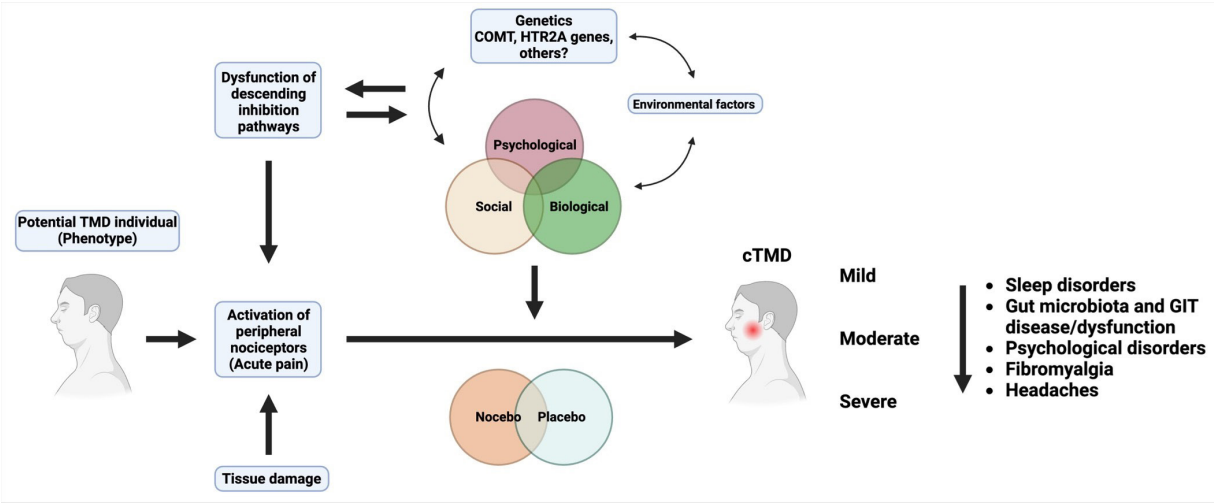
Genetics and epigenetics exert influence on both chronic pain and the psychological constitution of individuals, shaping neuroanatomical pathways and function. Chronic pain, characterised as pathological, is distinctly outlined in the ICD-11 classification, emphasising the absence of pathology at the pain site. Instead, the focus shifts to neural pathways, influenced by various factors such as psychological elements, sleep patterns, emotional trauma and immune system irregularities, which collectively contribute to the final experience of pain. Central sensitisation emerges as a pivotal factor in cTMD, playing a central role in amplifying pain perception and sensitivity among affected individuals. The supporting evidence for the biopsychosocial model in the context of cTMD is robust. We propose a tentative model illustrating the intricate interplay between biological and psychosocial factors, forming a cascade that culminates in cTMD as a disorder of neural and psychological pathways. This model underscores the imperative need to address biological, psychological and social factors comprehensively in the assessment and management of temporomandibular disorders.

Figure 1: Constructing the cTMD patient.



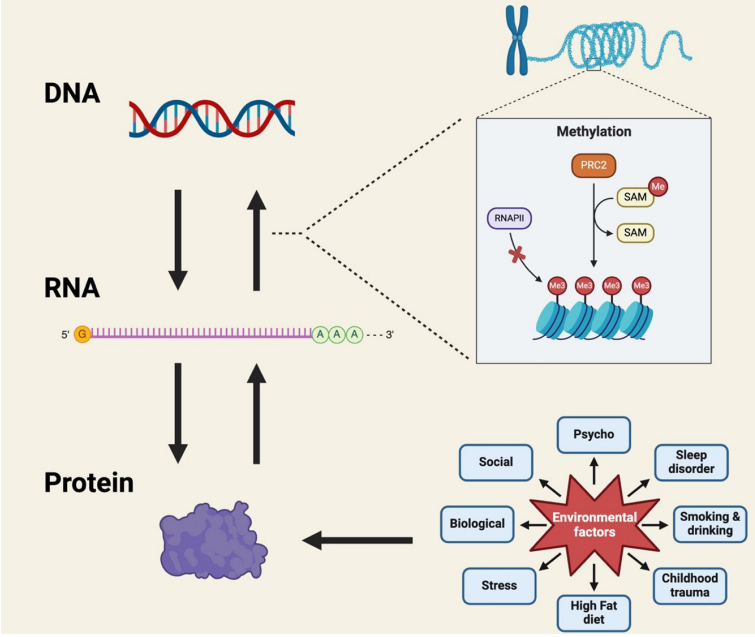
This model integrates both temporal (developmental) and biopsychosocial (construct) perspectives. It underscores the intricate interplay between genetic factors, which are influenced by psychosocial and other biological elements, culminating in noteworthy epigenetic changes. Created with BioRender.com

Figure 2: Transitioning to cTMD entails activation through diverse triggers.



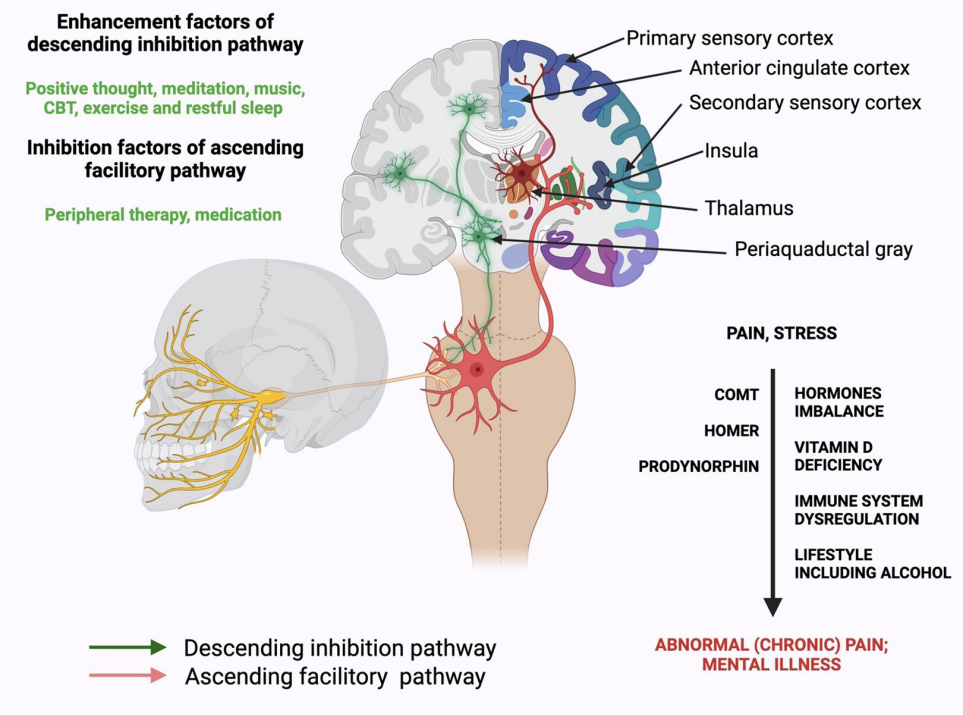
Transitioning to cTMD entails activation through diverse triggers. It elucidates the modelling of this intricate process, incorporating constructs from both the biopsychosocial model and the nocebo/placebo model. The severity of this transition is intricately shaped by a multitude of factors visually depicted in the figure. Created with BioRender.com

Figure 3: DNA, RNA and methylation.



