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Bridging the gap in trauma care across New Zealand

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By committing to data-driven audits and continuous quality improvement, New Zealand can effectively address the existing challenges in trauma care delivery. It is imperative that all residents—regardless of geographical location or ethnic background—receive access to world-class trauma care that meets international standards.

Te ara o Manawataki Fatu Fatu—Kaupapa Māori and Pacific qualitative co-design hui to explore cardiovascular disease care for Māori and Pacific peoples in Aotearoa New Zealand

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Cardiovascular disease continues to disproportionately affect Māori and Pacific peoples in Aotearoa New Zealand. In this study we spoke with 105 patients, whānau and kaimahi (healthcare workers) about their experiences of heart healthcare. Participants described deep barriers caused by racism, cost and access, but also shared clear solutions—care grounded in culture, trust and whanaungatanga. Delivering gold-standard, culturally safe and responsive care that reflects Māori and Pacific values, involves whānau and is delivered safely by people who understand their communities, is crucial to achieving heart health equity for Māori and Pacific peoples.

B4 School Check hearing screening and middle ear disease: a five-year analysis of prevalence and inequity

Thomas Oliver, Alexia Searchfield, Emmanuel Jo, Alehandrea Manuel, Alison Leversha, Suzanne Purdy, Daniel Exeter, Rebecca Garland

Otitis media with effusion, otherwise known as glue ear, is a common childhood condition that can lead to impaired hearing, with flow-on effects for language and social development as well as participation in education. If detected it is often able to be treated with grommets or other interventions. Data from the hearing component of the national health screening programme for four-year-olds (the B4School Check) were analysed to determine if certain groups of children were being missed by the screening programme and whether there were any differences in the rates of glue ear and in referral and access to care providers for further management. Māori and Pacific children and those living in higher deprivation were less likely to be screened, and when screened were significantly more likely to have glue ear. When glue ear was found, children from these groups were less likely to be immediately referred for management and less likely to be enrolled in GP practices to action treatment. These findings will help inform future redevelopment of the screening programme, to enable more equitable access to appropriate care and to improve hearing outcomes for these populations in particular.

Excess cancer incidence and mortality among patients with systemic lupus erythematosus, a population-based study in New Zealand

Chunhuan Lao, Nicola Tugnet, Ross Lawrenson, Douglas White

People with systemic lupus erythematosus (SLE) were about 50% more likely to get cancer compared to the general population. Around one in five of these cancers were haematologic cancers. Those who developed SLE before age 25 had two and a half times the risk of getting cancer compared with others their age. Male patients with SLE were twice as likely to have cancer than others without SLE.

Misclassified latent autoimmune diabetes in adults within Māori and Pacific adults with type 2 diabetes in Aotearoa New Zealand

Zanetta L L Toomata, Megan P Leask, Nicola Dalbeth, Lisa K Stamp, Janak de Zoysa, Tony R Merriman, Phillip Wilcox, Ofa Dewes, Rinki Murphy

Our study looked at whether Māori and Pacific adults in Aotearoa New Zealand who were diagnosed with type 2 diabetes (T2D) actually have a different form of diabetes called latent autoimmune diabetes in adults (LADA). LADA is a slower-developing autoimmune form of diabetes that often appears similar to T2D at first. We tested for antibodies that attack insulin-producing cells and assessed type 1 diabetes genetic risk scores (T1D GRS) to assess the likelihood of T1D. About 5% (14/262) of participants thought to have T2D were found to have LADA. However, their overall clinical characteristics and genetic risk were similar to others with T2D (and without T1D-associated autoantibodies). This study suggests that additional testing such as measuring C-peptide levels, which show how much insulin the body produces, could help clinicians decide who might benefit from earlier insulin treatment.

The incidence, prevalence and treatment of narcolepsy in New Zealand

Nathaniel Hutchinson-Wong, Alister Neill, Angela Campbell

Narcolepsy is a rare but serious sleep disorder where people suddenly fall asleep during the day, often without warning, and struggle with extreme fatigue, hallucinations and even muscle collapse. This New Zealand study is the first to show how often narcolepsy is diagnosed and treated nationwide, revealing big discrepancies in the number of people who are being treated relative to the number of people being formally diagnosed. That gap suggests problems in access to testing and possibly hidden numbers of people suffering without proper recognition. It also hints that another overlooked condition called idiopathic hypersomnolence may be going untreated because it doesn't qualify for funded medication. This research matters because it shines a light on invisible illnesses, challenges in our health system and explores where we can do better for people struggling just to stay awake.

Addressing rural mental health inequities for transgender communities in Aotearoa

Katie E McMenamin, Angie Enoka, Mel Meates

This paper looks at the mental health needs of transgender and gender-diverse people living in rural parts of Aotearoa New Zealand, with a focus on Whanganui. Many participants described struggling with anxiety, depression and suicidal thoughts, made worse by a lack of supportive services, long wait times, and experiences of stigma or discrimination. At the same time, having access to gender-affirming healthcare (for example, correct use of names and pronouns, hormones or surgery), supportive counselling (ideally with a transgender or gender-diverse therapist), Kaupapa Māori support and safe community spaces improved mental wellbeing. The study also found that neurodivergent people (for example, those with ADHD or autism) often faced extra barriers when trying to get help. The findings highlight the need for more accessible, affirming and culturally safe mental health support in rural areas, as well as stronger community-based initiatives to reduce inequities.

In vitro diagnostic devices need a robust regulatory framework

Geoffrey CE Herd, Samarina MA Musaad

Point-of-care testing (POCT) devices are used to obtain rapid medical diagnostic test results near to, or at, the patient's bedside. Examples of POCT devices include: rapid antigen test kits for COVID-19, urine pregnancy test kits and glucose meters for monitoring diabetes. These POCT devices are not regulated in New Zealand and faulty devices have been supplied to public hospitals (and may also be available on the internet). This paper calls for POCT devices to be regulated and checked for accuracy, so that quality assured devices are supplied to hospitals and to the public, in the interests of patient safety.

Non-traumatic rupture of the gluteus medius associated with fluoroquinolone use: a case report

Bernardo Martins Zonta, Júlia Locatteli Bet, Lauro Schweitzer Sebold, Franciani Rodrigues da Rocha, Caroline de Oliveira Fischer-Bacca, Guilherme Valdir Baldo

This report describes a rare case in which a commonly prescribed antibiotic, ciprofloxacin, led to a partial tear of a tendon in the hip called the gluteus medius. The patient developed sudden, severe pain and weakness in the hip about 2 weeks after finishing the antibiotic course. Magnetic resonance imaging scans confirmed the tendon injury, which improved after stopping the medication and starting physiotherapy. Although fluoroquinolones are known to occasionally damage the Achilles tendon, this case shows that other tendons can also be affected. The report aims to raise awareness among both doctors and patients about this uncommon but important side effect.

Pancreatic fallout: autoimmune pancreatitis post-mRNA COVID-19 vaccination

Justin Koh, Owain Blackwood, Bernard McEntee, Michael A Park, Grant Cave, Frank Weilert, Debra A Chalmers, Ariel Drori

Autoimmune pancreatitis is a rare form of inflammation of the pancreas, usually caused by the immune system attacking the organ. This case report describes a healthy 42-year-old woman in New Zealand who developed the condition days after her second Pfizer COVID-19 vaccine. Similar rare cases have been reported worldwide, and clinicians should be alert to this potential association.

A rare case of localised gastrointestinal vasculitis in a New Zealand patient

Josef Templeton, Clare French

This case report details a rare and fatal case of localised gastrointestinal vasculitis (LGVT) in a 51-year-old New Zealand male. The case highlights the challenges in diagnosing LGVT, which is considered an extremely rare and difficult diagnosis to make. Literature shows that LGVT commonly presents with general gastrointestinal symptoms, has some suggestive radiological features and is associated with high morbidity and mortality. Our case adds to the limited collection of literature on LGVT and shows that LGVT is an important diagnosis to consider for those with severe unexplained gastrointestinal symptoms.

Bridging the gap in trauma care across New Zealand

Luke Barker, Ruth Duncan, James McKay, Christopher Wakeman

Trauma or physical injury is a common presentation to New Zealand hospitals and results in many deaths and long-term consequences for those injured. Optimal trauma care can increase survival and improve outcomes. However, trauma care in New Zealand has historically been viewed as the "poor cousin" of healthcare, suffering from inadequate resource allocation and attention. This concern has been consistently raised in the *New Zealand Medical Journal* over the past decade, highlighting the significant disparities in trauma resource provision between the North and South Islands.[1–3]

The North Island boasts established trauma services in Auckland and Waikato, which have been functional for many years. However, even within this region, critical gaps remain. Wellington, recognised as the third-busiest trauma admitting hospital in the North Island, notably lacks a formal trauma admitting service (TAS). Instead, it defaults to intensive care unit resources, leading to potential delays in comprehensive trauma management. The availability and interest in specialised trauma surgeons have also been insufficient, limiting the hospital's ability to provide optimal care. Such systemic weaknesses have implications not only for Wellington but for the entire North Island trauma care landscape.[2]

Trauma verification

The Royal Australasian College of Surgeons (RACS) operates the Trauma Care Verification Program as an independent benchmarking process. This involves a team of multidisciplinary assessors reviewing a hospital's (or network's) trauma service against set international standards, providing a level of care verification (I–IV) or a review as unsuccessful at achieving the verification standard. A review also identifies strengths and weaknesses and recommends actionable improvements. This rigorous process is essential for ensuring high-quality trauma care and is increasingly focussing on entire trauma systems rather than individual centres.[4]

Historically, the North Island has been far more advanced in the RACS Trauma Care Verification Program: Waikato has successfully passed, Auckland is on track, and Central Regions underwent verification in 2024, with Masterton reaching the standard of Level IV (no others achieving this standard). By contrast, no South Island hospitals have undergone verification—a missed opportunity to benchmark services and drive improvement. If Health New Zealand – Te Whatu Ora is serious about ending the postcode lottery, then Christchurch should be expected to meet the same standards as Waikato. In reality, it would currently fail, but such a failure would provide valuable, unbiased identification of service gaps and help guide the allocation of resources to where they are most needed.

Current state of the South Island

Christchurch Hospital, despite being the South Island's busiest trauma facility, faces considerable operational challenges. Its TAS, which is only partially funded and staffed, operates under severe constraints, running from 8 am to 4 pm, Monday to Friday. Within this current roster there is inadequate clinical staffing to remain open consistently, let alone during the hours of peak trauma presentations—evenings and weekends. This pattern not only hampers immediate trauma care but further perpetuates disparities in treatment outcomes across the South Island.[1,5,6]

In 2015 the National Health Board required all district health boards to collect data for the National Minimum Dataset. In mid-2016 Christchurch Hospital established a trauma nurse coordinator (TNC) role to begin collecting data on admitted major trauma patients. The average caseload per TNC in the first few years was one full-time equivalent (FTE) per 190 major trauma patients. Currently, Christchurch Hospital has a TNC caseload of one FTE per 260, well in excess of the median caseload determined by the National Trauma Network (NTN) of 1:75. Christchurch Hospital has seen a significant increase in trauma admissions with no increase in resourcing for many years. When accounting for less severely

injured patients that are admitted to Christchurch Hospital, TNCs are unable to collect any meaningful data or offer any ongoing clinical support as this would more than double their workload. The incidence rate of major trauma by region shows the South Island at 64 per 100,000 people, well above the national average of 51. The Christchurch TAS has no FTE allocated for administration or data management support. When combined with the recent reviews of the TAS that highlighted the alarming lack of medical resourcing, we have a complete picture of a service under critical strain. Despite this, Christchurch Hospital has maintained its improved outcome measures; if we can be resourced out of our "treading water" situation, we can plan to make further improvements.

We now are seeing the fact that we have a service that is funded in a piecemeal fashion, which is, as a result, underperforming. Despite knowing that the Trauma Care Verification Program process would be a very important step forwards, we need to acknowledge the fiscal cost of verification running close to NZ$50,000, as well as the person-time required to prepare for the audit. However, given that the desired outcomes include decreased mortality, shorter hospital stays and less demand on ongoing care services, the cost should be viewed as an investment rather than an expense.

The path forwards

The Christchurch Hospital TAS is functioning on lesser funding than other major trauma centres across Australasia. Although this was previously highlighted in 2017 in the *New Zealand Medical Journal*, there has been no increase in funding for the Christchurch TAS, or any of the South Island trauma services.[3] As a result, we are not reaching the desired patient outcomes. To bridge the gaps in trauma care across New Zealand, several essential steps must be taken:

Standardising trauma admitting practices across tertiary centres, alongside targeted investment in regional resources, is essential for minimising treatment delays and addressing inequities in access to care. A key challenge in New Zealand's trauma care system is the inequitable funding allocation across hospitals and regions, which exacerbates variability in service delivery.[7] Smaller centres should be benchmarked against appropriate trauma service levels and supported by multidisciplinary oversight, with well-defined transfer pathways when higher-level care is required. However, variability in care and resource distribution remains a significant barrier to quality improvement, echoing Edwards Deming's assertion that "*variability is the enemy of quality*".[8] To better understand the long-term effects of trauma care disparities, it is critical that patient-reported outcome measures be collected systematically, as they are currently lacking and hinder the evaluation of outcomes across different regions. Strengthening the responsiveness and cultural competence of emergency medical services, particularly for rural and Māori communities, is another priority for enhancing system performance.[4,5] Additionally, initiating trauma service verification processes in South Island hospitals would provide an objective assessment of existing deficiencies, align services with national standards and inform the strategic prioritisation of future resource allocation. The NTN has an important role to play in addressing these issues, and a concerted push for its leadership could accelerate efforts to reduce disparities and improve overall system performance.

By committing to data-driven audits and continuous quality improvement, New Zealand can effectively address the existing challenges in trauma care delivery. It is imperative that all residents—regardless of geographical location or ethnic background—receive access to world-class trauma care that meets international standards.

In concluding, integrating these suggestions will not only bridge the gaps in trauma care but also foster a more equitable healthcare system for all New Zealanders. Let us aspire to elevate the standard of trauma care throughout the nation, ensuring that every patient receives timely and effective treatment in their moment of need.

COMPETING INTERESTS
Nil.

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Te ara o Manawataki Fatu Fatu—Kaupapa Māori and Pacific qualitative co-design hui to explore cardiovascular disease care for Māori and Pacific peoples in Aotearoa New Zealand

Jamie-Lee Rahiri, Jason Tuhoe, Sandra Hanchard, Alyssa Houma, Noah Appleby, Karen Brewer, Tua Taueetia-Su'a, Taria Tane, Shanthi Ameratunga, Vanessa Selak, Bridget Dicker, Corina Grey, Matire Harwood

ABSTRACT

AIM: Cardiovascular disease (CVD) inequities in Aotearoa New Zealand disproportionately affect Māori and Pacific peoples, who experience higher risk factors, hospitalisations and mortality than NZ Europeans. These disparities stem from the historical and contemporary effects of colonisation, including institutional racism, impacting access to healthcare and socio-economic resources. Despite guidelines for earlier CVD risk assessments (CVDRA), gaps in identification and management persist.

METHOD: The Manawataki Fatu Fatu (MFF) for Māori and Pacific Hearts in Unison for Achieving Cardiovascular Care in Equity Studies (ACCESS) is a Māori and Pacific-led research programme examining CVD inequities in Aotearoa New Zealand. This study presents phase three, focussing on qualitative co-design hui (meetings) across Aotearoa New Zealand to gather insights from Māori and Pacific patients, whānau (family/supports) and kaimahi (healthcare workers) engaged with CVD services spanning primary to secondary care.

RESULTS: A total of 105 participants attended four regional hui focussed on the heart healthcare experiences of Māori and Pacific peoples in Aotearoa New Zealand. Template analysis revealed four key themes for achieving equitable healthcare: the importance of the whānau/community, the need for providers to engage with patients at their level, the persistent barriers faced and a strong commitment to protecting Māori and Pacific communities and kaimahi.

CONCLUSION: This study is a comprehensive qualitative investigation into heart healthcare for Māori and Pacific peoples in Aotearoa New Zealand. The findings reiterate that care must align with the realities of Māori and Pacific peoples and that interventions must address long-standing systemic barriers to care.

Cardiovascular disease (CVD) inequities in Aotearoa New Zealand are evident in the health outcomes of Māori and Pacific peoples who face disproportionately higher rates of CVD risk factors, hospitalisations and mortality compared with New Zealand Europeans.[1,2] Most concerning is the premature onset and mortality from CVD among Māori and Pacific peoples.[3,4] These inequities are not coincidental but are instead rooted in the legacy of colonisation, which continues to be perpetuated through intergenerational institutional racism. This has resulted in systematic inequities across socio-economic determinants of health, including income, housing and education and manifests in reduced access to primary care and guideline-defined medical therapy for Māori and Pacific communities in Aotearoa New Zealand.[5]

Recognising these inequities, the Aotearoa New Zealand CVD primary prevention guidelines recommend earlier CVD risk assessments (CVDRA) for Māori, Pacific and South Asian peoples.[6] However, a recent systematic review highlighted significant gaps in CVDRA and management across all CVD care pathways for Māori and Pacific peoples in Aotearoa New Zealand.[5,7] Opportunities for reducing these gaps include providing adequate CVD literacy, involving whānau, fostering good patient-provider relationships, ensuring access to care and enhancing cultural safety. Therefore,

the findings of this study will inform the creation of an equity roadmap for Māori and Pacific CVD care.[5,8]

The Manawataki Fatu Fatu (MFF) for Māori and Pacific Hearts in Unison for Achieving Cardiovascular Care in Equity Studies (ACCESS) is a critical response to the known gaps in care. Launched in 2020, this Māori and Pacific-led mixed-methods research programme aims to investigate and address CVD inequities in Aotearoa New Zealand.[9] Following previous meetings and research with Māori and Pacific stakeholders, clinicians and researchers, it was cited that an integrated approach, incorporating both qualitative and quantitative methods that interrogate the available data and capture the stories of Māori and Pacific communities, their whānau and the wider community was required.[9] This study explored the experiences of Māori and Pacific peoples with CVD care in Aotearoa New Zealand by gathering insights from patients, their whānau and health workers involved in the complete care continuum, from primary care to secondary care.

Methods

Methodological stance

Kaupapa Māori and Pacific research methodologies informed this study, and the full methodological approach has been described and published.[9] This study was guided by governance and leadership provided by Māori and Samoan principal- and co-investigators, who oversaw a predominantly Māori and Pacific research team.[10–12] A co-design approach fostered community partnerships, aimed at systems-level change and improved equity for Māori and Pacific peoples. He Pikinga Waiora, a co-design framework for health intervention development, implementation and evaluation was utilised.[13] This framework emphasises cultural centeredness, community engagement, integrated knowledge translation, and systems thinking. The Fa'afaletui framework also guided the process, providing decolonising guidelines for reciprocal relationships between researchers and communities.[14] The Auckland Health Research Ethics Committee approved this study on 23 June 2021 (AH22609).

Māori and Pacific scholarship capacity

The MFF programme collaborates with Māori and Pacific researchers across various career stages.[9] Senior Māori and Pacific researchers oversee the programme, while emerging researchers facilitate research hui with community organisations like Hāpai Te Hauora and Moana Connect, conduct data analyses and produce final reports. This integration enhances research experience, mentorship and Māori and Pacific scholarship capacity.

Hui

Four hui facilitated by the research team in partnership with community organisations—Hāpai Te Hauora and Moana Connect—were held.[9] Three in-person hui in Kaitāia, Lower Hutt and South Auckland and one virtual hui (Zoom) (South Island) were held between 12 April and 25 July 2024 (Figure 1). The in-person hui were run as all-day sessions ranging from 4–6 hours and the virtual hui was conducted over 2 hours. All patients and whānau received the final report upon request. A follow-up virtual hui was held to allow participants to provide feedback to the research team (Figure 2). Māori and Pacific qualitative researchers with experience in facilitating Māori and Pacific hui and talanoa, and applying co-design principles, coordinated the hui. Hui schedules were semi-structured and were centred on three key questions:

1. How do optimal heart health services look and feel?
2. How can we improve the patient journey for you and your whānau?
3. What must we do to get from where we are now to where we want to go?

Participants and recruitment

Māori and Pacific patients who experienced CVD care, along with their whānau and kaimahi, were purposefully recruited. Recruitment involved invitations to communities known to the MFF research team and healthcare facilities specialising in CVD care. Purposive sampling ensured diverse representation of age, gender and geographical location across the four regions in Aotearoa New Zealand. A participant information video, produced by Hāpai Te Hauora, was available alongside the information sheet and consent form, featuring captions and audio. Participants received a koha of NZ$200 in the form of petrol or supermarket vouchers.

Data collection

Each hui was audio-recorded, with consent, to capture kōrero accurately. Researchers recorded field notes, and photos were taken with permission from participants to capture discussions (Figure 1). Self-reported demographic information

was also collected, and an artist was present at the South Auckland hui to capture the mood and atmosphere of the event.

Data analysis

Two researchers transcribed hui recordings and analysed the data using NVIVO 14 (Version 14.23.2).[15] They familiarised themselves with the data and created an initial coding template with *a priori* codes before applying it to the remaining transcripts and audio-visual texts.[15] The coding template was regularly revised to combine key themes identified *a priori* with new themes emerging *de novo*.[15] As new data were compared, the framework was refined, incorporating new codes and removing redundant ones. After analysing each of the four hui, a final report was sent to participants for feedback. A combined analysis of all four datasets produced the overall themes of this study.

Results

This study involved 105 participants and identified 18 themes across four hui, which were consolidated into four key themes. Separate reports were created for each region to honour the unique experiences shared and were returned to participants as requested (Figure 3).

1. Our village, our community, our whānau

This theme emphasises Māori and Pacific community values. Participants desired a culturally safe healthcare system with a responsive workforce, community connections and values like whānau, culture and equity.

> *"We want kaimahi with cultural intelligence working for us. Kaimahi who understand the meaning of equity and equity within the community. We want a system and services that do not operate in silos. A system that is connected to primary health, universities, hospitals, iwi, whānau and Pasifika. We want to be connected."* – South Auckland, Kaimahi Tāne 1.

Figure 1: Summary of regional hui and overview of participants.

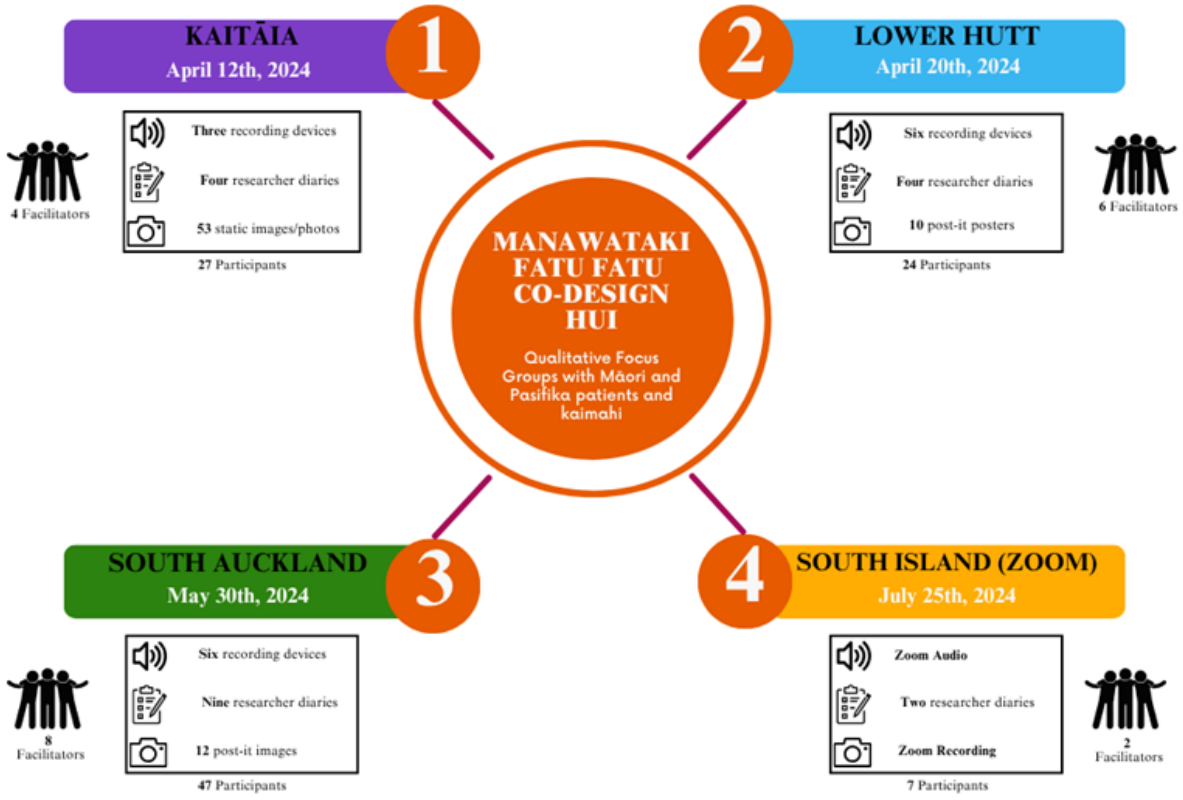
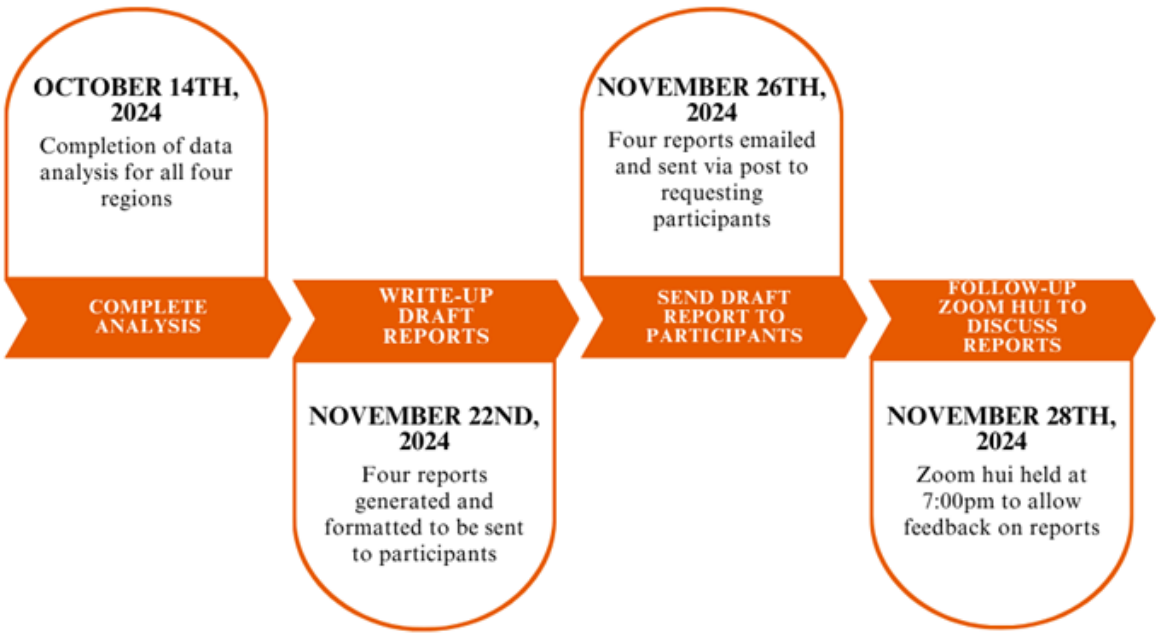


Figure 2: Dissemination strategy.



Pacific whānau emphasised the importance of their "village" amid challenges adapting to Aotearoa New Zealand life from the islands. They reflected that 4th generation Aotearoa New Zealand–born Pacific whānau have experienced changes in their socio-cultural context, yet family and community values persist.

> *"That's an attitude of us* [Pacific peoples], *who have just migrated. We still bring that village attitude here that you know if something goes wrong here, you want to go there. If you need help, that kind of thing. Whereas here* [in Aotearoa New Zealand], *it's different. You mind your own business, and you don't interfere with your next-door neighbour."* – Lower Hutt, Pacific 2.

The intergenerational family structure in Māori and Pacific communities emphasised by whānau and kaimahi highlights how connecting younger generations can support older members. This approach aligns with community cultural values. Whānau shared lessons from their heart health journeys, motivating them to educate themselves and ensure their mokopuna learn to maintain good heart health and avoid similar challenges.

> *"You start thinking about these things, and you start feeling vulnerable as if you never looked after yourself. So I will try to pass that on to my mokos. I sort of missed out with my children because I was too busy working. But my mokos, I try and pass on everything that I know so they don't make the same mistakes that I did."* – Lower Hutt, Kaimahi Tāne 1.

Engaging whānau in all aspects of care—from decision-making to recovery—is essential. Whānau members also reported needing support. Whānau play a vital role as advocates for heart health patients, helping them navigate the healthcare system. Empowering self-advocacy is key to enhancing patient and whānau agency.

> *"We found similar themes of needing in difficult times in the health system to learn how to, for our communities, learn how to be good advocates for themselves and the processes to learn how to become a good advocate."* – South Auckland, Kaimahi Tāne 2.

Participants also asserted that heart healthcare extends beyond the individual patient to encom-

pass the entire family unit, reaffirming that whānau require wrap-around support, especially when caring for a loved one hospitalised with CVD.

> *"We did not get that support, and at that same time, we were trying to look after this baby. So, I felt that the holistic approach to care wasn't taken care of. So whilst my baby was sick, the stress level on me and my husband and my other two children, that we're now part of this heart child, was intense."* – South Island, Pacific Wahine.

The lifestyle aspects of heart healthcare were widely discussed, including healthy homes, a balanced diet, regular exercise and a suitable job. However, these were only raised following extensive talanoa about family, identity, culture and values. Māori participants extensively discussed tino rangatiratanga (self-determination).

> *"Eating healthy and not smoking, vaping, drinking alcohol are all acts of resistance. Those are all acts of tino rangatiratanga. Start with ourselves. Meanwhile, we need all the health support too, but actually teaching our kids… Our Hauora could look like paddling waka ama, touch, kapa haka or could look like a raranga group. If we were doing raranga together or gardening, we could also learn about oranga whānau—all those kinds of things. We have to kill the racism though; it's got to go. It's the systemic stuff."* – Kaitāia, Wahine 5.

The current "system" is considered unacceptable due to recent pressures that are worsening already existing culturally unsafe and racist practices. In this light, rather than waiting for the system to be replaced or changed, whānau Māori described needing to take charge now.

> *"I think the biggest thing is that you have to be your own advocate. You have to take control of your health because really, honestly, nobody else is going to do it for you."* – South Auckland, Wahine Māori 4.

Participants emphasised the need for a culturally safe healthcare system that values community and whānau involvement. They called for a culturally intelligent workforce that respects individual autonomy while meeting community needs. Addressing the unique challenges of Māori and Pacific communities requires intergenerational learning, caregiver support and practical lifestyle interventions. Ultimately, heart healthcare should combine clinical excellence with cultural and familial foundations.

2. Nothing new

Numerous barriers and gaps in heart healthcare were identified, spanning areas such as CVDRA, discharge planning and transitioning from hospital to community care. This theme voices the frustration of patients, whānau and kaimahi about systemic racism and colonial values in healthcare. Shared experiences of culturally unsafe care have deepened mistrust and created barriers to accessing heart healthcare.