Typically, the genetic programme follows the sequence from DNA to RNA; however, it is noteworthy that RNA also has the capacity to modify DNA. The realm of epigenetics has illuminated an intriguing phenomenon wherein environmental factors, utilising processes such as histone methylation, can exert influence on gene expression. Created with BioRender.com

Figure 4: The trigeminal pathway.



Environmental triggers, including hormonal imbalances, vitamin deficiencies, immune system dysregulation and lifestyle choices, have the potential to adversely impact pain. The modulation of descending inhibition can be augmented through self-directed interventions and medications, ultimately leading to a reduction in pain. Created with BioRender.com

COMPETING INTERESTS
The authors have no conflict of interest to declare.

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Pae Ora (Disestablishment of Māori Health Authority) Amendment Act 2024: further Crown breaches of Te Tiriti o Waitangi

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ABSTRACT

The Waitangi Tribunal[1] in their Wai 2575 Report recommended the establishment of Te Aka Whai Ora (the Māori Health Authority) to remedy some of the contemporary breaches of Te Tiriti o Waitangi (Te Tiriti). Te Aka Whai Ora was the culmination of decades of Māori advocacy for the establishment of independent Māori health leadership, policymaking and commissioning.

Under urgency, the new National-led coalition Government passed the *Pae Ora (Disestablishment of Māori Health Authority) Amendment Act 2024* in February.

In this paper we use Critical Tiriti Analysis (CTA), a five-stage process, to review the extent to which the *Act* is compliant with the five elements of Te Tiriti (the authoritative Māori text), the preamble, the three written articles and the oral article.

We found that the *Act* had very limited Tiriti compliance and the potential to do great harm. We offered practical suggestions how this could have been avoided.

A major driver of recent health reforms[2] was the desire to reduce systemic ethnic health inequities and uphold Te Tiriti o Waitangi (Te Tiriti). Ethnic health inequities are longstanding within Aotearoa,[3] driven by uneven access to the determinants of health, intergenerational legacies of colonial trauma and institutional racism, which manifest in part as the quality and accessibility of healthcare and in health outcomes. The evidence is clear that culture and racism are key determinants of health.[4]

The Wai 2575 Waitangi Tribunal[1] enquiry, which occurred simultaneously with the 2020 health system review, examined allegations of breaches of Te Tiriti and the Treaty of Waitangi (English text) within the health system. The report recommended that the legislative and policy framework recognise and provide for "the Treaty of Waitangi and its principles". The Tribunal recommended stronger accountability mechanisms and processes for the manifest health policy failures since the signing of Te Tiriti, and in particular the two decades past, that there be a stand-alone commitment to achieving health equity and compensation for the historic underfunding of Hauora Māori. Likewise, co-governance in service design and delivery were seen as being essential to upholding respectful Tiriti relationships. The establishment of the Māori Health Authority (MHA) was fundamental to the Wai 2575 recommendations.

The urgent need to address long-standing inequities was recognised by previous governments, including the previous National Government, whose ministers Coleman and Goldsmith developed and signed off the *New Zealand Health Research Strategy*.[5] This strategy calls for partnerships with Māori and an assurance that the Treaty principles are part of all health research. The *Strategy* also calls for the promotion of rangatiratanga that enables whānau, hapū, iwi and Māori individuals to exercise control over their health and wellbeing.

Further reforms were enabled through the *Pae Ora (Healthy Futures) Act 2022*, which set up the structural components of the new health system, including Te Aka Whai Ora. Rae et al.[6] in their Critical Tiriti Analysis (CTA) of the *Pae Ora* Bill raised concerns about the legislation's lack of compliance with Te Tiriti, concerns that were amplified in our CTA of *Te Pae Tata Interim New Zealand Health Plan*.[7] We noted the problematic use of "treaty principles" rather than the authoritative Māori text and the failure to recognise that Māori never ceded sovereignty.

Te Aka Whai Ora was the pounamu within *Pae Ora*, enabling development of research and

health services that encapsulated the key principles of the *New Zealand Health Research Strategy*. Along with community engagement, developing community leadership and workforce capacity-building, Te Aka Whai Ora was addressing disparities and contributing to enhanced health and wellbeing. Extensive consultation with community researchers strengthened the level of support from Māori communities and held significant potential for improved Māori health outcomes.

Methodology

CTA, a methodology initially designed to monitor the Crown and inform policy writing,[8–9] is a collaborative way of assessing alignments of policy, strategy or plans to five elements of Te Tiriti —the preamble, three written articles and the oral article. CTA involves a five-stage process of i) orientation—a high level read of the document, ii) close reading—looking at content in relation to the five elements of Te Tiriti, iii) determination—applying indicators (see Table 1), iv) strengthening practice—ideas for improvements, and v) a Māori final overall word about how the document aligns with lived experience and lifeways. The authors HC and TM (Pākehā activist scholars) along with NC (Kāi Tahu scholar and CEO) and CA (Ngāti Maru, Ngāti Whanaunga and Ngāti Tamaterā scholar and health research activist) reviewed the *Disestablishment Act* separately and then reached a collective determination.

Findings

Stage one: orientation

The *Disestablishment Act* was introduced to Parliament under urgency on 27 February 2024 and received Royal Assent on 5 March 2024. This *Act* comes into force on 30 June 2024, at which stage Te Aka Whai Ora will cease to exist, with any residual authority/actions or aspirations subsumed elsewhere in the health sector.

The *Disestablishment Act* Part 1 makes amendments to the *Pae Ora Act*, while other parts deal with practical matters (pay, conditions, review requirements), consequential references to the MHA in other legislation. For our current application of CTA to the *Disestablishment Act*, we focus on Part 1, leaving these other components as implied or encompassed within our assessment.

Part 1 of the *Disestablishment Act* lists some 40 amendments or deletions (mainly the acronym MHA), along with a series of changes to specific clauses in *Pae Ora*. In addition, it creates a further 21 significant clauses to *Pae Ora*, inserted into its Schedule 1 as a new Part 2, to substantively change its direction. It is to these substantive changes that the lens of CTA is now applied. This initial reading of the text suggests the *Disestablishment Act* achieves a reversal of an approach to health justice that Māori have (albeit with caveats) defended, supported and collaborated with.

Stage two: close reading

Our close reading of the *Disestablishment Act* is dominated by the point that its sole reference to Te Tiriti is the remnant at Section 6 of the *Pae Ora Act*. Here the rendition is compromised by reference to the "principles of Te Tiriti o Waitangi (the Treaty of Waitangi)", which are non-existent. The problem here is that the *Treaty of Waitangi Act 1975*, through which the principles were legitimated, referenced only the English text, which bears scant relationship to Te Tiriti as the negotiated, signed, authoritative version of the agreement.[10]

As a result, we can see no evidence of the *Disestablishment Act* aligning to the preamble of Te Tiriti, which specifically promises to control the excesses of settler communities in Aotearoa and ensure that tangata whenua cultures and lifeways are guaranteed, supported and thriving. The *Act* ignores these Crown promises and expressions of intent and proceeds as if its only responsibility is to address what are deemed the excesses of the original *Pae Ora Act*.

In terms of kāwanatanga, the *Disestablishment Act* fails in the promise of good governance through its rejection of the provisions that Māori, in fulsome consultation, initiated and agreed to in the *Pae Ora Act*, which the current Government has dishonoured. There is no evidence of Māori community, provider or scholarly engagement with the *Disestablishment Act*, and it ignores Te Tiriti relations in a brazen expression of colonial power.

Further, rather than recognising tino rangatiratanga through embedding it into legislative development, as required by Article 2, the Government exerts the full stretch of its imagined sovereignty to destroy the enactment of even the limited form of mana motuhake envisaged within Te Aka Whai Ora. As a result, and in defiance of the WAI 2575 rulings, there are no mechanisms remaining by which independent Māori aspirations, as embodied in Te Tiriti, might be recognised, or realised. The various advisory groups and boards that persist

through the *Pae Ora Act* are without significant decision-making power and are hampered by the provisions that limit Crown resourcing of their roles.

Under the ōritetanga provisions of the Third Article, equity among the citizens of the country is a right and an aspiration to for a fair and just society, which is unlikely to be achieved within existing colonial structures. The *Disestablishment Act* shifts the dial away from Māori and population health achieved, reflected in the initiation of Te Aka Whai Ora alongside Te Whatu Ora – Health New Zealand. There is certainly no sense in which Māori communities are defining policy intent, as is their civil right and duty, so that as noted above they are not party to the decision to disestablish the strongest contemporary mechanism for achieving health equity.

The *Disestablishment Act* is entirely silent in terms of the Fourth Article of Te Tiriti around wairuatanga, which has come to be included in consideration of the oral promises made in Te Reo Māori at Waitangi and elsewhere, that all faiths of the country would be equally protected. Without acknowledgement of this dimension of holistic health, the *Act* breaches this vital domain of responsibility.

Stage three: determination

This *Act* seems to be firmly oriented to the destruction of tino rangatiratanga embodied in Te Aka Whai Ora, one of the only legislative initiatives within the health sector to give substance to Te Tiriti. Rather, we see the return to the colonial universalist tendencies of health policy found non-compliant with Te Tiriti within the WAI 2575[1] and Haumaru[11] Waitangi Tribunal reports. The continuities with policies that overtly excluded, marginalised and denigrated Māori within settler health systems show that the *Disestablishment Act* is inadequate in terms of Crown responsibilities under Te Tiriti. This move arguably steps health policy backwards into the colonial assumptions that have created and maintained disparities since the records of health outcomes began in the 1950s.

There is little evidence the health practice and leadership expertise within Māoridom has been conserved, except to dismiss it along with the community input and advice that went into the creation of Te Aka Whai Ora. We argue that ignoring such inputs in favour of an ideological position to disestablish Te Aka Whai Ora represents blatant institutional racism as well as a manifest breach of Te Tiriti.

The Fourth Article has rarely been adequately acknowledged by the Crown, although its existence was well documented by observers at Te Tiriti signings. Adherence to this article is of particular importance in the health arena, where Māori beliefs and practices around wairua are of signal importance to hauora in physical, mental and spiritual terms.[12]

The *Disestablishment Act* is not in keeping with tikanga Māori. Tikanga has been well described in research and scholarship. Tikanga has long been considered to have the character and authority of law. Tikanga is enshrined in Te Tiriti, elements of which reside across all the articles. Māori have

Table 1: Critical Tiriti Analysis (CTA) determination of Te Aka Whai Ora *Disestablishment Act* against indicators.

Critical Tiriti Analysis indicators	Silent	Poor	Fair	Good	Excel
Recognition that Te Tiriti is central, and Māori are equal or lead parties, and the legislation preserves Māori interests and contributes to peace and good order.	X				
Mechanisms to ensure Māori engagement and/or leadership in setting priorities, resourcing, implementing and evaluating the legislation.		X			
Evidence of the influence of Māori chiefly authority, values and worldviews.		X			
Māori exercising their rights and privileges of equitable citizenship as Māori.	X				
Recognition of wairuatanga and tikanga in legislation.	X				

always reserved the right to engage as partners with the Crown in a way that recognises tikanga, but at no point in this process has the Crown allowed for a discourse with Māori based on tikanga.

Discussion

Phase four: strengthening practice

A decision as big as the disestablishment of Te Aka Whai Ora required significant discussion with whānau, hapū, iwi and Māori health leaders. A political campaign trail is not respectful engagement with Māori or civil society. The public service is charged with developing policy and legislation and should have led engagement.

Legislative and policy development that is going to serve the public needs to be evidence-based. There is clear evidence in the health sector that "one size fits all" policy and interventions tend to fail to engage Māori.[13] We have a profoundly disappointing history of monocultural health policy that fails to fulfil Te Tiriti responsibilities. It is useful to remember that Te Aka Whai Ora was a response to the failure of monocultural practices and systemic institutional racism within the health sector.

Honourable kāwanatanga is about governance that serves everyone, but also particularly serves Māori as tangata whenua, as the Indigenous peoples of Aotearoa. The *Declaration on the Rights of Indigenous peoples*[14] outlines the unique collective human rights of Indigenous peoples, including the standards of how governments should engage with Indigenous peoples. These collective human rights were breached in relation to the *Disestablishment Act*.

The *Disestablishment Act* is likely to be profoundly damaging to Crown relationships with Māori. It failed to respect Māori tino rangatiratanga, Māori expertise and mātauranga Māori. Te Tiriti o Waitangi granted the Crown the right to govern their (non-Māori) people, which was affirmed in the Wai 1040 hearings. Māori health leaders needed to be at the table and be part of decision-making about the future of Te Aka Whai Ora in order to enact Māori tino rangatiratanga—that right for Māori to make decisions about things Māori.

Health inequities are driven by the legacies of colonisation, institutional racism and uneven access to the determinants of health. The burden of disease, and economic, educational and employment disadvantage is predominately held by Māori. The evidence shows that Māori receive less quality and quantity of healthcare, yet the greatest health need continues to be with Māori in this country. To achieve equity, health policy and legislation needs to embrace what Marmot[15] calls proportional universalism. That is, services need to be provided for everyone, but then targeted based on need.

That means prioritising Māori. That said, it is useful to remember that according to Manatū Hauora – Ministry of Health[16] figures, Māori health providers continue to receive only 1.91% of Vote Health. This does not recognise the burden of disease, recognise the legacies of colonisation and institutional racism, or align well with equity targets, Te Tiriti responsibilities nor international human rights obligations.

Conclusion

Phase five: Māori final word

From its establishment, Te Aka Whai Ora gained significant support from Māori communities across Aotearoa. Their investment in capacity building at local community levels, as well as their commitment to supporting Māori health research based on Te Tiriti, showed potential that could lead to advances in Māori health and equitable outcomes for all.[17] The disestablishment of this innovative strategic approach to improving health outcomes will likely contribute to Māori community disenchantment and further entrenchment of long-standing shameful health and social outcomes.

The health reforms initiated by the *Pae Ora* legislation, with Te Aka Whai Ora at the forefront, were a once in a lifetime opportunity to address Māori health inequities and enhance health outcomes for everyone who calls Aotearoa home. The call for rangatiratanga and kāwanatanga to underpin all aspects of the work of Te Aka Whai Ora was deliberately designed to give effect to ōritetanga. Given that there has been no indication of what will replace Te Aka Whai Ora, the disestablishment of this world-leading initiative is likely to lead to regressive measures and do nothing to improve the health and wellbeing of this country's Indigenous peoples. Moreover, the hasty non-Tiriti compliant measures taken by the coalition Government to disestablish Te Aka Whai Ora are likely to impede efforts to eliminate institutional racism and further disadvantage Māori for generations to come. Almost 200 years after the signing of Te Tiriti o Waitangi, both partners, and the Crown in particular, must commit to the original intentions of the agreement and implement innovative measures, such as a Māori-focussed entity to achieve equitable health outcomes for Māori and ensure social justice for all.