> *"Sometimes the interactions with doctors and nurses and other kaimahi are the interface* [through which] *we interact with the health system, and if that interface is not very good and the communication is poor, that really turns people off. And also … we're good at detecting bias and racism. We've all lived that experience, and when we detect it from the other side it undermines our trust in the system."* – South Auckland, Kaimahi Tāne 4.

Upon hearing this feedback, kaimahi were determined to hold themselves and their colleagues accountable. This was enshrouded in the notion that central to Māori and Pacific communities are their whānau, village and community.

> *"We should not, as providers, be hearing that kind of feedback from our community, our people, and it's my responsibility to make sure that I do better."* – Lower Hutt, Kaimahi 1.

Participants emphasised the need for systemic approaches by healthcare providers and policymakers to create an equitable and culturally safe healthcare system for Māori and Pacific communities. They noted that healthcare practitioners are the key link between health services and these communities and again expressed a desire for

more Māori and Pacific kaimahi to provide care.

> *"There's not enough of us in the system. So, whatever we can do to encourage more whānau to be involved in nursing or with doctors. Now, a lot of whānau are becoming doctors, but that was one thing that would, to me, make it better."* – South Island, Tāne Māori.

Discussions on critical shortages in primary healthcare were prevalent in all hui, focussing on cost, travel and access barriers. The inability to enrol with a local general practitioner (GP) was concerning, leaving participants and whānau feeling uneasy about that state of our healthcare system.

> *"Very often they still take up to 2 weeks to get an appointment, and I know when I was at Middlemore, especially during flu season, you just can't get to your GP. So all of these things, time, after hours, you know, it's really hard."* – South Auckland, Pacific Tāne 3.

Issues regarding the postcode lottery and subsequent food/alcohol/vape swamps were raised in all four regions. In Kaitāia, the concentration of vape and liquor stores has been an enduring concern among community members.

> *"I have not counted yet, but there are between nine and 15 vape shops on the main street in Kaitāia … that stuff is literally poisonous, so the companies have moved from tobacco to vaping. They're pushing that new stuff … vapes. We don't even know what's in that, but it won't be anything good."* – Kaitāia, Wahine 7.

Many valuable insights were shared regarding participants' desire for self-determination in their heart healthcare. However, addressing systemic barriers such as food, vape and liquor shop swamps must happen simultaneously. While facing many obstacles, including culturally unsafe or unpleasant interactions with healthcare providers, one participant described their desperation of going through six doctors before being heard.

> *"There is not enough information for new patients going through the heart experience for families to understand what's going on. We have a family of five, plus we have raised other kids, so our home has never been a home. It's always been a marae. So, to school our children up with what their father is going through, we couldn't explain that. 'Cause we didn't even know. We went through six doctors before we were able to find a doctor that would listen."* – Lower Hutt, Whānau Wahine 1.

Māori and Pacific kaimahi described overwhelming experiences of interpersonal and institutional racism coupled with the staggering amount of cultural loading.

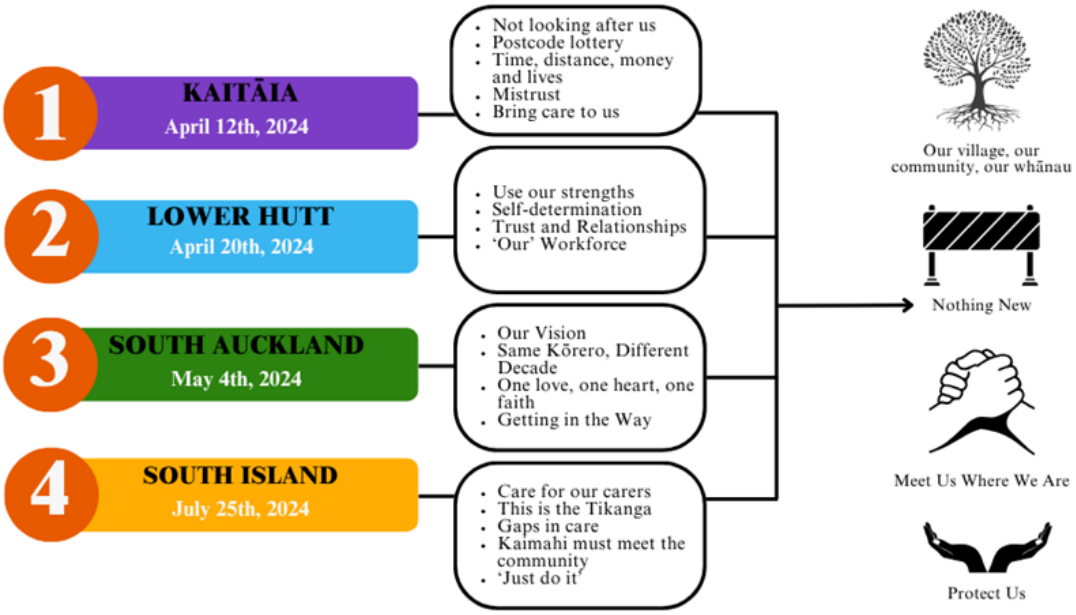
> *"I think the biggest barrier we have here is our own* [Pākehā]*… They are the biggest barrier. We can discuss this from experience. We throw around our overseas-trained* [staff]*, but I have found them to be the most open-minded. Whereas with our own, they're influenced by the racism that sits in this country. So they hold to that."* – South Auckland, Kaimahi Tāne 1.

Many experiences of racism were reported, and these were heightened in services and regions where a clear disconnect between the workforce and the community existed. Kaimahi were determined to push the responsibility of cultural safety training back onto non-Māori and non-Pacific providers as opposed to being further culturally loaded to provide this service in addition to their kaimahi roles.

> *"You know one of the doctors came up to me. She goes, 'we need some more of you* [Māori]*'. I thought to myself, no. You guys just need to get educated to have to look after our people because the education doesn't just come from me."* – South Island, Kaimahi Wahine 1.

This theme highlights ongoing challenges faced by whānau in accessing heart healthcare due to culturally unsafe systems, racism and systemic barriers. It emphasises the struggles of kaimahi who advocate for their whānau, often neglecting their own wellbeing. Participants pointed out the lack of Māori and Pacific healthcare workers and called for better representation and a more responsive system. The theme also outlines

Figure 3: Roadmap of themes drawn from all four hui.



the impacts of these barriers, including loss of lives and resources, compounded by long travel distances and limited cardiac services.

3. Meet us where we are

Participants showed enthusiasm for promoting heart healthcare in their communities. The theme "Meet us where we are" stresses the necessity for healthcare providers to connect with communities culturally. It aims to bridge Western healthcare and Māori and Pacific communities. Building trust between patients and providers involves creating safe spaces that embrace familiar cultural practices.

> *"But we've shared, I think, really in our discussions today, that it is about making sure that our spaces are familiar and are relevant to our people. Make it a space where they can feel comfortable coming in. And it's not just clinical, but we might offer them some kai. We might offer them some really good music."* – Lower Hutt, Kaimahi Tāne 1.

Secondly, bringing care to the community was also essential to bridge gaps.

> *"Having a remote heart health clinic that goes out to marae, goes out to the people. Actually goes out to see them instead of them coming in to see us. Having mobile clinics where we can go out and do echocardiograms, blood tests and electrocardiograms, especially in remote areas … to be able to provide the standard of care that should be given across the board and not just be given in the bigger areas. Earlier support for the heart patient before they have the heart attack and if someone has high blood pressure or angina they get referred instead of waiting until they have a heart attack or have heart failure."* – Kaitāia, Kaimahi 4.

However, having healthcare providers who look like and relate to them made participants feel safer and "like they belonged". As kaimahi, witnessing and being acknowledged by Māori patients significantly fostered a sense of belonging.

> *"I remember this young Māori man from out of nowhere. And when he saw me, it made all the difference when I was there with him because everyone else is not Māori."* – South Auckland, Kaimahi Tāne.

To realise aspirations such as equity, advocacy, Māori, Pacific and whānau-centric care, healthcare providers, leaders and managers must institute

genuine cultural safety training programmes and levers with a specific focus on upskilling non-Māori and non-Pacific healthcare providers. One kaimahi referred to the notion of non-Māori and non-Pacific providers doing some of this "heavy lifting" with respect to creating culturally safe and equity-centred heart healthcare.

> *"Something like changing the governance structure. Maybe that comes down to cultural-safety-training upskilling our non-Māori non-Pacific health workers in terms of cultural safety. You know, there are only so many of us who can do that work. We really need to use those who are not Māori and Pacific, who've got a Māori and Pacific heart."* – South Auckland, Kaimahi Wahine 3.

To ensure care for patients and whānau, it was essential to create a sense of "home". Supporting the healthcare workforce in Kaitāia was a priority, with examples given from other industries attracting Māori to healthcare. Keeping local healthcare providers involved included bonding to retain the local workforce.

> *"We should invest in our own doctors, in our own resources. So, we need the resources in Kaitāia … training our doctors and nurses here in Kaitāia. Invest in our own so that they stay here and they are not allowed to go anywhere!"* – Kaitāia, Tāne 4.

In Lower Hutt, Māori and Pacific kaimahi shared that in their experience, having a fit-for-purpose workforce with predominantly Māori and Pacific kaimahi facilitated stronger connections and acceptability within and by whānau.

> *"The number one thing for us is that we have staff that look like the people we serve. So Māori, Pacific, refugee staff so that when our whānau come to the clinic, they see people that look familiar, and they feel like we are there to help them. The people we employ reflect the community that we serve."* – Lower Hutt, Tāne 8.

Theme three recognised that smoother transitions between care facilities, particularly from hospitals to community settings, are needed. Key elements included medication reconciliation and follow-up. A digital health passport—a personal health record digital application for patients with long-term conditions that tracks and shares care plans—was raised as a potential solution.

> *"So when you get there* [to hospital/clinic]*, you say chur, instead of mucking around the bush. They know you've got a heart problem… Why have a heart attack when you've got to wait in line for nearly 4 hours? You'd be dead by the time you got in there."* – Kaitāia, Tāne 7.

While most participants acknowledged the benefits of digital health and its advancements in heart healthcare, they also reflected on the need to ensure that these advancements did not create further barriers.

> *"… especially with social media. I know with our people, it's a bit hard to access… So you have to look at other ways to reach our community, especially with English as a second language."* – South Island, Kaimahi Pacific Wahine.

In summary, this theme showcased where kaimahi are already making changes to bridge the gaps described earlier in theme two. These include growing the Māori and Pacific health workforce and adopting new approaches to delivering and receiving healthcare with the premise that no one should be left behind.

4. Protect us

Participants advocated for healthcare that was safe, urging kaimahi to protect Māori and Pacific communities from harm. Achieving this necessitates a culturally safe, responsive and compassionate workforce. Building trust and relationships is essential, which can be expedited by providers reflecting the communities whom they serve.

> *"As a provider, you need to tap into that skillset there because they are the springs of that community. They have a relationship with the people that you want to bring into the talanoa… Change the model of connecting to the community."* – Pacific Tāne 7.

There was a strong sense by whānau that having a Māori or Pacific GP meant you were more likely

to get what you needed out of the consult, be it referrals, culturally relevant advice and even straightforward but significant things like having your name pronounced correctly.

> *"Just that little bit of time makes a huge difference to everyone, to predominant culture patients as well as Māori, but especially for Māori and Pacific. We like to have that little moment of mihi before the process, or whatever the treatment might be, begins."* – South Island, Kaimahi Tāne 1.

Whānau Māori suggested improving access to healthcare training placements in Māori communities and increasing early community placements for new doctors. While it is preferable that these placements would target and support Māori and Pacific graduates, it was emphasised that achieving health equity for Māori and Pacific communities is everyone's responsibility, not just Māori and Pacific providers.

> *"Maybe those registrars and house officers could be coming out… Yeah, but I'm just thinking of going to those groups, going to these places, 'cause they are always doing some really good research out there."* – South Auckland, Wahine Māori 2.

Implementing legacy-building processes fosters connections, or whakawhanaungatanga, between healthcare providers and their patients. This strengthens engagement and helps providers understand their communities' needs and rights, while maintaining trust. Established trust enhances collaboration in and support towards research. Participants expressed a desire to conduct their own research to ensure relevance and cultural safety.

> *"We need more Māori and Pacific research that redefines our values for heart health… Māori and Pacific studies going into understanding the heart so we can reference our own, you know when treatment comes in, diagnosis, it's purely from a biobank of just Māori and Pasifika."* – South Auckland, Wahine Māori 2.

This reaffirmed the need to adapt heart healthcare pathways to the lived experiences and socio-cultural contexts of communities. Participants asserted that all healthcare providers must demonstrate compassion, regardless of their background.

> *"She's a European, and she comes into the centre once a week … and it's good because she's in environments with other Pacific Island and Māori and we can see the compassion. I think that's the word that's missing. You can become a professional, but if you're not compassionate, you miss it all."* – South Island, Pacific Tāne 1.

Similar to theme two, this theme also promotes the protection of Māori and Pacific kaimahi. Many kaimahi reported significant cultural loading and stressed the importance of adequate care and support.

> *"We could have a Māori network at* [redacted] *that would be so much stronger. If we call a hui and say we want to have a Māori network, then we can draw on each other and awhi* [support] *each other because we can't do it alone in our little areas because it's too difficult."* – South Auckland, Kaimahi Wahine 4.

Finally, a wero (challenge) was laid before the research team to ensure ongoing research is directly translatable and monitored to see if it makes an actual difference.

> *"So, this particular piece of research is only funded for a specific time. For our whānau sitting here, you know, is it going to make a difference? It's a huge question. Is it going to make a difference for us, for our generation and the generations to come?"* – Kaitāia, Kuia 1.

The research team was encouraged to advocate for action based on our findings, rather than conducting more research that is not translational. Whānau and kaimahi expressed a "no need to apologise, just get it done!" attitude towards implementing change using Māori and Pacific-specific stories.

> *"There is always a reason why it can't happen. You have to stop letting the system or the pathway dictate what*

to do; you just gotta go and do it!" – South Auckland, Kaimahi Tāne 1.

Kaimahi were also challenged by whānau about how healthcare providers and researchers engage with them to find solutions. The "gaze" was turned back on kaimahi to reflect on what needs to be done for Māori and Pacific whānau to be self-determining in their heart healthcare.

"Have you, the providers, ever asked yourselves the question, 'What can we do better' in order for the public to be receptive to what we are about to give?" – Lower Hutt, Pacific Wahine 5.

Others reiterated that the physical components of living, such as having land to belong to and tending gardens for good nutrition, have been hindered by colonisation and that the state of Te Taiao (environment) also affects how well Māori can be. Protecting patients, whānau and kaimahi in heart healthcare requires an empathetic and culturally safe workforce aware of the barriers faced by Māori and Pacific patients. Researchers must engage with these communities and be accountable for their work. Māori and Pacific representation as kaimahi fosters trust in Western healthcare settings. The need for culturally aligned healthcare providers underscores the importance of authenticity and connection with Māori and Pacific communities.

Discussion

Our study includes views from 105 Māori and Pacific patients, families and healthcare workers on heart healthcare. Using a Kaupapa Māori and Pacific approach, we identified four key themes: the importance of whānau/community, the need for providers to understand patients' contexts, ongoing barriers and a commitment to protecting Māori and Pacific communities.

Participants outlined the barriers they experienced in their heart healthcare journeys and a shared vision of a culturally safe, whānau/community-centred and equitable healthcare system that protects them in their pursuit of better heart health. These are not entirely new and exclusive to heart healthcare; however, they are consistent with related studies involving Māori[16–19] and Pacific peoples.[20–21] Overall, whānau proactively developed solutions to address the numerous barriers impeding their heart healthcare journeys. Brewer et al. reported that Māori and Pacific whānau who had experienced cardiovascular care were affected by the social determinants of health and experienced being deprived of accurate information about their condition and its management.[17] Achieving equity in heart healthcare and outcomes requires addressing the contextual factors, including the social determinants of health, fostering effective two-way communication that stems from strong provider-community relationships, and empowering individuals to take control of their heart health.[17,22]

The MFF research initiative addresses colonial aspects of co-design by being led by Māori and Pacific communities, promoting their advancement.[9] King argues that the co-design methodology reinforces whiteness by (re)producing white experts and white ways of knowing.[23] Government organisations have used these methods paternalistically towards Māori, portraying Indigenous peoples as "infantile" and undermining the value of Indigenous knowledge.[23] Utilising Kaupapa Māori and Pacific methodologies minimises the risk of further colonisation through research, while empowering transformative agency and aiming for equal power distribution between patients and providers.[24–25] In our hui, we divided participants into Māori whānau, Pacific whānau and kaimahi groups to present insights, recognising that Indigenous peoples are connected in a holistic network to their history, each other and their environments.[26] These relationships are primarily rooted in whakapapa (genealogy): an extensive and ever-evolving network of connections.[26–27] Additionally, some participants spoke from both provider and patient levels, offering valuable insider-outsider perspectives.[27]

In conclusion, this study is one of the most extensive qualitative studies conducted to date in understanding and improving heart healthcare for Māori and Pacific peoples in Aotearoa New Zealand. By utilising a combined Māori and Pacific methodological approach grounded in the foundational principles of the MFF programme, four regional hui facilitated the successful involvement of 105 Māori and Pacific patients, along with their whānau and kaimahi from different regions in Aotearoa New Zealand.[9] In presenting the study, we acknowledge the limitations in (re)presenting combined views of Māori and Pacific peoples and the potential dilution of unique perspectives shared concerning culture, identity and local

context. However, this study illustrates the strength of Indigenous-led, culturally responsive methodologies in generating robust qualitative evidence and provides a critical platform for informing policy, practice and future research aimed at advancing equity in cardiovascular health.

COMPETING INTERESTS
Nil.
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Appendix: Glossary

Kupu Māori or Pacific	Translation
awhi	Support
kaimahi	Māori or Pacific healthcare workers involved in CVD risk or CVD care for patients and their whānau (i.e., doctors, nurses, social workers, Māori/Pacific health liaison personnel etc.)
karakia whakakapi	Closing prayer
koha	Gift or token of appreciation (in voucher form for this study)
kōrero	Talk, discussion
hui/talanoa	Refers to a meeting and in specifically in this research refers to the four meetings held in the four regions of New Zealand
Māori	Indigenous peoples of New Zealand
mihi whakatau	Informal Māori welcome
mihimihi	Process of greeting/acknowledgement
mokopuna	Grandchild/grandchildren
oranga	To be well/wellness
Pacific	Indigenous peoples of the Pacific Islands (also referred to as Pacific peoples/ Pacific Islanders)
Pākehā	New Zealanders of European descent
paramanawa	Break
poroporoaki	Farewell
pōwhiri	Formal Māori welcome
raranga	Weaving
Te Taiao	Environment/natural world
tino rangatiratanga	Self-determination
waiata	Song
whakawātea	Exit song/item
whakawhanaungatanga	Process of making connections
whānau	Family/extended family or wider support persons including close friends

B4 School Check hearing screening and middle ear disease: a five-year analysis of prevalence and inequity

Thomas Oliver, Alexia Searchfield, Emmanuel Jo, Alehandrea Manuel, Alison Leversha, Suzanne Purdy, Daniel Exeter, Rebecca Garland

ABSTRACT

AIM: The B4 School Check includes hearing screening of four-year-old children in Aotearoa New Zealand. This study describes the prevalence and distribution of hearing loss, likely due to otitis media with effusion (OME), to determine if there is inequity in access to screening and primary healthcare, and to inform programme design and delivery.

METHOD: Hearing data over a five-year period were linked with demographic data and interrogated using regression analyses for differences in disease burden, access to screening and to primary healthcare.

RESULTS: Māori and Pacific children and those living with higher deprivation were less likely to be screened. When screened these children had higher rates of disease, were less likely to be referred immediately and had poorer access to primary healthcare to enable appropriate management.

CONCLUSION: The current delivery of hearing screening is inequitable, missing those that need it most and exacerbating an uneven distribution of disease burden. A redeveloped programme to enable identification and screening of all eligible children, differential delivery according to need and a more holistic provision of care is required. This includes support for speech and language concerns, ear health promotion and linkage with primary care and healthy housing programmes.

The B4 School Check (B4SC) is a national screening programme in Aotearoa New Zealand that identifies health and developmental issues at age four, before children begin school. Eligible children are identified via primary care, school and preschool enrolment data, with families also able to self-refer.[1] The programme is overseen by Health New Zealand – Te Whatu Ora across its regional branches, replacing the former 20 district health boards.

Detection of hearing loss is an integral component of early childhood screening, as timely management of ear disease supports optimal speech, language and learning outcomes.[2] In Aotearoa New Zealand hearing is formally screened at birth and again during the B4SC.[3] While newborn hearing results are routinely reported, data for the four-year screen are not.[4] Screening occurs in early childhood education settings or community clinics, and all children are offered this opportunity except those already managed by audiologists or otolaryngologists.

Children undergo pure-tone audiometry screening in each ear. Failure to respond to any screening tone triggers tympanometry testing, which assesses middle ear status as peaked (normal) or flat (abnormal, otherwise known as type B).[5] Type B tympanograms most often indicate otitis media with effusion (OME), but can also occur in the setting of wax impaction, a perforated drum or the presence of a ventilation tube. OME is a collection of middle ear fluid that may follow respiratory infection and can persist, diminishing hearing in home and classroom environments. Left undetected, chronic OME can delay speech, language and literacy development.[2]

Under B4SC protocols, children with abnormal tympanometry results are either rescreened after 3 months or referred to a primary care provider. This depends on the degree of hearing loss but is also based on the assessment of the technician conducting the screen as to whether the child is at high risk for developmental and learning difficulties. Further interventions beyond referral and their timing are not captured in B4SC data.

International guidelines recommend ventilation tube insertion for OME lasting beyond 3 months, especially if there are developmental concerns.[6,7] In Aotearoa New Zealand, higher rates of persistent middle ear disease are experienced by Māori and Pacific children and those living with higher deprivation.[8–11] Further, there is evidence

to suggest that OME can have a more significant impact on long term outcomes for children living in more deprived areas and that these impacts are not spread evenly across our specific populations.[12,13] These groups also have higher rates of hospitalisation for the related illness acute otitis media, yet have lower and later rates of elective grommet insertion.[14,15]

Access to both screening and follow-up services is not universal. Barriers include distance to clinics, appointment costs and primary health organisation enrolment status.[16,17] Although proposals for more targeted screening exist,[18] there is a lack of up-to-date, nationwide prevalence data for preschool OME which makes the design of such programmes difficult.

By linking national B4SC tympanometry and hearing results with individual level demographic data we can describe the true burden and distribution of OME and identify inequities in access and treatment. These insights can inform future screening strategies, resource allocation and service delivery models to deliver more equitable ear health outcomes for Aotearoa New Zealand children.

Methods

All B4SC individual hearing screening records from 1 January 2018 to 31 December 2022 were extracted, capturing pure-tone audiometry and tympanometry results for each ear (coded in the database as "Pass" or "Fail" for each ear and each metric without further detail), overall screening outcome and whether a child had been rescreened or was a direct referral after their first screen. These records were linked to individual level demographic data: ethnicity (Māori, Pacific, Other), gender and primary health organisation (PHO) enrolment status. We also collated each child's Health New Zealand – Te Whatu Ora region, rurality (as defined by the Health Services Accessibility Index [HAI] score[17]) and data required to grade each individual within the Index of Multiple Deprivation (IMD)[19].

To estimate the cohort either not identified or not successfully engaged by the screening programme, we identified children with any health services user activity recorded by Health New Zealand – Te Whatu Ora in their 4th year (1 January 2018 to 31 December 2021) who lacked a B4SC record, applying the same demographic linkages as above.

All data were de-identified and analysed in SPSS within The University of Auckland's secure data laboratory.

We compared demographic differences among screened versus unscreened cohorts using Chi-squared tests (α=0.05). Among screened children, we calculated the prevalence of flat (type B) tympanograms as a marker for OME. Sub-group Chi-squared tests and multivariable logistic regression models examined associations between flat tympanometry and demographic factors. In the subset with flat tympanograms, we evaluated whether children had been rescreened or were referred directly to primary care after their first encounter with the programme, stratified by ethnicity and deprivation. A final sub-group analysis assessed PHO enrolment disparities among those with flat tympanograms.

Ethics approval was granted by The University of Auckland Health Research Ethics Committee (AHREC ref AH27066).

Results

Screening uptake and tympanometry outcomes

Between 1 January 2018 and 31 December 2022 276,911 children were identified in the B4SC database. Of these, 261,986 (94.6%) underwent hearing screening and 13,507 (5.2% of those screened) had flat (type B) tympanograms. Among those with flat results, 7,774 (57.6%) were referred directly to primary care and 5,394 (39.9%) had undergone rescreening prior to referral; 286 (2.1%) were already under specialist care and 53 (0.3%) lacked outcome data.

Children not screened

We identified 38,035 four-year-olds (2018–2021) with health services user data but who were absent from B4SC records in the year they would have been eligible for screening. Māori (OR 0.58, $p<0.0001$) and Pacific (OR 0.49, $p<0.0001$) children were significantly less likely to be listed in the B4SC database. Higher deprivation was also associated with reduced inclusion: odds of screening declined progressively from IMD decile two through decile 10 relative to decile one (see Figure 1). Multivariable regression analysis confirmed that both ethnicity and IMD decile were independent predictors of unsuccessful identification and engagement by the screening programme.

Prevalence of flat tympanograms

Overall, 5.2% of screened children had flat tympanograms. Prevalence by ethnicity was 6.9% in Māori (OR 1.86, p<0.0001), 10.0% in Pacific (OR 2.79, p<0.0001) and 3.9% in other ethnicities. Analysis of IMD showed no difference across deciles one to five, but a significant increase in flat tympanograms from decile six onwards (see Figure 2). Rurality (HAI decile) had a minimal effect, with no significant difference between different deciles. Multivariable regression identified ethnicity and IMD as independent predictors of flat tympanometry.

Primary health organisation enrolment

Of the children with flat tympanograms, 741 (5.5%) were not enrolled in a PHO. Māori (OR 0.54, p<0.0001) and Pacific (OR 0.51, p<0.0001) children had significantly lower enrolment rates. Children in IMD deciles seven to 10 also showed reduced enrolment compared with decile one (p<0.05).

Rescreening versus referral

Māori (OR 0.87, p=0.0005) and Pacific (OR 0.81, p<0.0001) children with flat tympanograms were more likely to have undergone rescreening than be referred after their first screen (see Figure 3). Higher deprivation was similarly associated with rescreening; HAI showed no effect. Multivariable regression indicated ethnicity and deprivation effects on rescreening rates were not independent variables.

Discussion

There were inequities identified in our dataset in the B4SC screening process, as well as in underlying disease burden and the subsequent opportunity for management. An estimated 38,000 children over the study years 2018–2021 were potentially eligible for screening based on health services user activity but were not in the B4SC database; about half of these were Māori or Pacific children. When screened, these groups had significantly higher rates of type B tympanograms, indicating hearing loss from probable OME, with Māori being one and a half times more likely and Pacific peoples being two and a half times more likely than other ethnicities. These children were more likely to have undergone rescreening prior to referral and less likely to have access to primary care for consideration of intervention.

Increasing deprivation was also independently and strongly correlated with middle ear disease.

Figure 1: Estimate of the total children missed for screening in the B4 School Check by Index of Multiple Deprivation decile and ethnicity (2018–2021).

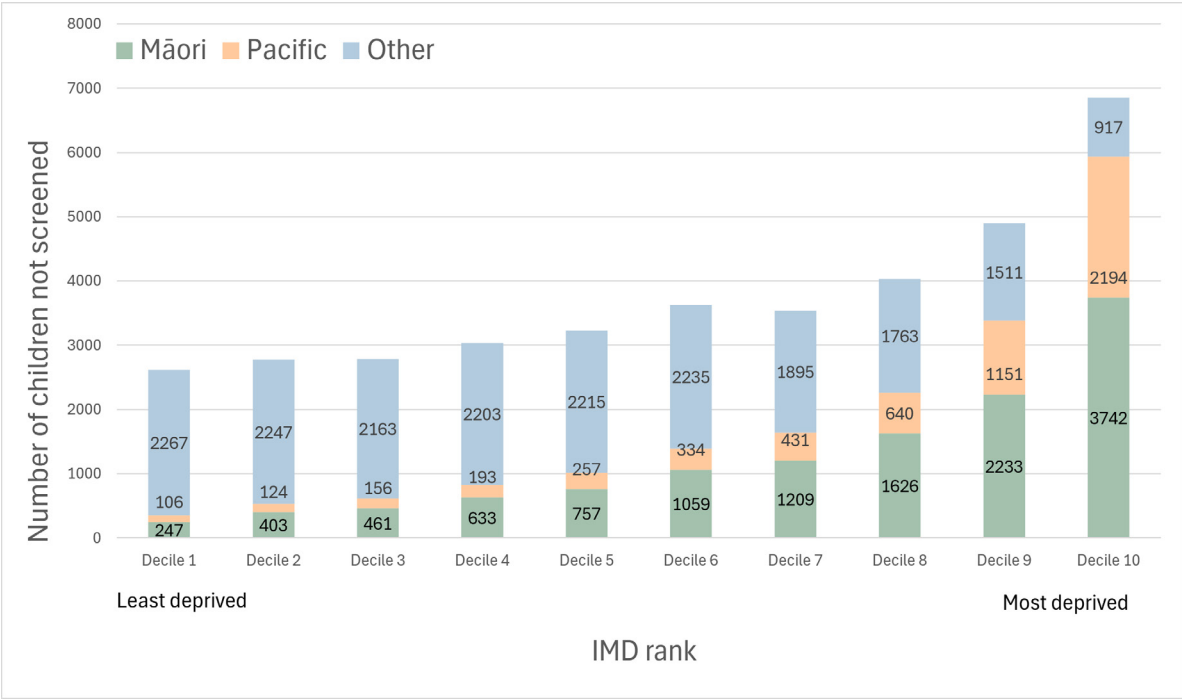


Figure 2: Total children with flat tympanograms in the B4 School Check by Index of Multiple Deprivation decile and ethnicity (2018–2022).

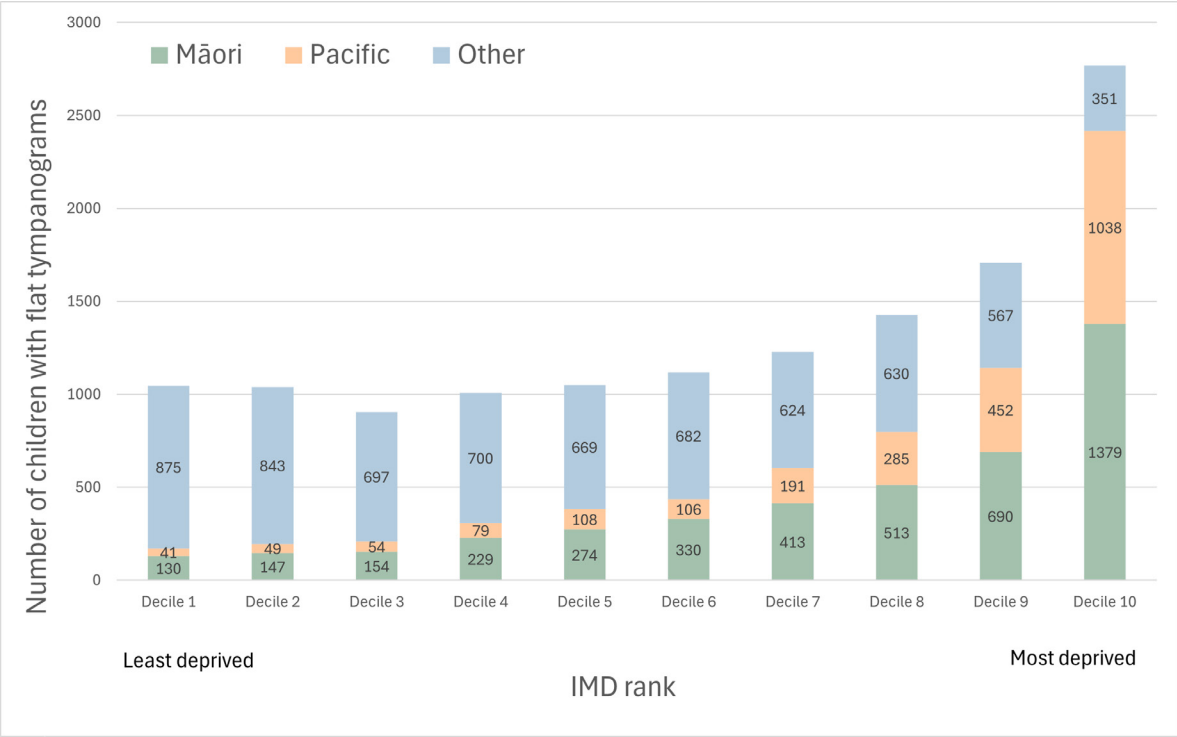
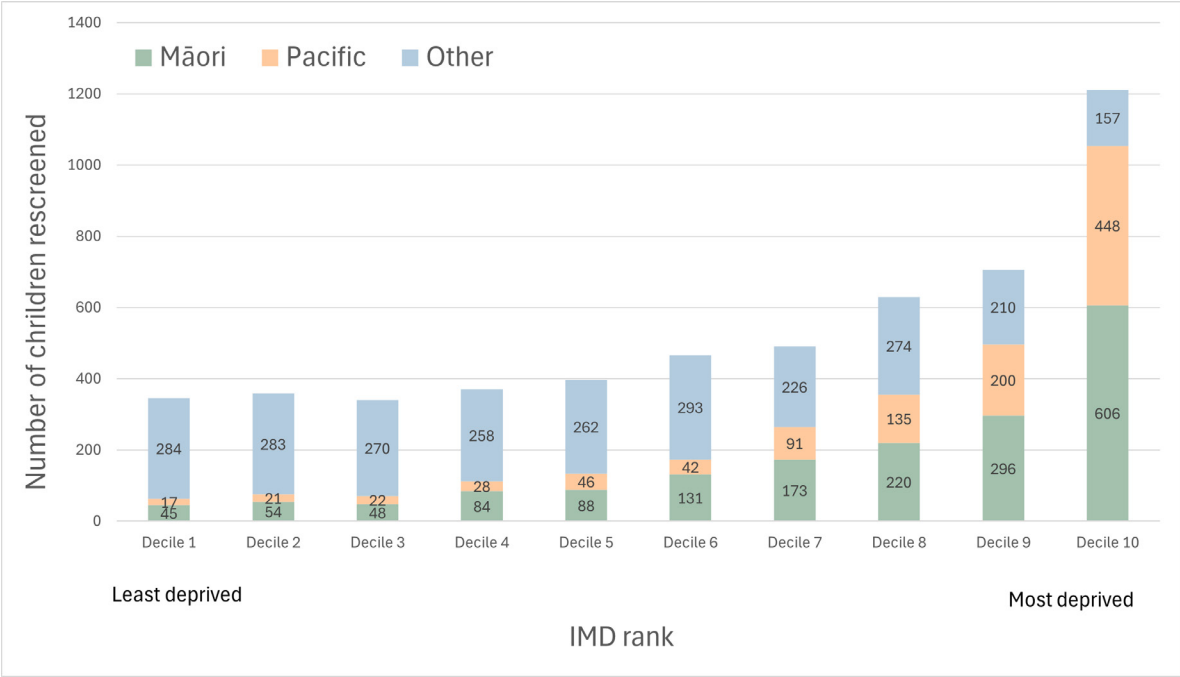


Figure 3: Total children coded as rescreened rather than referred directly with flat tympanograms by Index of Multiple Deprivation and ethnicity (2018–2022).



The cause of this is likely related to higher incidence of respiratory infections, and links have been made with poor housing conditions including dampness, poor heating and crowding.[20]

The higher rates of middle ear disease for Māori and Pacific children and those living in deprivation are broadly consistent with previous studies.[8–11] It is likely that the ongoing detrimental effects of colonisation as well as other social and economic determinants of health have a significant role in these ethnic disparities.[16] These determinants also contribute to healthcare access, affecting not only access to primary healthcare after the screening process, but also the opportunity to participate in the screen in the first place, compounding the inequity. It was interesting to note that ethnicity and deprivation were independent risk factors for OME in our dataset; more research would be useful to confirm this.

Māori and Pacific children are significantly disadvantaged by our health system in the cohort studied for this research, suggesting the screening programme is almost twice as likely to fail to offer hearing testing to these groups. As the B4SC database utilises the primary care enrolment database,[1] those not enrolled will possibly be less likely to be offered screening. The fact that a number of children were offered screening despite this suggests that the other pathways into the programme can be effective. This could be investigated more closely with further research to identify these successes. The health impact for this group of missed children can be inferred from other research including wide ranging short- and long-term effects of OME on child behaviours, learned social responses, language and cognition.[13,21]

A child may either be referred to primary healthcare or rescreened within the programme when found to have a flat tympanogram. Children in areas of higher deprivation and Māori and Pacific children are significantly more likely to be coded as having been rescreened, rather than as referred at first presentation, within the database. The decision to refer or rescreen is based initially on whether the child can cooperate and is developmentally ready for the test. Guidance from the audiometric results then subsequently directs decision making. For children who respond at higher thresholds but not at lower levels, the technicians then consider whether there are any concerns about speech and language development or other developmental difficulties. This is informed by diagnosed comorbidities but also by parental and educator concern and by the technician's impression of the child.

This partially subjective assessment potentially allows the introduction of bias and/or regional differences into decision making in the screening pathway. In many places around the country, significant efforts are being made by the regional screening units to engage with local populations, and children may be offered rescreening intentionally due to lack of access to primary care. Children who do undergo rescreening would potentially have a longer lead time from their first screening assessment to subsequent management of their underlying middle ear disease, which may exacerbate the negative impacts of the condition. If the findings in this study do reflect a systematic bias rather than simply underlying differences in audiometric findings, this may indicate an inequitable service which is prolonging pathways to management in those populations that need it most. Unfortunately, reasons for selecting rescreen or refer decision options after flat tympanometry are not well captured in data reporting and warrant further qualitative research to inform policy guidelines.

The data collected were robust as they are linked to each individual's national health number which does not change regardless of where the child is in the country. By analysing whole population data over a number of years, our findings are likely to be an accurate assessment of the state of middle ear disease in four-year-old children in Aotearoa New Zealand.

There are limitations in our dataset. We do not have the ability to determine why children underwent rescreening. It could be argued that different populations had different audiometric results including variable resolution rates in middle ear effusion between a first and second screening test, leading to a perceived variance in the rates of rescreening. Different practices in different regions may have also led to skewed results or affected the evidence of an inequitable provision of service. Our assessment of the population of children who did not undergo screening at all is an estimate which is probably based on incomplete data. We could not identify children who had no interactions with the health system at all, and transient populations may not have been identified.

Only children who demonstrated evidence of hearing loss proceeded for tympanometry in the screening programme. Type B, or flat tympanograms, were used as a marker for the presence of

OME; however, the results can be confounded by other conditions including wax impaction, canal blockage or eardrum perforation. Although some of these conditions can be differentiated by using tympanometry volume, this data is not input to the national database. This may mean that the overall prevalence of OME is different from what was estimated, although these children still required further assessment or intervention to manage their hearing loss.

Our dataset included the period that the country was under COVID-19 restrictions. This will have undoubtedly affected both access to the screening programme and the prevalence of middle ear disease. Internationally, prevalence of OME (and other non–COVID-19 infections) was lower during the pandemic period, hence these results may underestimate current OME statistics.[22]

OME related hearing loss remains a significantly prevalent health issue in Aotearoa New Zealand children approaching school age, affecting one in 10 Pacific children, one in 14 Māori children and one in 20 children overall. This analysis of Aotearoa New Zealand–specific nationwide whole-population OME prevalence data based on tympanometry screening should inform the ongoing redevelopment of the pre-school screening programmes, as the current screening programme design and implementation has gaps in the provision of equity. Redesign with proportionate universalism is critical, enabling the screening to be delivered in a different way to those that most need it.

Screening programme delivery

More accessible, culturally responsive, targeted programmes have been and are currently being trialled and implemented within select communities around the country.[23–26] These community programmes should be investigated further, and examples that are working well should be embedded in the nationwide delivery model. Ways of seeking enrolment for children currently being missed should be explored.

Screening programme design

Screening programmes need to have clear intervention pathways for children identified with problems. For children who have hearing deficits associated with type B tympanograms, pathways to assessment, referral and intervention should be clear and supported. This should include children who do not currently have access to primary care providers.

We recommend a timely and holistic approach which can be implemented directly from screening. This should include home and community-based speech and language support, online resources for parents and families, listening and communication activities and support for healthy housing and financial welfare.

In addition to data collection, database management and reporting of the B4SC should include type B tympanograms as a marker of middle ear disease in order for this to be monitored and inform future policy.

Alternative workforce models of care, including nurse practitioner community-based clinics, school-based outreach and advanced audiology-led programmes have been demonstrated in Aotearoa New Zealand to improve access for timely investigation and management of childhood ear disease.[25] These models are an example of an effective intervention pathway providing care which might occur after screening. Such programmes could be expanded to other targeted areas of the country and may especially improve access for children who are not enrolled with primary care providers after screening if referral pathways were streamlined.

Public health policy

Middle ear disease, including hearing loss from OME, has received little public health attention this century, with varying policy advice.[27] Calls for improved public health policy and intervention in the 1980s gained some traction with mobile nurse-led ear services such as “ear-vans”. Many of these services have now been eroded due to lack of funding.

Policy design needs to be based on contextually relevant research. The authors disagree with past Aotearoa New Zealand policymakers who have relied on overseas research concluding active intervention for OME has demonstrated little difference in long-term outcomes.[27–29] Much of that research was conducted in contexts that may not be generalisable, often excluding children who are most vulnerable due to deprivation and other socio-cultural factors such as multilingual households. Indeed, more recent local research suggests that standard follow-up models do not adequately manage children with middle ear disease, disproportionately impacting Māori children.[11] Current pathways and priorities have not been adapted for these specific contexts, compounding the impact of hearing loss for these groups.

This study supports other research demonstrating that middle ear disease remains an important cause of childhood hearing loss, especially affecting Māori and Pacific children and those living in deprivation.

We recommend an integrated policy programme with proportionate universalism through the journey from identification to intervention from the range of providers involved. This programme needs to be developed with co-design principles and communities, ideally with cross-sector collaboration such as between the education and health sectors and with support from the ministries of health, education and social development.

COMPETING INTERESTS
Nil.

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Excess cancer incidence and mortality among patients with systemic lupus erythematosus: a population-based study in New Zealand

Chunhuan Lao, Nicola Tugnet, Ross Lawrenson, Douglas White

ABSTRACT

AIM: This study aims to investigate the cancer incidence and mortality among patients with systemic lupus erythematosus (SLE) in New Zealand.

METHODS: SLE patients were linked to the New Zealand Cancer Registry to identify cancer cases. The cancer incidence rate and cancer mortality rate among SLE patients in 2010–2021 were age-standardised to the general population. The estimations were stratified by cancer site, sex, ethnicity and age group.

RESULTS: Among 2,656 SLE patients, 240 new cancer cases were identified, including 187 women and 53 men. Haematologic cancers accounted for 20% of cancer cases identified. The relative risk of cancer incidence for SLE patients compared to the general population was 1.48 (95% confidence interval [CI] 1.28–1.71) for women and 2.08 (95% CI 1.59–2.73) for men. The relative risk of cancer death for SLE patients compared to the general population was 1.75 (95% CI 1.40–2.19) for women and 2.27 (95% CI 1.49–3.44) for men. Younger patients had greater relative risks of cancer incidence and cancer mortality than older patients.

CONCLUSIONS: Patients with SLE in New Zealand experience a higher cancer burden compared to the general population, with greater disparity among younger patients and male patients. Haematologic cancers were especially prevalent among SLE patients.

Systemic lupus erythematosus (SLE) is a multisystem complex autoimmune disorder caused by immuno-dysregulation and presence of autoantibodies. This can result in widespread inflammation and damage to various organs, including the skin, joints, kidneys, heart and lungs.[1] In New Zealand, the age-standardised prevalence rate of SLE is 65.2 per 100,000 for women and 8.5 per 100,000 for men.[2] SLE predominantly affects women, with over 80% of those diagnosed being female. The majority of SLE cases are identified in individuals aged 15–45 years.[2,3] The outcomes of SLE can be highly variable, with the disease manifesting in a spectrum from complete and lasting remission to severe, life-threatening complications. Approximately 30% of SLE patients in New Zealand die of this disease.[4] Overall, patients with SLE experience worse outcomes compared to the general population, with a standardised mortality ratio (SMR) of 4.0 (95% confidence interval [CI] 3.7–4.3) in New Zealand.[4] Additionally, younger patients with SLE have higher SMRs compared with their older counterparts.[4]

Research has shown that patients with SLE have an increased risk of cancer compared to the general population.[5–8] A systematic review of 48 cohort studies involving 247,575 SLE patients found that the relative risk of overall cancer in this group is 1.62 (95% CI 1.47–1.79).[9] It is reported that SLE is a risk factor for 17 site-specific cancers, including six digestive cancers (oesophageal, colon, anal, hepatobiliary, liver and pancreatic), five haematologic cancers (lymphoma, Hodgkin lymphoma, non-Hodgkin lymphoma, leukaemia and multiple myeloma) and cancer in lung, larynx, cervix, vagina/vulva, kidney, bladder, skin and thyroid.[5–9] The underlying reasons for the association between SLE and an increased risk of cancer remain unclear. Several factors may contribute to this relationship, including chronic inflammation, use of certain immunosuppressive medications, genetic factors and the interaction between immune system dysregulation and cancer biology.[5–9] The impact of immunosuppressive medications on cancer development in patients with SLE remains controversial.[8,9] However, some research indicates that SLE patients may have a reduced risk of certain hormone-sensitive cancers, including breast, ovarian and endometrial cancers,[8,9] and antimalarial drugs might be protective

factors for cancer in SLE.[10]

SLE patients not only face higher cancer incidence rates but also experience increased cancer mortality rates compared to the general population. The relative risk of cancer-related death in SLE patients compared to the general population is 1.52 (95% CI 1.36–1.70).[9] In New Zealand, cancer represents the third most frequent cause of death among SLE patients, contributing to 19% of all deaths in this group.[4] This highlights the significant impact of cancer on the survival of SLE patients and underscores the need for targeted cancer screening and preventive measures within this vulnerable population. The incidence and mortality rates of cancer among patients with SLE have not yet been studied in New Zealand. This study represents the first effort to address this knowledge gap. It aims to investigate cancer incidence and mortality among SLE patients within New Zealand.

Methods

Study design, participants and data sources

This is a retrospective cohort study, including prevalent SLE patients in 2010–2021 in New Zealand. These patients were identified by searching the National Minimum Dataset (NMDS) and the Mortality Collection using the International Statistical Classification of Diseases 10th revision (ICD-10) code "M32" and by searching death certificates with the keyword "systemic lupus erythematosus". More details on patient selection and verification have been reported previously.[4] The NMDS is a national database that compiles discharge information from both public and private hospitals, including coded clinical data for both same-day and multi-day inpatient stays. The Mortality Collection contains coded mortality data (date and cause of death), while death certificates record the latest uncoded mortality information. The date of initial SLE identification was determined as the earliest occurrence of an inpatient event with the ICD-10 code "M32" in the NMDS, or the first outpatient event recorded in the National Non-Admitted Patient Collection (NNAPC) within a rheumatology department or renal service. NNAPC includes event-based purchase units associated with medical and surgical procedures in outpatient settings, as well as emergency department events. SLE cases were then linked to the New Zealand Cancer Registry (NZCR) to identify when and what cancer was diagnosed, using patients' National Health Index (NHI) numbers. The NHI number is a unique identifier for individuals accessing health and disability services in New Zealand. The NZCR is a population-based register of all primary malignant diseases diagnosed in New Zealand.

Statistical analyses

The characteristics, including cancer site, ethnicity (Māori and non-Māori) and age at cancer diagnosis (0–24, 25–44, 45–64, 65–74 and 75+ years), of SLE patients who were diagnosed with cancer in 2010–2021 were described by sex (women and men). In New Zealand, sex and ethnicity data in national administrative datasets are self-identified by patients when they engage with health services. The cancer incidence rate among patients with SLE was stratified by sex and age-standardised to the general population in New Zealand as of the year 2019. This was because detailed cancer incidence data categorised by cancer site and age group for the general population were only available for 2019 for comparisons with the age-standardised cancer incidence rate among SLE patients, and such data were not updated beyond this year.[11] The age-standardised registration rates of cancer have been stable in New Zealand, so it was reasonable to use the 2019 data for comparison. The relative risk of developing cancer among SLE patients compared to the general population was also computed. This measure indicates how much more likely SLE patients are to develop cancer relative to the general population. Along with the relative risk, a 95% CI was calculated for each cancer site, age group and ethnicity.

The Mortality Collection provides the underlying cause of death, which is indicated and coded using ICD-10 codes. However, this underlying cause is not explicitly stated on the death certificates, which list up to seven causes of death. To determine the primary cause of death, the causes listed on the death certificates were examined by the authors to identify the underlying cause. The underlying cause of death is defined by the World Health Organization as "*(a) the disease or injury which initiated the train of events leading directly to death, or (b) the circumstances of the accident or violence which produced the fatal injury*."[12] The underlying causes of death were classified into two groups: cancer deaths and non-cancer deaths. Characteristics of patients who died from cancer were described by sex. The overall cancer death rate among SLE patients, stratified by sex and ethnicity,

was age-standardised to the general population based on the 2019 cancer mortality data. The relative risk of cancer death for SLE patients compared to the general population was calculated, with adjustments made for age and ethnicity. The data analyses were conducted using R 4.0 (R Institute, Vienna, Austria). Approval for the study's ethics was obtained from the Northern B Health and Disability Ethics Committee, with the reference number 2022 EXP 13741.

Results

Patient characteristics

During the study period, 2,656 prevalent SLE patients were identified, including 2,305 women and 353 men. The total person-years of follow-up were 20,591 for women and 2,721 for men (Table 1). There were 187 new cancer cases identified among women and 53 among men. The most common cancer diagnosed among SLE patients was non-Hodgkin lymphoma (33, 13.8%), followed by breast cancer (28, 11.7%), lung cancer (25, 10.4%), colorectal cancer (24, 10.0%) and melanoma (19, 7.9%) (Table 2). In total, haematologic cancer (including leukaemia, lymphomas, multiple myeloma and malignant plasma cell neoplasms, other specified and unspecified malignant neoplasms of lymphoid, haematopoietic and related tissue) accounted for 20% of cancer cases identified (38 cases among women and 10 cases among men). Fifteen percent of the cancers were identified among Māori patients, and 85.0% were found among non-Māori patients. Half of the cancers were diagnosed at under 65 years of age, including 10.4% at the age of 25–44 years and 40.8% at the age of 45–64 years. Women were diagnosed with cancer at a younger age than men. Specifically, 12.3% of cancers in women were diagnosed between the ages of 25 and 44, while 18.7% were diagnosed at age 75 or older. In contrast, for men, these figures were 3.8% and 32.1%, respectively.

Cancer incidence

The age-standardised cancer incidence rate was 797 per 100,000 person-years for patients with SLE, compared to 541 for the general population in 2019 (Table 3). Male patients with SLE (1,209 per 100,000 person-years) had a higher cancer incidence rate than female patients (742 per 100,000 person-years), while there was no substantial difference between sex in the general population. Overall, the relative risk of cancer incidence for SLE patients compared to the general population was 1.47 (95% CI 1.30–1.67) for all SLE patients, 1.48 (95% CI 1.28–1.71) for women and 2.08 (95% CI 1.59–2.73) for men. The relative risk was 1.52 (95% CI 1.10–2.11) for Māori and 1.46 (95% CI 1.27–1.68) for non-Māori patients. There were differences in relative risk by cancer sites. For example, the relative risk was 5.41 (95% CI 3.82–7.65) for non-Hodgkin lymphoma, 1.70 (95% CI 1.14–2.52) for lung cancer, 4.55 (95% CI 1.70–12.19) for leukaemia and 3.03 (95% CI 1.43–6.40) for thyroid cancer. Greater disparity in cancer incidence was observed in younger patients, with a relative risk from 2.48 (95% CI 1.67–3.68) for those aged 25–44 years to 1.35 (95% CI 1.03–1.77) for patients aged 75 years or older.

Cancer mortality

In 2010–2021, 100 SLE patients died of cancer, including 78 women and 22 men (Table 4). Six patients died of cancer before the age of 25 years and 31 at the age of 25–44 years. Lung cancer (20.0%) was the most common cancer death, followed by non-Hodgkin lymphoma (17.0%), colorectal cancer (9.0%), breast cancer (7.0%) and leukaemia (6.0%). The relative risk of cancer death for SLE patients compared to the general population was 1.70 (95% CI 1.39–2.07) for all SLE patients, 1.75 (95% CI 1.40–2.19) for women and 2.27 (95% CI 1.49–3.44) for men (Table 5). The difference in cancer deaths between SLE patients and the general population was observed in patients under the age of 75 years with a relative risk from 4.11 (95% CI 1.83–9.24) for those aged 25–44 years to 2.20 (95% CI 1.58–3.07) for patients aged 65–74 years, but not in those aged 75 years or older (relative risk 1.17, 95% CI 0.80–1.69). SLE patients had a much greater risk of dying of haematologic cancer than the general population, with a relative risk of 4.99 (95% CI 3.38–7.37). Non-Hodgkin lymphoma contributed most of the differences, with a relative risk of 8.53 (95% CI 5.24–13.89).

Discussion

This is the first comprehensive study examining the cancer incidence and mortality among patients with SLE in New Zealand. Patients with SLE experience a significantly higher risk of developing cancer and cancer mortality compared to the general population. These findings align with existing research conducted in other countries.[9,13–15] Specifically, the relative risks calculated in this study reveal that patients

with SLE in New Zealand have a cancer incidence risk of 1.47 and a cancer mortality risk of 1.70. Similarly, a Finnish study comparing 1,006 SLE patients and 3,005 population controls estimated an incidence rate ratio for any malignancy of 1.41 (95% CI 1.08–1.85) and an adjusted hazard ratio for death of 1.68 (95% CI 1.17–2.43).[13] These findings underscore the critical need for awareness of cancer risk and enhanced cancer screening to SLE patients, as well as the potential benefits of early detection and tailored treatment strategies to improve survival outcomes in this vulnerable group.

The increased risk of cancer in patients with SLE is influenced by several factors, including chronic inflammation, the use of immunosuppressive medications and genetic predispositions.[5–9] Chronic inflammation can promote tumour growth, while immunosuppressive therapies weaken the immune system's ability to detect and eliminate cancer cells.[5–9] High cumulative cyclophosphamide dose increases the risk of cancer.[16] When using cyclophosphamide, careful monitoring and consideration for alternate therapies are recommended. In contrast, hydroxychloroquine was reported to be a protective factor for cancer in SLE patients.[10,16] It is important for SLE patients to use preventative measures, including smoking cessation, vaccines and regular cancer screening programmes (particularly in cervical cancer).[17,18] Future research should explore treatment regimens for SLE that minimise cancer risk without compromising disease control.

The risk of cancer among patients with SLE varies substantially by cancer type, with certain malignancies showing increased incidence, while others may be less common. It has been found that the risk of non-Hodgkin lymphoma and Hodgkin lymphoma was increased by over threefold, while the risk for myeloma and liver cancer was more than doubled.[19,20] Additionally, the risk for cervical, lung, bladder and thyroid cancers was elevated by at least 1.5-fold, and stomach and brain cancers showed a greater than 1.3-fold increase.[19,20] A Canadian study also reported a four-fold increased risk specifically for non-Hodgkin lymphoma and a three-fold increased risk of haematologic cancers among SLE patients.[14] These are consistent with what we found in our study, which showed that non-Hodgkin lymphoma was the most common cancer diagnosed among SLE patients (33% of all cancers), with a relative risk of 5.41 compared to the general population. Additionally, patients with SLE faced a relative risk of 3.39 for developing haematologic cancers overall. Our study also identified an elevated risk of certain cancer types, such as lung cancer, leukaemia and thyroid cancer. Lung cancer ranked as the third most common cancer and was the leading cause of cancer-related death among SLE patients in New Zealand. SLE patients had a 78% higher risk of dying from lung cancer.[4] These findings are consistent with prior research indicating that autoimmune rheumatologic conditions, such as SLE, rheumatoid arthritis and scleroderma, are associated with an elevated risk of not only developing lung cancer but also dying from it.[14,21]

It has been reported that the cancer-specific standardised mortality ratio was higher in younger SLE patients,[22] a finding consistent with our study. This increased risk suggests that younger SLE patients are more likely to die of cancer compared with their counterparts in the general population. This may be partly because younger patients had a higher relative risk of developing cancer (2.48 for those aged 25–44 years compared with 1.35 for those aged 75+ years in our study). Other contributing factors may include more severe disease manifestations in younger patients, leading to the need for aggressive treatments like immunosuppressive therapies, which can weaken the immune response and elevate cancer risk. While we lacked information on the disease severity of the SLE patients in our study, our prior research indicated that younger patients were over 10 times more likely to have SLE listed as the underlying cause of death.[4] This finding suggests that younger patients may experience more severe forms of the disease.

Our study demonstrated a higher relative risk of both developing and dying from cancer for male patients compared with females. A meta-analysis also showed a slightly higher relative risk for men (1.59, 95% CI 1.18–2.14) than women (1.49, 95% CI 1.15–1.93).[8] However, the gap was much greater in our study, with a relative risk of 2.08 (95% CI 1.59–2.73) for men and 1.48 (95% CI 1.28–1.71) for women. This may be related to the different clinical characteristics between male and female SLE patients, including more renal and haematological involvement in male patients.[23] Male patients were also reported to be diagnosed with more late-onset SLE, which may lead to increased reliance on immunosuppressive therapies that compromise their immune systems.[24]

This study has some strengths. This is the first population-based study examining the cancer

incidence among SLE patients in New Zealand. The linkage of multiple national datasets through the unique NHI numbers allowed for a thorough analysis of cancer incidence and mortality among patients with SLE compared to the general population. The analyses with detailed stratification by cancer site, sex, age group and ethnicity also revealed specific patterns and disparities in cancer risk. Such insights are crucial for developing targeted screening and prevention strategies to improve outcomes for diverse patient populations. Despite the valuable information provided by this study, there are notable limitations that need to be acknowledged. One limitation is the relatively small number of cancer cases available for analysis, especially when data were further stratified by cancer site, sex, age group and ethnicity. This small sample size can lead to greater variability and uncertainty in the cancer incidence/mortality rates and relative risk estimates. Another limitation is the lack of data on smoking, body mass index/obesity, disease activity and medications received by these SLE patients, limiting our ability to adjust for their potential effects. Future research with more comprehensive data is needed to address these factors. We did not have data on cancer screening uptake in our cohort either. Previous research showed less uptake of cancer screening by SLE patients, e.g., not having regular cervical screening etc.[25]

Conclusions

Patients with SLE in New Zealand experience a higher cancer burden compared to the general population, with greater disparity among younger patients and male patients. Haematologic cancers were especially prevalent among SLE patients. It is crucial to stay alert for the possibility of cancer and carefully evaluate any new symptoms that might indicate its presence, even in younger patients. Further research is needed to explore potential associations between SLE treatments and cancer risk.

Table 1: Number of new cancer cases among systemic lupus erythematosus (SLE) patients in 2010–2021.

Year	2010	2011	2012	2013	2014	2015	2016	2017	2018	2019	2020	2021	2010–2021
Women													
Person-years	1,444	1,502	1,560	1,607	1,664	1,708	1,770	1,810	1,835	1,871	1,896	1,924	20,591
New cancer cases	17	15	11	15	19	23	18	25	13	10	13	8	187
New cancer deaths	7	7	7	3	7	12	6	6	10	6	4	3	78
Men													
Person-years	180	193	197	206	218	229	233	240	247	252	260	266	2,721
New cancer cases	6	5	3	4	4	10	5	1	1	6	5	3	53
New cancer deaths	0	3	2	1	1	4	3	1	0	1	4	2	22

Table 2: Characteristics of cancer cases among systemic lupus erythematosus (SLE) patients in 2010–2021.

Sub-group	Women	Men	Total
Cancer site			
Non-Hodgkin lymphoma	27 (14.4%)	6 (11.3%)	33 (13.8%)
Breast	28 (15.0%)	0	28 (11.7%)
Lung	19 (10.2%)	6 (11.3%)	25 (10.4%)
Colorectal	19 (10.2%)	5 (9.4%)	24 (10.0%)
Melanoma	13 (7.0%)	6 (11.3%)	19 (7.9%)
Leukaemia	6 (3.2%)	4 (7.5%)	10 (4.2%)
Kidney	3 (1.6%)	4 (7.5%)	7 (2.9%)
Thyroid	6 (3.2%)	1 (1.9%)	7 (2.9%)
Others	66 (35.3%)	21 (39.6%)	87 (36.3%)
Ethnicity			
Māori	29 (15.5%)	7 (13.2%)	36 (15.0%)
Non-Māori	158 (84.5%)	46 (86.8%)	204(85.0%)
Age at cancer diagnosis			
0–24	0	0	0
25–44	23 (12.3%)	2 (3.8%)	25 (10.4%)
45–64	77 (41.2%)	21 (39.6%)	98 (40.8%)
65–74	52 (27.8%)	13 (24.5%)	65 (27.1%)
75+	35 (18.7%)	17 (32.1%)	52 (21.7%)
Total	**187**	**53**	**240**

Table 3: Cancer incidence rate and relative risk for systemic lupus erythematosus (SLE) patients compared to the general population.

Sub-group	Cancer incidence rate (per 100,000 person-years)						Relative risk (95% CI)		
	General population			SLE patients					
	Women	Men	Total	Women	Men	Total	Women	Men	Total
Cancer site									
Haematologic cancer‡	38	57	48	150	239	161	**3.95 (2.86–5.47)**	**4.16 (2.23–7.75)**	**3.39 (2.54–4.51)**
Non-Hodgkin lymphoma	24	16	20	107	151	109	**4.45 (3.03–6.54)**	**9.31 (4.16–20.85)**	**5.41 (3.82–7.65)**
Breast	140	1	71	104	0	88	0.74 (0.51–1.08)	0.00	1.24 (0.85–1.80)
Colorectal	64	72	68	82	109	84	1.27 (0.81–1.99)	1.52 (0.63–3.67)	1.24 (0.83–1.86)
Lung	48	48	48	74	149	81	1.53 (0.97–2.41)	**3.12 (1.40–6.95)**	**1.70 (1.14–2.52)**
Melanoma	51	61	56	54	132	65	1.06 (0.61–1.83)	2.16 (0.97–4.81)	1.16 (0.74–1.82)
Leukaemia	13	19	16	25	88	36	1.93 (0.86–4.32)	**4.55 (1.70–12.19)**	**2.24 (1.20–4.18)**
Kidney	8	16	12	10	91	21	1.35 (0.43–4.24)	**5.85 (2.18–15.66)**	1.82 (0.86–3.83)
Thyroid	10	4	7	23	23	23	2.24 (1.00–5.02)	5.23 (0.73–37.48)	3.03 (1.43–6.40)

Table 3 (continued): Cancer incidence rate and relative risk for systemic lupus erythematosus (SLE) patients compared to the general population.