COMPETING INTERESTS
HC has received payment for expert testimony for the Waitangi Tribunal evidence brief.

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A case report of successful dual external defibrillation in cardiac arrest

Anna G Bergin, Chamé C Blackburn, Eric Chong, Ankur Gupta

Sudden cardiac arrest is the most common fatal manifestation of cardiovascular disease and a leading cause of death worldwide.[1] Ventricular fibrillation (VF) is an indication for external defibrillation. When combined with good-quality cardiopulmonary resuscitation (CPR), VF has the highest likelihood of sustained return of spontaneous circulation (ROSC) and neurologically intact survival compared to other rhythms.[2] However, up to 50% of patients do not respond to initial defibrillations and are considered to be in refractory VF (rVF).[3,4] Further defibrillation without modification of defibrillation method is usually unsuccessful and associated with worse outcomes.[4]

There is growing evidence behind dual external defibrillation (DED) as an augmented treatment option for rVF.[5] DED is the use of two defibrillators to deliver two shocks. The second set of pads may be placed adjacent to the first in the anterior-lateral position or they may be placed in an anterior-posterior position. Shocks may be delivered simultaneously or sequentially. Currently, there are no recommendations on the use of DED in the New Zealand Advanced Life Support (ALS) guidelines.

Case report

A 45-year-old 150kg Māori man presented to Whakatāne Emergency Department with chest pain persisting for 50 minutes. Soon after arrival, the patient became unresponsive. Medical staff promptly initiated CPR. Cardiac monitoring showed VF. Defibrillation pads were placed in the anterior-lateral position and the first 200J biphasic defibrillation shock was given. The patient briefly regained responsiveness and was transferred to a resuscitation bay.

In the resuscitation bay, he returned to VF. CPR was resumed. A further four cycles of single external defibrillation were given. Between defibrillations, intravenous adrenaline and amiodarone at standard doses were given to the patient as per ALS guidelines. After five cycles of single external defibrillation, the decision was made to attempt DED. The rationale behind this was despite adequate pad placement, there was concern the patient's large body habitus could be causing high chest wall impedance. Therefore, a second set of defibrillation pads could help overcome this. At this stage, CPR had been ongoing for over 30 minutes and DED as a last-resort therapeutic option felt worth trialling. The second pair of defibrillator pads were placed alongside the first pair in the anterior-lateral configuration with no overlapping of the pads. Each defibrillator was charged to 200J and the lead clinician delivered the shocks simultaneously. The first DED shock was unsuccessful, and CPR was continued for a further 2 minutes. The second DED shock led to sustained ROSC. A 12-lead ECG showed an inferior myocardial infarction. Post-resuscitation care was commenced, and the patient was transferred to a tertiary centre. The patient was later discharged with a good neurologic outcome. Phone consults at 6 and 12 months revealed that he had returned to work and had not suffered any adverse effects from DED in this time.

Discussion

There are several theories behind the effectiveness of DED. The first hypothesis is the use of two sets of defibrillation pads has an additive effect that depolarises a larger mass of myocardium.[6,7] ROSC occurs when a critical mass of myocardium is depolarised to enable the resumption of organised electrical activity.[1] Another theory is that DED increases the number of shock vectors across the myocardium. Defibrillation threshold is the lowest when defibrillation takes place along the longitudinal axis of the cell.[6] The use of two vectors likely exposes many more myocytes to a defibrillation shock along their longitudinal axis and therefore increases the number of myocytes

in which VF is terminated. Lastly, DED may be effective at lowering transthoracic impendence, increasing the density of current at the cardiac surface and thus the chance of successful defibrillation.[8,9]

In a recent trial, DED was compared to vector-change (VC) defibrillation and standard defibrillation in adult patients with rVF during out-of-hospital cardiac arrest.[5] Their technique of DED differed from ours, with rapid sequential shocks rather than simultaneous shocks and the second set of pads placed in the anterior-posterior position. The trial showed that survival to hospital discharge was more common in the DED group (30.4%) and VC group (21.7%) compared to the standard group (13.33%). DED but not VC defibrillation was also associated with a higher percentage of patients having a good neurologic outcome than standard defibrillation. However, consideration should be made that this trial was stopped early due to the COVID-19 pandemic limiting the number of participants. Furthermore, an earlier systematic review found that there were no differences in neurological outcome, survival to hospital discharge, survival to hospital admission, ROSC or termination of rVF between DED and a standard defibrillation strategy.[10]

Here, we report on the first successful case of DED for rVF recorded in New Zealand. With rates of cardiac disease and cardiac arrest increasing, DED may be a promising resuscitation technique to consider. However, more research is required to optimise the technique of DED before it becomes part of ALS guidelines, including identifying optimal pad placement, energy dose and timing of shocks delivered. Practical considerations, such as the availability of two defibrillators and the risk of defibrillator damage, need to be considered in any treatment recommendation.

COMPETING INTERESTS
Nil.

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An act of desperation: self-attempted gender-affirming mastectomy

Mairarangi Haimona, Sue Hui Ong, Scott Diamond

Gender-affirming surgery utilises surgical intervention to affirm a person's identity. In Aotearoa New Zealand, there is an overwhelming unmet need for masculinising chest reconstruction for transgender men. Transgender people, who identify differently to the sex assigned to them at birth, can have significant distress known as gender dysphoria. There are multiple avenues of gender affirmation; however, gender-affirming surgery remains the most challenging to access.[1]

Case report

Patient Z, an 18-year-old female-to-male transgender patient, presented to the emergency department after attempting a partial left mastectomy at home. He had a background of gender dysphoria on testosterone treatment and was awaiting gender-affirmation surgery. However, due to the long wait times of referral in the public healthcare system, an inability to afford a private consultation and the significant psychological stress of having breasts at an upcoming pool party he planned to complete a bilateral self-mastectomy at home. He had demonstrated premeditation by watching a "how to" video on YouTube, preparing appropriate equipment, marking the incision and considering analgesia and haemostasis. He sought medical attention several hours through his self-attempted left mastectomy due to concerns of damaging a nerve. He was reviewed by the acute mental health team and was deemed to have capacity to consent, with no active mood disorder, psychosis or suicidality. After discussing the risks, including irreversibility, poor cosmetic outcome/asymmetry and loss of both nipples, he proceeded to have a completion left mastectomy and symmetrising right mastectomy. The operation was uncomplicated and the patient was discharged home day 1 post-operatively. At clinic 4 weeks post-operatively, his bilateral mastectomy scars had healed well and he reported improvement in self-esteem and self-confidence and his ability to complete school work, and was looking forward to enrolling at university.

Discussion

Gender dysphoria causes significant distress for transgender people.[2] It is well documented that transgender people experience higher levels of discrimination, bullying and violence compared to the general public.[1] Additionally, a study reviewed patients prior to gender-affirming surgery and identified a significant proportion of patients with undiagnosed anxiety and depression.[3] While not all transgender people want gender-affirmation surgery, individuals that do should be able to access this service and express their authentic gender.