Sub-group	Cancer incidence rate (per 100,000 person-years)						Relative risk (95% CI)		
	General population			SLE patients					
	Women	Men	Total	Women	Men	Total	Women	Men	Total
Ethnicity									
Māori	383	344	364	518	920	554	1.35 (0.94–1.95)	**2.67 (1.27–5.61)**	**1.52 (1.10–2.11)**
Non-Māori	525	628	576	783	1,276	842	**1.49 (1.27–1.74)**	**2.03 (1.52–2.71)**	**1.46 (1.27–1.68)**
Age group (years)									
0–24	22	20	21	0	0	0	0.00	0.00	0.00
25–44	164	82	123	308	285	306	**1.88 (1.24–2.84)**	3.46 (0.86–13.88)	**2.48 (1.67–3.68)**
45–64	708	710	709	990	1,955	1,108	**1.40 (1.12–1.75)**	**2.75 (1.79–4.23)**	**1.56 (1.28–1.91)**
65–74	1,331	2,223	1,766	2,266	3,125	2,398	**1.70 (1.29–2.24)**	1.41 (0.82–2.42)	**1.36 (1.06–1.73)**
75+	2,032	3,064	2,485	2,726	6,367	3,353	1.34 (0.96–1.87)	**2.08 (1.29–3.35)**	**1.35 (1.03–1.77)**
Overall	502	581	541	742	1,209	797	**1.48 (1.28–1.71)**	**2.08 (1.59–2.73)**	**1.47 (1.30–1.67)**

‡Including leukaemia, lymphomas, multiple myeloma and malignant plasma cell neoplasms, other specified and unspecified malignant neoplasms of lymphoid, haematopoietic and related tissue.
SLE = systemic lupus erythematosus; 95% CI = 95% confidence interval.
The bolded relative risks indicate statistical significance.

Table 4: Number of cancer deaths by sub-group.

Sub-group	Women	Men	Total
Cancer site			
Lung	15 (19.2%)	5 (22.7%)	20 (20.0%)
Non-Hodgkin lymphoma	15 (19.2%)	2 (9.1%)	17 (17.0%)
Colorectal	8 (10.3%)	1 (4.5%)	9 (9.0%)
Breast	7 (9.0%)	0	7 (7.0%)
Leukaemia	5 (6.4%)	1 (4.5%)	6 (6.0%)
Others	28 (35.9%)	13 (59.1%)	41 (41.0%)
Ethnicity			
Māori	7 (9.0%)	3 (13.6%)	10 (10.0%)
Non-Māori	71 (91.0%)	19 (86.4%)	90 (90.0%)
Age at death			
0–24	0	0	0
25–44	5 (6.4%)	1 (4.5%)	6 (6.0%)
45–64	27 (34.6%)	4 (18.2%)	31 (31.0%)
65–74	26 (33.3%)	9 (40.9%)	35 (35.0%)
75+	20 (25.6%)	8 (36.4%)	28 (28.0%)
Total	**78**	**22**	**100**

Table 5: Cancer death rate and relative risk for systemic lupus erythematosus (SLE) patients compared to the general population.

Sub-group	Cancer death rate (per 100,000 person-years)						Relative risk (95% CI)		
	General population			SLE patients					
	Women	Men	Total	Women	Men	Total	Women	Men	Total
Cancer site									
Haematologic cancer‡	16	21	18	88	108	92	**5.58 (3.60–8.66)**	**5.12 (2.12–12.36)**	**4.99 (3.38–7.37)**
Lung	37	38	38	60	126	67	1.65 (0.99–2.75)	**3.27 (1.36–7.88)**	**1.78 (1.15–2.76)**
Non-Hodgkin lymphoma	5	8	7	58	43	57	**10.85 (6.36–18.51)**	**5.38 (1.34–21.67)**	**8.53 (5.24–13.89)**
Colorectal	22	27	25	39	22	33	1.73 (0.86–3.47)	0.79 (0.11–5.59)	1.34 (0.70–2.58)
Breast	28	0	14	24	0	21	0.87 (0.41–1.83)	-	1.49 (0.71–3.14)
Leukaemia	6	8	7	25	22	23	**4.22 (1.73–10.30)**	2.66 (0.37–19.00)	**3.36 (1.50–7.53)**
Age group (years)									
0–24	2	3	3	0	0	0	0.00	0.00	0.00
25–44	21	15	18	67	143	73	**3.26 (1.33–7.95)**	**9.42 (1.31–67.53)**	**4.11 (1.83–9.24)**
45–64	164	172	168	347	372	350	**2.11 (1.44–3.09)**	2.17 (0.81–5.79)	**2.09 (1.46–2.97)**

Table 5 (continued): Cancer death rate and relative risk for systemic lupus erythematosus (SLE) patients compared to the general population.

Sub-group	Cancer death rate (per 100,000 person-years)						Relative risk (95% CI)		
	General population			SLE patients					
	Women	Men	Total	Women	Men	Total	Women	Men	Total
65–74	493	686	587	1,133	2,163	1,291	**2.30 (1.56–3.39)**	**3.15 (1.64–6.07)**	**2.20 (1.58–3.07)**
75+	1,259	1,918	1,548	1,558	2,996	1,805	1.24 (0.80–1.92)	1.56 (0.78–3.13)	1.17 (0.80–1.69)
Overall	185	219	202	324	497	343	**1.75 (1.40–2.19)**	**2.27 (1.49–3.44)**	**1.70 (1.39–2.07)**

‡Including leukaemia, lymphomas, multiple myeloma and malignant plasma cell neoplasms, other specified and unspecified malignant neoplasms of lymphoid, haematopoietic and related tissue.
SLE = systemic lupus erythematosus; 95% CI = 95% confidence interval.
The bolded relative risks indicate statistical significance.

COMPETING INTERESTS
Nil.

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Misclassified latent autoimmune diabetes in adults within Māori and Pacific adults with type 2 diabetes in Aotearoa New Zealand

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ABSTRACT

AIM: We investigated Māori and Pacific adults with type 2 diabetes (T2D) to determine the prevalence of latent autoimmune diabetes in adults (LADA), assess the type 1 diabetes (T1D) genetic risk score (GRS) distribution in those with and without autoantibodies and investigate differences in clinical diabetes characteristics based on autoantibody presence or a high T1D GRS.

METHOD: A total of 2,538 Māori and Pacific participants from the Genetics of Gout, Diabetes, and Kidney Disease study in Aotearoa New Zealand were included (830 with T2D, 1,708 without). LADA was defined as age of diabetes onset >30 years, presence of autoantibodies and no insulin treatment within the first 6 months. Clinical characteristics were extracted from medical records. T1D-associated autoantibodies (glutamic acid decarboxylase, islet antigen 2, zinc transporter 8) were measured from stored blood samples from 293 participants (262 T2D, 31 without). A T1D GRS consisting of 30 single-nucleotide polymorphisms was calculated for all participants.

RESULTS: Autoantibodies were detected in 8.8% (23/262) of individuals with T2D, with 5.3% (14/262) meeting the criteria for LADA. No significant difference in T1D GRS or clinical characteristics was observed between T2D cases with and without autoantibodies. Autoantibodies were also detected in 3.2% (1/31) of participants without diabetes.

CONCLUSION: LADA is present in a subset of Māori and Pacific individuals with T2D. Autoantibody presence was not associated with differences in T1D GRS or clinical features. Further research is needed to assess whether C-peptide monitoring could guide treatment decisions in those with LADA.

Correctly classifying diabetes is essential for ensuring appropriate and effective treatment strategies.[1] While type 1 diabetes mellitus (T1D) results from autoimmune destruction of pancreatic beta cells, leading to severe insulin deficiency, type 2 diabetes mellitus (T2D) is primarily characterised by insulin resistance.[2] This distinction has significant treatment implications, as T1D necessitates lifelong basal-bolus insulin therapy, while T2D is initially managed with lifestyle modifications and non-insulin medications, with later progression to simpler insulin regimens in some.[3] However, the existence of latent autoimmune diabetes in adults (LADA), characterised by slowly progressive autoimmune beta cell destruction, adds complexity to this classification.[2]

Diagnosing LADA presents significant challenges due to its clinical similarity with features of both T1D and T2D.[4–6] The key definition of LADA includes adults diagnosed with diabetes at 30 years of age who test positive for T1D-associated autoantibodies and do not require insulin for at least 6 months after their diabetes diagnosis.[7,8] Those with a slowly progressive form of autoimmune diabetes at ages younger than 30 years are classified as latent autoimmune diabetes in the young (LADY).[9] Those with LADY are likely to have more aggressive autoimmune destruction of beta cells and even more rapid progression to insulin requirement than LADA, given that youth with T2D have more rapid progression to insulin requirement than older adults with T2D.[10] However, research comparing progression to insulin between LADY and LADA has not been done.

Studies, primarily conducted in European populations, show similarities in genetic risk scores (GRS) between LADA and T1D,[11,12] with the human leukocyte antigen (HLA) region playing a dominant role in T1D risk.[13] This HLA influence on T1D GRS differentiation is also observed in Japanese and African ancestry populations, where incorporating both HLA and non-HLA variants enhances T1D risk prediction.[14,15] Interest-

ingly, a European-derived T1D diagnostic model incorporating age, body mass index (BMI), autoantibodies (i.e., glutamic acid decarboxylase [GAD], islet antigen 2 [IA2] or zinc transporter 8 [ZnT8]) and a T1D GRS demonstrated good performance (area under the receiver operating characteristic curve [AUC] = 0.84) even in an Indian population, suggesting a broader applicability.[16,17]

The prevalence of LADA has been reported to be high in Northern Europe and China compared to African American and Hispanic populations.[11,18,19] However, the prevalence among Māori and Pacific peoples living in Aotearoa New Zealand remains unknown. This study, leveraging data from the Genetics of Gout, Diabetes, and Kidney Disease (GoGDK) cohort,[20,21] aimed to: 1) determine the prevalence of LADA among Māori and Pacific adults clinically classified with T2D, 2) assess the distribution of a T1D GRS in those with T2D who did and did not have detectable autoantibodies, and 3) investigate any potential clinical differences, including glycaemic control and progression to insulin therapy, based on autoantibody presence or a high T1D GRS.

Methods

Study participants

All participants provided written informed consent for the collection of their samples and subsequent analyses. Ethical approval for this study was granted by the New Zealand Multi-Region Ethics Committee (MEC/05/10/130; MEC/10/09/092; MEC/11/04/036). The GoGDK study was initiated in 2005 to investigate the genetic and environmental contributors of gout, diabetes and kidney disease in adults from Aotearoa New Zealand. Participants were recruited through primary care clinics and community outreach programmes, aiming for broad population-level representation. Various clinical, demographic and biochemical measurements were collected from these participants at the time of recruitment. Longitudinal data for glucose lowering medications were collected from routine electronic healthcare dispensing records covering the period from 2002 to November 2022. A total of 2,538 participants who identified as Māori and/or Pacific from the GoGDK study of Aotearoa New Zealand were included in this analysis due to the availability of their genetic data for calculating their T1D GRS.[20,21] T2D diagnosis was ascertained in this cohort based on clinical records.

For this study, participants were reclassified with LADA according to the guidelines proposed by the Immunology of Diabetes Society (IDS): 1) over 30 years of age at diabetes onset, 2) positive titre for at least one T1D-associated autoantibody, such as GAD, IA2 and ZnT8, and 3) not treated with insulin the first 6 months after their diabetes diagnosis.[22]

Autoantibody measurement

Autoantibodies associated with T1D, including GAD, IA2, and ZnT8, were measured simultaneously in available serum samples using the ElisaRSR™ 3 Screen ICA™ kit (RSR Limited, Cardiff, United Kingdom). Individuals were deemed positive for autoantibodies if their combined GAD/IA2/ZnT8 concentration was ≥20u/mL and/or if their sample yielded an index value ≥30. Samples were tested in duplicate, and a repeat test was conducted to confirm a positive autoantibody status.

Clinical diabetes characteristics

Demographic characteristics, BMI, diabetes, age of diabetes diagnosis, diabetes treatment and glycated haemoglobin (HbA_{1c}) were obtained from linked electronic health records retrieved from the TestSafe data repository. Access to this data was approved by the Regional Éclair Change Management Group. Individuals with diabetes were categorised into either a "high" or "low" clinical risk group for T1D based on their time to insulin initiation. Those who started insulin treatment within 3 years of their diabetes diagnosis were placed in the "high" risk group; all others were assigned to the "low" risk group. Achieving an HbA_{1c} ≤55mmol/mol indicated "good" control of blood glucose levels.

T1D GRS calculation

The T1D GRS consists of 30 variants from both HLA and non-HLA regions, accounting for DR3/DR4-DQ8 contribution.[16] Specifically, there are five variants in the HLA region and 25 variants in other genes, including insulin (*INS*), interleukin genes (i.e., *IL2*, *IL2RA*, *IL10*, *IL27)*, and protein kinase D2 (*PRKD2*). Assuming that non-DR3/DR4 risk alleles have a log-additive effect on T1D risk, the T1D GRS was calculated by summing the dosages across risk alleles multiplied by the weight (ln[odds ratio]) for each allele, divided by the number of variants (Appendix Table 1). HLA class II DR3/DR4-DQ8 haplotypes were inferred from two single-nucleotide polymorphisms (SNPs), rs2187668 and rs7454108, with corresponding

weights assigned to each individual's score.[22] The whole-genome imputed sequence data constructed for GoGDK participants detailed in previous work was used to extract genotypes from a VCF file using BCFtools (v1.9-94-g9589876).[23,24] The ped files for these SNPs were imported into R v4.3.1 for processing and simple allelic scoring.[25] The Wellcome Trust Case Control Consortium GRS centiles were used as a guideline threshold, such that a T1D GRS >0.28 (>50th centile) suggests T1D.[16]

Statistical analysis

The Kruskal–Wallis test was employed to examine significant differences among group means of the T1D GRS. Subsequently, a Dunn's test was used to identify pairwise groups with significant differences. A false discovery rate correction was applied for p-value adjustment. When comparing only the LADA and true T2D (autoantibody negative) groups, the Wilcoxon Rank-Sum Test was used for continuous variables, while the Pearson's Chi-squared test was used for categorical variables. All analyses were performed using R software v4.3.1.[25]

Results

Serum sample availability allowed testing for T1D autoantibodies in a subset of 293 participants: 262 with clinically classified T2D (age of diabetes diagnosis ranged 3–64 years) and 31 without T2D (age at testing 19–73 years). Among those with T2D, 23 (8.8%) tested positive for at least one autoantibody in the triple autoantibody panel (i.e., GAD/IA2/ZnT8). Of these, 14 (5.3% of the total T2D subset) met the criteria for LADA based on the IDS guidelines of age at diagnosis over 30 years, autoantibody positivity and absence of insulin treatment within the first 6 months of diagnosis (Table 1). Eight (3.1%) individuals that tested positive for autoantibodies met the criteria for LADY. Among the 31 controls without diabetes tested, one individual tested positive for autoantibodies and was recruited at 50 years of age, with a negative screening HbA_{1c} for diabetes at 58 years of age.

Insulin initiation data were available for 10 of the 14 LADA participants. Of these, six (60%) eventually required insulin, with a mean time to insulin initiation of 13.75 (95% confidence interval [CI] 9.26–8.24) years. None of the 10 participants initiated insulin within the first 3 years of diagnosis, and the HbA_{1c} ranged from 34 to 96mmol/mol. Of the eight out of 10 who initiated insulin, these were at 9–26 years after their diabetes diagnosis. Seven of the eight participants with LADY initiated insulin within the observation period of 2002 to November 2022, with an average time to insulin of 18.29 years (95% CI 10.19–26.38). No significant differences in clinical diabetes characteristics (such as age at diagnosis, BMI, time to insulin, proportion on insulin or glycaemic control) were found between the LADA group and T2D group without T1D-associated autoantibodies.

The T1D GRS was calculated for a total of 2,538 participants, including 830 with clinically classified T2D and 1,708 without diabetes at the time of recruitment. Although 31 individuals met the criteria for "high" clinical risk for T1D due to insulin treatment within 3 years of diagnosis (age of diabetes diagnosis ranged from 12 to 78 years), none of these individuals tested positive for T1D autoantibodies. Among the 30 variants in the T1D GRS, 23 variants were present in the GoGDK cohort (Table 1). There was no significant difference in the T1D GRS between T1D autoantibody negative and positive groups (mean GRS [standard deviation] was 0.146 [0.016], n=269 and 0.148 [0.014], n=23, respectively; adjusted p=0.73; Figure 1) or between those with and without insulin treatment within 3 years of diagnosis (0.143 [0.017], n=31 and 0.147 [0.016], n=339, respectively; adjusted p=0.19; Figure 2 and Table 2).

Discussion

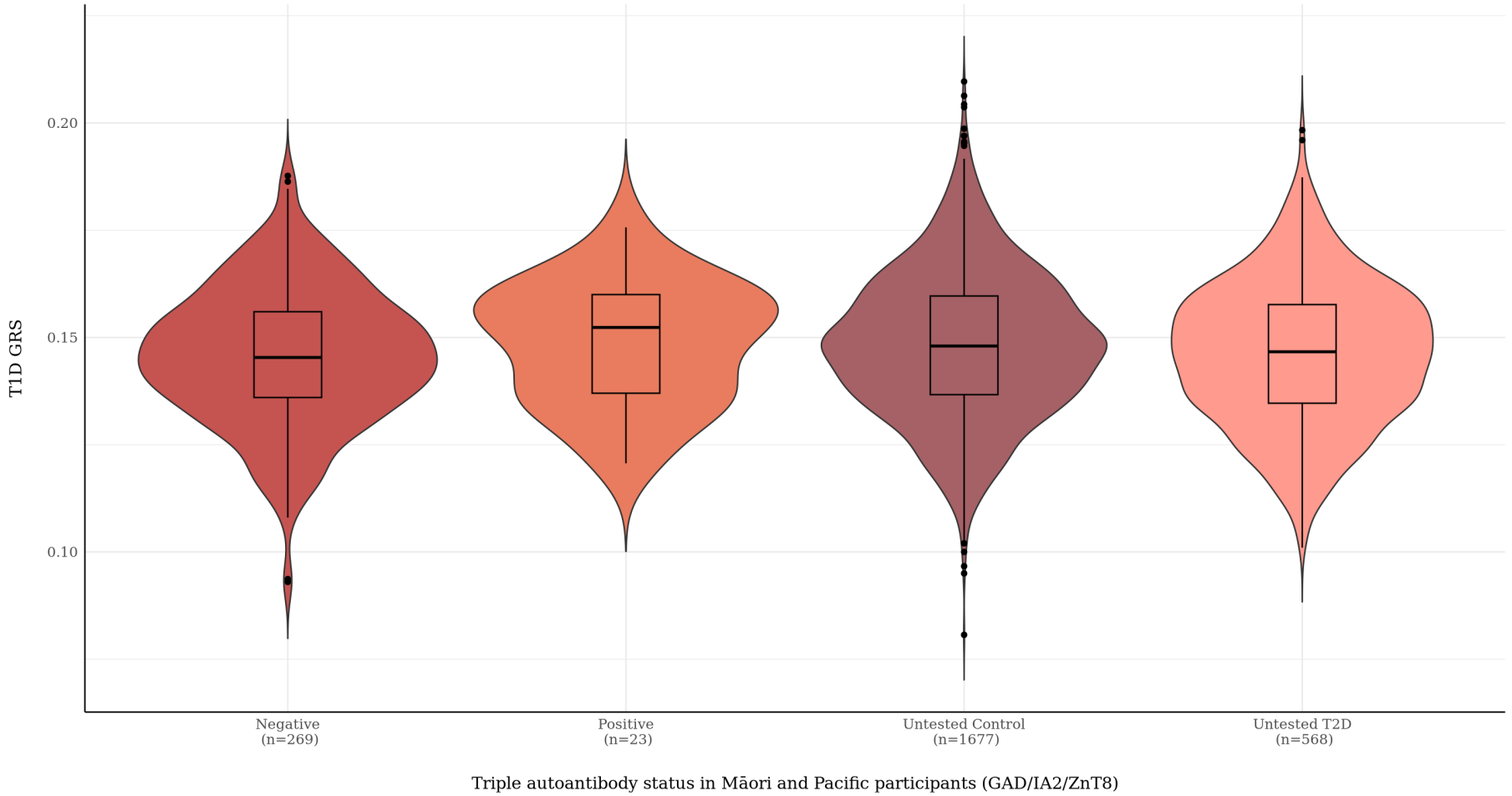
This study found that 5.3% (14/262) of the Māori and Pacific peoples in the GoGDK study that were clinically classified with T2D met the criteria for LADA. This was based on the presence of T1D-associated autoantibodies (GAD/IA2/ZnT8), age of diabetes diagnosis over 30 years and no insulin treatment within the first 6 months from diabetes diagnosis.[26] Additionally, 3.1% (8/262) of participants met the criteria for LADY, having tested positive for T1D-associated autoantibodies, age of diabetes diagnosis at an age younger than 30 years and not requiring insulin within the first 6 months of their diagnosis.[11] The LADA prevalence of 5.3% in our study is lower than the pooled global estimate of approximately 8.9%. Regional analysis shows that the Western Pacific Region has a prevalence of 8.3%, which is second lowest to Europe with a prevalence of 7%. This study is consistent with previous studies that have detected latent autoimmune diabetes in a subset of individuals diagnosed with T2D.[17,19,27,28] However, there were no significant differences in clinical characteristics, including the need

Table 1: Demographic and clinical characteristics of GoGDK participants with LADA and T2D (confirmed autoantibody negative).

Characteristic	N	LADA Mean (95% CI)	N	T2D Mean (95% CI)	p-value
N	14		239		
Continuous					
Age (years)	14	48.64 (43.90–53.38)	239	48.07 (46.43–49.71)	0.95
BMI (kg/m^2)	14	36.49 (32.52–40.46)	229	37.11 (35.91–38.31)	0.74
Age at diabetes diagnosis (years)	14	36.29 (33.54–39.04)	223	32.59 (31.09–34.09)	0.06
Time to insulin (years)	8	13.75 (9.26–18.24)	163	13.45 (11.89–15.01)	0.57
HbA_{1c} (mmol/mol)	8	67.87 (52.30–83.44)	133	66.44 (62.42–70.46)	0.17
T1D GRS	14	0.15 (0.14–0.15)	239	0.15 (0.14–0.15)	0.64
Categorical n(%)					
Female	14	8 (57.14)	239	144 (60.25)	1.00
Insulin therapy	10	6 (60)	239	163 (68.2)	0.84
Achieved good control 55mmol/mol	8	5 (62.5)	133	53 (39.85)	0.37
Insulin within 3 years of diagnosis	10	0 (0)	239	24 (10)	0.44

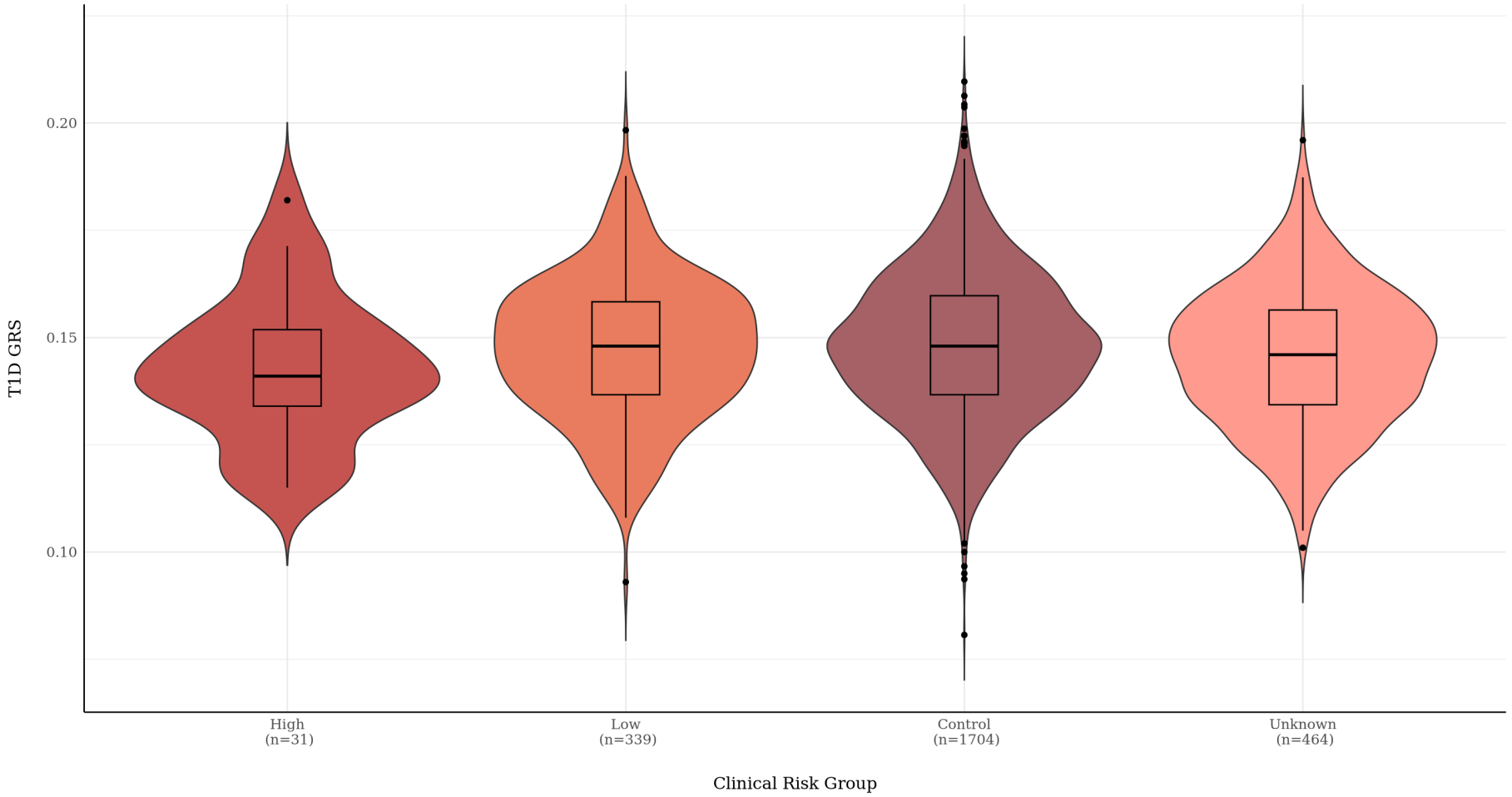
P-value comes from a Wilcoxon Rank-Sum Test for continuous values and Pearson's Chi-squared test for categorical values. GoGDK = Genetics of Gout, Diabetes, and Kidney Disease; LADA = latent autoimmune diabetes in adults; T2D = type 2 diabetes; CI = confidence interval; BMI = body mass index; HbA_{1c} = glycated haemoglobin; T1D = type 1 diabetes; GRS = genetic risk score.

Figure 1: Distribution of T1D GRS by autoantibody status.



Positive autoantibody result comes from a combined triple autoantibody test for GAD/IA-2/ZnT8.
T1D = type 1 diabetes; GRS = genetic risk score; T2D = type 2 diabetes; GAD = glutamic acid decarboxylase 65; IA2 = islet antigen 2; ZnT8 = zinc transporter 8.

Figure 2: Distribution of T1D GRS by clinical risk group.



The high-risk group consists of those who started insulin treatment within 3 years of their diabetes diagnosis, while all others were assigned to the low-risk group.
T1D = type 1 diabetes; GRS = genetic risk score.

Table 2: Pairwise group comparisons of T1D GRS by autoantibody status and clinical risk group.

	Group 1	Group 2	N (Group 1)	N (Group 2)	p-value	*Adjusted p-value
Autoantibodies	Negative	Positive	269	23	0.42	0.73
	Negative	Unknown control	269	1,677	0.034	0.102
	Negative	Unknown T2D	269	568	0.715	0.857
	Positive	Unknown control	23	1,677	0.863	0.863
	Positive	Unknown T2D	23	568	0.486	0.728
	Unknown Control	Unknown T2D	1,677	568	0.021	0.102
Clinical risk	High	Low	31	339	0.106	0.192
	High	Control	31	1,704	0.058	0.173
	High	Unknown	31	464	0.294	0.352
	Low	Control	339	1,704	0.497	0.497
	Low	Unknown	339	464	0.128	0.192
	Control	Unknown	1,704	464	0.004	0.026

*P-value and adjusted p-value come from a Dunn's test for pairwise group comparisons.
P-values for statistically significant differences are shown in bold (p<0.05).
T2D = type 2 diabetes; GRS = genetic risk score; T2D =type 2 diabetes.

for insulin therapy within 3 years of diagnosis, between those with LADA and those with autoantibody-negative T2D. Additional monitoring of C-peptide levels in the LADA and LADY group may have identified a sub-group with a rapid decline in beta cell function, who could have benefited from earlier insulin therapy.[8,29] While C-peptide measurements were not available for these participants, rapid decline in endogenous insulin secretion would be signalled by high HbA_{1c}, which is generally what is used in routine clinical practice decision making for when to initiate insulin therapy.

The observed prevalence of T1D-associated autoantibodies in the clinically ascertained T2D group was higher (8.8%; 23/262) than the false-positive rate observed in the control group (3.2%; 1/31). It has been suggested that identifying LADA in clinically defined T2D populations may lead to excess false-positive autoantibody results.[5] Increasing the titre threshold for a single autoantibody and considering the presence of multiple autoantibodies may better align with clinical, biochemical and genetic characteristics of LADA/T1D.[5] This requires further testing in larger cohorts of Māori and Pacific peoples with and without T1D or T2D, and soon after diagnosis since autoantibodies generally decline with time.[30]

The T1D GRS aggregates the effects of multiple genetic variants associated with T1D risk and has shown promise in differentiating T1D from T2D in several populations,[16,31] but is less useful for differentiating LADA from T2D given the intermediate distribution of the T1D GRS in LADA between

that seen in T1D and T2D.[32] This study confirms the lack of utility of the T1D GRS in distinguishing LADA from T2D in this cohort of Māori and Pacific peoples. The absence of T1D cases in this study cohort means we cannot determine the utility of the T1D GRS assessment in Māori and Pacific peoples in distinguishing between T1D and T2D.