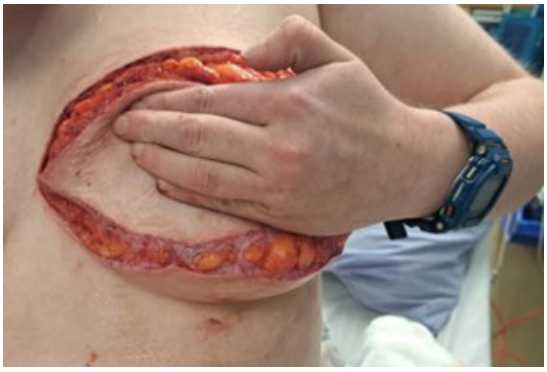
In Aotearoa New Zealand, limited access to gender-affirming surgery due to resources is an increasing issue. Currently, there is only one surgeon performing gender-affirmation operations.[4] Additionally, the publicly funded gender-affirming surgery eligibility assessment is a lengthy process that requires a multidisciplinary approach.[2] Regardless, many patients who meet the eligibility criteria are declined due to the lack of resource and financial capacity, or are wait-listed on a decade-long wait list.[5,6] A recent report estimated that only 13% have had access to this service via the public system.[1] This is a significant under-service and likely an under-representation of the unmet need due to poor documentation and lack of information of how to access services.

Cost is another barrier to this service, with insurance companies excluding gender-affirmation surgery, resulting in patients needing to either self-fund or fundraise.[7] In extreme cases, transgender men may even perform a self-mastectomy. Previous cases of self-amputation of breast tissue have been described in literature. However, these patients demonstrated an active psychiatric disorder and acute triggers that likely initiated self-mutilation.[8,9] Patient Z did not have a psychiatric disorder and had been considering gender-affirming surgery for years. A lack of access to gender-affirming surgery led to this act of desperation.

Transgender people often need to self-advocate for care in the public health system,

but with increasing demand and associated psychological and possible physical harm it is crucial for public services to be more accessible to an under-served population.

Figure 1a and 1b: Left self-attempted mastectomy.



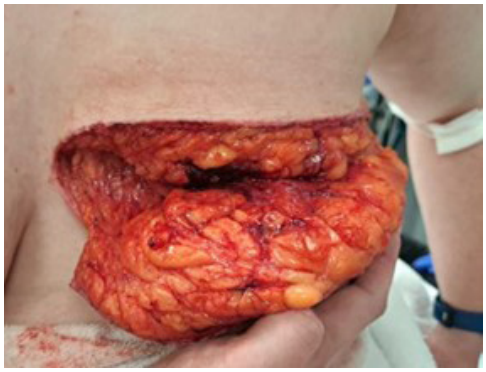
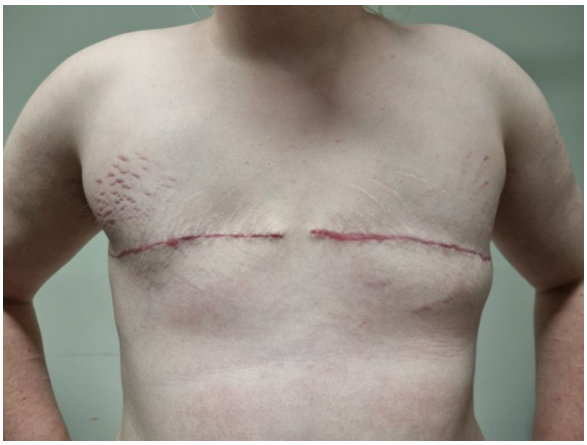
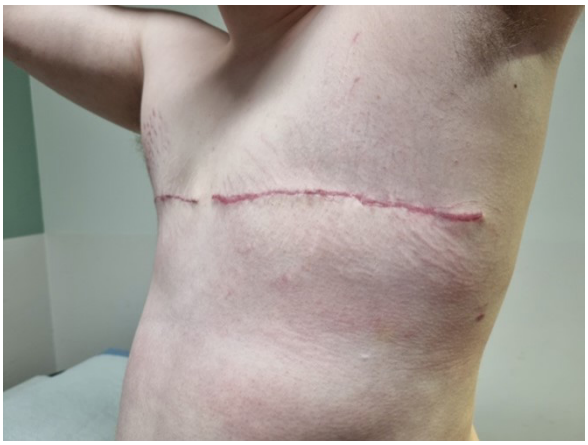


Figure 2a and 2b: Post left completion mastectomy and symmetrising right mastectomy.





COMPETING INTERESTS
None.
Written consent was obtained from the patient in order to publish this case report.

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Management of early dysglycaemia in pregnancy varies by region in Aotearoa New Zealand with risks of widening inequities

Rosemary M Hall, Ruth CE Hughes, Elizabeth Lewis-Hills, Janet A Rowan

A guideline for screening, diagnosis and management of gestational diabetes in Aotearoa New Zealand was published in 2014.[1] The guideline was based on evidence that active management and treatment of gestational diabetes had been demonstrated to reduce adverse pregnancy outcomes.[2,3] The recommendation around early diagnosis of previously unrecognised diabetes and prediabetes was debated, as there were no trial data that identified the glucose threshold at which early treatment improved pregnancy outcomes. The guideline recommended HbA_{1c} to be included routinely with the first antenatal blood tests. Those with an HbA_{1c} ≥50mmol/mol were to be referred for early treatment as having "previously unrecognised diabetes." Those with an HbA_{1c} of 41–49mmol/mol were to be given dietary advice and screened for gestational diabetes with a 75g oral glucose tolerance test (OGTT) at 24–28 weeks gestation.

At the time the national guideline was published, several centres were already treating people with an HbA_{1c} of 41–49mmol/mol from early pregnancy without requiring a second screening test (OGTT). The guideline noted this and stated that centres that were following this practice could continue to do so where resources and capacity were available. Further research was recommended. Subsequent published data support early treatment. People with an early HbA_{1c} of 41–46mmol/mol are at higher risk for pregnancy complications compared with those who have a lower HbA_{1c}[4,5] and early treatment of these people is associated with improved pregnancy outcomes, compared with those managed according to the national guideline.[6,7] Moreover, early treatment for those with an HbA_{1c} >40mmol/mol addresses some inequities in diagnosis of Māori and Pacific people.[8] A randomised controlled trial comparing different approaches was difficult to achieve.[9] Since 2014, more specialist diabetes in pregnancy services around Aotearoa New Zealand have adapted their local referral guidelines to include people with an early pregnancy HbA1c >40 mmol/mol.

We sought to understand what referral criteria were being used in each region, and how they followed the 2014 national guideline. These data will help inform the diabetes in pregnancy guideline currently under review.

Methods

Members of the New Zealand Society for the Study of Diabetes, the leading clinical network of diabetes health professionals in Aotearoa New Zealand, were invited to complete a survey via Survey Monkey.[10] The survey was sent to all 600 members for completeness; however, we knew that many members did not work with pregnant people. Questions were designed to identify those members who cared for pregnant people and who worked within a multidisciplinary specialist diabetes in pregnancy service. Participants were asked to identify their role in the specialist team and which region within the country they worked in, as defined by Health New Zealand – Te Whatu Ora. Participants were then asked about the process within their region for referrals less than 24 weeks gestation and specifically for those with an elevated HbA_{1c}. Responses were grouped by region to identify the pattern of care in each region around the country.

Results

Eighty-two people completed the survey, of whom 71 cared for pregnant people with diabetes. The role of the respondents is presented in Table 1. All regions were represented by the respondents.

In total, 97% of respondents reported that

Table 1: Members of the multidisciplinary team who completed the survey.

Role in the multidisciplinary team	Numbers of responses
Physician	18
Obstetrician	2
Nurse practitioner	5
Diabetes nurse specialist	27
Midwife	3
Dietitian	13
Other	3

Table 2: Criteria for acceptance of referrals to a specialist diabetes in pregnancy clinic after an early HbA_{1c}, by Health New Zealand – Te Whatu Ora region.

Current criteria	Prevalence of diabetes (n/1,000)[g]
Accept referrals with HbA_{1c} 41–49 mmol/mol[a]	
Auckland	50.6
Waikato	45.6
Tairāwhiti	56.2
Taranaki	40.8
MidCentral	40.5
Capital and Coast	37.3
Hawke's Bay	41.4
Wairarapa	32.8
Nelson Marlborough	26.9
Canterbury	26.9
West Coast	30.8
Southern	32.9
Accept referrals with HbA_{1c} 41–49 mmol/mol with risk factors[b]	
Waitematā	42.8
Whanganui	43.8
Hutt Valley	44.3
South Canterbury	34.7

Table 2 (continued): Criteria for acceptance of referrals to a specialist diabetes in pregnancy clinic after an early HbA_{1c}, by Health New Zealand – Te Whatu Ora region.

Accept referrals only with HbA_{1c} ≥50 mmol/mol[c]	
Counties Manukau	73.4
Lakes	43.6
Mixed/other response	
Northland[d]	42.0
Bay of Plenty[e]	32.6

[a] Accept referral, offer nutrition advice, start home blood glucose testing and initiate therapy based on blood glucose levels. No oral glucose tolerance test (OGTT) is required.
[b] Accept the referral with significant other risk factors such as previous gestational diabetes, obesity or strong family history of diabetes.
[c] Decline the referral until they have a diagnosis of gestational diabetes based on OGTT.
[d] Arrange a hospital dietitian appointment and recommend an OGTT at 24–28 weeks gestation.
[e] Refer back to lead maternity carer for lifestyle advice and recommend an OGTT before 24 weeks gestation.
[g] From Health New Zealand – Te Whatu Ora Virtual Diabetes Register.

their region accepts referrals for pregnant people <24 weeks gestation. The criteria under which the referrals are accepted varies by region, as presented in Table 2. Ten of the 20 regions accept referrals to a specialist diabetes in pregnancy clinic when an HbA_{1c} measured <24 weeks gestation is 41–49 mmol/mol. Four regions accept referrals for HbA_{1c} 41–49mmol/mol only when other risk factors are present, and two regions decline a referral until there is a positive OGTT at 24–28 weeks gestation. Four centres reported different referral acceptance criteria from different healthcare professionals within the same clinic. All centres accept referrals when an early Hba_{1c} is ≥50 mmol/mol, as per national guidance.

Discussion

This Aotearoa New Zealand-wide survey of health professionals working within a diabetes in pregnancy specialist clinic identified that early acceptance into a specialist clinic varies considerably by region, despite the presence of a national guideline. Of the 20 Health New Zealand – Te Whatu Ora regions, half accept referrals <24 weeks gestation when an early HbA_{1c} is 41–49 mmol/mol without additional screening, only two regions follow national guidance and the rest accept early referrals if they meet additional screening requirements.

Since the national guideline was published in 2014, publications have reported that an early HbA_{1c} ≥41 mmol/mol identifies those at higher risk,[5,6] and treating this group early is associated with lower rates of pre-eclampsia, pre-term birth and large-for-gestational-age babies[7], particularly for Māori and Pacific people. Beneficial effects of treating gestational diabetes diagnosed in early pregnancy has additionally been observed using other diagnostic criteria.[11,12] The current study shows that a number of centres in Aotearoa New Zealand are now seeing these women early, but this is not uniform, which suggests that the national guideline recommendations may be contributing to inequitable care and outcomes. It is likely that some centres are still only resourced to see women with an early pregnancy HbA_{1c} of 41–49mmol/mol if they subsequently have a diagnostic OGTT at 24–28 weeks.

The Aotearoa New Zealand guideline is currently under review. It is important that the guideline recommendations are fiscally responsible and practically implemented, and consider the impact of any change on resources. Most centres are already seeing these women. The additional resources required for the remaining centres to see these women earlier will depend on the underlying prevalence of women with an early HbA_{1c} in that area. One publication has reported that 1.7% of a population of 5,084 pregnant people had an early HbA_{1c} 41–49mmol/mol,[13] but it is likely

to differ depending on the prevalence of hyperglycaemia in the community, as noted in Table 2. However, to balance this, centres will need to consider potential savings related to lower rates of pregnancy complications in these women if they are treated early.

Strengths of this survey are that representatives from all regions within Aotearoa New Zealand completed the survey, including a diverse range from the multidisciplinary team. A limitation was that we did not specify whether the respondent was the team member primarily responsible for accepting referrals. We assumed that all members of the team would know the regional referral practice, but there may have been different interpretations within the team.

Conclusion

It is essential that the new diabetes in pregnancy guideline reflects the evidence that an early HbA_{1c} ≥41 mmol/mol identifies a group at higher risk for pregnancy complications, which can be reduced by initiating early treatment. Importantly, all pregnant people must have equitable access to the same care across the country.

COMPETING INTERESTS
Nil.

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Note on the Pathology of Goitre in New Zealand

By A. M. Drennan, *from the Department of Pathology, Otago University, Dunedin*

The complete data are not yet available so that this must necessarily be merely a preliminary and general sketch.

In 1917, I was impressed by the number of enlarged thyroids to be seen any day in the street in women, particularly young women. Apparently this was so general that no particular notice was taken of it, even by medical men. On enquiry at the hospital one found that admissions for goitre were common and operations for it frequent. At my request the surgeons kindly sent the thyroids to the laboratory for routine examination and one realised then that, compared with what went through the pathological department of the Edinburgh Royal Infirmary, the proportion of goitres here was much greater than one had been accustomed to at Home.

Certain fundamental questions naturally arose:—(1) Were the goitres here of the same pathological types as those met with elsewhere? (2) What was the age and sex distribution of goitre here? (3) What was the distribution with regard to different areas? (4) What was the iodine content of thyroids here? (5) Did the distribution of iodine in soil bear any relation to the distribution of goitre, and what is the vehicle of iodine to the human organism? (6) Were animals affected? A number of other questions also require an answer, some purely pathological, some etiological, but it is perhaps better to confine attention to the above points first.

At this date (1917), no statistical data were available as to the distribution of goitre, and an attempt was made to get information by making goitre voluntarily notifiable. Unfortunately, from the nature of the condition, the results were disappointing, but they served to show at least that it was a very prevalent condition among adults.

About 1920, *Dr. Hercus* made the first definite move to get data as to goitre among school children in the Canterbury area, and got some very valuable information which has already been recorded in this JOURNAL (April, 1920, p. 116; April, 1923, p. 79; June, 1923, p. 169). In the Otago area, *Dr. Mecredy*, working on the same lines mapped out the distribution along certain valleys (see NEW ZEALAND MEDICAL JOURNAL, August, 1923, p. 236). This work is still in progress.

So far for distribution as to age and area. The cases that come for treatment are, in the great majority of cases adults, but the common history is one of goitre of years standing, and this corresponds usually with the pathological changes. The female sex, in adults, far outnumber the male, in cases getting treatment and therefore in pathological specimens.

In answer to the first general question I can say definitely that goitre as seen here shows the same types as described in other countries. There are hyperplastic types, diffuse or localised; colloid types; so-called adenomatous types, which may be either hyperplastic or colloid—included in this type are the "foetal" adenomas. Malignant goitres are remarkably infrequent, only one occurring in several hundred specimens. Pathologically this is an extremely interesting fact. The thyroid is a constantly-changing organ, and in the goitres one sees evidence of recurring activity over long periods, with regression of some parts and hyperplasia of other parts—apparently all the requirements for cells to run riot and become malignant and yet this seldom occurs. In this respect the thyroid affords a sharp contrast to the breast.