Our study has several limitations. The limited sample size, resulting in a small number of participants with LADA, may have limited our power to detect subtle differences in clinical characteristics or genetic risk. Serum samples were not available for all participants with T2D and genetic data to accurately test for the presence of T1D-associated autoantibodies. Additionally, autoantibody measurement on serum samples collected at recruitment, rather than at the time of diagnosis, could have underestimated the true prevalence of LADA due to the recognised decline of autoantibodies over time.[30] The use of the combined ElisaRSR™ 3 Screen ICA™ kit thresholds (combined GAD/IA2/ZnT8 concentration ≥20u/mL and/or ≥30 index value) is a potential limitation in not being able to differentiate titre or number of positive autoantibodies, given that lower titre single autoantibodies are more likely to be false positives than high titre of multiple autoantibodies. Finally, we recognise the absence of C-peptide measurements, which could better differentiate individuals with slowly progressive beta cell loss who may benefit from earlier insulin initiation, although in clinical practice HbA_{1c} is used to inform decisions around insulin initiation.[33]

Despite these limitations, these findings contribute to the understanding of LADA in Māori and Pacific peoples. Larger studies with T1D GRS and comprehensive autoantibody testing at diagnosis of clinically classified T1D and T2D, and among controls, along with monitoring of C-peptide over time are warranted to clarify the utility of the T1D GRS and T1D autoantibody results in Māori and Pacific peoples. Such research will be crucial for effective strategies in diabetes prevention, screening and management for these populations.

COMPETING INTERESTS

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Appendix

Appendix Table 1: T1D SNPs included in the genetic risk score with weights.

SNP	Gene	OR	Weight	Effect allele	MAF GoGDK	MAF European*
rs2187668, rs7454108	DR3/DR4	48.18	3.87	NA	0.00	0.03
	DR3/DR3	21.12	3.05	NA	0.00	0.01
	DR4/DR4	21.98	3.09	NA	0.00	0.02
	DR4/X	7.03	1.95	NA	0.00	0.17
	DR3/X	4.53	1.51	NA	0.00	0.21
rs1264813	HLA_A_24	1.54	0.43	T	0.00	0.10
rs2395029	HLA_B_5701	2.50	0.92	T	1.00	0.963
rs3129889	HLA_DRB1_15	14.88	2.70	A	0.00	0.872
rs2476601	PTPN22	1.96	0.67	A	0.023	0.095
rs689	IN	1.75	0.56	T	0.00	0.730
rs12722495	IL2RA	1.58	0.46	T	0.979	0.902
rs2292239	ERBB3	1.35	0.30	T	0.205	0.334
rs10509540	C10orf59	1.33	0.29	T	0.795	0.733
rs4948088	COBL	1.30	0.26	C	0.989	0.960
rs7202877	NA	1.20	0.25	G	0.285	0.096
rs12708716	CLEC16	1.23	0.21	A	0.285	0.636
rs3087243	CTLA4	1.22	0.20	G	0.467	0.546
rs1893217	PTPN2	1.20	0.18	G	0.241	0.157
rs11594656	IL2RA	1.19	0.17	T	1.00	0.749
rs3024505	IL10	1.19	0.17	G	0.902	0.845
rs9388489	C6orf173	1.17	0.16	G	0.683	0.469
rs1465788	NA	1.16	0.15	C	0.645	0.725
rs1990760	IFIH1	1.16	0.15	T	0.309	0.599
rs3825932	CTSH	1.16	0.15	C	0.383	0.678
rs425105	NA	1.16	0.15	T	0.772	0.839
rs763361	CD226	1.16	0.15	T	0.482	0.479
rs4788084	IL27	1.16	0.15	C	0.537	0.592

Appendix Table 1 (continued): T1D SNPs included in the genetic risk score with weights.

rs17574546	NA	1.14	0.13	C	0.046	0.198
rs11755527	BACH2	1.13	0.12	G	0.313	0.448
rs3788013	UBASH3A	1.13	0.12	A	0.309	0.427
rs2069762	IL2	1.12	0.11	A	0.453	0.698
rs2281808	NA	1.11	0.10	C	0.699	0.659
rs5753037	NA	1.10	0.10	T	0.267	0.379

*MAF European was retrieved from the gnomAD genome browser v4.1.0 (https://gnomad.broadinstitute.org/), except for DR3/DR4-DQ8 haplotypes, which were retrieved from the Wellcome Trust Case Control Consortium (WTCCC) study's control subset of 2,938 people without diabetes.
T1D = type 1 diabetes; SNP = single-nucleotide polymorphism; OR = odds ratio; MAF = minor allele frequency; GoGDK = Genetics of Gout, Diabetes, Kidney disease; NA = not applicable.

The incidence, prevalence and treatment of narcolepsy in New Zealand

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ABSTRACT

AIM: No previous research has assessed the epidemiology or treatment of narcolepsy in New Zealand. This study aimed to estimate its national incidence and prevalence and examine demographic trends in the prescribing of narcolepsy-related medications.

METHOD: From 2021 to 2023, diagnostic data from all centres conducting multiple sleep latency tests (MSLTs) were analysed to estimate incidence and prevalence. Concurrently, data on all special authority (SA) approvals for narcolepsy medications were obtained from Pharmac and analysed by medication type, region, age, gender and ethnicity.

RESULTS: Among 342 MSLTs, 57 cases of narcolepsy were identified, giving an incidence of 0.36 per 100,000 person-years and a prevalence of 21.9 per 100,000 people. Over the same period, 223 new and 762 total SA applications were approved. The average number of new approvals (74.3 per year) was 3.9 times higher than the number of new diagnoses (19 per year). Demographic variations were observed in the SA data. Generally, methylphenidate hydrochloride was prescribed more than modafinil.

CONCLUSIONS: This is the first national estimate of the incidence and prevalence of narcolepsy in New Zealand. The mismatch between diagnosis and treatment data likely reflects limited diagnostic access, multiple medication use, the existence of imported cases with established diagnoses and the treatment of idiopathic hypersomnolence (IH) under the guise of narcolepsy. Policy and funding changes are needed to improve care access and reporting accuracy.

Narcolepsy is a chronic sleep disorder characterised by the irrepressible need to sleep during daytime hours (excessive daytime sleepiness [EDS]).[1] It is clinically divided into two forms: type 1 and type 2. Type 1, affecting up to 60% of patients, is further characterised by cataplexy—presenting as sudden, brief episodes of muscle weakness or loss of muscle tone occurring during wakefulness.[2] All patients may otherwise experience hallucinations on falling asleep/waking, disrupted nighttime sleep and/or episodes of sleep paralysis.[2] Global prevalence estimates suggest that narcolepsy affects 0.025–0.05% of the world's population.[3] The condition typically onsets between 10 and 20 years of age, with an average diagnostic delay of 8–12 years.[3]

The gold-standard diagnostic test for narcolepsy remains the multiple sleep latency test (MSLT).[4] This measures how fast patients fall asleep under controlled conditions as well as the speed at which they enter REM sleep—a phase of sleep normally observed at the end of a 60-to-90-minute sleep cycle.[4] Primarily, a diagnosis of narcolepsy requires EDS to be present every day for at least 3 months and an MSLT result of ≤8 minutes with ≥2 sleep-onset entries into the REM sleep phase (SOREMPs).[2] Because other sleep disorders can produce EDS, patients also require baseline polysomnography to rule out other conditions. This is an overnight sleep test that measures brain activity, blood oxygen levels, heart rate, breathing and movements of the eyes and legs.[4] Idiopathic hypersomnolence (IH), obstructive sleep apnoea (OSA) and chronic sleep restriction are the key differentials to exclude before diagnosing narcolepsy.[4]

A combination of symptom-managing medications and lifestyle changes can help improve patient wellbeing.[5] However, even with treatment, many feel disabled by their condition.[6] EDS and the other symptoms of narcolepsy can have significant impacts on the psycho-social-behavioural outcomes of patients. Patients often face barriers in achieving their social, educational, career and financial potential, especially if delays in accessing diagnosis and effective treatment occur.[7] Furthermore, due to the presence of involuntary sleep episodes, patients are at high risk for car accidents and other injuries.[8] Considering the young age of onset, it is clear that prompt recognition, diagnosis and treatment are important for improving patient wellbeing in the long term.

In this regard, the last 5 years have shown significant advancements in the medications

available for symptom control.[5] As research in this area accelerates, it is important to understand where each country stands in diagnosing and treating this condition. While several international studies have explored the prevalence, incidence and treatment of narcolepsy, minimal research has been conducted in New Zealand. Therefore, an up-to-date perspective is highly necessary. To achieve this, national data regarding testing and diagnosis between 2021 and 2023 were collated at the WellSleep centre in Wellington from all New Zealand centres offering formal testing for narcolepsy. This data was then compared to the prescription funding requests—special authorities (SAs)—processed by Pharmac (the national regulatory authority for medical funding) in the same period. Demographic trends in these SA applications were further described according to region, age, gender and ethnicity. This was done to help identify at-risk populations and to ensure that healthcare resources are being allocated appropriately based on specific community needs. This is particularly important for Māori, who often face inequities in healthcare access and outcomes, making it essential to understand how narcolepsy may uniquely affect them.

Method

Testing and diagnostic outcomes data for 2021–2023

All centres that conduct testing for narcolepsy (the NZ Respiratory & Sleep Institute [NZRSI], Auckland; WellSleep, Wellington; and the hospitals of Auckland City, Waikato, Christchurch and Dunedin) were contacted and asked to answer the following:

1. How many MSLTs did you perform in 2021, 2022 and 2023?
2. Based on the national MSLT reporting criteria, how many tests were positive for narcolepsy versus idiopathic hypersomnolence (IH) versus OSA/other diagnoses/non-diagnostic outcomes?

These data were used to estimate the incidence and prevalence of narcolepsy. These values were calculated as follows:

1. Incidence = (number of cases ÷ [total New Zealand population × study period in years]) × 100,000.
2. Prevalence = ([incidence ÷ 100,000] × [New Zealand average lifespan − average age of onset for narcolepsy in New Zealand]) × 100,000.

All of the sleep disorders mentioned in this study were diagnosed based on clinical criteria and objective sleep measures according to the *International Classification of Sleep Disorders-Third Edition* (ICSD-3).[2] All labs provided finalised diagnoses alongside their MSLT results.

Narcolepsy-related prescription data for 2021–2023

Pharmac was contacted by email to obtain data regarding medications used in the treatment of narcolepsy. A formal request under the *Official Information Act 1982* was sent to enquiry@pharmac.govt.nz in accordance with their protocols. The email asked the following:

For these medications:

- Modafinil (MF)
- Methylphenidate hydrochloride (MPHC)
- Dexamphetamine sulfate
- Clomipramine hydrochloride

Could you please provide:

1. The number of SAs for **each** medication, applied for **and** approved in relation to **narcolepsy** from 2021 to 2023. Both:

- Overall (new, repeats, and renewals).
- New (excluding repeats and renewals).

2. Demographic data (and associated sublevels provided by Pharmac) for the information in (1), broken down by:

- **Region:** further divided into Northern, Midland, Central and Southern.
- **Age:** ranging from 10 to 84-years-of-age, plus an "other" category, recorded in 5-year increments (e.g., 10–14, 15–19, etc.).
- **Gender:** female, male and unknown (encapsulating gender-diverse and non-reported individuals).
- **Ethnicity:** divided into NZ European, Māori, Pacific peoples, Asian, Indian, Middle Eastern or Latin American (MELAA), other and unspecified.

For each sublevel, the overall and new SA applications were broken down by medication type and provided in a spreadsheet by Pharmac.

Of note, Pharmac requires that all stimulant medications used in the treatment of narcolepsy are funded via the SA application process. Prior to December 2024, these SAs had to be renewed every 2 years. This restriction has since been lifted.[9] All data used in this study were collected before this change was implemented.

Statistical analysis

As this is a descriptive report, no statistical analyses were performed.

Results

Between 2021 and 2023, New Zealand conducted a total of 346 MSLTs (Table 1). Four of these tests, conducted by Waikato Hospital, had unknown diagnostic outcomes and were therefore excluded, leaving 342 useable outcomes. Among the remaining tests, 57 (16.7%, averaging 19 per year) were positive for narcolepsy, 106 (31.0%, averaging 35.3 per year) for IH and 179 (52.3%, averaging 59.7 per year) for OSA and other diagnoses.

Based on the average number of new narcolepsy cases diagnosed annually, the incidence of narcolepsy between 2021 and 2023 was 0.36 per 100,000 people per year. This was calculated as: Incidence = (57 cases ÷ [5,223,100 total New Zealand population × 3 years]) × 100,000. The prevalence of narcolepsy was therefore 21.9 cases per 100,000 people, calculated as: Prevalence = ([0.36 ÷ 100,000] × [New Zealand average lifespan of 82 years − average age of onset for narcolepsy in New Zealand of 20.7 years[10]]) × 100,000.

Overall, Auckland NZRSI (39.2%) and WellSleep Wellington (30.7%) conducted the highest proportions of MSLTs, followed by Auckland City Hospital (13.2%), Christchurch Hospital (11.7%) and Dunedin Hospital (5.3%). In terms of narcolepsy case detection, Auckland NZRSI (18/57; 31.6%), Auckland City Hospital (16/57; 28.1%) and WellSleep Wellington (14/57; 24.6%) diagnosed the most cases. Christchurch (3/57; 5.3%) and Dunedin (6/57; 10.5%) made smaller contributions.

Between 2021 and 2023, a total of 223 new and 762 overall SA applications were submitted to Pharmac, all of which were approved (Table 2). The annual average number of new SA applications was 74.3 per year, which was 3.9 times higher than the average annual case number for narcolepsy (19 cases per year). In comparison, the annual average of overall SA applications (254 per year) was 13.4 times higher than the average annual case number for narcolepsy. MPHC was prescribed slightly more than MF, representing 53.8% of new and 57.9% of overall applications.

When testing centres were grouped by their Health New Zealand – Te Whatu Ora regions, the total number of new applications was lower than overall new SA applications provided in Table 2 (193 vs 223), while the total number of overall applications was higher (839 vs 762) (Table 3). The greatest contribution to new SA applications came from Central (34.2%, covered by WellSleep Wellington), followed by Southern (27.5%, covered by Christchurch and Dunedin Hospitals), Northern (25.4%, covered by Auckland NZRSI and Auckland City Hospital) and Midland (9.8%, covered by the Auckland centres, with a small minority from Waikato Hospital). Of the applications, 3.1% did not have a region associated with them. In the overall data, this distribution shifted to Southern (31.2%), Northern (28.4%), Central (24.7%), Midland (13.7%) and unknown (2.0%). In terms of new SA applications, the Northern and Central regions prescribed proportionally more MF at 57.1% and 53.0%, respectively, while the Midland and Southern regions used more MPHC at 84.2% and 52.8%. By comparison, for overall SA applications, all regions prescribed more MPHC (58.4% in Southern, 56.0% in Northern, 60.9% in Central and 56.1% in Midland).

When the SA application numbers were broken down by age groups, both the total number of new applications and the total number of overall applications were lower than overall new SA provided in Table 2 (148 vs 223 and 710 vs 762, respectively) (Table 4). For new applications, there were three notable peaks in the data: 20–24 years (25.7%), 35–39 years (11.5%) and 75–79 years (3.4%). In contrast, the overall SA applications peaked at 25–29 years (15.1%), 50–54 years (8.2%) and 70–74 years (4.4%). In the new SA group, there was a slightly larger proportion of MPHC used, accounting for 50.7% of applications. This was more pronounced in the overall SA group, where MPHC accounted for 58.9% of applications.

When the SA application numbers were broken down by gender, the total number of new applications was lower than the total new SA number provided in Table 2 (215 vs 223), while the total number of overall applications was accurately reported (762 vs 762) (Table 5). In both the new and overall groups, more SA applications were made for females (68.4% and 58.4%, respectively) compared

Table 1: National MSLT diagnostic outcomes across 2021–2023.

MSLT outcomes	Testing centres					
	Auckland NZRSI	Auckland City Hospital	WellSleep Wellington	Christchurch Hospital	Dunedin Hospital	Total
Total MSLT numbers 2021–2023	134.0	45.0	105.0	40.0	18.0	342.0
% Contribution to total MSLT number	39.2	13.2	30.7	11.7	5.3	100.0
Average narcolepsy case numbers	6.0	5.3	4.7	1.0	2.0	19.0
Total narcolepsy cases	18.0	16.0	14.0	3.0	6.0	57.0
% Contribution to total narcolepsy cases	31.6	28.1	24.6	5.3	10.5	100.0
% Of total MSLTs resulting in narcolepsy diagnoses	13.4	35.6	13.3	7.5	33.3	16.7
Average IH case numbers	13.0	5.3	13.3	2.7	1.0	35.3
Total IH cases	39.0	16.0	40.0	8.0	3.0	106.0
% Contribution to total IH cases	36.8	15.1	37.7	7.5	2.8	100.0
% Of total MSLTs resulting in IH diagnoses	29.1	35.6	38.1	20.0	16.7	31.0
Average OSA + other case numbers	25.7	4.3	17.0	9.7	3.0	59.7
Total OSA + other cases	77.0	13.0	51.0	29.0	9.0	179.0
% Contribution to total OSA + other cases	43.0	7.3	28.5	16.2	5.0	100.0
% Of total MSLTs resulting in OSA + other diagnoses	57.5	28.9	48.6	72.5	50.0	52.3

MSLT = mean sleep latency test; IH = idiopathic hypersomnolence; OSA = obstructive sleep apnoea.

Table 2: The new and overall numbers of special authority (SA) applications approved by Pharmac across 2021–2023.

SA application numbers	Total
Total new	223.0
Average new	74.3
% for MPHC	53.8
% for MF	46.2
Total overall	762.0
Average overall	254.0
% for MPHC	57.9
% for MF	42.1

SA = special authority; MPHC = methylphenidate hydrochloride; MF = modafinil.

Table 3: The new and overall numbers of special authority (SA) applications approved by Pharmac by Health New Zealand –Te Whatu Ora region across 2021–2023.

SA application numbers	Health New Zealand – Te Whatu Ora region					
	Northern	Midland	Central	Southern	Unknown	Total
Total new	49.0	19.0	66.0	53.0	6.0	193.0
Average new	16.3	6.3	22.0	17.7	2.0	64.3
% for MPHC	42.9	84.2	47.0	52.8	50.0	51.3
% for MF	57.1	15.8	53.0	47.2	50.0	48.7
% contribution of region	25.4	9.8	34.2	27.5	3.1	100.0
Total overall	238.0	115.0	207.0	262.0	17.0	839.0
Average overall	79.3	38.3	69.0	87.3	5.7	279.7
% for MPHC	58.4	60.9	56.0	56.1	82.4	57.9
% for MF	41.6	39.1	44.0	43.9	17.6	42.1
% contribution of region	28.4	13.7	24.7	31.2	2.0	100.0

SA = special authority; MPHC = methylphenidate hydrochloride; MF = modafinil.

to males (30.7% and 41.3%) and individuals with unknown gender (0.9% and 0.3%). In both groups, MPHC was prescribed more than MF, with MPHC accounting for 51.2% of new applications and 57.9% of overall applications.

When the SA application numbers were broken down by ethnicity, both the total number of new applications and the total number of overall applications were again lower than that provided for all new SAs in Table 2 (188 vs 223 and 732 vs 762, respectively) (Table 6). In both the new and overall application groups, most applications were made for the NZ European group (78.2% and 78.7%). This was followed by the Māori (8.5% and 13.1%) and Asian (5.3% and 3.8%) groups. The remaining ethnic groups contributed smaller proportions, ranging from 0.4 to 2.1%. In all three main groups, MPHC was prescribed more than MF, with MPHC respectively accounting for 54.4%, 81.3% and 80.0% of new applications for the NZ European, Māori and Asian groups. For overall applications, MPHC accounted for 57.8%, 58.3% and 53.6% of

Table 4: The new and overall numbers of special authority (SA) applications approved by Pharmac by age group across 2021–2023.

SA application numbers	Age group																
	10–14	15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80–84	Other	Total
Total new	1.0	5.0	38.0	26.0	12.0	17.0	11.0	10.0	5.0	4.0	2.0	4.0	4.0	5.0	1.0	3.0	148.0
Average new	0.3	1.7	12.7	8.7	4.0	5.7	3.7	3.3	1.7	1.3	0.7	1.3	1.3	1.7	0.3	1.0	49.3
% for MPHC	100.0	60.0	52.6	53.8	25.0	47.1	72.7	20.0	60.0	50.0	50.0	50.0	25.0	60.0	100.0	33.3	49.3
% for MF	0.0	40.0	47.4	46.2	75.0	52.9	27.3	80.0	40.0	50.0	50.0	50.0	75.0	40.0	0.0	66.7	50.7
% contribution of age group	0.7	3.4	25.7	17.6	8.1	11.5	7.4	6.8	3.4	2.7	1.4	2.7	2.7	3.4	0.7	2.0	100.0
Total overall	2.0	41.0	74.0	107.0	85.0	86.0	55.0	37.0	58.0	53.0	29.0	11.0	31.0	27.0	9.0	5.0	710.0
Average overall	0.7	13.7	24.7	35.7	28.3	28.7	18.3	12.3	19.3	17.7	9.7	3.7	10.3	9.0	3.0	1.7	236.7
% for MPHC	100.0	61.0	56.8	53.3	56.5	51.2	67.3	54.1	55.2	62.3	65.5	72.7	54.8	88.9	88.9	40.0	58.9
% for MF	0.0	39.0	43.2	46.7	43.5	48.8	32.7	45.9	44.8	37.7	34.5	27.3	45.2	11.1	11.1	60.0	41.1
% contribution of age group	0.3	5.8	10.4	15.1	12.0	12.1	7.7	5.2	8.2	7.5	4.1	1.5	4.4	3.8	1.3	0.7	100.0

SA = special authority; MPHC = methylphenidate hydrochloride; MF = modafinil.

Table 5: The new and overall numbers of special authority (SA) applications approved by Pharmac by gender group across 2021–2023.

Total SA numbers	Gender			
	Female	Male	Unknown	Total
Total new	147.0	66.0	2.0	215.0
Average new	49.0	22.0	0.7	71.7
% for MPHC	55.8	42.4	0.0	51.2
% for MF	44.2	57.6	100.0	48.8
% contribution of gender group	68.4	30.7	0.9	100.0
Total overall	445.0	315.0	2.0	762.0
Average overall	148.3	105.0	0.7	254.0
% for MPHC	58.0	58.1	0.0	57.9
% for MF	42.0	41.9	100.0	42.1
% contribution of gender group	58.4	41.3	0.3	100.0

SA = special authority; MPHC = methylphenidate hydrochloride; MF = modafinil.

applications in these same groups. The remaining ethnic groups showed more variation in medication use.

Discussion

Between 2021 and 2023 New Zealand conducted 346 MSLTs. Of the 342 tests with diagnostic outcomes, 57 were positive for narcolepsy (16.7%), 106 for IH (31.0%) and 179 for OSA and other diagnoses (52.3%). In these 3 years, Pharmac approved all 762 narcolepsy-associated SA applications, of which 223 were new (i.e., not repeats or renewals). Overall, 52.9% of applications were for MF and 47.1% were for MPHC. Notably, no new, repeat or renewal SA applications have been made for dexamphetamine sulphate since 2021, and clomipramine hydrochloride (a tricyclic antidepressant) is not processed under special authority, meaning Pharmac could not supply data on its use. As such, discussion on both medication types has been omitted.

The global incidence of narcolepsy ranges between 0.3 and 1.3 per 100,000 person-years, while its prevalence sits between 25 and 50 cases per 100,000 people.[11] Based on the national diagnostic MSLT data of 2021 to 2023 (Table 1), New Zealand's estimated incidence (0.36 cases per 100,000 person-years) is comparable to other countries, while our prevalence (21.9 cases per 100,000 people) is slightly lower. These estimates are the first ever calculated for the New Zealand context based on New Zealand's diagnostic data. However, for two reasons, these measures may be underestimated. Firstly, Pharmac allows physicians to diagnose and prescribe for narcolepsy based on clinical criteria alone, suggesting that some cases may not be represented in the MSLT-based data set. Secondly, the MSLT data used in this study were collected soon after the COVID-19 pandemic. During this time, there were drastic burdens on New Zealand's healthcare system, reducing access for non-urgent referrals across many disciplines.[12] This likely decreased the number of people receiving MSLT testing. Future work exploring narcolepsy in New Zealand will need to address these factors to ensure a wholly representative sample.

Beyond this, it was surprising to see that the average number of new SAs approved across 2021–2023 was 3.9 times higher than the average number of new cases diagnosed (Table 2). Logically speaking, there should be minimal differences between these values, as each patient should be treated under a single SA. Four sources for this discrepancy are suggested: 1) there are

Table 6: The new and overall numbers of special authority applications approved by Pharmac by ethnicity group across 2021–2023.

Total SA numbers	Ethnicity								
	NZ European	Māori	Pacific peoples	Asian	Indian	MELAA	Other	Unspecified	Total
Total new	147.0	16.0	4.0	10.0	2.0	2.0	1.0	6.0	188.0
Average new	49.0	5.3	1.3	3.3	0.7	0.7	0.3	2.0	62.7
% for MPHC	54.4	81.3	50.0	80.0	0.0	50.0	0.0	50.0	56.9
% for MF	45.6	18.8	50.0	20.0	100.0	50.0	100.0	50.0	43.1
% contribution of ethnicity group	78.2	8.5	2.1	5.3	1.1	1.1	0.5	3.2	100.0
Total overall	576.0	96.0	5.0	28.0	4.0	3.0	4.0	16.0	732.0
Average overall	192.0	32.0	1.7	9.3	1.3	1.0	1.3	5.3	244.0
% for MPHC	57.8	58.3	60.0	53.6	50.0	33.3	25.0	81.3	57.9
% for MF	42.2	41.7	40.0	46.4	50.0	66.7	75.0	18.8	42.1
% contribution of ethnicity group	78.7	13.1	0.7	3.8	0.5	0.4	0.5	2.2	100.0

SA = special authority; MPHC = methylphenidate hydrochloride; MF = modafinil.

cases being diagnosed and treated based on clinical criteria alone (as above), 2) there are cases arriving from overseas with existing diagnoses, 3) patients are receiving both MPHC and MF in the same year, and 4) patients who are diagnosed with IH are being treated under the guise of narcolepsy. With the data collected in this study it is not possible to estimate the proportion of cases diagnosed using clinical criteria alone. Similarly, it is difficult to use these data when estimating the number of cases coming and going from New Zealand via migration. As such, points three and four are of primary interest.

For point three, the number of new applications for MF and MPHC were roughly proportional. This could suggest that patients are receiving both medications in the same year. Pharmac requires patients to have trialled MPHC before being prescribed MF.[13] As MPHC is a second-line medication with little evidence for its use in narcolepsy,[14,15] physicians may be rapidly switching their patients onto MF due to inefficient treatment responses, indicating a redundancy in the SA application process. Other countries often combine MF with other first-line pharmaceuticals to properly manage the symptoms of patients.[14,15] If physicians in New Zealand were switching to MF from MPHC in the absence of access to these other options, hoping for better control, this would suggest a clear need to explore whether new medications should be introduced.

Then, regarding point four, IH and narcolepsy are both central hypersomnolence disorders.[16] IH is not only similar to narcolepsy in clinical presentation, but it shares the same first-line treatment (i.e., MF).[16] However, IH lacks the recognition and treatment avenues afforded to narcolepsy in New Zealand, despite our data revealing it to be the more common of the two (based on MSLT results). That is, there is no option to provide MPHC or MF to patients with IH as SA applications for MPHC and MF are only approved for narcolepsy or attention deficit hyperactivity disorder.[17] Therefore it seems likely that part of the discrepancy between the number of new cases and the number of new applications for SA could come from applications for medications to treat IH being listed as narcolepsy-related. If this were the case, it would be clear that Pharmac should amend the SA criteria to allow funding for IH as well, thereby helping a significantly under-represented cohort of patients and allowing accurate data collection on central hypersomnolence disorders.

However, by assuming this is the case, one must assume the demographic data supplied by Pharmac do not truly represent narcolepsy. Instead, they are likely to represent central hypersomnolence disorders overall. Regardless, inferences for the demographic distribution of narcolepsy can likely be pulled from these data due to the similarities between both conditions. Within this, it should also be noted that the international prevalence of IH has been described as being four times less than that of narcolepsy.[18] Although all IH cases reported in this study are confirmed diagnoses, diagnosed according to clinical and testing criteria from the ICSD-3, the discrepancy between New Zealand and the global population should be queried.

Further difficulties in interpreting the Pharmac data also come from the fact that Pharmac's privacy policies prevented them from releasing data points with fewer than six values (these were conservatively estimated as one). As such, nearly all of the demographic totals—both new and overall—were underestimated when compared to the reported total for each group. The only exceptions were the overall totals for Health New Zealand – Te Whatu Ora regions and ethnicity, where Pharmac indicated that a single patient could belong to multiple groups. Since these underestimations for each demographic were significantly smaller in the overall group, trends and averages in the medications used to treat patients with central hypersomnolence disorders (and by extension, narcolepsy) are discussed based on these data. This decision was made to minimise the bias caused by unknown values. As a result, several interesting findings emerge.

Firstly, for geographic distribution, most new cases of narcolepsy diagnosed in 2021–2023 were identified by NZRSI and Auckland City Hospital (59.7%). This was followed by WellSleep Wellington (24.6%), Dunedin Hospital (10.5%) and then Christchurch Hospital (5.3%) (Table 3). Yet, in this same period, the Northern and Midland Health New Zealand – Te Whatu Ora regions (covered by the Auckland testing centres) only applied for 42.1% of the overall SAs, while Central (covered by WellSleep) applied for 24.7% and Southern (covered by the Christchurch and Dunedin Hospitals) for 33.2%. This suggests that despite diagnosing the minority of cases by MSLT, the Southern testing centres are applying for a relatively large proportion of narcolepsy-related SAs. Most likely, this would represent the South Island clinicians applying for a higher number of SAs based on clinical criteria alone. A possible reason as to why

this might be happening could be the fact that there are only two testing centres on the island despite its large area. There could be a significant geographical barrier reducing access to testing, promoting clinicians to offer clinical diagnoses instead of formal testing. It may also be possible that a small percentage of established cases are being imported to the Southern regions within the student cohort. However, the data presented cannot be used to quantify this variable.

Age-wise, three peaks were seen in the overall SA data (Table 4). The first and largest was seen in the 25-to-29-year-old cohort. Its distribution was relatively wide, spanning from 20 to 39 years (all values above 10.0%). The second largest was seen at 50–54 years, and the smallest was seen at 75–79. The first two peaks seem to mirror what is seen internationally. That is, previous research has noted that the incidence of narcolepsy peaks between 14.7 and 18.1 years and again at 35 years.[18,19] However, due to a diagnostic lag of up to 13 years, many patients do not start treatment until years after.[20] Therefore, it makes sense that New Zealand prescriptions are peaking in these decades. Comparatively, the third peak is unexpected. As narcolepsy becomes increasingly recognised, we would expect the peak age of diagnosis/prescription to converge on the ages of onset. Given the fact we see peaks in the 60+ cohort, there may be some level of diagnostic catch-up occurring as knowledge of narcolepsy increases among healthcare professionals—consistent with data showing that some patients still report diagnostic lags of 16–28 years.[21] Beyond this, as a point of interest, the use of MPHC over MF appears to increase with age. In this regard, MF only became funded in New Zealand from 2004.[22] This pattern may therefore reflect the long-term, continued use of MPHC by 50-to-70-year-old patients who were initiated on this medication and preferred not to switch to MF over time.