The pathological pictures presented by the numerous specimens are extremely varied. In a few, such as some exophthalmic goitres, there is a frank primary hyperplasia with no pathological evidence of older, pre-existing change. In the majority, including the majority of exophthalmic cases clinically, the pathological changes are those of old standing hyperplasia with secondary change, especially hæmorrhage and subsequent fibrosis and even calcification, together with more recent activity in the form of hyperplastic areas. Any one goitre also may show hyperplastic areas in one part, with resting colloid areas in other parts. It is therefore necessary in examining specimens to take several samples from different parts of the gland.

Toxic symptoms are usually associated with

evidences of activity histologically, but some specimens show only resting colloid thyroid although clinically the cases are "toxic" goitres.

The question of "adenomas" is an interesting one. The naked eye appearance of the "adenoma" is frequently much more striking than the histological appearance. One feels much more inclined to regard the adenomas not as tumours in the strict sense but merely as localised active areas, localised because of being less damaged, or less exhausted, than adjacent areas. It is the same principle that obtains in all pathological processes, if the damage—be it mechanical or otherwise—is localised the damaged part regresses and the undamaged, or less damaged, adjacent parts hypertrophy to compensate. In the case of the thyroid localised areas may be damaged, or exhausted by previous activity, or destroyed by secondary changes, and these either atrophy or lag behind the less stressed areas which respond to the next recurring call for activity by enlarging and proliferating. This very enlarging further embarrasses the less efficient parts and may even crush them out of existence. All these changes can be demonstrated in any series of goitre specimens.

In comparatively few cases, as seen in the pathological laboratory, is there uniform change. As mentioned, a few exophthalmic (hyperplastic) goitres show it, and a few large colloid goitres show it. One case occurs particular in this latter connection. It was that of an elderly woman who had had a large goitre for many years, with pressure symptoms latterly. The goitre was removed and consisted of uniformly large colloid areas lined by flat epithelium. Though many parts were examined all showed the same resting colloid picture—a uniform colloid goitre. As Marine says, every goitre is hyperplastic to begin with. All the varieties are merely variants of the activity or results of previous activity.

Examination of thyroids at post mortem has proved instructive. In many cases, although there is no question of goitre clinically and no obvious enlargement of the thyroid, there are pathological changes similar to those found in the clinical cases of goitre but less in degree.

Proceedings of the Waikato Clinical Campus Research Seminar, Thursday, 28th March 2024

Tōku Ara OraNGA (TAONGA): Kaupapa Māori informed co-design of outpatient care for thyrotoxicosis—stage 2, qualitative review

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Situated within a multi-stage project, stage 2 of TAONGA focusses on a qualitative review of the experience of thyrotoxicosis for Māori. Largely focussing on completing qualitative sessions with whānau and stakeholders—kōrero mai—this stage actively looked at what the thyrotoxicosis journey through the health system looks like for those navigating, their whānau and stakeholders.

Three focus groups with whānau and stakeholders were conducted (whānau recognised as rights-holders) creating a safe space fostering an environment for open lines of communication. Each focus group was presented with compact quantitative data gathered from previous research stages and originating Whakangungu Rākau Study. These data were used to help frame what we currently know about the journey, and provide a basis for the conceptualisation of the journey from the perspective of the participating group. Drawing on Kaupapa Māori methodology, participants were given the space to shape the session so that it was reflective of their reality.

Thought-provoking conversation was observed in the focus groups, in a space that allowed for the recognition, building and maintaining of meaningful relationships. Over the time spent with participants' key hotspots, areas of tension, as well as areas of success, were highlighted, supporting the understanding from quantitative data. The creation of a thyrotoxicosis journey utilising these conversations, and prior data, is crucial to helping the research team garner understanding.

These findings of the focus groups will inform the subsequent stages of TAONGA, and ultimately the production of a new model of care.

Exploring seasonal and geographic variation in rural hospital use and bypass behaviour

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AIM

To improve our understanding of how demand for rural hospital services changes over time. We use routinely collected health data to examine whether rural hospitals have seasonal "surges" in emergency department presentations and hospital discharges, the extent to which these are driven by "non-catchment residents", and the possible reasons why people living in rural areas travel past their local rural hospital to access tertiary hospital services.

METHOD

Two years of linked anonymised health data from the National Minimum Dataset (NMDS) and the Primary Health Organisation (PHO) dataset were used to identify the total number of hospital admissions per month for each rural hospital

examined over a 24-month period. Trends for all hospitals pooled together were examined, including comparing average monthly admissions to determine the presence or absence of any seasonal effect on hospitalisations using several tests for statistical significance.

RESULTS

An upward trend across the study period indicates growing demand for hospital services. While nationally hospitals have a winter peak in admissions around August, rural hospital admissions peaked in January. This appears to be largely driven by visitors to rural regions. A detailed examination of rural Waikato hospitals revealed differences between admission patterns for individual hospitals. Admissions at Taumarunui and Te Kūiti hospitals peaked in winter, while Thames exhibited a summer peak.

Estimating the incidence of dementia in New Zealand using capture-recapture analysis on routinely collected health data

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BACKGROUND

Issues of under-diagnosis and under-coding of dementia in routinely collected health data limit their utility for estimating dementia prevalence and incidence in Aotearoa New Zealand. Capture-recapture techniques can be used to estimate the number of dementia cases missing from health datasets by modelling the relationships and interactions between linked data sources.

METHODS

All incident cases of dementia in the Aotearoa New Zealand 60+ population were identified in three linked national health datasets—interRAI, Public hospital discharges and Pharmacy. Capture-recapture analysis fitted eight log-linear models to the data, with the best-fitting model used to estimate the number of cases missing from all three datasets, and thereby estimate the "true" incidence of dementia. Incidence rates were calculated by 5-year age bands, sex and ethnicity.

FINDINGS

Modelled estimates indicate 36% of incident cases are not present in any of the datasets. Modelled incidence rates in the 60+ age group were 18.0 (95% CI 15.8–21.5)/1,000 per year, with an incident rate ratio of 1.93 (95% CI 1.87–1.98) per 5-year age band. There was no difference in incidence rates between males and females. Incidence rates in Asian ($p<.001$) but not Māori ($p=.974$) or Pacific peoples ($p=.110$) were significantly lower compared to Europeans, even after inclusion of missing cases.

INTERPRETATION

This is the first study to provide estimates of age 60+ dementia incidence in Aotearoa New Zealand and for the four main ethnic groups and suggests over a third of incident dementia cases are undiagnosed. This highlights the need for better access to dementia assessment and diagnosis so that appropriate supports and interventions can be put in place to improve outcomes for people living with dementia and their families.

Change in patients' opinions on participating in a proposed clinical trial before and after cardiac surgery

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BACKGROUND

Clinical trial recruitment can be challenging, especially in trials with restrictive eligibility criteria. For an upcoming pacemaker trial, we analysed patients' perspectives around proposed trial elements before and after cardiac surgery to understand how this affected willingness to participate.

METHODS

Potential patients completed questionnaires before and after cardiac surgery regarding health literacy, pacemaker technology and trial structure. Echocardiography was performed to evaluate its feasibility after cardiac surgery. Time to hospital

and length of inpatient stay were also collected. The primary outcome was willingness to participate in a hypothetical pacemaker trial.

RESULTS

Of 30 patients recruited, 21 wanted to participate pre-operation. Post-operation, seven patients (33%) were no longer willing; their main objection was staying in hospital longer than standard of care. Before surgery, patients were willing to remain 2.7+/-2.0SD days extra, dropping to 1.5+/-0.7SD days after surgery. We found no relationship between this and length of inpatient stay before surgery. Proposing regional hospitals closer to the patients' homes as follow-up sites increased acceptance of follow-up visits from 80% to 100%. Discomfort from ultrasound posed a barrier to participate in one patient. Two patients would not participate because the trial would be first in human. While only three patients would strictly fit the future trial eligibility criteria, we believe our results remain relevant to the proposed trial.

CONCLUSION

While we found most patients willing to trial novel technology, our findings suggest that keeping patients for an extended period (>1.5 days) post-operatively might be challenging. Having follow-up sites closer to patients' homes improved acceptance.

Anal botox injection in the outpatient clinic is an effective alternative to injection under general anaesthesia for chronic anal fissure

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PURPOSE

Chronic anal fissures can be a disabling condition. One established treatment option is botulinum A toxin (botox) injection to the anal sphincter. However, in many centres in New Zealand and Australia, botox injection is performed under general anaesthesia (GA), which results in significant resource consumption. To investigate the efficacy of healing chronic anal fissures with botox, we studied the outcomes of its use in the outpatient clinic (OPC) setting without sedation.

METHODS

Between September 2011 and September 2013, a retrospective audit was carried out at Waikato Hospital, New Zealand. During this time, patients who were diagnosed with a chronic anal fissure underwent an anorectal examination in a general surgery OPC, and botox injection was performed accordingly. The study recorded demographic data as well as any comorbidities that might have affected fissure healing rates. Patients were followed up with a phone call or in-person visit after at least 2 weeks to assess the success of the treatment. Success was defined as patient-reported symptom resolution or improvement to the extent that no further treatment was necessary.

RESULTS

During the study period, a total of 163 patients were treated, out of which 54 were male, and the median age was 40.5 years (with a range of 15–88 years). After one botox injection, 106 patients (65.0%) reported satisfactory symptom resolution at the 2-week or subsequent evaluation. After two or fewer botox injections, 134 patients (82.2%) reported satisfactory symptom resolution. However, 29 patients (17.8%) reported refractory symptoms after two injections, and they were offered a third botox injection in the outpatient clinic or examination under GA.

CONCLUSION

Botox injections can effectively treat chronic anal fissures in an outpatient setting, with similar success rates as injections performed under general anaesthesia. Widespread adoption of this technique could lead to significant cost savings in healthcare resources.

An observational study of patients with anti-glomerular basement membrane disease in the Midland Region

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INTRODUCTION

This subgroup analysis is part of a nation-wide study of anti-glomerular basement membrane (anti-GBM) disease. Data were collected to improve understanding of incidence, baseline characteristics, treatment and outcomes of patients with this rare

but high morbidity and mortality disease.

METHODS

This is a retrospective observational study of all patients in the Midland Region diagnosed with anti-GBM disease diagnosed between January 2009 and December 2020. Patients were identified using ICD codes, biopsy and serology data. Electronic health records and clinical notes were reviewed to collect patient baseline characteristics, treatments and outcomes.

RESULTS

Fourteen patients were included in our study, seven males and seven females. Mean age was 42.7 years old and mean follow-up time was 4.98 years. A high proportion (57.1%) of patients were Māori or Pacific people. A very high proportion (85.7%) of patients were current or ex-smokers. There was a higher incidence in summer months over the course of the study of 6 compared to 2–3 in other months. At presentation the mean creatinine was 501 and of those tested, 84.6% had proteinuria and 100% had haematuria. All patients were treated with PLEX and a form of steroid, with a majority (85.7%) also receiving cyclophosphamide. Ten patients (71.4%) required dialysis following diagnosis and three patients (21.4%) died during the follow-up period. Significant sequelae of the disease and treatment also included six patients admitted with infection, one patient developing cancer, one patient developing type 2 diabetes mellitus and one patient suffering a cardiovascular event.

CONCLUSION

In conclusion our study supports the established data that anti-GBM remains a high morbidity and mortality disease despite appropriate treatment. Our study demonstrated a strong correlation between smoking history and development of anti-GBM disease. Patients with Māori/Pacific ethnicity were over-represented in the affected population, which is a new finding compared to recent national evidence.

The burden of paediatric supracondylar humeral fractures at a level one trauma centre in New Zealand

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AIMS

The aim of this study was to determine the incidence and outcomes of paediatric supracondylar humeral fractures (SCHF) in Te Manawa Taki (TMT)/Midland Region of Aotearoa New Zealand.

METHODS

Prospective data from TMT Trauma Registry were extracted for all paediatric trauma hospitalisations (aged 0–14 years) across an 11-year period from 1 January 2012 to 31 December 2022. Patients were identified using the International Classification of Disease-10 cause codes. Demographic, injury and hospital information were analysed.

RESULTS

Paediatric SCHF (n=1,563) occurred with an incidence of 73.7/100,000 (CI 61.5–85.8). Majority of those hospitalised (62.8%) were aged 5–9 years with an incidence rate of 131.5/100,000 (CI 104.2–158.8). Five- to nine-year-olds were 6.6 and 2.0 times more frequently hospitalised than 10–14- and 0–4-year-olds. The most common place for injury was home (40.9%) followed by school, other institution and public administrative areas (32.5%). Ninety-one point seven percent of injuries occurred by falls with the majority (52.4%) occurring at a height of less than 1 metre.

Majority (76.2%) of patients spent 1 day in hospital and over three quarters had operative management. Estimated hospital costs were $9,842,587 total with an average cost of $6,354 per admission. There was a peak of total hospitalisations from 2016–2019; however, there was no significant difference in annual incidence over time.

CONCLUSION

Paediatric SCHF are a significant issue in Aotearoa New Zealand, particularly in those aged 5–9 years. There is a clear need for focussed interventions in the home and school to minimise falls and reduce the costs of this issue.

Tōku Ara OraNGA (TAONGA): Kaupapa Māori informed co-design of outpatient care for thyrotoxicosis—reimagining dissemination, a student internship

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Research provides important insights to posed questions. Critical to research is dissemination, as recognising how we distribute knowledge holds influence over who has access to it. Looking deeper into the area of knowledge sharing, the TAONGA project, alongside Ngā Pae o te Māramatanga, funded a student to reimagine dissemination processes used in TAONGA employing a Kaupapa Māori methodology.

Although dissemination has always been considered a vital aspect of TAONGA, the processes surrounding it were less structured and formulated. This studentship was tasked with understanding how research findings are generally disseminated back to participants, and how this may look different within the TAONGA research cohort. Under a Kaupapa Māori methodological framework, TAONGA wanted the voices of whānau prioritised in dissemination. Therefore, this project focussed on making contact with whānau and understanding how, what, where and when they would like information presented back. By placing the priority with whānau, TAONGA recognises the expertise whānau hold in their own realities.

Discussions were held with willing whānau regarding dissemination. Key themes from this were using language that is accessible to all, framing discussions with a non-deficit approach and consideration of key audiences to increase translatability. Two outputs were created (a brochure for patients/whānau and poster for key decision-makers), which highlight the key themes of the focus groups in simple terms, with emphasis given to information pertaining to the strength/importance of having whānau involved in the thyrotoxicosis health pathway.

It is expected that this dissemination model and the prioritisation of whānau voices will be carried throughout the remainder of the TAONGA project, helping ground the research team and recentre outcomes for whānau. This studentship highlights the importance of enduring theory and methodology informs all research processes, dissemination.