Then, regarding gender equity, our data indicate there may be gender-related biases in diagnosis and/or prescribing (Table 5). At the time of data collection, New Zealand's population was 99.3% cisgender and 0.7% transgender or non-binary.[18] Of the cisgender population, 49.3% were male, while 50.3% were female.[18] However, the percentage of females receiving SAs overall was high (58.4%) when compared to males (41.3%). The higher number of SAs approved for women is consistent with some international data showing that women are diagnosed with narcolepsy more than men—although such findings vary between studies, and the risk of narcolepsy among both genders has been shown to be equivocal with variations in symptom profiles and diagnostic access causing biases in the scarce, gender-related findings.[24] By comparison, the 0.3% patients with unknown gender may closely represent the number of gender diverse individuals within New Zealand's population—especially when factoring in the conservative estimation of one for values less than six.

Finally, New Zealand has historically struggled to meet the health needs of its minority populations, especially for the Indigenous Māori.[25] Historically, Māori have been less likely to seek healthcare and less likely to receive appropriate management when they do. This has resulted in long-standing health outcome disparities, especially when compared to NZ Europeans.[25] These disparities have been a major public health focus in New Zealand for several years now. The percentage of overall SAs approved for NZ Europeans (78.7%) was high compared to the percentage of people identifying as NZ European (67.8%) but low in Māori (13.1%) compared to those identifying as Māori (17.8%) (Table 6).[26] It should be considered that the number of Māori accessing diagnosis and treatment for narcolepsy may be under-representative of the group's needs. This is especially true when considering that Māori suffer from more frequent and severe viral and bacterial infections when compared to NZ Europeans,[27] which is argued to be a trigger for narcolepsy.[28] Māori also tend to live in more rural communities with lower access to healthcare,[29] potentially compounding any disparities. However, it is also known that globally, different ethnicities have different propensities for narcolepsy.[11] Therefore, New Zealand's prescription rates could still be ethnically representative. More research is needed to confirm.

Limitations and future directions

Overall, this study has several limitations that need to be considered when interpreting its results. Firstly, the case numbers presented may be incomplete as some individuals with narcolepsy may not have sought diagnosis or may have been diagnosed based on clinical criteria alone. Secondly, regarding the SA applications, it is likely that the data presented actually represent central hypersomnolence disorders, reducing the specificity for narcolepsy. This is further complicated by the privacy policies of Pharmac—a system that naturally causes underestimations in the data for

rarer disorders. To overcome these limitations, it is recommended that future research focusses on prospective diagnostic data collection encompassing both objective and clinical cases. Access to diagnostic testing services needs to be improved through Health New Zealand – Te Whatu Ora sleep services. It is also recommended that Pharmac develop and fund separate SAs for the treatment of IH—allowing proper reporting of both disorders, guiding future management.

Conclusion

This study provides the first estimates of narcolepsy incidence and prevalence in New Zealand and describes potential disparities in the medications used to treat it. Changes in policy and future prospective data collection are recommended.

COMPETING INTERESTS
Nil.

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Addressing rural mental health inequities for transgender communities in Aotearoa

Katie E McMenamin, Angie Enoka, Mel Meates

ABSTRACT

AIM: We aimed to examine barriers and enablers to mental health support for transgender and gender-diverse individuals in rural Aotearoa New Zealand, drawing on research conducted in the Whanganui Region.

METHOD: Findings were drawn from a qualitative study involving interviews with transgender and gender-diverse participants in Whanganui, where mental health concerns consistently arose despite not being the study's primary focus.

RESULTS: Participants reported high levels of psychological distress, shaped by intersecting factors such as gender dysphoria, neurodivergence, financial hardship and social isolation. Major barriers to accessing support included a lack of affirming and knowledgeable mental health providers, limited service availability and experiences of discrimination—both systemic and interpersonal. Some participants described additional difficulty related to provider biases or the ineligibility of publicly funded therapy for gender-related issues. At the same time, protective factors included access to gender-affirming care, culturally safe counselling and peer or community-based support. For neurodivergent participants, inflexible service design and diagnostic barriers further impacted mental health access.

CONCLUSION: Strengthening culturally safe, affirming and accessible mental health services is essential for improving outcomes for transgender and gender-diverse communities in rural Aotearoa. Strategies such as increasing provider training, supporting community-led initiatives, expanding telehealth and creating clearer care pathways may help address persistent inequities.

Gender diversity is expressed through a wide range of terms, including culturally grounded identities used within Māori and Pacific communities in Aotearoa New Zealand and across the Pacific.[1,2] In this commentary, the term "transgender" is used to describe individuals whose gender identity does not align with the sex assigned to them at birth. For clarity and inclusivity, we use transgender as an umbrella term throughout, encompassing identities such as non-binary, whakawahine, fa'afafine and genderqueer, among others. For perspective, the 2023 New Zealand Census reported that around 26,000 adults (0.7% of the adult population) identified as transgender;[3] however, currently there are no reliable data on rural versus urban distribution.

Access to mental health services is a well-recognised challenge in rural Aotearoa, where specialist services are limited, wait times are often longer and distance creates additional barriers.[4,5] These challenges have been linked to inequities in service access and persistently higher suicide rates in rural communities. This broader context highlights the additional barriers faced by transgender and gender-diverse people who live in rural areas of Aotearoa.

As a minority group, people from the transgender community face significant mental health challenges, exacerbated by ongoing barriers to accessing appropriate healthcare.[6–8] These barriers include discrimination, stigma, lack of transgender-competent healthcare providers and insufficient access to gender-affirming care (GAC)—an affirming approach to healthcare that may include puberty blockers, hormones, surgery and psychosocial support—particularly in rural regions like Whanganui.[8–15] Access issues are associated with higher levels of psychological distress, self-harm and suicidality among transgender people, with Indigenous populations experiencing even greater disparities.[9] The recent Aotearoa-based Counting Ourselves survey[7,8]—a nationwide community-led health survey of transgender and non-binary people—found that over half of the participants had seriously considered suicide in the past year, with more than a third having attempted it at some point.[7,8] These insights point to an ongoing need for inclusive, accessible healthcare that better meets

the specific needs of transgender communities.

In rural areas, such as Whanganui, access to GAC is hindered by a lack of designated gender health services and a limited number of healthcare providers with adequate knowledge of transgender issues.[9,14,16] Moreover, many transgender individuals face navigating their healthcare alone, further increasing their risk of negative mental health outcomes.[16] Historically, gender diversity was pathologised, with transgender identities often framed as disorders rather than recognised as valid expressions of self.[13,15] This history has shaped a health system where transgender people still encounter systemic barriers to care and support. While there is no single agreed definition of "transgender competence" comparable to cultural competence frameworks, several key factors are consistently described. These include using correct names and pronouns, creating inclusive environments, understanding GAC pathways and recognising the impact of stigma and minority stress on health. Both international standards[17] and Aotearoa guidelines[18] emphasise that this competence is ongoing and built through clinical knowledge, respectful communication and culturally safe practice.

Positive mental health outcomes are closely tied to the presence of supportive environments and affirming care practices.[7,11,13] For instance, transgender individuals within Aotearoa who receive strong support from their whānau are nearly half as likely to attempt suicide compared with those who lack such support.[7] Of concern, a Canadian study found that only 13% of transgender youth reported having supportive parents, highlighting the critical role of non-familial external support networks.[13] Primary care providers, including general practitioners (GPs), play a crucial role in initial and ongoing GAC access. Yet many transgender individuals report negative experiences within primary care, such as having to educate their providers, being misunderstood or stigmatised and facing long wait times for transgender-friendly services.[11,16,19]

This commentary draws on findings from a qualitative study conducted in the Whanganui Region in 2023–2024, which explored healthcare experiences of transgender and gender-diverse people and their whānau, alongside perspectives from primary care clinicians.[9] Although the study was designed to examine access to gender-affirming healthcare more broadly, mental health needs and barriers to support emerged as dominant themes throughout the interviews. This commentary draws attention to mental health concerns raised throughout the study and outlines practical steps to strengthen access to appropriate support for transgender and neurodiverse individuals. Recently, the Whanganui Region has introduced a specific transgender health clinical pathway and opened a clinic that specialises in GAC, addressing critical gaps in transgender healthcare; however, there remains a pressing need for further action. For example, enhancing the availability of mental health support and safe spaces for physical activity is essential to the overall wellbeing of transgender individuals. This commentary will explore these challenges and emphasise the importance of adopting an asset-based approach that leverages community strengths to improve healthcare access and outcomes for transgender populations in Whanganui.

Mental health challenges and barriers

Transgender people living in rural areas, such as Whanganui, face numerous challenges that significantly impact their mental health and wellbeing. Recent research with transgender participants from the Whanganui Region highlighted the significant role of mental health on the transgender journey.[9]

Negative impacts on mental health

The Whanganui-based research revealed high rates of mental health–related problems, including anxiety, depression, post-traumatic stress disorder (PTSD), dissociation and suicidal ideation.[9] These mental health challenges stemmed from both transgender-related and non-transgender-related circumstances, such as early childhood trauma, neurodiversity and difficulties with family acceptance. Together, these factors create a complex landscape in which accessing appropriate support is often difficult and inconsistent. Participants discussed the profound impact of gender dysphoria, where the discrepancy between the physical body and gender identification was often intertwined with mental health, highlighting, as in previous research, the importance of access to GAC.[15]

One of the most prominent barriers identified was the limited availability of accessible mental health services in the Whanganui Region. Participants reported that access to counselling or

therapy is limited, with those who were able to find support often receiving it for reasons unrelated to their gender identity. Many participants found that their counselling sessions could not be used for gender-related support due to funding restrictions (e.g., Accident Compensation Corporation [ACC] sexual abuse claims) or the counsellor's lack of expertise in transgender issues. As a result, individuals were often unable to fully engage in therapy, limiting its effectiveness and adversely impacting their mental health.

Additionally, participants faced challenges in finding transgender-competent mental health providers. A strong preference for counsellors with lived experience was emphasised, but the absence of transgender and gender-diverse counsellors in the Whanganui Region limited this potential access to gender-related mental health care. Participants differed in opinion around how to choose a counsellor, with a strong preference for face-to-face, in-person appointments. For some, this meant seeing a counsellor with minimal knowledge or education about transgender issues. For others, it meant compromising on their preference for in-person sessions and resorting to online appointments with transgender clinicians.

Some participants reported that therapy was further complicated by therapists' personal or religious beliefs, which at times undermined the therapeutic relationship. Participants described feeling judged or misunderstood, particularly when their therapist held conservative views on gender or attempted to pathologise transgender identities. Such experiences left some participants feeling alienated or stigmatised, leading to disengagement from care.

Another significant impact on mental health was the experience of living "stealth"—concealing one's transgender identity. This lifestyle often resulted in heightened stress due to the secrecy and the need to conform to cisgender norms. Participants reported that, while they enjoyed gender-affirming activities in safe spaces, returning to a non-affirming environment afterwards led to negative thoughts and low mood, exacerbating their mental health struggles.

Finally, financial stressors were highlighted as significant contributors to poor mental health. Many transgender individuals struggled to afford basic needs (e.g., clothing or haircuts), let alone the additional costs associated with GAC, such as hormone therapy or surgeries. The cost of accessing GAC, alongside employment challenges linked to discrimination or gendered expectations, contributed to financial instability and added strain on mental wellbeing.

Positive impacts on mental health

Conversely, participants noted several factors that positively impacted their mental health and wellbeing. Access to supportive GAC, including hormone therapy and surgeries, was frequently cited as a key contributor to improved mental health. Having access to GAC allowed individuals to align their physical appearance with their gender identity, significantly alleviating gender dysphoria and improving overall wellbeing. Whether through social transition, hormone therapy or surgery, participants described a strong desire for access to these services and noted poorer outcomes when care was unavailable.

Participants also emphasised the importance of "rainbow safe" counselling and trans-friendly sport and recreational activities. Access to such inclusive activities in Whanganui was acknowledged to be poor. Engaging in environments that supported their gender identity provided a sense of safety and connection. Community support—including peer connection and belonging within LGBTQI+ spaces—was described as vital to mental wellbeing. Peer support, especially in early stages of transition, offered lived experience, practical advice and emotional connection often missing in formal services. This is in line with previous Aotearoa findings.

Preferences around therapy and challenges

The interviews revealed a clear preference among transgender individuals for therapy that is affirming, accessible and delivered by someone who understands the unique challenges of being transgender. In-person therapy was valued for its direct human connection, seen as essential for building trust and rapport. However, due to the scarcity of qualified transgender therapists in rural areas, many participants turned to online sessions, which, while helpful, did not always meet their needs for personal connection and depth.

Neurodivergent participants, including those with attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD) or other cognitive differences, described additional barriers in accessing GAC. Many lacked a formal diagnosis due to fears it could jeopardise their access to GAC

or because assessments were financially out of reach. Neurodivergence shaped how participants engaged with healthcare, with challenges in planning ahead to avoid prescription lapses, attending appointments and navigating social interactions often resulting in delays or avoidance. These findings highlight the need for flexible, inclusive care pathways that accommodate the diverse needs of neurodivergent transgender individuals.

Implications for policies, legislation, and future directions

There is a need for greater investment in transgender-affirming mental health services that are accessible across both urban and rural areas. Improvements could be made by broadening telehealth access, enhancing rural clinician training and ensuring streamlined pathways to GAC without imposing unnecessary psychological assessments.[15,20]

Future research and advances in rural health practice

Further research is needed to examine how locally tailored GAC models affect mental health outcomes in rural settings. In particular, studies should investigate the impact of culturally grounded, community-led services on rates of distress, disengagement and suicidality among transgender populations. Future research should focus on evaluating sustainable mental health support models that incorporate Kaupapa Māori values, peer-led strategies and digital delivery tailored to areas with limited clinical infrastructure.

Recommendations

- **Fund and integrate affirming mental health services in rural care models:** Ensure mental health support is not treated as an adjunct, but as a core component of GAC pathways.
- **Build capacity through training and shared learning:** Develop and deliver targeted mental health education for rural clinicians, incorporating transgender-specific and culturally responsive competencies.
- **Support peer and community-based mental health initiatives:** Expand roles for peer support workers and community navigators in providing early, easy to access mental health assistance.
- **Strengthen cross-sector and iwi partnerships:** Foster collaboration between primary care, iwi providers and community groups to expand culturally grounded mental health services.
- **Prioritise service integration and sustainability:** Develop enduring rural mental health strategies that include transgender-specific pathways, informed by both clinical and lived experience.
- **Expand access to inclusive and affirming physical activity environments:** Support the development of safe, community-based spaces that enable transgender individuals to engage in physical activity without fear of discrimination or harm. Recognise the role of such spaces in promoting mental health, social connection and long-term wellbeing, particularly in regions where access to clinical support may be limited.

Conclusion

Mental health emerged as a central concern for transgender participants and clinicians in this rural study, highlighting the importance of improving accessibility, affordability and GAC. We propose a community-informed approach for improving mental health support for transgender people in rural Aotearoa. It highlights ongoing service gaps, including the limited availability of affirming, culturally appropriate care. Addressing these gaps through regional health planning, integrated service delivery and targeted mental health initiatives is essential to reducing inequities. With greater investment and collaborative planning, rural health systems can improve access to timely, affirming mental health support for transgender communities.

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KEM is the past president of Deaf Children New Zealand (August 2025).
MM is an interim committee member for New Zealand Nurses Organisation (NZNO) special interest Rainbow Nursing group.

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In vitro diagnostic devices need a robust regulatory framework

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ABSTRACT

AIMS: To discuss the regulatory scope of *Medicines Act 1981* related to point-of-care testing (POCT) *in vitro* diagnostic (IVD) devices, the implications of the now repealed *Therapeutic Products Act 2023* and the regulatory requirements which will be needed in the proposed *Medical Products Bill*.

METHODS: This review includes the relevant sections on regulation of IVD devices under the *Medicines Act 1981*, the role of Medsafe, the relevant sections of the *Therapeutic Products Act 2023*, the cabinet papers on the proposed *Medical Products Bill* and published literature on regulation for POCT devices in New Zealand and overseas.

RESULTS: IVD devices are not regulated under the *Medicines Act 1981*. Faulty devices have been supplied to health services and direct to the public. New Zealand is currently behind international regulatory standards. Cabinet papers and the proposed *Medical Products Bill* state that IVD devices should be regulated and subject to a risk classification system.

CONCLUSION: A comprehensive regulatory framework for POCT IVD devices is required to ensure the supply of high-quality devices to health services and consumers. The proposed *Medical Products Bill* must include a regulatory framework for POCT IVD devices in the interests of patient safety. Implemented wisely, the advantages of regulation outweigh disadvantages.

Point-of-care testing (POCT) devices are *in vitro* diagnostic (IVD) devices that produce medical laboratory test results near to a patient, or at the point-of-care (POC).[1,2] POCT can help improve clinical decision making by providing test results faster than conventional laboratory-based testing. Examples of POCT IVD devices include urine pregnancy test kits, rapid antigen test (RAT) kits, capillary blood glucose meters, nucleic acid amplification testing and portable or bench top multi-analyte blood analysers.

The scope and scale of POCT in New Zealand is extensive. POCT is performed in many settings including public and private hospitals, pharmacies, general practice, rural and remote settings, ambulance services, marae and schools. POCT devices are used by patients at home and in the community for many reasons, including managing glycaemia using simple glucose meters or wearable devices with result trending, alarm systems, remote monitoring and automated insulin dosing or warfarin anticoagulation. POCT is very important for decision making in rural and remote settings;[3] it can improve outcomes[4] and improve access to testing.[5]

Medical laboratory testing is essential but tests, whether laboratory-based or POCT, are associated with risk. Inaccurate or clinically unreliable test results can occur due to pre-analytical errors, e.g., incorrect specimen collection; analytical errors, e.g., the device or analyser is not properly calibrated and quality controlled; or post-analytical errors, e.g., incorrect reporting or interpretation of results. Clinical risks are amplified at the POC where testing is usually performed by non-laboratory trained individuals or health professionals, outside of the controlled medical laboratory environment, with results that are actioned swiftly. This is most relevant for results of high clinical risk, e.g., troponin or neonatal lactate results.

Conventional medical laboratory testing is governed by registered pathologists, medical scientists and technicians, using diligently selected IVD devices and tests that are, for the most part, accredited in New Zealand by International Accreditation New Zealand (IANZ). The choice of devices, platforms and tests in the laboratory is based on thorough established processes. POCT in accredited public hospital laboratories is also subject to clinical governance and oversight and is performed by trained and certified staff using verified IVD devices to minimise clinical risk.

POCT devices used outside accredited laboratory settings or supplied to the public are accessible without appropriate clinical governance, oversight or local laboratory verification. This is compounded by the fact that currently, there is no pre-market assessment or regulation even though POC tests carry no less a risk, if not more, than laboratory-based tests. It is in the public interest that these

IVD devices are subject to a national regulatory and clinical governance framework.[6,7]

This viewpoint article outlines the lack of regulation for POCT IVD devices under the *Medicines Act 1981* (*MA 1981*)[8] and the limitations of the current state, with real world examples. Now that the *Therapeutic Products Act 2023* (*TPA 2023*)[9] has been repealed, the Ministry of Health – Manatū Hauora proposed a *Medical Products Bill* to Cabinet.[10] The article calls for an effective regulatory framework for publicly funded POCT and, where relevant, direct-to-consumer (DTC) or over-the-counter (OTC) IVD devices. The regulatory implications for conventional laboratory testing, medicines, medical devices and natural health products are not discussed.

Methods

The limitations of the role of Medsafe and relevant sections on the regulation of IVD devices under the *MA 1981*[8] are discussed, with examples of faulty IVD devices in the public domain. The article also reviews relevant sections of the *TPA 2023*,[9] the cabinet papers on the proposed *Medical Products Bill*[10] and discusses literature on POCT regulation in New Zealand and overseas.

Results

The regulatory status of POCT devices in New Zealand

At the time the *MA 1981*[8] was enacted, POCT IVD devices were limited to urine glucose and ketone tests and urine pregnancy test kits. Handheld glucose meters for personal use became available internationally from the mid-1980s. The *MA 1981* includes legal definitions for the terms "therapeutic purpose" and "medical devices".[8] In terms of POCT, the interpretation of the Act is difficult. However, the definition of "therapeutic purpose" in the *MA 1981* includes some functions of POCT,[8] such as:

> *"(a) … diagnosing* [and] *monitoring* [e.g., COVID-19] …
>
> *"(c) testing the susceptibility of persons to a disease or ailment* [e.g., pre-diabetes] …
>
> *"(e) testing for pregnancy."*[8]

This legal definition of a therapeutic purpose does not imply that Medsafe regulates IVD devices. In its Regulatory Guidance, Medsafe states that IVD devices are medical devices under the *MA 1981* and must comply with requirements of the Act and its regulations.[11] It lists a range of medical devices for diagnosing or monitoring a disease, such as thermometers, heart rate monitors, medical imaging systems and urine pregnancy test kits. Medsafe also has a mechanism to report adverse events.[11]

Urine pregnancy test kits being the only POCT IVD devices listed may still not be subject to approval by Medsafe.[12] Also, Medsafe notes on its website that: *"As the pre-market legislative/regulatory requirements are minimal, this places even more responsibility on the manufacturer/importer to ensure the medical devices supplied are safe and effective."*[12]

Medsafe has a risk classification system for medical devices such as drug eluting stents or cardiac pacemakers, but as of July 2014 it states that it has no risk classification system for IVD devices,[11] which is not clinically effective. Medsafe operates the Web Assisted Notification of Devices Database (WAND). Suppliers are encouraged, but not obligated, to notify WAND of their IVDs.[13]

COVID-19 public health response point-of-care tests order 2021

On 22 April 2021 an order—"COVID-19 Public Health Response (Point-of-care Tests) Order restricting import, manufacture, supply, sale, packing and use"—was created in response to the SARS-CoV-2 pandemic.[14] The New Zealand Point of Care Testing Advisory Group (NZPOCTAG) provided advice on this order, which stated that the director-general may exempt POC tests from prohibitions provided that the "*point-of-care test or class of point-of-care tests is sufficiently accurate and reliable so as not to pose a material risk to the public health response to COVID-19*".[14] This was an exemplar of regulation limiting the use of potentially unsafe POCT hence mitigating clinical risk and supporting public health measures. The order was revoked on 28 April 2023.[14]

Adverse events management for POCT

The director-general of health has power to investigate unsafe devices under Section 38 of the *MA 1981*.[11] Examples of problematic IVD POCT devices recorded on the Medsafe website include:

1. 24 July 2020; the Yes! urine pregnancy test kit showed a high rate of false positive and inconclusive results; this test kit was

withdrawn on 2 March 2021.
2. 26 May 2016; the Easy Check pregnancy test kits, for professional use, showed an unacceptable rate of invalid (inconclusive) and false negative results.
3. 5 September 2022; recall of a batch of the KetoSens Test Strips owing to a high rate of clinically unsafe falsely elevated ketone test results.[15]

POCT devices evaluated in New Zealand and found not to be fit-for-purpose include a device for the detection of Group A *Streptococcus* in throat swabs[16] and a handheld device for testing capillary blood glycated haemoglobin (HbA1c).[17] In April 2024, at-home OTC RAT kits for chlamydia, gonorrhoea and herpes-2 were vigorously advertised and sold in New Zealand. In response, a multidisciplinary position paper was published cautioning against their use, indicating that these devices have not been verified and that overseas evidence on similar kits showed unacceptable clinical performance.[18] These examples of faulty devices demonstrate the need for regulation in New Zealand.

POCT pathologists and scientists recognise the technical and analytical limitations, and therapeutic implications associated with IVD devices. A national adverse events management system specifically for POCT devices was proposed in 2015[19] to address the need for a robust, easily accessible system to monitor trends in performance, record near-misses or clinical incidents and develop alerts where devices are found to be faulty or produce clinically misleading results. Such a system would be bolstered by connectivity to improve operator and device management, reduce transcription errors, improve safety and assist with adverse events management.[20]

IVD device regulation in other jurisdictions

Lack of a regulatory system for IVD devices in New Zealand is not consistent with other jurisdictions. Therapeutic products including IVD devices are regulated in Canada,[21] and in Australia they are regulated by the Therapeutic Goods Administration (TGA).[22]

The TGA provides guidance for manufacturers on meeting clinical evidence requirements for IVD devices and states in Essential Principle 15 that an IVD medical device must meet the "*analytical and clinical requirements to support its intended use, based on appropriate scientific and technical methods*".[23]

The TGA provides comprehensive information on the regulation of medical and IVD devices which ensures that the level of regulation is appropriate and consistent with clinical risk.[24] The risk classification system is based on the intended use and clinical risk, to person or public, associated with inaccurate results. The TGA lists classes one to four for IVD devices; the higher the clinical risk, the higher the risk classification. A pregnancy test kit is classified as class two, with a low personal health risk or associated risk level. Class three includes tests used to detect sexually transmitted disease, with a moderate public health or high personal risk, and a kit with analyser and test strips for international normalised ratio testing.[25]

The *Therapeutic Products Bill* 204-1 (2022) and the *Therapeutic Products Act 2023*

The *Therapeutic Products Bill* 204-1 was introduced to parliament on 30 November 2022.[26] The NZPOCTAG, in its 2023 submission on this bill, advised that POCT IVD devices should be regulated along with appropriate verification, quality assurance, adverse events management and recall systems.[27] The *TPA 2023* received royal assent in July 2023 and was due to be enacted by September 2026.[9]

The *TPA 2023*[9] had its limitations but was a step in the right direction. Section three stated that the purpose of the Act "*is to protect, promote and improve the health of all New Zealanders*" and in this context, provides for the "*acceptable safety, quality, and performance of medical devices across their lifecycle*".[9] Section eight stated that medical devices are therapeutic products covered by the regulatory scheme and these ranged from tongue depressors to robotic surgery machines.[9]

Section 15, of the *TPA 2023*, lists up to 11 therapeutic purposes. The following stated therapeutic purposes are relevant to this topic:

> *"(a) preventing, diagnosing, monitoring … for a disease, ailment* [e.g., COVID-19] …
>
> *"(c) testing the susceptibility of humans to a disease or an ailment* [e.g., human papilloma virus] …
>
> *"(e) testing for human pregnancy …*
>
> *"(g) investigating a human physiological process …* [e.g., diabetic ketoacidosis]."[9]

The *TPA 2023* requires the regulator to evaluate a medical device to determine its safety and fitness-for-purpose and its likely benefits and risks.[9] It includes provisions for recall orders for faulty devices and states that the regulator can designate a testing entity to carry out tests on therapeutic devices.[9] These sections are consistent with the advice provided in the NZPOCTAG submission on this bill.[27]

The new coalition government revoked the Act within the first 100 days of government. The cabinet paper CAB-24-MIN-0154 presented on 23 May 2024 states that reasons for repeal were to improve timely approval for medicines, avoid over regulation of natural health products and low-risk medical devices and that the regulatory framework would be cost-prohibitive for exporters.[28]

Ministry of Health – Manatū Hauora Social Outcomes Committee proposed *Medical Products Bill*

On 24 September 2024, a Cabinet Social Outcomes Committee paper titled "Modernising the Regulation of Medicines and Medical Devices"[10] authorised the associate minister of health to issue drafting instructions for a *Medical Products Bill* to replace the *TPA 2023*.[10] The proposed Bill covers medicines and medical devices and recognises the regulatory differences between them. The purpose of the Bill is to improve health outcomes by enabling access to effective medical products, which must meet acceptable standards of safety, quality and clinical efficacy or performance.[10] An example of a regulatory and authorisation schema for IVD POCT devices in New Zealand was proposed in 2019.[6]

The proposed *Medical Products Bill* includes a risk proportionate regulatory framework and is harmonised with international good practice.[10] The framework included the use of assessments and decisions from trusted overseas regulators. Medical devices range in complexity and risk; devices which expose staff or patients to minimal risk would have minimal regulation.[10]

The cabinet paper notes that "*clinically worthless tests are being sold to patients at chemists including tests for sexually transmitted diseases*".[28] These test kits are not publicly funded, but in the interest of public health, government should assume a role in regulating them.

The proposed Bill would regulate advanced technology, such as artificial intelligence or software as a medical device (SaMD), as medical products.[10] An example of a SaMD was the QUiPP App, a cell phone–based application. This application is used in conjunction with clinical parameters and POCT results for the prediction of preterm labour. In this context, the QUiPP App is recognised as a medical device. However, it is essential that POC IVD devices used to produce quantitative results (e.g., in ng/L), are also subject to appropriate regulation, together with clinical governance and quality assurance, to ensure the actual test results are clinically reliable for use with the QUiPP App.

Discussion

The *MA 1981* does not provide a regulatory framework for POCT IVD devices. The scope, scale and complexity of POCT has increased dramatically since the 1980s; the regulatory framework needs to reflect these advancements. Public hospitals and health services may not be aware that publicly funded health IVD devices have not been subject to pre-market assessment, are not required to be notified to the WAND database and have no risk classification. Since the repeal of *TPA 2023*,[9] New Zealand has been left with a regulatory void for POCT devices.

There are currently significant limitations to the extent of oversight for medical devices. Relying on manufacturers to ensure safety of devices and tests is not satisfactory due to commercial conflict of interest, lack of transparency of information supplied, and known and unknown limitations in clinical performance data supplied by the manufacturer.

Clinically reliable POC test information relies on carefully selected devices and tests that are fit for purpose in the intended clinical setting. From a best practice perspective, regulation is inextricably linked with risk management. It is the first tier of risk mitigation that supports clinical governance performed at the healthcare provider level. This assumes higher importance considering the pressures and limited resources in the health sector. By reducing the availability and use of unreliable and potentially unsafe devices and tests, regulation can reduce the risk of misdiagnosis or inappropriate management, the risk of serious harm, and healthcare costs.

Previous experience with faulty IVD devices suggests that the regulator must have the legislated power to initiate recalls and request additional verification and reassessment of device performance. To that end, a user-friendly accessible

adverse events management system needs to be implemented.[19]

Like any decree, while regulation has advantages, it would have disadvantages. Table 1 lists pros and cons of regulating POCT IVDs and proposes risk mitigating measures. The list is not exhaustive.

The regulatory process should be transparent to allow policymakers to review and improve the process. Regulation should be accompanied by regular clinical audits and research to facilitate continuous improvement.[29]

Harmonisation and alignment with international regulatory frameworks could avoid inadvertent over regulation of IVD devices. New Zealand can learn from these countries, and we in turn can share our expertise with IVD devices and POCT.

Right four of the Code of Health and Disability Consumers' Rights 1996[30] states that consumers/patients have the right to reasonable standards of care, in this case accurate POCT results. Clinical staff must be able to rely on the accuracy of POCT information for decision making and in turn inform and communicate this test information to the patient in accordance with right five and right six of the code.[30]

Where possible, the regulator should ensure that more than one brand or model of an IVD device with reliable analytical performance are selected so that in the event of a recall, manufacturing problem or global logistical supply

Table 1: Pros and cons of regulating POCT IVDs and risk mitigating measures.

Advantages	Disadvantages, risks and mitigation measures
Improved safety of devices, particularly high-risk devices, and tests.	Risk of overregulation inhibiting competition and innovation, reducing consumer choice, and potentially falling behind international standard of care. **Mitigation:** risk-appropriate regulation.
Supports better health outcomes.	
Aligns with international regulatory frameworks.	Need to resource the regulatory machinery, which if under resourced can create an inefficient system with no true benefits. **Mitigation:** risk-appropriate regulation, scoping, building on existing systems.
Supports clinical governance of POCT in the community and in hospitals.	
Supports funding bodies fund devices (and tests) that are fit for purpose, using tax-payer money judiciously.	
Complements pro-equity measures.	Potentially increased cost to manufacturers and suppliers.
Aligns with Code of Health and Disability Services Consumers' Rights, in particular right four: "Right to services of an appropriate standard".	Potential delays in availability and access to the New Zealand consumer/s. **Mitigation:** an agile and responsive regulatory system.
Informs commercial entities, such as pharmacies, deciding on choice of devices and tests.	
Gives the clinicians and consumers a measure of confidence.	Potentially disproportionate delays in availability and access of devices/tests for rare disorders. **Mitigation:** an agile and responsive regulatory system.
Improved traceability and supply chain management.	
Maximises efficiencies in the healthcare sector, which has broader societal benefits.	Research and development leaving New Zealand. **Mitigation:** support innovation within an accountable and transparent regulatory system.
Patient-centric focus, which is responsive to unmet needs of New Zealanders	

problem, an alternative device is available. In the past year there have been two disruptions to the supply of the foetal fibronectin (fFN) test kits, used in the prediction of preterm labour, due to manufacturing issues. Obstetric units replaced fFN with one of two alternative tests in New Zealand. Addressing disruption to supply chains for medical devices was particularly relevant during the COVID-19 pandemic.

A regulator must have overall responsibility for the regulation of publicly funded IVD devices and, where appropriate, DTC and OTC IVD devices, with advice from POCT experts.[6] Purchasing and on-going funding of POCT devices is not a simple monetary decision. In the absence of regulation, health providers and users of POCT devices are encouraged to be vigilant and critical and consult with their local accredited laboratory for advice. The NZPOCTAG New Zealand Best Practice Guidelines for Point-of-Care Testing is also a valuable resource to ensure safety, quality and clinical and operational effectiveness.[2]

In conclusion, the current regulatory state for IVD devices is unsafe, does not serve our clinicians and consumers, is not aligned with international standards and fosters a reactive approach. Structured risk-based regulation would be a huge milestone for New Zealand, and with a considered approach its risks can be mitigated, if not avoided. This viewpoint articulates measures that would help establish an effective regulatory framework for POCT IVD devices that is tailored to the population's needs.

COMPETING INTERESTS
Nil.

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Non-traumatic rupture of the gluteus medius associated with fluoroquinolone use: a case report

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Fluoroquinolones are commonly prescribed for the treatment of bacterial infections due to their efficacy and broad antimicrobial spectrum. However, their use is associated with rare but serious adverse events, including tendinopathies and atraumatic musculotendinous ruptures.[1,2] Studies indicate that the risk of tendon injury is increased by up to 41.9% within the first 30 days of fluoroquinolone therapy compared with other antimicrobial classes.[3] Although Achilles tendon ruptures are the most frequently reported, accounting for approximately 90% of cases,[4] this case report presents a rare and previously undocumented adverse reaction: a partial rupture of the gluteus medius tendon associated with ciprofloxacin use, with no prior cases identified in the literature to date. This unusual presentation broadens the recognised spectrum of fluoroquinolone-related musculoskeletal injuries and underscores the importance of clinical vigilance for early diagnosis and appropriate management.

Methods

The present work is characterised as an observational and descriptive study in the form of a case report. This report has been prepared in accordance with the principles of the Consensus-based Clinical Case Reporting (CARE) guidelines.

Case report

A 41-year-old Brazilian woman, eutrophic, presented with sudden and severe pain in the left sacroiliac and gluteal region, radiating to the posterior thigh, which began on 3 March 2024. The pain was described as an incapacitating "twinge", not associated with any trauma, fall or physical exertion. The patient also reported associated paresis in the left lower limb and antalgic claudication, with significant functional impairment.

Her medical history included thrombophilia, polycystic ovary syndrome, endometriosis and nephrolithiasis. She denied any allergies, smoking or alcohol consumption. In October 2023, 5 months prior to the current event, she had undergone cervical discectomy and arthrodesis for disc protrusion with radicular compression, treated with corticosteroids and physiotherapy. Crucially, at the time of the gluteal pain onset, she was still in the final phase of her post-operative recovery and had been instructed to avoid physical activities, which reinforces the absence of a mechanical trigger for the injury. The patient denied any personal or family history of primary osteoarticular disorders.

With regard to her recent history, the patient had completed a 5-day course of ciprofloxacin 500mg twice daily on 18 February 2024 for a urinary tract infection. Fourteen days after completing the treatment, the painful condition began.

On 4 March 2024, due to worsening pain and functional limitation, she sought emergency care and was assessed by a neurosurgeon. On physical examination she was haemodynamically stable and scored 15 on the Glasgow Coma Scale. Localised pain was noted in the left gluteal region and posterior thigh, with no signs of radicular compression on the Lasègue manoeuvre.

Magnetic resonance imaging (MRI) of the left hip was requested, revealing a partial tear at the insertion of the gluteus medius tendon at the greater trochanter, associated with trochanteric bursitis (Figure 1).

Based on the clear temporal correlation with the antibiotic use, the absence of other evident causes and the MRI findings, a diagnosis of fluoroquinolone-induced tendinopathy (FIT) was established. The causal relationship between ciprofloxacin use and the development of tendinopathy was supported by the Naranjo Adverse Drug Reaction Probability Scale,[5] a validated tool

Figure 1: Magnetic resonance imaging (MRI). a) Coronal slice, short tau inversion recovery (STIR) weighted sequence, showing hyperintensity in the gluteal region (as marked), predominantly involving the gluteus medius and minimus muscles, with extension to adjacent musculature. b) Axial T2-weighted sequence, demonstrating hyperintensity and partial discontinuity of the tendinous fibres at the trochanteric insertion of the posterosuperior portion of the gluteus medius tendon (indicated by the arrow), associated with trochanteric bursitis.

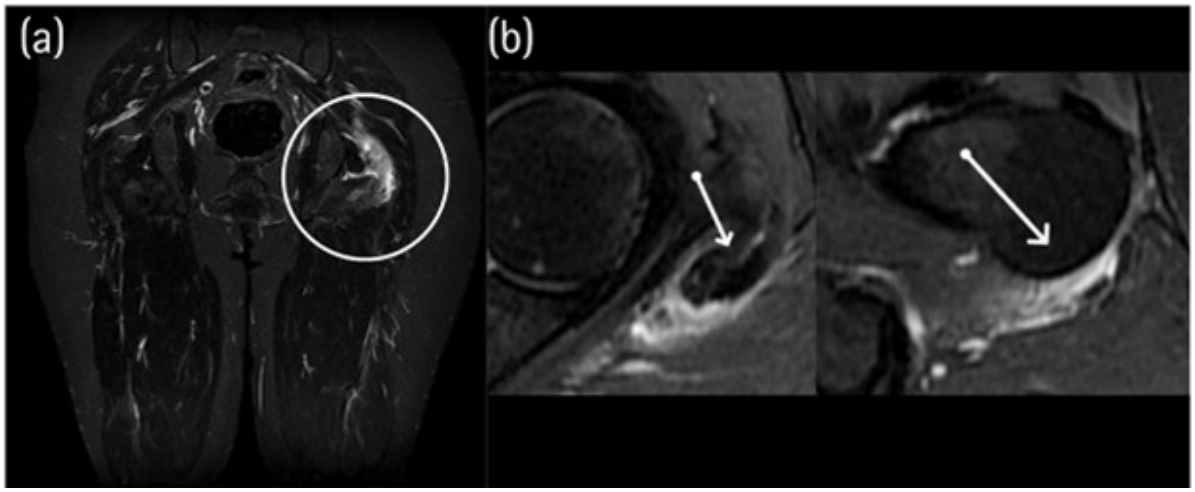

Table 1: Naranjo Adverse Drug Reaction Probability Scale.[5]

Naranjo score				
	Yes	**No**	**Do not know**	**Patient score**
1. Are there previous conclusive reports on this reaction?	+1	0	0	+1
2. Did the adverse event appear after the suspected drug was administered?	+2	−1	0	+2
3. Did the adverse reaction improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	0
4. Did the adverse reaction reappear when the drug was readministered?	+2	−1	0	0
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	−1	+2	0	+2
6. Did the reaction reappear when a placebo was given?	−1	+1	0	0
7. Was the drug detected in the blood (or other fluids) in concentrations known to be toxic?	+1	0	0	0
8. Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	+1	0	0	0
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	+1	0	0	0
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	+1
Total score				6

used to assess the likelihood of an adverse drug reaction compared with alternative explanations (Table 1). In this case, the calculated score was 6, indicating that the adverse reaction was considered probable.

On 5 March 2024, the patient was assessed by an orthopaedic specialist, and complementary laboratory investigations revealed no significant abnormalities. Treatment included analgesics, anti-inflammatory medication, the use of a supportive orthosis for 1 week and physiotherapy aimed at pain relief and functional recovery. The patient demonstrated good adherence to the prescribed regimen, which contributed to significant clinical improvement, with progressive pain reduction and full restoration of strength and mobility. She was able to resume her daily activities without limitations or recurrence of symptoms.

Discussion

FIT was one of the first adverse effects identified following the administration of this class of antimicrobials. It is characterised by acute tendon injury or rupture, most commonly affecting the Achilles tendon. This condition may lead to severe and permanent disability, representing a significant clinical challenge.[6–8] Since its initial description in 1983 as a rheumatic disorder induced by norfloxacin,[9] FIT has been extensively studied and has drawn regulatory attention from agencies such as the United States Food and Drug Administration (FDA).[10,11]

FIT should be considered in any patient presenting with new musculoskeletal symptoms and a history of fluoroquinolone use within the preceding 6 months.[12] These antimicrobials increase the risk of acute tendinopathy by a factor of two to four, with an incidence of up to 2% among patients aged ≥65 years. For Achilles tendon involvement specifically, the incidence is estimated at three to four cases per 100,000 people.[6,13]

The onset of symptoms most commonly occurs within the first month following exposure to the antimicrobial, with a median latency period of 6 to 14 days,[11,14] as observed in our patient (14 days), reinforcing the temporal association. However, symptoms may also emerge weeks to months after treatment discontinuation.[4,8]

Risk factors associated with an increased incidence of FIT include concomitant corticosteroid use (associated with up to a 14-fold increased risk),[15,16] age over 60 years, renal insufficiency, cardiovascular disease, solid organ transplantation, rheumatic disorders, diabetes mellitus, hyperparathyroidism, obesity, lipid metabolism disorders, participation in sports, hypothyroidism, duration of fluoroquinolone therapy and high dosages.[17,18]

However, our case did not involve any classic risk factors, which highlights the importance of considering FIT even in patients without comorbidities. Although the patient had previously received corticosteroids, their discontinuation prior to symptom onset reduces the likelihood of a direct contribution—despite their known impact on collagen synthesis and tendon regeneration in the long term. Furthermore, our case supports existing literature indicating a higher prevalence of FIT among biological females.[8,16,19] Regarding risk factors, it is noteworthy that the patient had a history of corticosteroid use 5 months prior. Although not concomitant, prior exposure to corticosteroids is known to alter collagen synthesis and tendon structure, which could have acted as a predisposing factor, synergistically increasing the patient's susceptibility to the fluoroquinolone's toxic effects.[8,16,19]

Although 90% of cases involve the Achilles tendon, other tendons may also be affected.[4,6,10] While there are reports of gluteus medius tendinopathy associated with ciprofloxacin use,[7,12] an extensive literature review suggests that rupture of the gluteus medius tendon has not previously been described, as observed in our patient. This may be attributed to insufficient recognition or under-reporting of FIT cases.

Fluoroquinolones exhibit favourable pharmacokinetics, with good oral absorption and wide tissue distribution owing to low plasma protein binding. These properties enable the drugs to reach high concentrations in muscle and tendon tissues, particularly via the myotendinous junction, resulting in substantial exposure of tendinous structures to elevated antimicrobial levels.[2]

The exact pathogenesis of FIT remains unclear. However, five main mechanisms have been proposed: inhibition of tendon cell proliferation through cell cycle arrest at the G2/M phase; reduced tenocyte migration due to decreased phosphorylation of focal adhesion kinase; diminished type I collagen production as a result of increased matrix metalloproteinases; iron chelation impairing collagen synthesis; and oxidative stress induced by reactive oxygen species.[4,6,14,20,21]

Typical symptoms of FIT include sudden and acute pain, localised tenderness and pain on movement of the affected area, often accompanied by functional impairment and loss of strength. Inflammatory signs may also be present.[18,21,22] In our case, the patient presented with abrupt, severe pain, associated with strength loss and worsening during movement of the affected limb, without any identifiable triggering factors such as previous trauma.

The diagnosis of tendinopathy is primarily clinical but can be supported by imaging studies such as ultrasound or MRI. As in our patient's case, MRI can reveal findings consistent with tendinopathy or musculotendinous avulsion, such as T2 hyperintensity, tendon thickening and architectural distortion, indicating a recent inflammatory process compatible with an acute injury. Furthermore, the absence of signs of chronicity on the imaging—such as tendon retraction, muscle atrophy or calcifications—reinforces the hypothesis of a recent and acute event.[23,24]

The main diagnostic challenge in this case lies in distinguishing an acute drug-induced injury from a pre-existing chronic rupture of the gluteus medius tendon. While partial-thickness tears of the gluteus medius are more prevalent in middle-aged and older women, they are predominantly symptomatic, with asymptomatic cases being relatively rare. In a large cross-sectional MRI-based study, only 1.7% of asymptomatic hips presented with partial-thickness tears, and no full-thickness tears were identified without symptoms. Moreover, most under-surface partial-thickness tears are difficult to detect clinically and radiologically, often requiring direct visualisation through advanced endoscopic techniques. These findings, combined with the acute onset and clinical course in our case, argue against a chronic degenerative process and instead support the hypothesis of an acute adverse event potentially associated with fluoroquinolone exposure.[25,26]

First, the sudden onset of intense pain and the presence of significant functional impairment are not typical features of chronic degenerative tendinopathy and are more consistent with an acute rupture. Second, the absence of mechanical triggers—such as trauma or intense physical exertion—further supports this interpretation. Additionally, the patient had no history of musculoskeletal comorbidities, no previous locomotor deficits in the affected region and no prior episodes of hip pain. Lastly, symptom onset occurred exactly 14 days after the completion of ciprofloxacin therapy—a latency period consistent with that described in the literature for FIT—which increases the plausibility of a drug-related aetiology in this case.

The likelihood of an adverse drug reaction and the causal relationship between fluoroquinolone use and the development of tendinopathy can be evaluated using the Naranjo Adverse Drug Reaction Probability Scale.[5] In the present case, this tool yielded a score of 6, classifying the event as a "probable" adverse reaction. This score was derived from the positive identification of FIT as a previously documented reaction (+1), the correct temporal sequence between drug administration and symptom onset (+2) and objective confirmation of the tendon tear by MRI (+1).

A crucial score of +2 was assigned for the absence of alternative aetiologies (question 5), a determination based on a thorough clinical assessment. Specifically, we identified no alternative cause that could, on its own, explain the event due to: 1) the hyperacute, non-traumatic onset of debilitating pain; 2) the patient's explicit lack of physical exertion, as she was in a post-operative recovery period from cervical surgery; and 3) the absence of pre-existing musculoskeletal comorbidities or prior history of hip pain. While acknowledging the prevalence of asymptomatic degenerative tears in this demographic, the combination of a plausible pharmacological trigger acting within a known latency period and the lack of any identifiable mechanical cause renders FIT the most compelling and parsimonious diagnosis.

When symptoms such as pain and tendon inflammation arise, it is crucial to discontinue the antimicrobial and avoid physical activity involving the affected limb for a period of 2–6 weeks.[27] Non-surgical treatments, including analgesics, physiotherapy, immobilisation, orthoses and rest, should be tailored to the severity of the injury.[28] Surgical repair is a viable option for appropriately selected candidates.[29] In our case, the patient, no longer exposed to the antimicrobial, was treated with analgesics, anti-inflammatory medication, orthoses and physiotherapy, achieving full recovery. This typically occurs within a mean period of 1–2 months, although long-term sequelae have been reported in approximately 10% of patients.[18] To prevent recurrence, we advised lifelong avoidance of fluoroquinolones, as recurrent cases have been documented.[30]

Our case underscores the need to consider FIT as a differential diagnosis in patients presenting with musculoskeletal complaints and a history

of fluoroquinolone use within the preceding 6 months. It also highlights a previously undocumented complication: partial rupture of the gluteus medius tendon.

Conclusion

FIT is a serious adverse reaction associated with considerable morbidity and functional impairment. This case report presents a novel instance of gluteus medius tendon rupture, thereby expanding the recognised spectrum of musculoskeletal injuries attributed to this class of antimicrobials, which are traditionally associated with Achilles tendon rupture. The objective is to underscore the importance of clinical vigilance and prompt diagnosis, considering FIT as a differential diagnosis in patients presenting with musculoskeletal symptoms and recent fluoroquinolone exposure. Recognising the potential involvement of less commonly affected structures is essential for optimising management and preventing further complications.

COMPETING INTERESTS
The authors declare that they have no conflicts of interest related to the content of this manuscript.

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Ethical approval to report this case was obtained from the Research Ethics Committee of the University Centre for the Development of the Alto Vale do Itajaí (UNIDAVI), under protocol number 7.034.154, on 27 August 2024. The patient provided written informed consent for the use of her medical data and imaging in this case report. No identifiable information has been included in the submitted manuscript. All efforts were made to ensure the patient's privacy and anonymity, and no data or images that could compromise her identity have been presented.

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Pancreatic fallout: autoimmune pancreatitis post-mRNA COVID-19 vaccination

Justin Koh, Owain Blackwood, Bernard McEntee, Michael A Park, Grant Cave, Frank Weilert, Debra A Chalmers, Ariel Drori

Autoimmune pancreatitis (AIP) is a distinct and rare form of pancreatitis, typically presenting with obstructive jaundice, weight loss and abdominal pain. Diagnosis is based on clinical evaluation, biochemical and radiological investigations, histological findings and, in some instances, the response to glucocorticoids.[1]

During the COVID-19 pandemic, New Zealand began vaccinating at-risk groups with the Pfizer-BioNTech Comirnaty mRNA vaccine in February 2021. Although phase-II/III trials (~44,000 participants) demonstrated safety, very rare effects were unable to be excluded and may only come to light during post-marketing surveillance.[2]

Emerging case reports and small series suggest a temporal association between this mRNA vaccine and new-onset autoimmune manifestations.[3,4]

We describe the first Australasian case of autoimmune pancreatitis developing shortly after a second Pfizer vaccine dose.

Case report

A 42-year-old previously healthy female healthcare worker received a second Pfizer mRNA COVID-19 vaccine in April 2021. Four days later, she developed abdominal pain and malaise. After multiple primary care visits, she presented to the emergency department in May 2021 with 6 weeks of worsening pain, early satiety and weight loss.

An abdominal computed tomography (CT) scan showed inflammatory changes of the pancreatic head in keeping with focal pancreatitis (Figure 1). Biochemical results were remarkable for serum lipase 212U/L (0–70U/L), AST 237U/L (10–50U/L), ALT 249U/L (0–30U/L), CRP 26mg/L (<5mg/L). Serum bilirubin, ALP and electrolytes were within normal limits. SARS-CoV-2 RNA real-time PCR was negative.

Endoscopic ultrasound (EUS) in June 2021 showed a diffusely bulky, inflamed pancreas without ductal dilatation. EUS-guided fine needle biopsy revealed lymphoplasmacytic infiltrate and perilobular fibrosis with no malignancy. IgG4 staining was negative.

High-dose corticosteroids were initiated with rapid symptom relief. Follow-up imaging and blood tests confirmed complete radiological and biochemical remission, fulfilling international AIP diagnostic criteria.[1] A likely aetiology was the Pfizer-BioNTech Comirnaty mRNA vaccine due to temporal association and absence of other concomitant autoimmune disease or infectious triggers, including COVID-19 infection.

Discussion

AIP after COVID-19 vaccination is rare but increasingly recognised. To our knowledge, only eight similar cases have been published to date. Among these, Patel et al. describe a 63-year-old man who developed type 1 AIP, 2 months after mRNA-based COVID-19 vaccination.[5] The patient presented with weight loss, fatigue and insulin-dependent diabetes. Treatment with glucocorticoids led to complete clinical and radiological remission after 6 weeks of therapy.

Rodrigues et al. report a case of a 65-year-old man who developed seronegative type 1 AIP 2 weeks following Pfizer-BioNTech COVID-19 vaccination.[6] Despite normal serum IgG4 levels, the patient exhibited extra-pancreatic manifestations and achieved complete biochemical and radiological remission within 6 weeks of initiating glucocorticoid therapy.

Surveillance data suggests COVID-19 vaccine-associated autoimmune events occur in ~1.5 per 100,000 vaccine recipients.[7] Given its rarity, it is unsurprising that only isolated case reports have temporally associated AIP to COVID-19 vaccination.

The presumed mechanism is molecular mimicry combined with the immune response triggered by the vaccine.[4,8] Structural similarities between the

Figure 1: Abdominal computed tomography (CT) demonstrating findings of focal pancreatitis. Arrow pointing toward inflamed head of pancreas.

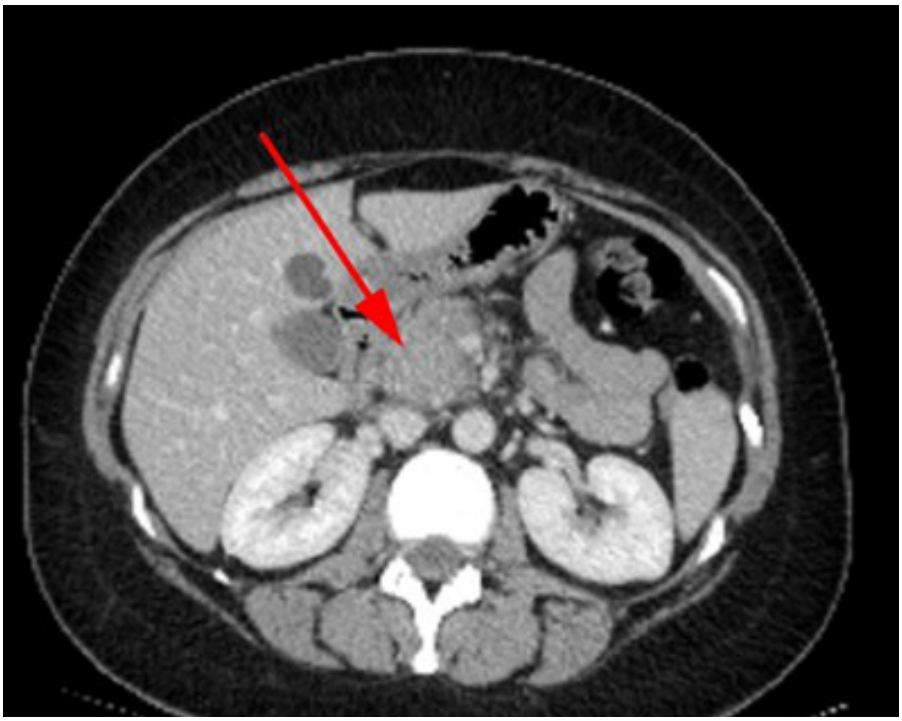
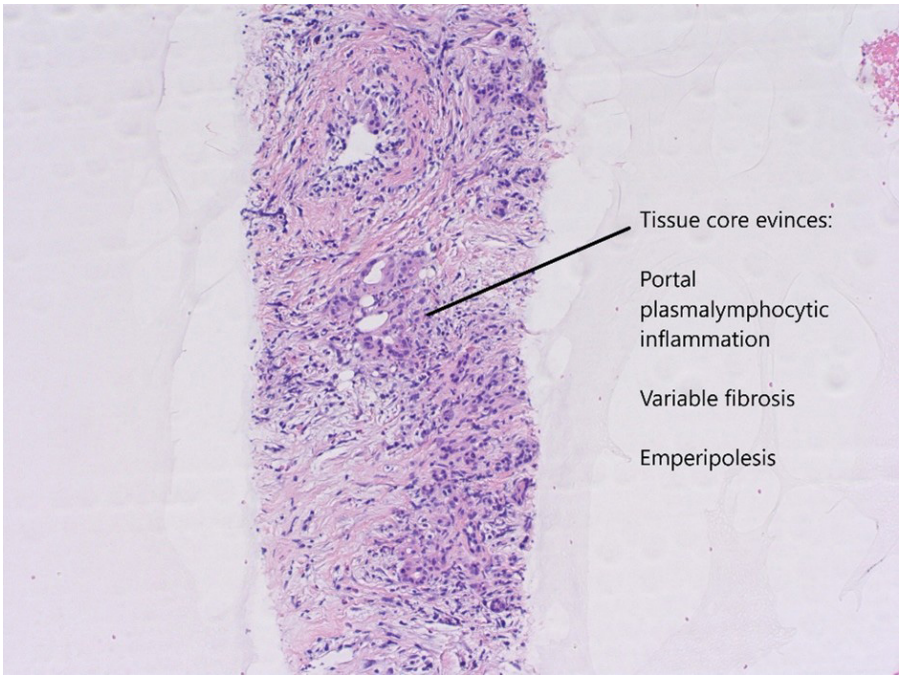


Figure 2: Histological section (Haematoxylin and eosin stain, x200 magnification) of pancreatic head core biopsy, showing features in keeping with autoimmune pancreatitis, particularly portal plasma lymphocytic inflammation and variable fibrosis.



SARS-CoV-2 spike protein (expressed after mRNA vaccination) and host peptides may activate T cells and B cells, reducing tolerance to self-antigens and triggering organ-specific inflammation.[8,9] A COVID-19 mRNA vaccination may therefore deliver a potent innate stimulus for spike antigen, promoting the pathogenesis of autoimmune pancreatitis.

This mechanism mirrors reports of AIP after natural SARS-CoV-2 infection,[3] lending biological plausibility to a shared pathway between infection and vaccination in autoimmune disease.

Certain HLA genotypes are associated with AIP, both in the Japanese and Caucasian population.[10] However, genetic susceptibility for vaccine-associated AIP is unknown.

Conclusion

This case adds to the small amount of data demonstrating a temporal association between new onset AIP and COVID-19 vaccination. Clinicians should remain aware of this potential association in order to facilitate earlier diagnosis and management. Continued surveillance and detailed case reporting are essential to better understand the epidemiology and potential mechanisms of vaccine-associated autoimmune conditions.

COMPETING INTERESTS
Nil.

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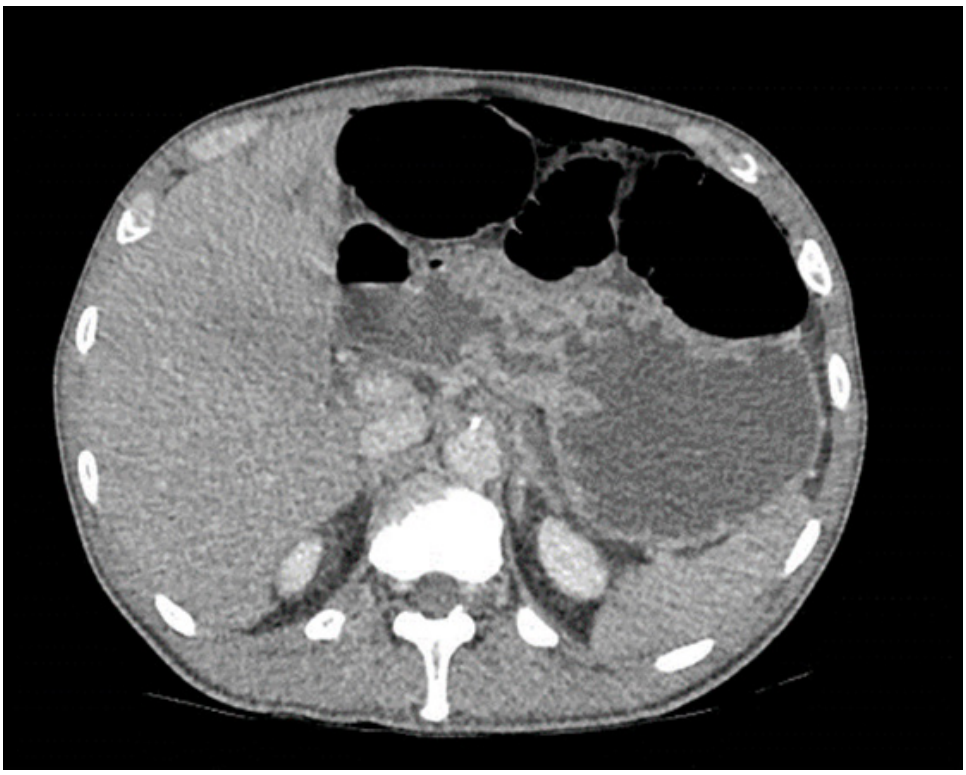
A rare case of localised gastrointestinal vasculitis in a New Zealand patient

Josef Templeton, Clare French

A 51-year-old man with *JAK2* positive essential thrombocythaemia diagnosed a year prior, presented to Wairarapa Hospital Emergency Department with severe right sided abdominal and flank pain with a history of 3 months of similar pain. He had been discharged from Wellington Regional Hospital 5 days prior. He also presented with 13kg of weight loss over 3 months and watery diarrhoea. On physical examination he was tender to abdominal palpation and was found to be in urinary retention. An indwelling catheter was subsequently placed with moderate relief. Initial blood tests showed raised inflammatory markers (WBC 26x10^9/L, Neutrophils 23x10^9/L, CRP 94mg/L), an acute kidney injury (Creatinine 134mcmol/L), raised platelets (1700x10^9/L, which were felt to be reactive) and a normal lactate on venous blood gas. Computed tomography (CT) imaging showed non-specific dilation of the small bowel and no specific cause for his symptoms was identified. He was admitted to hospital for analgesia and observation.

Later that night, despite intravenous analgesia, the patient had further escalation of his abdominal pain. On physical examination he had developed gross peritonism and a venous blood gas revealed a lactate of 8.0mmol/L. He proceeded to an urgent CT-abdominal angiogram. This showed new free air and fluid, but with no clear point of the perforation (Figure 1). No large vessel thrombus or emboli was identified; however, it was noted that the SMA was attenuated with no filling defect.

Figure 1: CT-Angiography showing bowel perforation.



The patient proceeded to the acute operating theatre for an explorative laparoscopy identifying ischaemic bowel in the terminal ileum. A lower midline laparotomy incision was made, and he was found to have an ischaemic ileum. His ileum had multiple ulcerative lesions, with two areas of full thickness perforations (Figure 2). Pus and small bowel contents were seen in the abdomen and pelvis. Scattered luminal ulcerative disease was also seen in the proximal ileum and jejunum, and this region of bowel was dilated but pink and alive. The transition point between ischaemic and healthy bowel can be seen in the intraoperative photo (Figure 3). The terminal ileum was resected with staples. As the surgeon proceeded to forming a loop ileostomy, the patient became progressively hypotensive with increasing noradrenaline requirements and then became unstable in a ventricular tachycardia. Ileostomy formation was abandoned, and the bowel was left stapled in discontinuity with a nasogastric tube *in situ*.

The patient was transferred via emergency flight to Wellington. He was admitted to the intensive care unit (ICU) and reviewed by the Wellington surgical team. On arrival he was in profound septic shock with his lactate rising to 15mmol/L. Laboratory tests on arrival showed multi-organ failure with further acute kidney and now liver injury (Creatinine 250mcmol/L, ALT 5722U/L). A repeat CT scan with arterial contrast showed multiple ischaemic infarcts with no arterial thrombus or embolism, suspected to be from hypoperfusion. The Wellington surgical team decided to proceed for an urgent re-look explorative laparotomy. This operation started 12 hours after the initial laparotomy. Exploration revealed extensive solid organ, large bowel and small bowel ischaemia. The surgical team deemed this ischaemia unsurvivable, and the abdomen was closed. Upon returning to the ICU, the operative findings were discussed in a meeting with his whanau, the ICU team and the surgical team. He was then extubated and died later that night.

Within the 3 months leading up to his death, this patient had undergone extensive work-up for his abdominal symptoms. These symptoms initially began with upper abdominal pain, diarrhoea and weight loss. He visited his GP twice; at first he was given a trial of oral antibiotics, and then on the second occasion was referred to the emergency department (ED). In ED he was clinically evaluated with blood tests and a CT scan. The only remarkable finding was a raised lipase (418U/L) which was felt to be non-diagnostic. He was discharged and referred to outpatient gastroenterology. His symptoms continued to progress and over the following 2 months he had a further ED presentation as well as a second normal CT scan, and he was discharged to the community with no diagnosis identified.

Following concern from his haematologist about the severity of ongoing symptoms, the patient was admitted to Wellington Regional Hospital to facilitate work up. Wide infective, autoimmune, gastrointestinal and haematological panels were unremarkable. Notably, faecal calprotectin was normal at 33ug/g lowering the suspicion for IBD. The patient underwent colonoscopy, gastroendoscopy and CT enterography with findings as follows. Colonoscopy macroscopically found diffuse mild inflammation of the ileum with sparing of the terminal ileum (Figure 4). Histology suggested active mild ileal inflammation. His colon was normal on endoscopic appearance and showed lymphocytic colitis on biopsy. Upper gastrointestinal endoscopy was normal on endoscopic appearance and biopsy. CT enterography was limited by suboptimal bowel distention, but no gross abnormalities were seen despite this.

Given these findings were not typical for Crohn's disease, the differential included gastrointestinal vasculitis, and the patient was briefly started on IV methylprednisolone after his colonoscopy. Vasculitis screening was non-specific: ANA was positive (titre of 1:1320 with homogenous pattern) but the remaining panel including ENA, anti-dsDNA, anti-GBM antibodies and ANCA were normal. Both haematology and gastroenterology noted that the patient's symptoms were disproportionate to that expected in lymphocytic colitis. However, given minimal improvement on IV methylprednisolone he was swapped to oral budesonide, and his omeprazole changed to famotidine due to the possibility of medication-induced lymphocytic colitis. With a subsequent improvement to his pain and bowel motions, the patient was discharged on oral budesonide and analgesics.

Five days after this discharge from Wellington Regional Hospital, the patient re-presented to Wairarapa ED, with the presentation as described above, and passed away. After his death, histology from the resected terminal ileum was reviewed and identified small artery vasculitis in the terminal ileum.

Figure 2: Operative specimen photo showing bowel perforations in ischaemic ileum.

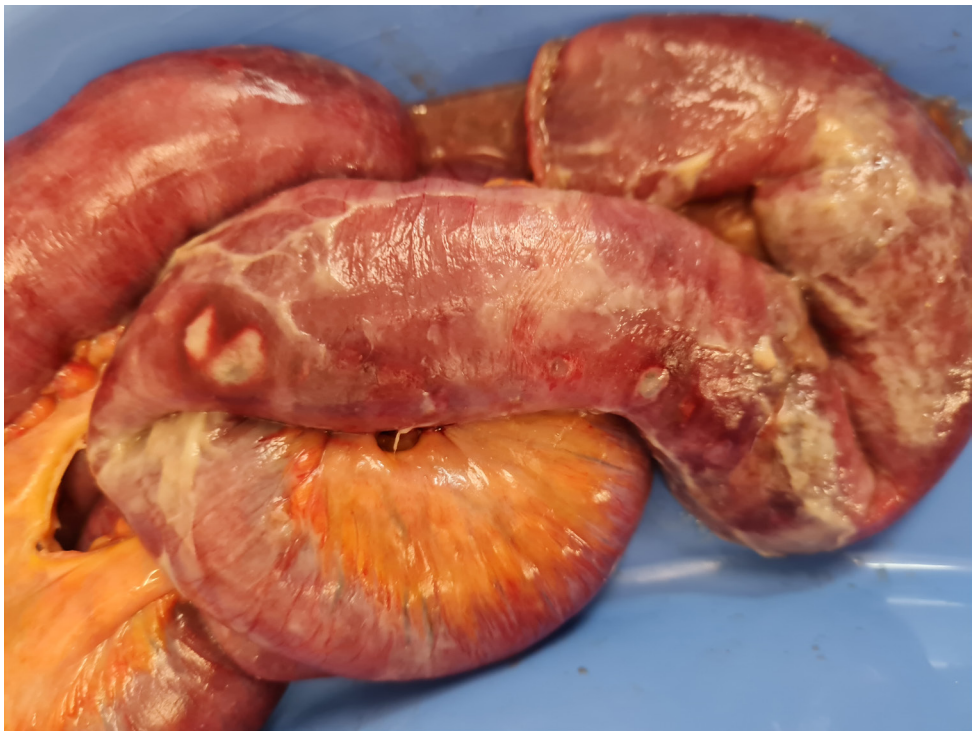


Figure 3: Transition point of ischaemic—dilated bowel.

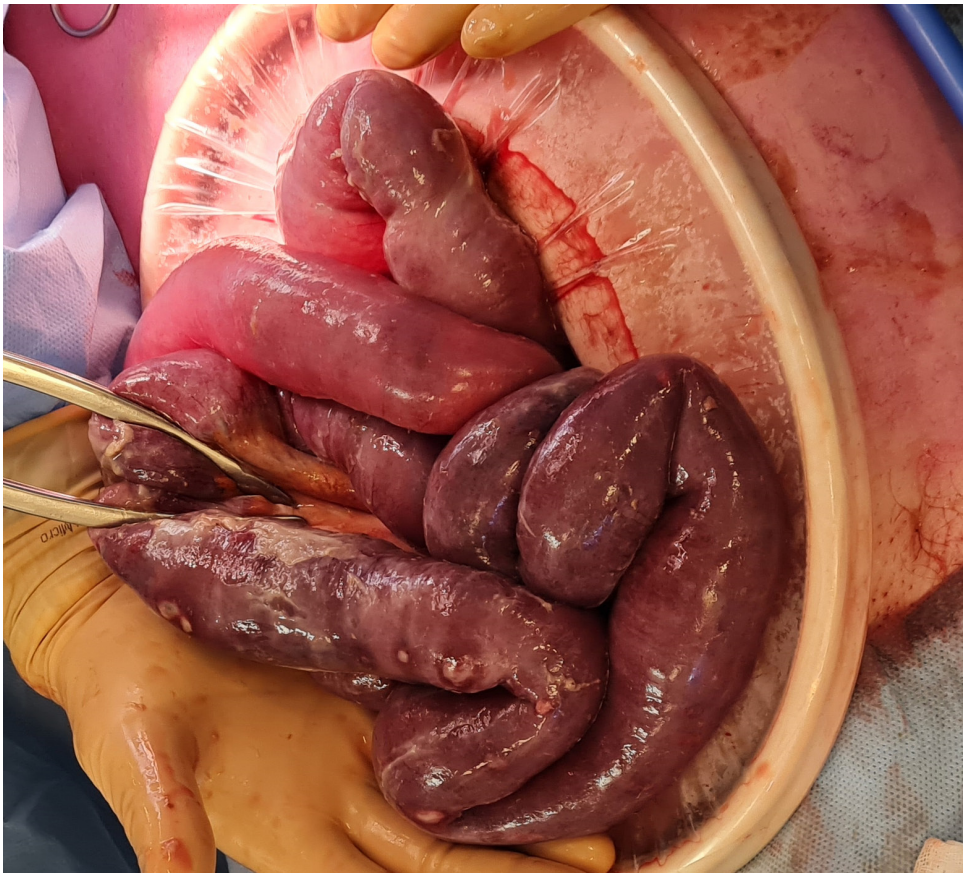


Figure 4: Colonoscopy showing mild ileitis.

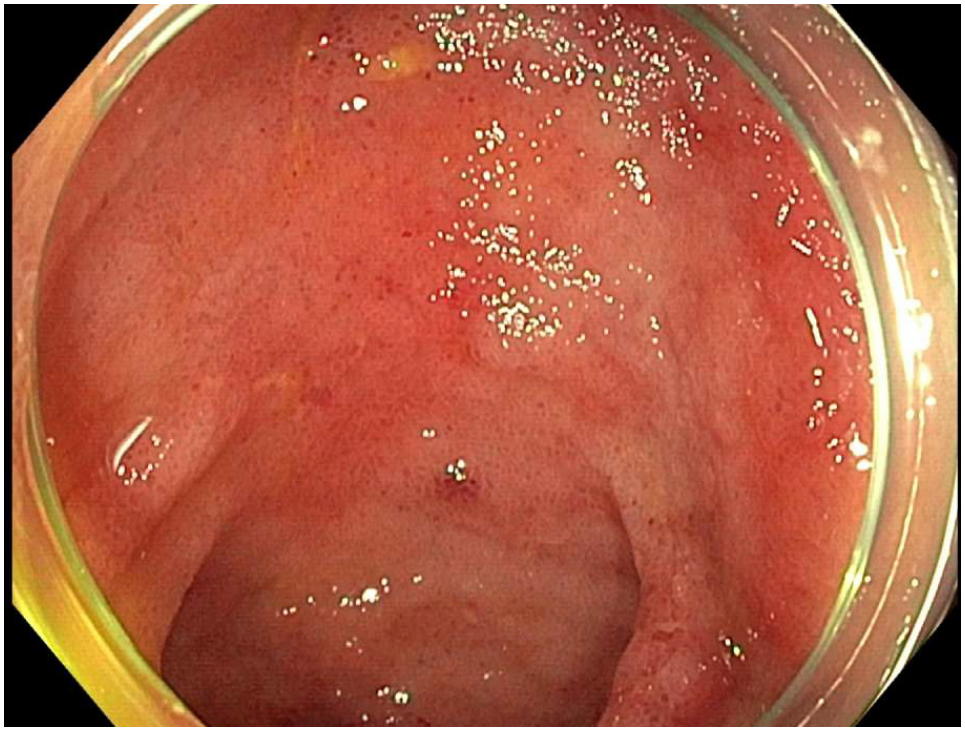
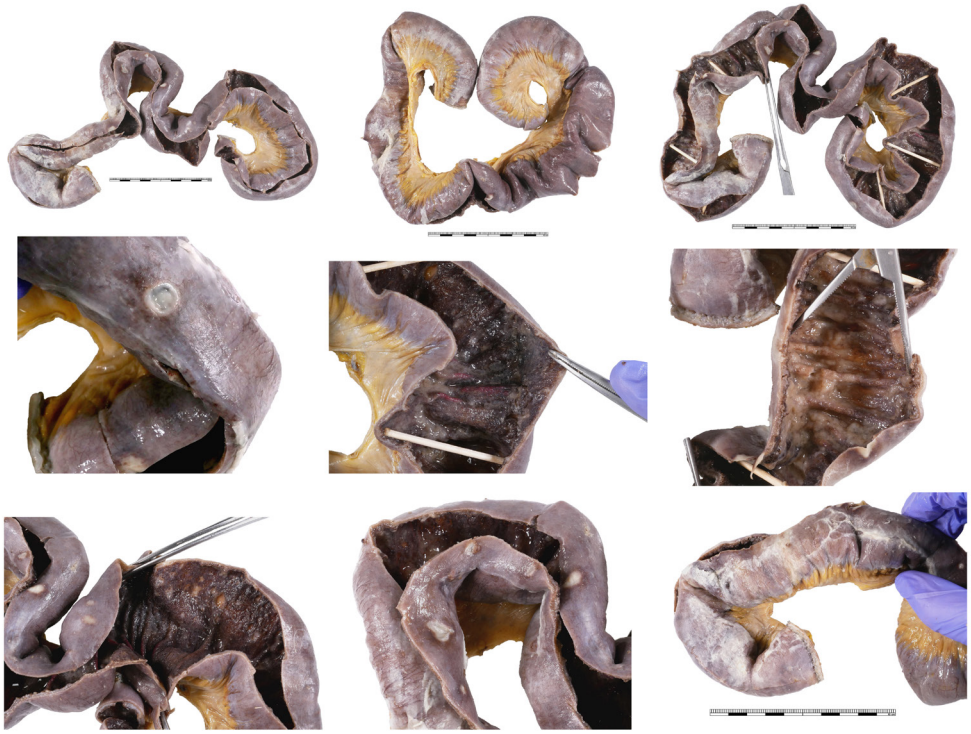


Figure 5: Terminal ileum specimen—showed small artery vasculitis on histology.



Discussion

Gastrointestinal involvement is common in systemic vasculitic conditions such as Henoch-Schönlein purpura, Polyarteritis nodosa and ANCA-associated vasculitis; however, cases of localised gastrointestinal vasculitis are rare and documented in only a few case reports and small case series. Localised gastrointestinal vasculitis (LVGT) is considered to be extremely rare, a difficult diagnosis to make and is associated with high morbidity and mortality.[1,2]

Salvarani et al. published a series on 18 patients with confirmed LVGT via histology or highly suspicious radiological features between 1996 and 2007 at the Mayo Clinic.[3] The study found abdominal pain as the most common symptom, with 17 of 18 patients complaining of abdominal pain, usually severe. Other common symptoms included nausea, vomiting, diarrhoea, weight loss, melaena, haematochezia and abdominal angina. Salvarani et al. found no specific laboratory findings that were consistently abnormal for their patients. Radiologically, 15 of the 18 patients had undergone some form of abdominal angiography (catheter-based, MRI-based or CT-based), and 14 of these patients had radiological features suggestive of gastrointestinal vasculitis. However, it must be noted that these features were also used in the inclusion criteria for the study. Of their 18 patients, seven died as a result of their illness during the period of the study; 10 of the 18 patients received medical therapy with immunosuppression and three (30%) of these patients died, whereas four (50%) of those patients who did not receive medical therapy died.[3] Other case series would also suggest LVGT can progress to systemic multi-organ vasculitis. Burke et al. studied 63 patients with LVGT, and during longitudinal follow up, six patients developed systemic vasculitis.[4]

In summary, our patient died from a bowel perforation caused by LVGT of the ileum, confirmed by histology. He had no evidence of other organ involvement in his disease process. LVGT is a rare diagnosis and could only have been confirmed by a full thickness biopsy or indicated towards by abdominal angiography. CT angiography during his acute illness showed only subtle changes to the SMA. His presentation is in keeping with the small case series published on this topic; however, the rarity of this condition and sizes of these case series make it difficult to relate their findings on management and prognosis to this case.

COMPETING INTERESTS
Nil.

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Endemic Goitre in certain parts of Auckland

NZMJ, 1925
By R. J. Mecredy, M.B.

This survey of the incidence of goitre in parts of the Auckland Health District makes no pretence at being complete and exhaustive. It does, I believe, represent approximately the amount of goitre present in certain localities in North Auckland, Coromandel, the Bay of Plenty and the City of Auckland as measured by the incidence in school children. The accompanying table of results and graphs is based upon the examination of 10,725 children.

Basis of Classification.—The classification used was that laid down by Drs. Hercus and Baker as described in this Journal for April, 1921, and was used by me in a previous report on goitre in Otago, which appeared in this Journal for August, 1923. I have, however, relied throughout on palpation of the thyroid in preference to inspection, and I believe that the resultant classification is more accurate.

For convenience in recording the results I have grouped the enlargement of the thyroid found in percentages and under two headings. "Total goitre" is self-explanatory, while "visible goitre" includes goitres classed as small, medium, and large. The difference between the "total goitre" and "visible goitre" represents the percentage of children with "incipient goitre."

I may say that I entirely agree with Drs. Hercus and Baker that this slight enlargement of the thyroid is pathological.

In certain areas the number of children examined was too small to allow of much weight being attached to the findings. This is particularly the case in the Maori children examined in the Tauranga and Opotiki districts, though the figures show a certain relative agreement with those for the European children in the same areas. In order to give a clearer picture of the incidence I have separated the records for boys and girls and those for Europeans and Maoris.

In the country districts all the children were examined in the smaller schools, while in the latter schools and in the city only those in the primers, standard 2 and standard 6 were examined. This, however, forms a fair cross-section of the school population. In Bayfield every child was examined. In goitrous areas such as the Urewera and Whakatane every child was examined.

Districts Covered.—In the Russel-Kaitaia area practically every school is included to the north of Russel and south of Waipapakauri. Whangarei area includes all schools within a radius of about fifteen miles of that town. The Upper Wairoa group comprises seven small schools all situated upon the watershed of the Upper Wairoa in the near neighbourhood of Whangarei.

The Hokianga group is made up of three small schools close to Rawene on the Hokianga River. Rawene is not included in this group for the reason that all the children with goitre in Rawene came daily from other localities to attend the High School.

All the schools visited in the City of Auckland lay between the Waitemata and Manukau Harbours. They are grouped for the sake of convenience, but the incidence of goitre varied from 2.3 per cent in Beresford Street to 20.3 per cent. in Bayfield. Grey Lynn with 10.6 per cent., Curran Street with 8.4 per cent., and Ponsonby with 8.1 per cent., rank next to Bayfield, while Pt. Chevalier with 2.1 per cent., Napier Street with 2.9 per cent., and Newton East with 3.3 per cent, approach most closely the figures for Beresford Street.

The eight schools on the Coromandel Peninsula comprise all the larger schools north of Thames itself. Here, as in the case of other areas with a low goitre incidence, a part only of the children were examined in each school.

The schools in the Tauranga area are fairly representative of the country from Tauranga to Te Puke.

The Whakatane area includes all schools from Matata to Kutarere, and inland to Te Teko in the Bay of Plenty. This area represents roughly the deltas of the Rangitaiki and Whakatane rivers.

The Opotiki area includes two schools in the town and one a few miles away. All Maori school children in the Urewera country were examined.

The amount of goitre recorded in the individual schools included in these various groups showed

no appreciable variation from the average for the group. Any exceptions have been noted separately.

Results of Survey.—No attempt was made to eliminate from the figures recent immigrants to any district. As a result a proportionately large number of cases of goitre in the areas of low incidence, such as the City of Auckland, had a history of a recent arrival from a known goitrous area elsewhere.

It should be noted that all the city schools examined and found to have a relatively high incidence of goitre lie more or less contiguous to each other. I was unable to find any local cause to explain the high incidence at Bayfield. If the forty-six girls from a nearby orphanage are excluded from the numbers for this school, the goitre incidence is reduced to 17.9 per cent., which is still much higher than any of the neighbouring schools.

Proceedings of the Waikato Clinical Campus Research Seminar, Thursday 11 September 2025

BOWELING OVER BOWEL CANCER BY EARLY DETECTION, AN AUDIT OF THE NATIONAL BOWEL SCREENING PROGRAM IN THE WAIKATO REGION

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BACKGROUND

New Zealand has one of the highest incidence of bowel cancers globally.[1] Hence a national bowel screening program was implemented for 60–74 year olds in 2017, and Waikato DHB was inducted in March 2021.[2] Since December 2023 Waikato lowered its age for Māori/Pacific people to start screening from the age of 50, as a higher proportion of bowel cancer occurs in Māori and Pacific people before reaching 60.[2]

AIM

To analyse the effectiveness of the bowel screening program, and its implication for Māori/Pacific people.

METHOD

Retrospective chart audit of polyps removed following positive faecal immunochemical test (FIT), with subgroup analysis of high grade dysplasia (HGD), adenocarcinomas and adenocarcinomas arising from polyps.

RESULTS

From March 2021 to June 2025, FIT positive patients were scoped and 11,407 polyps/masses were sampled.

Of these, 273 (2.39%) showed high grade dysplasia, and 211 (1.85%) were adenocarcinoma of which 60 (28%) arose from polyps.

HGD and adenocarcinoma diagnoses (154) from December 2023 to June 2025 comprised 39 (25%) Māori and 18 (46%) of these were in the 50–59 age group.

CONCLUSION

A significant proportion of early colorectal cancer is detected in Māori by commencing screening at age 50. This justifies the need to implement this screening strategy nationwide to achieve early detection for this high risk group.

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PHASE 1B DOSE-ESCALATION TRIAL OF SELENIUM COMPOUNDS IN CANCER PATIENTS

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AIMS

Selenium (Se) compounds have shown therapeutic synergy with anticancer treatments (including radiotherapy and chemotherapy) while reducing off-target toxicities in experimental models. This trial aimed to evaluate safety and pharmacokinetic–pharmacodynamic (PK–PD) relationships of three Se compounds in cancer patients, to inform optimal compound selection and dosing for future clinical trials.

STUDY METHODS

This double-blinded, intra-subject dose-escalation trial enrolled nine patients with metastatic cancer not currently receiving cytotoxic treatment. Patients were randomised to receive one of three

Se compounds orally: Se-methylselenocysteine, L-selenomethionine or sodium selenite, dosed at 1,600µg of elemental Se/day for 4 weeks followed by 6,400µg/day for 4 weeks. An earlier 400µg/day dose cohort has been reported previously. Safety, PK and PD parameters were assessed at baseline, after each dose period, and 1 month post-dosing. PD investigations in peripheral blood mononuclear cells focused on ER stress, angiogenesis, plasma antioxidant activity, DNA damage and repair, methylation and gene expression pathways.

RESULTS

All three selenium compounds were well tolerated, with no treatment-related toxicities exceeding grade 2. Pharmacokinetic analyses indicated that L-selenomethionine produced the greatest increases in plasma selenium concentrations at both dose levels. Nuclear DNA (nDNA) and mitochondrial DNA (mtDNA) damage demonstrated variability without consistent trends. Analyses of ER stress and angiogenesis by Western blot, as well as intracellular glutathione assays, are ongoing.

CONCLUSIONS

All three Se compounds were well tolerated at 1,600 and 6,400µg/day. L-selenomethionine accumulated most strongly in plasma, but there was no evidence of significant genotoxicity. These findings will contribute to an understanding of the safety and PK–PD relationships of Se compounds, supporting rational selection of agents for future clinical trials in combination with cancer therapies.

WHIRI WĀHINE HAPŪ: A HOLISTIC, CULTURALLY SAFE MODEL ENHANCING CARE FOR PREGNANT MĀORI WOMEN

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INTRODUCTION

Midwife shortages and the complexity of navigating maternity services has contributed toward stark and significant health inequities in birth and maternal outcomes for Māori wāhine hapū/pregnant women. Compared to non-Māori, wāhine hapū have poorer access to lead maternity carers and young wāhine hapū report many systemic barriers from their first health contact. Wāhine hapū are at increased risk of adverse mental health outcomes, and maternal experiences of discrimination are associated with lower birth weight and shorter gestation length. We aimed to explore wellbeing needs of pregnant Māori women, then codesign and test a wellbeing assessment and clinical support to enhance wellbeing.

METHODOLOGY AND METHODS

Using Kaupapa Māori theory, the He Pikinga Waiora framework and a mixed method approach, our team codesigned, piloted and evaluated a Whiri model of care for wāhine hapū. This was codesigned with >100 stakeholders including whānau/family, community, researchers and healthcare providers in the Waikato region. The Whiri model of care is structured around navigators and a holistic wellbeing assessment that identifies unmet needs and has support referral pathways, and links to a multidisciplinary, nurse-led clinical team to address clinical need. Overseen by Indigenous clinical governance, the model of care was piloted with wāhine hapū in the Waikato region.

OUTCOMES

Codesign stakeholders identified four health domains in Te Whare Tapa Whā as an important approach for the Whiri model of care, adding the importance of Te Ao Māori (Māori worldview) and Kaupapa Māori. We recruited 31 wāhine hapū participants (aged 16–44 years, Ɱ=29, 15 urban/14 rural) into the pilot, between August 2023 and June 2024. Significant need (345 unmet needs; 11/wāhine) was identified and supported by the navigator and nurse. The most common unmet needs supported: mental health support (21; 75%), social support, referrals to healthy home, information on the National Travel Assistance scheme and food nutrition and access to more kai/food for the whānau. Over half (19; 61%) were referred to the Whiri nurse for clinical support for a variety of health concerns: mental health support, diet and nutrition information, pregnancy or medication advice or support for gestational diabetes. When asked in a phone survey, wāhine valued the reassurance and advice the nurse provided.

CONCLUSIONS

Significant social and clinical unmet needs were identified and supported using the Whiri approach. Engagement with maternity service end-users is essential to understand the wellbeing needs of wāhine hapū, providing valuable insight to inform health policy and practice.

A SURVEY EVALUATING THE ATTITUDES OF CANCER PATIENTS IN AOTEAROA NEW ZEALAND TO FAECAL MICROBIOTA TRANSFER

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AIM

Faecal microbiota transfer (FMT), also known as gut microbiome transfer, is intended to favourably change the gut microbiome in patients with various health disorders. Early studies in cancer patients suggest that FMT might improve efficacy of treatment and reduce some toxicities, but little is known about the patients' perspectives. The primary aim of this study of patients with cancer was to describe their awareness of, and potential willingness to undergo, FMT in conjunction with cancer therapies. Secondary aims were to evaluate differences in responses according to patient demographic, cancer type and treatment, and to compare the characteristics of participants versus non-participants in the survey.

METHOD

This study was conducted through online surveys accessed by posters displayed at oncology treatment facilities. Patients were questioned about awareness of FMT, willingness to undergo FMT, attitudes to FMT administration method and donor preferences.

RESULTS

Thirty-four surveys were submitted from participants in Auckland (n=19) and Waikato (n=15). Twenty-one participants (62%) were female, 27 (79%) were >45 years old and 26 (76%) had a post-secondary educational qualification. Self-reported ethnicity was 27 (79%) NZ European, three (9%) Māori, one (3%) Pacific people and three (9%) other participants. The most common cancers were bowel and breast, each with eight participants (24%). Seventeen (50%) of survey participants had heard of a "microbiome transfer", of whom 14 (82%) reported knowing "a little" about FMT. Overall 97% of survey respondents were receptive to having a microbiome transfer if it was proven effective, 14/21 (67%) preferred FMT via oral capsules and 28/33 (85%) had no preference for donor characteristics. Of 317 potentially eligible Chemotherapy Daystay attendees at Waikato Hospital during the survey period, 15 (4.7%) participated in the survey; two of 26 (7.7%) Māori participated. Patient characteristics were similar in those who did and did not participate in the survey.

CONCLUSION

This study indicates that many cancer patients in Aotearoa New Zealand would be willing to undergo FMT for treatment benefit. Our study was limited by the low number of survey responses, indicating the importance of addressing recruitment strategies in future intervention clinical trials.

ACCURACY AND PRECISION OF THE CLEARSIGHT™ NON-INVASIVE MONITORING DEVICE IN THE NEW ZEALAND POPULATION UNDERGOING TRANSCATHETER AORTIC VALVE INTERVENTION

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BACKGROUND

Transcatheter aortic valve implantation (TAVI) is a rapidly expanding treatment for severe aortic stenosis (AS), the most common valvular pathology in the ageing population. Accurate, continuous haemodynamic monitoring is crucial during TAVI to guide decision-making and ensure patient safety. Invasive arterial pressure monitoring is the gold standard but carries risks of complications. The ClearSight™ (CS) device (Edwards Lifesciences, Irvine, CA) offers a non-invasive alternative based on the volume clamp method, but its accuracy in the TAVI setting remains uncertain, with mixed results in prior studies. No published data exists in the New Zealand population. This study evaluated the clinical equivalence of CS compared with invasive arterial monitoring in patients undergoing TAVI for true severe native valve AS. The primary hypothesis was that CS provides accurate and precise alternatives to invasive measurements.

METHOD

This single-centre, prospective observational study was conducted at Health New Zealand – Te Whatu Ora Waikato, enrolling 25 consecutive patients with severe high-gradient AS undergoing

elective TAVI between March and June 2023. Patients with low-flow, low-gradient AS or valve-in-valve procedures were excluded. Simultaneous blood pressure measurements were obtained from CS and invasive monitoring via femoral arterial sheath or pigtail catheter. CS was applied using a finger cuff and HemoSphere™ monitor. Measurements of systolic (sBP), diastolic (dBP) and mean arterial pressure (MAP) were recorded at 20-second intervals. Statistical analysis included Bland–Altman and error grid assessments. Clinical equivalence was defined as a bias of ±5mmHg and percentage error ≤30%. Error grid thresholds were: zone A >90%, zones B and C <5%, zone D <4% and zone E <2%.

RESULTS

A total of 4,063 paired measurements were analysed. The mean age was 80.2 years, with near-equal sex distribution. Most participants were New Zealand European, with one Māori patient. CS showed poor correlation with invasive sBP and dBP, with Bland–Altman biases of −7.98mmHg and −5.34mmHg, and percentage errors of 39% and 37%, respectively. MAP correlation was considered equivalent, with a bias of −1.74mmHg and a percentage error of 28.8%, meeting equivalence criteria. Error grid analysis showed 71.1% of MAP points in zone A, 25% in zone B and 3% in zone C. In hypotension (MAP ≤65mmHg), CS overestimated MAP with large negative biases and unacceptable error grid performance.

CONCLUSION

CS demonstrated acceptable accuracy for MAP but not for sBP or dBP in New Zealand TAVI patients. Overestimation of MAP during hypotension is a key limitation with potential clinical risk. CS may serve as a non-invasive alternative for MAP monitoring, but reliance on systolic or diastolic readings is discouraged. Further investigation is required in physiological hypotension and additional derived haemodynamic parameters provided by this device.

Mairi Alice Sewell

Dr Mairi Alice Sewell (née McCaskill) was born on 7 October 1929 in Wellington, of Scottish parents. At age 6 weeks, her parents moved to Sydney for employment. She graduated from The University of Sydney in 1953 with a degree in medicine, along with 49 other girls and 300 men, the majority of whom were returned servicemen.

In 1953, Mairi returned to New Zealand looking for work and adventure. Her first jobs as a house surgeon were at Gore and Whangārei hospitals, followed by general practitioner (GP) locums in Pahiatua and the Westland District.

Mairi married Tom Sewell, an agriculturalist with the Department of Agriculture (now the Ministry for Primary Industries), in 1955.

She worked in general practice as a sole practitioner and had part-time work in public health as a school and Plunket doctor while raising their family of seven children.

In January 1967, the family moved to Levin when her husband was transferred.

For the following 35 years, Mairi lived in Levin and worked at Tararua Medical Centre, then at the Horowhenua hospital as the senior medical officer, being responsible for geriatric rehabilitation, palliative care and oncology. She also had close links with Kimberley Hospital as their staff medical officer.

In 2003, she and Tom moved to Auckland for retirement. Tom passed away in 2005, and Mairi continued relief medical work until she finally hung up her stethoscope in 2007. She gave the valedictory speech at her 60th university medical school reunion in 2013, flying to Sydney on a day return.

Mairi played a full role in community life with energy and enthusiasm. She was in demand as a wise counsellor, educator and speaker for many women's and church groups across New Zealand.

Throughout Mairi's life, she cared for people, supporting those in need with words of wisdom and encouragement.

Until her vision was lost, Mairi delighted in reading her bible daily, praying for her long list of Christian missionaries and doing the cryptic crosswords. On 23 August 2025, Mairi passed away peacefully in her sleep aged 95 years. She is survived by 7 children and their spouses, 23 grandchildren and 20 great-grandchildren.

AUTHOR INFORMATION

This obituary was written by Dr Graham Sewell, FRNZCGP, RACGP.